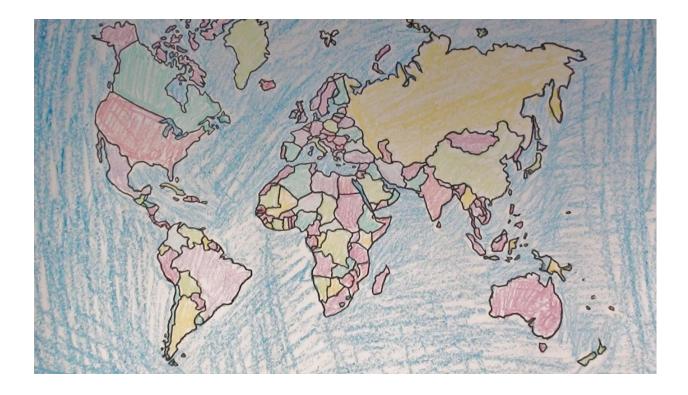
CRITICAL CARE HANDBOOK FOR GLOBAL SURGERY

Edited by Jacob S Dreyer & David R Ball Assisted by Abebe Bekele & Andrew Howard



Alba CCCD SCIO (2015)

CRITICAL CARE HANDBOOK FOR GLOBAL SURGERY

Edited by Jacob S Dreyer & David R Ball, Assisted by Abebe Bekele & Andrew Howard.

For supporting teaching of Surgical Critical Care in Sub-Saharan Africa and other global surgery regions where teaching and training in essential and emergency surgery has become a priority.

Alba Critical Care Course Design SCIO (Alba CCCD)

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Cover picture by the children in Ward 15, Dumfries & Galloway Royal Infirmary.

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THIS BOOK IS FOR ALL THOSE WHO HAVE TO DEAL WITH CRITICALLY ILL PATIENTS IN SURGERY, OFTEN AT NIGHT AND ALONE, IN HOSPITALS WITH FEW STAFF AND LIMITED RESOURCES.

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A major impetus to the programme came from Andrew Howard, Director of the Office of International Surgery at the University of Toronto, who asked us to write a series of review papers on essential critical care topics for the Ptolemy Project, an online learning resource delivered in partnership between the University of Toronto, the College of Surgeons of East, Central and Southern Africa (COSECSA) and the Canadian Institutes of Health Research. Writing the review papers brought in many more potential faculty for teaching and clarified what we wanted to teach. My sincere thanks to Dr Howard for his support.

There would have been no progress if it was not for the vision of Mr Bob Lane, pastpresident of ASGBI, in seeing the need for a course on managing surgical emergencies (MSE) for surgical trainees within the nine countries (now 10) of the COSECSA region. This vision was supported by strong leadership and perseverance to develop the MSE course with the support of COSECSA. I sincerely thank Mr Lane for asking me to develop the two day critical care curriculum and giving me a free hand to develop course content, delivery methods and assessment tools, and recruit co-authors and tutors.

My sincere thanks to all tutors who contributed to the contents of our critical care course and to the authors of this book. Some were still trainees or students and obviously felt under pressure to deliver but all did an excellent job. My thanks also to all tutors who have travelled to teach on MSE and on independent critical care courses run for surgical societies in the COSECSA region. It was to enable such travel for teaching that Alba CCCD SCIO was founded and registered as a Scottish Charity; Alba CCCD was also registered as a publisher in the UK to facilitate publication our handbooks at low cost. The MSE course was fully funded by the Surgical Foundation of ASGBI, mainly through a major grant from the UK government's Department for International Development (DfID) and managed through the Tropical Health and Education Trust (THET), which was invaluable. Again Mr Lane played the major role in acquiring this grant.

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I would never have embarked on this critical care journey without the influence of two people. In my first year as a junior doctor in surgery a senior registrar, Andries Retief, told me *"if a patient is good enough to operate on, that patient is good enough to look after afterwards"*. Sadly he died a year later after being in contact with a patient with Crimean-Congo haemorrhagic fever. The process that made me understand critical illness, however, was the quality teaching in surgical critical care I received in 1987 as a trainee from Prof Andre Coetzee, Head of the Department of Anaesthesiology at the University of Stellenbosch and Tygerberg Hospital. Weekly and weekend ward rounds drilled home some principles that are still evident in the contents of this book. I have used his concepts unashamedly and where I have deviated from them, the errors are mine alone.

Lastly, a special thanks to my family, who have allowed themselves to be pulled into this project in numerous ways, and for their ongoing support.

JS (Fanus) Dreyer Dumfries, May 2015.

FOREWORD

JACOB S DREYER, ROBERT H S LANE

The 1978 Declaration of Alma Ata stated basic health goals in primary health care. It has become the ethical cornerstone of global public health. In section VII.3 it addressed issues such as "education concerning prevailing health problems,...maternal and child health care, ...appropriate treatment of common diseases and injuries" [1]. Clearly some of these primary health care problems have always needed surgical care but for many years surgery had been the "neglected step child of global health" [2]. In the last few years this started to change. The increase in non-communicable diseases (NCDs) [3], the costs of the international trauma epidemic in lives and disability [4], and ongoing maternal mortality due to complications of pregnancy and childbirth that could be managed surgically [5] have raised awareness of the need for surgery to be recognised as part of primary health care. NCDs are expected to increase by 45% by 2030, with 80% of cancer deaths expected to occur in low and middle income countries (LMICs) [3]. Surveys in Rwanda and Sierra Leone (before Ebola) showed that the annual surgical need affects 15-25% of the population [6, 7]. The term "Emergency and Essential Surgery" (EES) was coined [8] and advocacy for EES in global surgery has been driven by groups such as the Global Initiative for Emergency and Essential Surgical Care at the WHO (GIEESC), the International Collaboration for Essential Surgery (ICES), the Global Alliance for Surgical, Obstetric, Trauma and Anaesthesia Care, and the Lancet Commission on Global Surgery (LCoGS) [9]. In January 2014 Jim Kim, President of the World Bank, urged the global health community to challenge the injustice of global inequity in surgical care, stating that "surgery is an indivisible, indispensable part of health care and of progress towards universal health coverage" [10]. At the time of publication of this book the World Health Assembly (WHA) unaminously passed a resolution from the WHA Health Board To include strengthening emergency and essential surgical care and anaesthesia as a component of universal health coverage [11].

The implication of this is that the world population will need 143 million more operations per year especially in low income countries [12]. WHO figures have put the surgical burden of disease at 11%, with >2 billion people with no access to surgery [13], but the LCoGS have recalculated that almost 5 billion people cannot access affordable, safe and timely surgical care [12]. The World Bank has recently published their definition of Essential Surgery [14]. The reality is that international surgery will need many more surgeons, whether trained through classical pathways as medical specialists or as trained non-physician surgeons such as health or clinical officers, assistant medical officers or surgical technicians. Many people in LMICs depend on non-surgeon physicians (medical doctors who are not formally trained as surgeons) for emergency operations; these surgeons by experience also need support from the surgical training community in order to improve access to EES to the same global standard.

To paraphrase Sir Ian Aird, a surgeon needs to know three things: Anatomy, Physiology and Pathology [15]. Pathology teaches us when to operate, Anatomy how to operate and Physiology how to look after the patient. Critical care is about Physiology. A recent study in Uganda showed that patients who get to the intensive care units (ICU) in Africa are generally young, still within the economically active part of their lives, have reversible conditions such as major injuries and surgical sepsis, and generally spend only a few days in ICU with good recovery and return to economic activity in most [16]. In a recent editorial for the New England Journal of Medicine Bill Gates discussed lessons from Ebola and how the world should be better prepared for the next epidemic; this includes better availability of critical care facilities and trained staff [17]. In the first place this book, however, is not about ICU care. It is for those who work "at the coalface" of emergency surgical care, often junior doctors and non-physician clinicians, who need to recognise critical illness early when they see patients in surgical wards or admissions units, to know how to start simple measures of physiological support early and to know how to support critical cell function until they can institute definitive therapy or get help.

It is easy to misunderstand critical care as a science that belongs in the ICU and needs complex skills such as ventilation, inotropic drugs and invasive monitoring. Critical care actually starts in any clinical area, by using your eyes, ears, hands and brain to collect easily available clinical information, decide with every patient you see whether he/she is stable or unstable and to ask "Why?". It teaches you not to ignore signs that do not fit but to step back from unconscious competence and to re-evaluate your decision making. Through critical care courses developed through Alba CCCD SCIO, the Association of Surgeons of Great Britain and Ireland, and the International Federation of Surgical Colleges, we teach that simple actions completed early prevent a cascade of complications and save lives. Some of these concepts are also taught in courses like CCrISP (© RCSEng) and others, but we adapt course content to suit local needs, using participants' feedback. What we therefore teach are physiological principles of deterioration due to loss of airway, breathing and circulating volume, and how to support these ABC systems. We teach how disability is caused by neurological injury, burns, sepsis, pain, anaesthesia, the psychological impact of critical illness and end-of-life events. In the end all we teach is that air must go in and out and blood must go round and round to keep essential cells alive. All this is done with a minimum of props, a CPR and Airway manikin, a few laptops for small group teaching and regular breaks for caffeine refuelling. Surgical critical care is simple to teach and simple to practice.

The same principles of critical care can therefore be taught to consultants in surgery, surgical trainees, health or clinical officers who do surgery, non-specialist surgeons who work in small hospitals and nurses who look after surgical patients in high-care units, theatre recovery rooms or surgical admission wards or units. The pathways of physiological deterioration are the same, irrespective of whether the patient has had multiple fractures from a car crash, had a stab wound to the abdomen, has postpartum haemorrhage or renal sepsis, had a typhoid perforation or has a strangulated hernia. Therefore the principles of support and specific management are the same, whether that patient needs rapid resuscitation, a laparotomy or evacuation of the uterus. It does not matter who the first surgical service provider for that patient is, as long as they know what to do and can do it well within their particular practice environment.

Learning critical care had been identified as an important aspect of surgical training by the council of the College of Surgeons of East, Central and Southern Africa (COSECSA). We were approached in 2011 to help develop such training. Since 2011 we have had >200 participants on our courses (consultants, surgical trainees, anaesthetic assistants, health/clinical officers and nurses) and we have trained >20 tutors [18]. This two-day course has now been taken over by COSECSA and from 2016 will be compulsory for all their trainees before fellowship examinations [Personal Communication]. One problem in teaching a relatively new entity is making reading material available. From December 2011 to April 2013 we therefore published a series of critical care review articles through the Ptolemy Project, a University of Toronto open access online resource primarily for

surgical trainees in low-income countries [19]. These articles have formed the core of this handbook but we have added chapters on topics that affect surgical practice in critically ill patients.

As medicine has shifted from modernist to post-modernist over the last few decades, it has become clear that modern medicine cannot cure everything [20, 21]. In many ways "Care" is more important than "Cure". For that reason we have included an extensive section on Surgical Humanities in critical care. When we teach critical care these are the topics that are often most difficult to discuss openly but always bring the most gratifying responses from participants. African philosophy has always recognised "Ubuntu" (Nguni: *"I am because you are"*), and it is in that spirit of African humanism that we present this handbook on critical care for global surgery.

The LCoGS have defined Global Surgery as "an area of study, research, practice, and advocacy that seeks to improve health outcomes and achieve health equity for all people who require surgical care, with a special emphasis on underserved populations and populations in crisis. It uses collaborative, cross-sectoral, and transnational approaches and is a synthesis of population-based strategies with individual surgical care" [12]. So, as surgery advocacy groups propagate the concept of global surgery, with a certain minimum number of essential operations to be available to everyone worldwide, we are addressing the "practice" part, developing and supporting systems that teach the optimisation of patients pre-operatively and how to manage ill patients, whether due to major injury, planned major operations or surgical sepsis. The physiological support of critically ill surgical patients makes a difference to outcomes and is an indispensable component of essential and emergency surgery in any environment. We trust that this book will contribute to that understanding.

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1. INTRODUCTION TO SURGICAL CRITICAL CARE

STEPHAN B DREYER, ANDREW J JACKSON, JACOB S DREYER.

INTRODUCTION

Surgical patients who become critically ill almost always fall in one of three categories: major trauma, major surgery or sepsis. Major trauma relates to significant injury of a single organ system or anatomical part, or multiple injuries, often of varying severity, of different body parts. Major surgery usually implies surgery where a body cavity (chest, abdomen or pelvis) is entered or extensive muscular-skeletal or soft-tissue surgery. Sepsis is a term that is often used more loosely but implies that the Systemic Inflammatory Response Syndrome (SIRS) is triggered; this can happen without infection e.g. in pancreatitis, or with serious infection e.g. perforation of the colon [1,2]. In all three patient categories the pathophysiological processes that make patients ill and lead to cellular injury and organ dysfunction are essentially the same, and therefore the way that patients need support of critical organ function is the same.

Trauma needs special attention in Africa. As energy has become cheaper and more easily acquired, trauma has become endemic in parallel with economic development [Imre Loeffler. Personal communication 2005]. Transport has become faster and more dangerous, leading to high velocity deceleration injuries, e.g. from road traffic crashes. As recently as a 100 years ago young men had to work hard to acquire enough food to support their families but now have leisure time due to easy access to energy; they need outlets for extra energy, fuelled by testosterone, which leads to violent tests of strength and athleticism. Collateral damage of violent behaviour often includes women, children and the elderly.

DEATH AFTER TRAUMA FOLLOWS A TRIMODAL DISTRIBUTION [3]:

The first peak occurs within seconds to minutes due to injuries to e.g. the brain stem, high spinal cord, heart and major vessels. Very few patients with such injuries are salvageable although military medical triage and retrieval systems have recently addressed some of these.

The second peak occurs minutes to hours after injury and can be due to manageable conditions such as tension pneumothorax, massive haemothorax, injuries to the liver, spleen and pelvis, intracranial bleeding. The Advanced Trauma Life Support (ATLS) [©ACS] and definitive trauma management courses address management of these trauma complications.

The third peak of death occurs days to weeks after the initial injury and is usually due to sepsis, surgical complications and multi-organ failure. Minimising these complications is addressed through training in critical care e.g. through CCrISP [©RCSEng]. Critical care in this context is not the same as intensive care. ICU care is only a component of critical care where staff provision, monitoring and organ support are provided at an intense level. Critical care actually starts in the emergency unit, surgical admissions unit or

surgical ward and is ideally delivered by surgical trainees and other junior staff, well before intensivists become involved.

EXAMPLE: An elderly patient who is a smoker undergoes successful operation for a strangulated inguinal hernia. He is still behind on fluids due to pre-admission vomiting; nobody thinks that he might have low potassium. He has severe wound pain and does not cough properly, develops atelectasis but because it is Saturday night it is not noticed. On Sunday morning the patient is not on the handover sheet to the new on-call team because it has been a busy night in A&E and there are three patients waiting to get into theatre. By Monday morning the patient has lobar pneumonia. He is given antibiotics and somebody is asked to give him physiotherapy. He develops atrial fibrillation from a combination of hypoxia and electrolyte disturbances; the resident says he will come and see him as soon as theatre finishes. He stays tachypnoeic and by early evening "suddenly" arrests.

This could have been prevented by prediction of risks, good basic care such as pain relief, effective fluid therapy, structured clinical review, early physiotherapy and patient support, and effective communication and handover.

EFFECTIVE CRITICAL CARE IS THUS BASED ON:

- a) sound knowledge of the physiology of the critical organ systems along the pathway of tissue oxygenation, i.e. that Airway, Breathing and Circulation exist to support cellular oxygenation in the Tissues (**A**, **B**, **C** →**T**) [ETCC, ©RCSEd];
- knowledge of the pathophysiology of tissue injury, and how cellular injury leads to a cascade of chemical, hormonal, cellular and immunological responses which exist to prevent early death but which can lead to progressive further cellular damage and dysfunction and failure of essential organ systems;
- c) understanding that the sequence ABCD is followed in organ support as it is in resuscitation. Critical care is in essence about keeping air going in-and-out and blood going round-and-round to keep the tissues alive. A systematic approach gives the best chance of keeping essential cells alive;
- d) remembering that most of us are bad at managing unexpected events and it is therefore better to prevent morbidity through
 - prediction,
 - repeated clinical assessment,
 - early detection of deterioration or failure to progress.
- e) gathering all available information about a patient yourself through
 - good clinical observations;
 - rapid clinical assessment of deteriorating patients, using ABCDE;
 - emergency support of ABCD to allow time for more thorough assessment and treatment;
 - thorough further assessment using all available information;
- f) understanding that critical care starts with prompt, simple actions that save lives and prevent complications [1];
- g) using specific interventions to support critical organ function and prevent physiological deterioration;

- h) taking active decisions about patient management;
- i) and asking for help early in an assertive way, e.g. through using the **SBAR** system (clearly state the <u>Situation</u>, <u>Background</u>, <u>Actions or Assessment</u> undertaken, and your <u>Recommendation</u> for help, or the <u>Results of your Assessment</u>).

PATHOPHYSIOLOGY OF TRAUMA:

In 1942 Cuthbertson first proposed the 'ebb' and 'flow' phases modelling the physiological response to major trauma [4]. Since then, the factors influencing this response have been extensively investigated and characterised. Trauma leads to alterations in haemodynamic, metabolic, cellular and immune responses of patients [5]. This is driven by a primary systemic inflammatory response (ebb) and a subsequent antiinflammatory response, with immunosuppression and multiple organ dysfunction [6]. The net goal of the physiological response to trauma is to maintain cardiovascular haemostasis, to retain salt and water to maintain intravascular volume and to enter a catabolic state to mobilise energy substrate to provide energy.

The initial systemic inflammatory response is characterised by [7]:

- Sympathetic nervous system activation
- Activation of specific endocrine stress hormones
- Microvascular disturbance
- Cytokine and Acute Phase reaction.

None of these systems act independently of one another, and responses are augmented by complex interactions between these systems. The trigger may not necessarily be accidental trauma – a planned surgical procedure of overwhelming infection with sepsis produce the same physiological response.

SYMPATHETIC RESPONSE:

The sympathetic response is characterised by the release of noradrenaline (also known as norepinephrine) from the sympathetic autonomic nervous system, which is amplified by secretion of adrenaline (epinephrine) from the adrenal medulla under control of ACTH. This serves to create direct cardiovascular effects as well as modifying endocrine pancreatic secretion and hepatic function:

Direct cardiovascular effects manifest as:

- Increased Heart Rate
- Increased Blood Pressure
- Increased Myocardial Contractility
- Diversion of blood from skin and visceral organs
- Bronchodilation
- Reduced gastro intestinal motility.

Other hormonal effects are [8]:

- Reduced insulin production
- Increased glucagon production

- Increased gluconeogenesis
- Increased blood sugar.

The net effect of hormonal modulation of is to increase available energy from catabolism of carbohydrate, fat and protein. The patient enters a catabolic state, utilising available body stores as a source of energy. Insulin is the primary anabolic hormone which inhibits protein catabolism and lipolysis; therefore inhibition allows catabolism of body substrate to provide energy. Glucagon increases gluconeogenesis in the liver and facilitates lipolysis. These effects are augmented by cortisol secretion from the adrenal medulla.

ACTIVATION OF ENDOCRINE STRESS HORMONES:

In conjunction with the sympathetic response, endocrine stress hormones are activated, primarily under control of the hypothalamic-pituitary axis [9].

Trauma stimulates the anterior pituitary (under hypothalamic control) to synthesise and release ACTH and growth hormone [10]. Concentrations of TSH, FSH and LH do not appear to change following trauma.

In response to ACTH, cortisol is rapidly released from the adrenal cortex [11]. Its effect is to promote protein breakdown and gluconeogenesis in the liver. Glucose utilisation by cells is also inhibited and lipolysis is facilitated. ACTH further inhibits insulin secretion and promotes glucagon production. Thus utilisation of all body energy stores is promoted.

Other key endocrine changes are stimulation of anti-diuretic hormone (ADH) release from the posterior pituitary and increased aldosterone production from the adrenal cortex. ADH secretion promotes retention of fluid volume by direct effect on the kidney. Increased aldosterone production is an effect of stimulation of the renin-angiotensin system (stimulated by reduced perfusion of the kidney), which similarly promotes sodium and water resorption in the distal tubules of the kidney [12].

Overall the net hormonal effect is again to provide energy substrate for tissue repair and to preserve circulating plasma volume to aid cardiovascular homeostasis.

MICROVASCULAR DISTURBANCE

The initial microvascular response to trauma is characterised by vasoconstriction of arterioles under control of the sympathetic nervous system. This results in reduced capillary flow and an increased hydrostatic pressure. A local, microcirculatory inflammatory response follows, initiated by local leukocyte endothelial adherence [13]. A cascade of local responses is initiated, characterised by TNF-alpha production, other pro-inflammatory cytokines release, and increased nitric oxide production [14]. All this leads to microcirculatory vasodilatation and severe endothelial dysfunction with tissue and cell damage resulting from accumulation of metabolites and tissue hypoxia [15].

CYTOKINE AND ACUTE PHASE REACTION:

Cytokines are immune mediators that direct the inflammatory response to the site of injury. An exaggerated cytokine response can lead to homeostatic instability and metabolic derangement [8].

Pro-inflammatory cytokines released at the site of injury include TNF-alpha and interleukin 1beta [16]. These direct the immune reaction, stimulating production of interleukin-6 (which directly promotes hepatic C-reactive protein synthesis) and other acute phase proteins [17]. These direct immune responses, but imbalances at microcirculatory levels can cause impaired oxygen transport to organs. Exaggerated systemic release can lead to pulmonary damage via activation of lung macrophages and recruitment of neutrophils which, in conjunction with microcirculatory disturbance, can cause ARDS [18]. A combination of local tissue hypoxia and exaggerated inflammatory response increases intestinal permeability to bacteria and endotoxin which can further aggravate and augment the response of the already hyper-reactive inflammatory response [19]. A 10% decrease in circulating volume can lead to a 50% fall in mesenteric perfusion which contributes to translocation, an important immunological trigger of physiological deterioration.

Overall it can be seen that the systemic response to trauma is a complex series of interactions between autonomic, hormonal, cellular and immune responses. The beneficial effect of these processes can be clearly seen in maintaining homeostasis and nutrition as well as directing the inflammatory and immune response to trauma, but there comes a point where, if left unchecked, these responses can become counter-productive to patient survival.

PHYSIOLOGICAL DETERIORATION OF THE TRAUMA PATIENT

The physiological responses can maintain homeostasis for a certain period of time (in relation to the degree of trauma), but there comes a point where the response becomes harmful. This occurs as a complex interaction between the physiological response to trauma and intervention, whether operative or supportive management. Without adequate intervention the primary homeostatic responses of increased heart rate and blood pressure, and volume supplementation with sodium and water conservation, will not be sufficient to maintain plasma volume. Even if adequate intervention is performed and circulating volumes normalised, the cytokine response can quite rapidly become deleterious to patient survival. Inappropriate amplification causes micro-circulatory disturbance which impairs oxygen transport to tissues and waste product excretion. Organs can quickly become dysfunctional, with failure of cardiac, respiratory, renal and gastro-intestinal systems (multiple organ dysfunction syndrome). This can occur despite adequate attention to the primary insult [5].

Even the shift to a catabolic state can be detrimental to patient survival. Whilst it is an advantage for an injured animal to be able to catabolise its own stores when food is not readily available, this catabolic state has a number of disadvantages when nutrition is inadequate. A patient becomes hyperglycaemic and ketotic which predisposes to infection.

In addition to these responses, patients with exsanguinating trauma can develop the 'triad of death': hypothermia, coagulopathy and acidosis. Like the inflammatory response to trauma, this can develop quickly into a vicious cycle from which it is difficult to recover. If left unchecked and not dealt with aggressively (see DCR below), the 'triad of death' amplifies rapidly, further haemorrhage occurs, the systemic inflammatory response is further exaggerated and multiple organ dysfunction takes hold. Even with adequate

resuscitation and intervention, mortality can approach 50% if all three factors are present [20].

DAMAGE CONTROL RESUSCITATION:

Damage control resuscitation (DCR) is directed at preventing secondary organ dysfunction after tissue injury by controlling haemorrhage and infection whilst avoiding the lethal triad of coagulopathy, acidosis and hypothermia [21,22].

Damage Control Resuscitation was first introduced into military emergency care in 2007 in order to co-ordinate several new advances in military medical care [23]. DCR represents a systematic method of trauma care integrating the <C>ABC approach to trauma with more invasive management to ultimately improve outcome. This involves haemostatic techniques early on in management, and the three stages of damage control surgery. The ultimate aim of these measures, apart from the resuscitation of the patient, is to avoid coagulopathy, acidosis and hypothermia.

Coagulopathy is a well known complication following severe trauma, haemorrhage or critical illness. This is associated with a poorer prognosis than patients with similar injuries without evidence of coagulopathy. Recent evidence from a 5-year retrospective review of a major trauma centre's experience demonstrated that trauma patients with an established coagulopathy upon arrival had their mortality increased from 10% to 46% [24]. This data correlates with that of the recent military experience in the Gulf conflict. Injured soldiers that required massive transfusion and an international normalised ratio (INR) > 1.5 upon admission had a mortality rate of 30%. In those casualties who required massive transfusion, but in whom the INR was <1.5 the mortality rate was as low as 5% [24].

There are several pathophysiological causes of coagulopathy in the critically ill patient. There is immediate trauma induced coagulopathy (ETIC) [see chapter 8]. Massive haemorrhage causes rapid depletion of the body's relatively small store of fibrinogen and platelets [24]. This is further exacerbated by the haemodilutional effect of hypotensive resuscitation with non-blood fluids such as crystalloids and colloids [25]. Large volume transfusion with packed red blood cells have a similar effect in diluting the quantity of clotting factors within the blood, thus perpetuating the coagulopathic effect. The early use of blood products is advocated and combining hypotensive with haemostatic resuscitation. Hypotensive resuscitation refers to treating shock by IV fluid therapy to increase intravascular volume. Haemostatic resuscitation, on the other hand, uses blood products early to restore tissue perfusion and normal coagulation [24]. Fluid resuscitation is aimed at maintaining the systolic blood pressure at around 90mmHg, thereby maintaining end organ function, until surgical control can be achieved, whilst reducing the risk of re-bleeding by dislodging any clot. There is evidence to support the early use of fresh frozen plasma (FFP) in resuscitation. Experience from recent military conflicts supports the early use of coagulation factors and FFP to address coagulopathy [25]. Current military protocol suggests infusion of FFP with red cells upon arrival in all patients that suffer major injury. Using FFP in a 1:1 or 1:2 ratios with red cells early in resuscitation has reduced mortality in military casualties with similar injuries.

In patients who suffer severe trauma, or those who are critically ill, hypothermia and acidosis often co-exist and exacerbate coagulopathy. Hypothermia is caused by several factors. Exposure to elements at the time of injury and haemorrhagic loss causes reduced tissue perfusion which results in a major drop in the body's oxidative metabolism. This effect is then exacerbated by aggressive volume resuscitation with room temperature fluids. The resultant hypothermic state causes major disruption to the body's coagulation pathways. The decreased body temperature causes decreased platelet activation, inhibition of Von Willebrand factor interaction and reduced enzyme activation of coagulation factor enzymes. Recent evidence suggests that these processes can double mortality [24]. Despite replacement of platelets and clotting factors, a body temperature of < 34 C increased mortality from 36% to 80%.

Metabolic acidosis is a common sequel after trauma or during critical illness. Reduced tissue perfusion increases lactic acid formation resulting in a fall in pH. This is often exacerbated by aggressive resuscitation with crystalloid solutions containing a chloride. is supraphysiological concentration of Normal saline known to cause/exacerbate hyperchloraemic acidosis by disruption of the Stewart model of acid base equilibrium. Acidosis affects the coagulation system by mainly inhibiting the function of clotting factors' interaction with cell lipid surfaces. Laboratory studies have shown that a significant reduction in pH can cause a reduction in the activity of Factor VIIa by 90% [24]. The co-existence of hypothermia and acidosis thus result in significant decreases in function of coagulation pathways.

Damage control resuscitation and surgery aim to restore physiological, rather than anatomical, function as soon as possible. This is done by reducing any haemorrhage and contamination, and reversing coagulopathy, acidosis and hypothermia promptly. Damage control surgery (DCS) forms part of the resuscitation in patients that are severely injured. The aim of DCS is to deliver the patient to the intensive care unit in a more stable physiological state [22]. This was first described in the 1990's and has replaced traditional 'all-in-one' surgery for military trauma [22,23]. The length of surgery is limited to 1 hour and aims at minimising haemorrhage and preventing secondary infection. Combined with good DCR, patients arrive at the intensive care unit with a near normal INR, stable haemodynamic parameters, warm, and non-acidotic [23].

SUMMARY

Critical care thus depends on good clinical observation and effective decision making, leading to rapid intervention to support organ function and prevent further physiological deterioration.

In patients who had major surgery, with serious trauma or surgical sepsis the physiological pathways of deterioration are the same and therefore the principles of physiological support is similar. Physiology often is different in pregnancy, young children and the elderly.

Rapid assessment and life saving interventions, followed by systematic review and organ support, all based on the ATLS principle of ABCDE, prevent later complications. The efficiency with which a patient is managed within the first few minutes or "golden hour"

will help determine the need for later complex salvage procedures and advanced critical care.

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2. PATIENT ASSESSMENT IN TRAUMA AND CRITICAL CARE

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ASSESSMENT OF THE TRAUMA PATIENT

Trauma care was revolutionised in the 1970's after the inadequacies of initial trauma care were publicised. This resulted in the start of the Advanced Trauma Life Support (ATLS) course [1]. Since then the 'golden hour' of trauma care has been based around an organised approach that aims at identifying and treating life threatening injuries. This approach can be applied to all critically ill surgical patients. Assessment follows the pattern of initial (**Primary**) assessment and **Resuscitation**, followed by a more detailed **Secondary** survey and further **Definitive** decision making and treatment.

The **Primary Assessment** constitutes the basis of trauma care and adheres to the following sequence:

Airway maintenance and C-spine control Breathing and ventilation Circulation with haemorrhage control Disability – address neurological status Exposure

This algorithm was developed to identify the conditions that will cause death most rapidly. This applies to all age groups, with some minor adjustments when dealing with paediatric or older patients. The principle of **Resuscitation** after major trauma or other surgical critical illness is to fix what you find as you do the primary survey, in the ABCD sequence (except where C-ABCD applies), because the systems below do not work without the systems above.

AIRWAY WITH C-SPINE CONTROL

It is vital that, upon initial assessment, the trauma patient's airway is assessed first for patency. This can be done as soon as the clinician sees the patient for the first time by addressing two questions: Is the patient communicating verbally? Are the airway sounds normal? If there is any concern the airway can be opened and inspected for foreign bodies. Soft tissue obstruction can be overcome with the chin lift or jaw thrust technique. The head tilt is not recommended as hyper-extension of the neck can cause damage to the spinal cord in cervical spine injuries. If there is any concern over airway patency it must be addressed immediately. This can range from simple airway manoeuvres or adjuncts to a definitive endo-tracheal tube or surgical airway.

Mandibular, facial and laryngeal trauma must be considered when performing the airway assessment. In certain circumstances the airway may be at high risk of developing progressive obstruction. These include burns or laryngeal trauma and the need for continued re-assessment is paramount.

In the patient with a reduced Glasgow Coma Scale (GCS) score, usually \leq 8, a more definitive airway is almost always required. This must be inserted by somebody with sufficient training and experience, as poor technique can precipitate further airway problems.

When assessing the trauma patient's airway, great care must be taken to protect the cervical spine from injury. All patients that sustained significant trauma must be assumed to have sustained a cervical spine injury until excluded. The patient's head and neck must be immobilised either manually by a member of the trauma team or through adjuncts such as a hard collar and blocks. A missed spinal injury can turn into an absolute disaster.

BREATHING

Assessing the trauma patient's breathing aims at identifying thoracic injuries that will cause rapid respiratory failure. These include tension pneumothorax, open pneumothorax, massive haemothorax and flail chest. Non-life threatening injuries such as simple pneumothorax or pulmonary contusion may not be identified until the secondary survey.

The patient is assessed by fully exposing the chest and excluding evidence of penetrating or blunt trauma. The clinician assesses air entry by visualising chest wall movements and auscultation. This allows flail chest to be identified. A flail chest occurs due to multiple rib fractures, and paradoxical inward movement of the flail segment of the chest wall will be seen with inspiration. Flail chest can be associated with significant pulmonary contusion that further impairs gas exchange.

Hyper-resonance on percussion of the chest wall can suggest tension pneumothorax. This results from a 1-way valve within the chest cavity causing air to build up in the pleural cavity. The resultant increase in pressure causes mediastinal shift and compression of the intra-thoracic structures. This results in obstructive shock by decreasing venous return to the heart and needs urgent decompression.

Massive haemothorax can be identified by decreased breath sounds and dullness to percussion on the affected side. Initial treatment consists of an inter-costal chest drain to improve ventilation and urgent restoration of intra-vascular blood volume. Drainage volume and tempo need to be monitored to indicate whether thoracotomy is required (>1500ml immediate blood loss [or one third of patient's intra-vascular volume], ongoing loss of >200 ml/h x 3 hours or >100ml/h x 6 hours, or haemodynamic compromise are almost always indications for thoracotomy).

There are instances where airway compromise can be difficult to distinguish from breathing problems; in that case intervene to support both. After major trauma all patients should receive a chest radiograph when safe and practical to do so. This should not halt or delay any resuscitative effort or initial assessment.

CIRCULATION

The largest number of preventable deaths in trauma victims is related to haemorrhage. Identifying haemorrhage and controlling it is the basis of circulation care. Always assume that hypotension in the trauma patient is secondary to haemorrhage until proven otherwise.

The patient's haemodynamic stability can be assessed by pulse rate and blood pressure. The clinician must use his clinical ability to further assess the patient's full circulation status. Level of consciousness and skin perfusion are useful measurements of perfusion that can be assessed quickly in the emergency situation. The character and rate of the pulse will also give the clinician vital information on the haemodynamic status of the patient. Urine output is another valuable marker of end organ perfusion, but requires catheterisation which may not be appropriate during the initial assessment.

If a patient's haemodynamic stability is compromised it essential to correct it before proceeding with the rest of the initial assessment. This involves restoring any intravascular volume with fluid or ideally blood. Haemorrhage control is crucial. In recent times, the military have replaced the traditional ABC method of assessment with <C>ABC, where <C> represents catastrophic haemorrhage. Recent evidence has shown that deaths due to major ballistic trauma can be reduced by controlling extremity haemorrhage by tourniquet application. This principle can be applied to civilian trauma with severe extremity injuries. A tourniquet on an extremity can, however, compromise all distal viable tissue and in many civilian circumstances direct pressure over the wound may be a preferred means of immediate control of haemorrhage.

The early use of blood products is advocated in improving haemodynamic resuscitation and avoiding coagulopathy, as discussed under DCR.

DISABILITY

The assessment of neurological status of the trauma patient is done at the end of the initial assessment. This establishes a measure of the patient's conscious level, pupillary size and reaction and evidence of spinal cord injury.

The Glasgow Coma Scale (GCS) score is widely used to measure a patient's level of consciousness. This is particularly useful to measure progress or deterioration after the injury. Some clinicians advocate the use of a quick alert score, such as **AVPU**, in the initial assessment and performing GCS during the secondary survey. (AVPU stands for the patient being <u>A</u>lert, responding to <u>V</u>oice or <u>P</u>ain stimuli, or being <u>U</u>nresponsive). It is crucial that the patient's blood glucose is determined and documented at the time of assessing neurological status, as this is often overlooked in the trauma scenario.

EXPOSURE / ENVIRONMENT

This is the final stage of the initial assessment and involves completely exposing the patient to identify any obvious injuries. At this stage it is important to ensure that there

are no haemorrhagic or penetrating injuries that were missed. The patient's temperature needs to be assessed and, if hypothermic, be corrected. This is crucial to improve survival. Mild hypothermia can be corrected using blankets or surface-warmers and warmed IV fluids. In severe hypothermia thoracic, bladder or naso-gastric lavage and ultimately cardio-pulmonary bypass may be necessary, where such facilities are exist.

ADJUNCTS TO PRIMARY SURVEY

This depends on availability in local hospitals:

 Imaging: X-ray of the chest / pelvis / cervical and thoracolumbar spine should always be performed, and make sure that the films are checked thoroughly by an experienced enough person. For the cervical spine the minimum requirement is a lateral film that shows all seven cervical vertebrae and T1.
 In some situations a patient will go to CT rapidly before a secondary survey can

In some situations a patient will go to CT rapidly before a secondary survey can be completed.

- 2) Take blood for:
 - a. Cross match of urgent blood products;
 - Electrolytes, basic haematology, clotting screen, arterial blood gases, serum amylase or lactate: when resources are limited these should not be done routinely but ask what the clinical usefulness of each result will be in this patient;
 - c. Blood cultures (or pus for gram stain and culture) if the patient is septic.
- 3) Urine catheter, but always check for possible urethral injury first.
- 4) Severely injured patients at risk of rapid exsanguination might need emergency damage control surgery as part of resuscitation.

SECONDARY SURVEY IN TRAUMA

The secondary survey should not be performed until the primary survey has been completed and the patient is in a physiologically stable condition. It allows the clinician to identify any further injuries and get an accurate history of the event; it is crucial to know the mechanism of injury. Physical examination is very important in patients that have a reduced conscious level and include

- Head and Neurological status
- Maxillofacial
- Full spine assessment not complete until the patient's back has been examined
- Chest
- Abdomen (including use of FAST / CT scan)
- Perineum / Rectum
- Musculoskeletal system.

Identification of occult injuries, such as of the hands and feet, is often not possible until the patient is of a suitable conscious level. Occult abdominal injuries can be missed with catastrophic consequences. Patients with unexplained hypotension and those that have reduced GCS scores must be evaluated with great care. The use of FAST ultrasound scan and CT imaging is extremely useful in identifying intra-abdominal injuries. The importance is not in diagnosing the specific injury but identifying that an injury exists early. Surgical advice is crucial.

SECONDARY SURVEY IN CRITICAL CARE

In a critically ill surgical patient it is important to remember that a secondary survey depends on much more than just examining the patient [2]. Information has to be gathered from:

- a) Pre-hospital care: speak to paramedics, police, family or friends, whoever brought the patient in. Use **MIST**
 - M = Mechanism of injury;

I = Injuries noted pre-arrival;

- S= Systems affected or in danger;
- T = Therapy given/started pre-hospital.
- b) Take a thorough patient history:
 - Speak to the patient.
 - Check the case notes: current notes, previous history, drug history, operation notes.
- c) Check the Charts:
 - Observation chart/HDU/A&E Charts.
 - Fluid balance chart: Intake-Output; think of unrecorded or hidden losses.
 - Check the trends, not single values.
 - Drug chart: Did the patient receive the prescribed drugs? At the correct time? Is the dose correct? What is the risk of interaction or side effects? (look it up in the formulary or MIMS or online yourself).
- d) Examination:
 - Examine the patient systematically (head to toe) yourself.
 - Remember spinal column and rectal examination in trauma.
 - Remember to look at drains and bags.
 - Think what you are looking for and why.
- e) Extra Information:
 - Check on all results yourself (Imaging, biochemistry, haematology, microbiology).
 - Speak to colleagues: microbiology, laboratories, radiology, pharmacy.
 - Recheck the charts for missing information.

f) If things don't fit, re-examine the patient or ask for senior review.

Once you have completed your thorough patient assessment it is essential to **DECIDE** and **PLAN** [©CCrISP, RCSEng] on what to do next (2):

- 1) **DECIDE** whether your patient is Stable or Unstable or whether you are Unsure:
- 2) If the patient is STABLE you must have a management plan for the day, i.e. a patient's care must not be allowed to simply drift through the day. A management plan can include that a patient is simply given a day to recuperate or for further rehabilitation but then it has to be recorded as such.

- 3) You must write down your management plan and this will typically include
 - diet, fluids, drugs, mobilisation;
 - physiotherapy if necessary;
 - discharge planning;
 - where you will be or whom staff can contact if necessary;
 - AND
 - you must tell the patient and relatives/guardian what is happening next and record what you have told them.
- 4) If the patient is **UNSTABLE** or if you are **UNSURE** it is essential to:
- a) Make a Diagnosis:
 - Ask WHY is the patient unstable or are things not clear?
 - Get the quickest and simplest investigation to give a definite answer.
 - Ask for help: senior review, other specialties, tertiary hospital.
- b) Start Definitive Treatment as quickly as possible:

Drugs/Surgery/Drainage/Refer/Transfer.

SUMMARY

Assessment of patients who have sustained serious trauma or are critically ill for other reasons e.g. after major surgery or with sepsis, need thorough assessment. This depends on a primary survey of critical organ organs, Airway, Breathing, Circulation and Disability, with immediate resuscitation to correct abnormalities found in any of these systems that are critical for survival. Once the patient is stabilised this is followed by a systematic secondary survey, which includes gathering information from pre-hospital care, meticulous history taking, review of all written patient information in case notes, letters, drug records, charts and results of imaging and laboratory investigations. Only then a decision is taken on whether a patient is stable or unstable. If the clinical picture is unclear it is not just accepted that a patient will somehow get better but the patient is regarded as unstable. For unstable patients there should be a definite plan to get to a diagnosis quickly and to start definitive therapy. If the situation is unsure a definite plan could be to re-assess a patient in 1-2 hours, but it should be no later than that. Once a patient is stable a definite management plan for the day should be written down so that care is never allowed to drift (2).

This basic approach to patient assessment is aimed at predicting which patients are at risk of deterioration, picking up signs of potential deterioration early and intervening early to support essential organ systems and maintain tissue oxygenation, and prevent later complications that will threaten patient survival and need more complex and expensive care. These are the principles that effective surgical critical care are based upon.

If, at any stage, the patient deteriorates or if things don't fit, one must go back to ABCDE, Resuscitation and Review of the secondary survey and treatment plan.

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An <u>Assessment Checklist</u> has been trialled in surgical emergencies in Ndola, Zambia, and Lilongwe, Malawi, and found to be very valuable in directing management [http://www.surgicalneed.nl/wp-content/uploads/2014/05/Dr.-Musowoya-Zambia.pdf].

ASSESSMENT CHECKLIST FOR CRITICALLY ILL SURGICAL PATIENTS

- Did I complete primary survey (ABCDE)?
- □ Have I completed resuscitation?
 - \Box O_2 ?
 - □ IV fluids?
- Did I complete secondary assessment?
 - □ History (Notes; Reports)?
 - Complete physical examination?
 - □ Chart review (Vital signs/MEWS; Fluid balance; Drugs)
 - □ Results?
 - □ Anyone I still wanted to speak to?
- □ Is my patient...
 - □ STABLE?
 - □ UNSTABLE?
 - □ Am I unsure?
- □ Is the problem...
 - Diagnostic
 - □ Therapeutic
 - Both
- Do I need to intervene...
 - Diagnostic?
 - □ Therapeutic?
 - □ Ask for help?
- □ Have I informed all parties what will happen next?
- □ Meanwhile, am I supporting ABC-T optimally?

(Anyone is allowed to use this checklist in their Emergency Rooms or Surgical Admission Units, with recognition to Alba CCCD)

3. RESUSCITATION ALGORITHMS

DAVID R BALL

INTRODUCTION

RESUSCITATION MAY BE DEFINED AS TO "RESTORE OR REVIVE".

The first definition is broad, where "resuscitation" is directed towards restoring acceptable physiological activity with a variety of treatments, such as oxygen, fluid, blood and electrolyte therapy, antibiotic administration *etc*. This may be a preparatory step for surgery, the resuscitation phase is aimed at optimising the patient's condition to reduce risk and therefore improve outcome.

The second definition applies to cardiac arrest, where therapeutic efforts are directed at achieving "return of spontaneous circulation" resulting from a life-threatening cardiac event. This event may be from a primary cardiac cause or secondary to other pathologies which have been grouped into the "4Hs and 4Ts":

- Hypoxia
- Hypovolaemia
- Hypo- or Hyperkalaemia (and other profound metabolic disturbances)
- Hypothermia
- **T**ension (pneumothorax)
- Tamponade
- Toxins (and overdose)
- Thrombosis (coronary or pulmonary)

For any form of resuscitation to succeed, it must be done as part of a series or rapid therapeutic *responses* which maximise the chance of a successful outcome. One guide uses the "5Rs".

- Recognise the problem and its severity.
- Review the patient in a rapid, time-sensitive manner.
- Request help.
- Resuscitate, aiming to "revive or restore" the patient
- **R**esource the situation, with drugs, equipment and/or people.

Life support involves two phases, Basic Life Support (BLS), followed by Advanced Life Support (ALS).

BLS involves chest compressions with ventilation. ALS adds to this with the administration of drugs (especially adrenaline) and (if indicated) defibrillation. ALS requires monitoring with an electrocardiogram (ECG) to inform decision-making. Provision of BLS with ALS must be done with management of potentially reversible pathology (the "4Hs and 4Ts")

Provision of BLS and ALS has been standardised into a series of "Life Support Guidelines", which aid decision-making in the highly time-sensitive situation of actual or

impending cardiac arrest. These include algorithms which use "*If-Then*" rules. For example *If* the patient has Ventricular Fibrillation, *Then* defibrillation is indicated. The 2010 algorithms are reproduced below (for adults, children and the newborn), with acknowledgement to the Resuscitation Council (www.resus.org.uk) These are under review and change every five years.

Outcome from resuscitation is very variable, since patients and their pathologies are variable. Attempts are worthwhile when three general criteria are applicable:

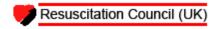
- 1. There is a reasonable chance of a successful outcome.
- 2. Attempts do not lead to prolonged suffering from treatment failure or futility.
- 3. There is no evidence available that the patient would not wish to be resuscitated.

2010 RESUSCITATION ALGORITHMS

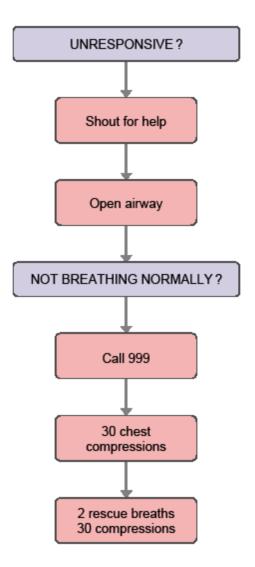
Reproduced with the kind permission of the Resuscitation Council (UK).

- 1. Adult Basic Life Support
- 2. In-hospital Resuscitation
- 3. Adult Advanced Life Support
- 4. Paediatric Basic Life Support
- 5. Paediatric Advanced Life Support
- 6. Newborn Life Support

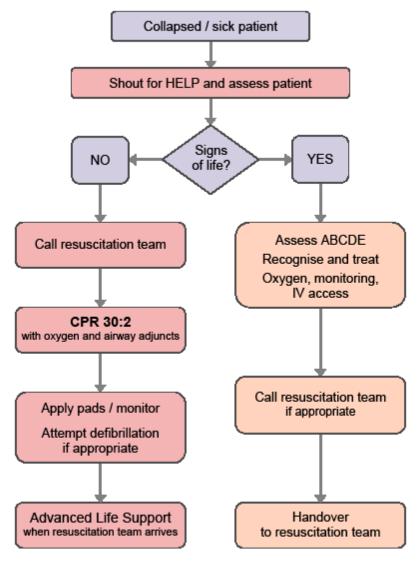


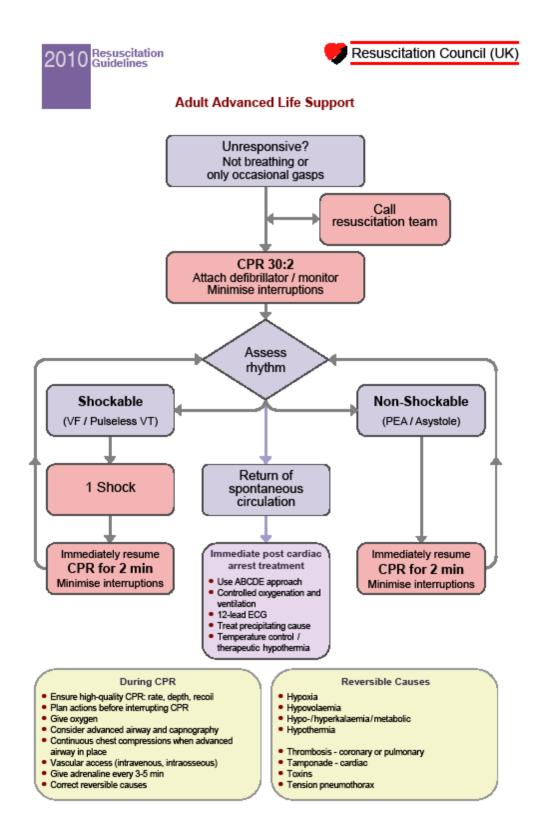


Adult Basic Life Support





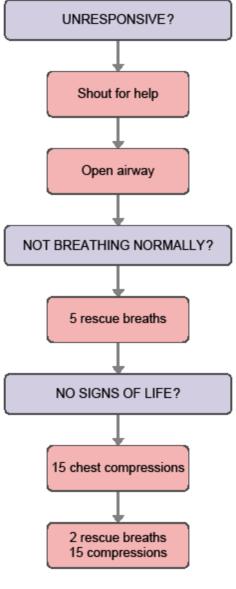




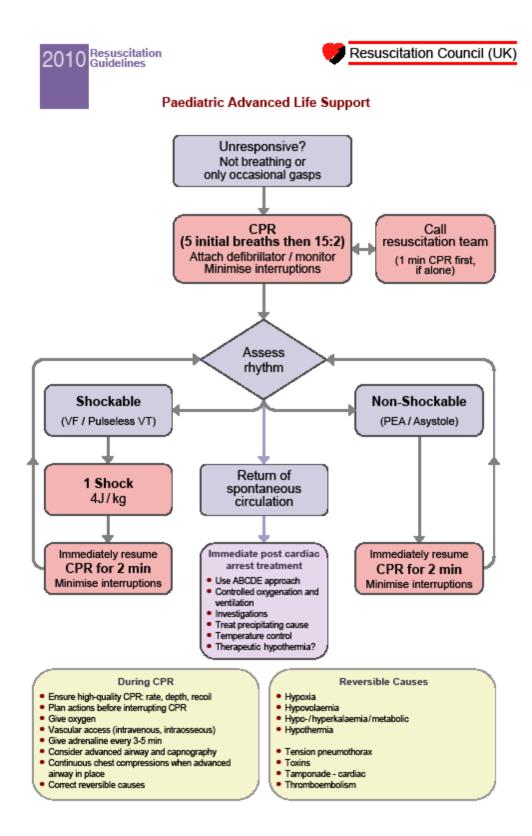


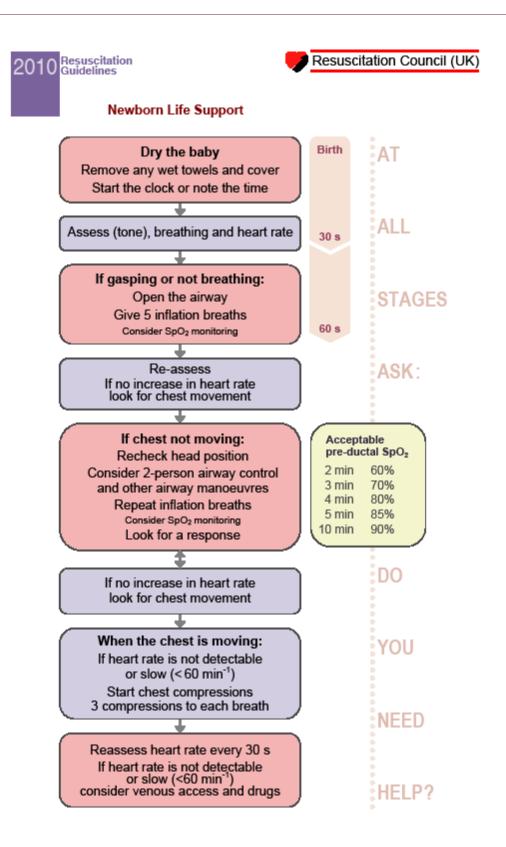


Paediatric Basic Life Support (Healthcare professionals with a duty to respond)



Call resuscitation team





4. AIRWAY MANAGEMENT IN TRAUMA AND CRITICAL CARE

DAVID R BALL

INTRODUCTION

Airway management gives benefit to the critically ill or injured patient in three ways. When done well, management allows *provision* of gas exchange, *protects* the lungs from aspiration injury and *permits* safe and effective treatments.

THE THREE BENEFITS OF AIRWAY MANAGEMENT:

1. PROVISION OF GAS EXCHANGE, DELIVERING OXYGEN AND REMOVING CARBON DIOXIDE.

Humans have an absolute need for oxygen, suffering organ dysfunction, damage and death if deprived of oxygen for more than a few minutes.

Carbon dioxide removal (technically clearance) is also necessary, but priority is less than for oxygen provision. Hypercarbia (increased CO_2 in the blood) is generally tolerated, perhaps for up to 1 hour. The exception is for a closed brain injury, where hypercarbia increases cerebral blood volume through vasodilatation, contributing to a raised intracranial pressure [1].

Oxygenation can be measured by cutaneous pulse oximetry but this may be difficult in patients with vasoconstriction when cold or shocked. Arterial blood gas, where available, is an alternative. Ventilation (carbon dioxide clearance) may be measured by continuous *capnography*, sampling CO₂ in expiratory gases. Detection of expiratory CO₂ provides simultaneous information on the *airway* (patency, since CO₂ leaves the lungs through the patient's airway), the *breathing* (ventilator efficiency), and the *circulation* (the ability of the cardiovascular system to transport CO₂ from the tissues to the lungs). Capnography provides information *rapidly* and in *real-time*. The CO₂ value at the end of expiration is called the end-tidal value. In healthy patients, the end-tidal CO₂ is about 0.5 – 1.0 kilopascal (4-7 mmHg) less than arterial CO₂. In ill or injured patients, especially with severe shock or chest injury, this linkage is lost and the discrepancy widens. This is due to increased dead-space ventilation of non-perfused lung tissue.

2. PROTECTS THE AIRWAY AND LUNGS FROM FURTHER INJURY.

There are two types of injury – *inhalational* and *iatrogenic*. Inhalational injury (aspiration) results from the entry of gastric content, blood, secretions, or debris into the lungs. This interferes with gas exchange directly (by physical obstruction) or indirectly (by provoking acute bronchospasm or delayed inflammation). Gastric content is acidic, highly toxic and contains particulates which can block the airway. Blood clots and debris from airway trauma also damage the lungs.

latrogenic injury due to misapplied instrumentation such as traumatic intubation attempts can cause upper airway obstruction from oedema. It can also result in contamination of the lower airway through aspiration of blood and debris. The lower airway and lungs may suffer instrumental damage.

3. PERMITS TREATMENT.

Safe and effective airway management allows interventions such as surgery and mechanical ventilation.

WHEN AIRWAY MANAGEMENT IS NEEDED

Airway management is required in two broad situations:

- 1. When the airway is directly injured.
- 2. When treatment of other organ systems is needed.

Sometimes both airway and organs need simultaneous treatment.

FACTORS INFLUENCING SUCCESS

Airway management has to be safe, effective and reliable. In trauma and critical care this can be challenging, requiring high standards of technical and non-technical competency. Technical skills are related to effective performance of tasks. Non-technical skills encompass communication, teamwork and decision-making which contribute to effective *judgement*. Judgement is increasingly appreciated as a dominant factor in success or failure, life or death. The National Audit Project 4 analysed all serious airway incidents reported in the UK over a year (2008-9). These included any unplanned tracheostomy or cricothyroidotomy, death or unplanned admission to intensive care because of an airway management problem [2]. The majority of the events resulted from poor judgement, followed by problems resulting from lack of education and training. The report highlighted the increased relative risk of an airway-related death occurring in the Emergency Room and Critical Care compared to the Operating Room: about 35 and 55 times more, respectively. These are the most challenging areas in which to provide airway management, because of four interacting factors: *complexity, risk, uncertainty* and *dynamism* [3].

Complexity arises from multiple interactions between the patient, his/her problems, and the procedures and personnel involved in treatment. Added to these are the provision of drugs and equipment, and transportation.

Risk arises from the severity of illness or injuries, and possible complications of interventions.

Uncertainty arises from the unpredictable nature of interacting factors and from the possibility of a hidden pathology.

Dynamism describes the rapid, time-sensitive changes in patient physiology and the recognition and response to these by care providers. As mentioned, the time course of

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complications resulting from airway interventions, most importantly hypoxia, is measured in minutes.

THE BALANCE OF BENEFIT TO HARM

All interventions in medicine and surgery involve an assessment of expected benefit versus potential for harm. The balance of these is risk. The common, recognized **risks** of airway management include:

- 1) Failure to plan and prepare, leading to unexpected or unmanaged complications.
- Failure to achieve airway control, leading to life-threatening or lethal loss of gas exchange (especially hypoxia) and loss of airway protection with aspiration lung injury.
- 3) Success, but with immediate complications such as dental damage, airway injury with bleeding, swelling or injury to the larynx, especially the vocal cords.
- 4) Success, followed by delayed complications such as intubation-related pneumonia, tube blockages or unplanned extubation of the trachea.
- 5) Each of the above can affect future confidence, with future problems arising from performance anxiety, aversion, or avoidance of responsibility.

THE SYSTEMATIC APPROACH

Outcomes improve with safe, sensible and systematic approaches to all aspects of patient management. The systematic approach prioritises treatment responses in the order *Airway*, *Breathing*, *Circulation*, *Disability* and *Exposure*. Airway management is the first priority. The rare exception is for patients with catastrophic exsanguination, for whom the "<C> ABC approach" is necessary [4].

For all trauma patients, airway management is co-incident with restriction of cervical spine movement, since instability of the cervical column risks catastrophic spinal cord damage if the head is moved. All trauma patients with actual or possible neck injury should therefore be managed with neck immobilisation by rigid cervical collar, with further restriction of head movement (e.g. by use of head blocks and tape) and initial treatment on a spinal board. These initial interventions (which aim to immobilise the head in a neutral position), place restrictions on access to and mobility of the patient's airway. When tracheal intubation is planned, the collar, tapes and blocks should be temporarily removed, the head held in neutral by an assistant, providing "manual in-line stabilization" (MILS). Airway interventions should be carried out with the head held in this neutral position, which usually reduces airway patency and increases difficulty in achieving tracheal intubation. Once intubation is achieved and confirmed, the immobilisation should be restored until the neck is 'cleared', (deemed stable and not a danger to the patient).

Safe and effective airway management depends on three factors: *Personnel, Provision* and *Planning.*

- 1) **Personnel:** wherever possible, management should be provided by skilled, trained staff with dedicated, trained assistants.
- Provision: appropriate equipment should be available at all times. Drugs are necessary for some types of airway control, notably rapid sequence induction (RSI) for oro-tracheal intubation.

3) **Planning:** this includes the rostering of staff, resourcing of drugs, teaching and training. Both individual and group planning for the management of expected (and unexpected) injuries should be an ongoing, fixed part of every care provider`s job.

The positive interaction of these factors allows the formulation of a safe "Airway Management Strategy" for your patient [2]. This is a logical, co-ordinated sequence of plans aimed at providing good gas exchange, protection from injury and permitting further treatment. Have a series of plans in a sequence of potential approaches to airway management: use Plan A, Plan B, Plan C then Plan D if needed. These plans vary according to patient need and practitioner competence, but the priorities (providing gas exchange, protection from aspiration and permitting further treatment) would apply to all patients. This type of management forms the basis of the Difficult Airway Society's (DAS) guidelines on the management of unanticipated difficult tracheal intubation [5]. The Guidelines apply Plans to three clinical scenarios: unexpected difficult intubation for elective surgery, unexpected difficulty during an RSI and failed tracheal intubation with failed facemask ventilation, the so-called "CICV" situation ("Cannot Intubate Cannot Ventilate"), more correctly called "CICO" ("Cannot Intubate Cannot Oxygenate"). The second and third scenarios are relevant to airway management in trauma and critical care. For example, in a patient with facial injuries who needs a life-saving laparotomy for traumatic shock, Airway management is: Plan A is RSI tracheal intubation, Plan B temporary use of a Supraglottic Airway Device (SAD) e.g. a laryngeal mask airway and Plan C a rescue tracheostomy or cricothyroidotomy. If the surgical indication was less critical, another sequence would be that Plan A is RSI tracheal intubation, Plan B abandoning the anaesthetic and using facemask ventilation (until the patient woke up), Plan C a SAD if ventilation fails and Plan D a tracheostomy or cricothyroidotomy if CICO develops.

These guidelines stress the need for a Strategy (planning), early and prompt recognition of failure, calling for help early and limiting attempts at tracheal intubation. DAS currently advices no more than three attempts at tracheal intubation for an emergency case. Others advise no more than two attempts for a critical care patient, since major complication such as cardiac arrest and aspiration risk increases to about 20% with more than two intubation attempts [6].

DECIDING ON AN AIRWAY MANAGEMENT STRATEGY

Planning an Airway Management Strategy for each patient is *aided* by asking the following questions. ("the five As of Airway management")

1) Assess immediate risks. The two big risks are anoxia and aspiration. Anoxia (the desperate form of hypoxia) may be evident from the colour of mucous membranes, but this sign is lost in severe anaemia or bleeding. Oximetry is more accurate, but may be lost with vasoconstriction, hypothermia or in hypovolaemic, obstructive or cardiogenic shock. Hypoxia requires immediate treatment. All trauma patients have an aspiration risk, where gastric content can enter the lungs. Those with depressed airway reflexes or facial injuries are especially at risk. Risk of aspiration can be reduced through a tilting table, access to suction and the application of cricoid pressure during tracheal intubation.

- A, B, C and D? What plans will form your strategy? These determine steps 4 and 5.
- Awake or asleep? e.g. tracheostomy under local anaesthesia (=awake) or orotracheal intubation following Rapid Sequence Induction of anaesthesia (RSI) (=asleep). The choice is informed by considering the *safest* option.
- 4) **Above or below the vocal cords?** For patients with severe facial injury, airway control below the vocal cords by temporary cricothyroidotomy or tracheostomy is a reasonable choice, probably under local anaesthesia.
- 5) *Afterwards?* Once airway control has been achieved, ask "What next for your patient?" Consider when and where airway devices can be safely removed.

Your Airway Management Strategy may be *altered* by asking these questions ("**SLADE**"). These factors are also known as *context modifiers* [7],:

- 1) Skills? What skills do you and your team possess?
- 2) **Location?** Where is your patient? Management in the ward may have to be different from the Emergency Room or Operating Theatre because your resources, drugs, equipment and personnel will most likely be different.

3) **Assistance?** Teamwork with good communication and decision-making improves outcome.

4) Destination? Should your patient be moved to another site for treatment?

5) *Equipment?* What airway devices are available to you immediately or from elsewhere in the hospital?

THE FIVE APPROACHES TO AIRWAY MANAGEMENT

Five approaches can be incorporated into a series of plans (A,B,C,D) as part of an individualized airway strategy tailored to patient need. These approaches require a variety of skills, equipment [8], drugs and assistance to achieve successful outcome. Each requirement should be considered in a *proactive way*, since the complex, risky, dynamic and uncertain nature of airway management makes it hard to safely salvage a situation later.

These principles apply to airway management for adults and children, bearing in mind that children are different in *Physiology, Psychology* and *Physical status* (size and shape).

They also apply to the pregnant patient. Note that the third trimester brings restricted lung volumes, higher volumes of gastric content and a greater risk of regurgitation. Importantly, a pregnant patient must be managed with at least a 15 degree tilt when lying down to reduce the risk of supine hypotension resulting from inferior caval compression.

To repeat - airway management is carried out together with measures to protect from cervical spine injury. If cervical spine precautions are removed to permit airway intervention such as tracheal intubation *above* the vocal cords (oral intubation) or *below*

the cords (tracheostomy or cricothyroidotomy), an alternative form of immobilization, namely MILS should be applied by a staff member dedicated to the task.

1. FACEMASK VENTILATION

- A. *Open* type mask, i.e. there is no effective mask seal with the patient. A lightweight plastic mask with reservoir bag (so-called `trauma mask`) is usually used. Oxygen should be supplied at at least 10l/min. Expiration is *via* the expiratory ports in the mask.
- B. Closed type mask, used to provide facemask anaesthesia. The mask has an edge (often with an inflated cuff), a body, and a mount. A connector on the mount joins the mask to a breathing system (or circuit), which is in turn linked to either an anaesthetic machine or oxygen source. It is important to pull the patient's face up into the mask and not press the mask down onto the face.

Breathing systems are usually of the Bag-Valve-Mask variety (systems for children lack the valve). A seal between the mask edge and the patient's face produces most efficient oxygenation, with expiration *via* the valve. The reservoir bag provides a volume of oxygen for inspiratory flow. A patient may breathe spontaneously through this device (with the expiratory valve open) or receive *assisted* positive pressure breaths by variably closing the expiratory valve. This is sometimes called manual ventilation.

A common form of bag-valve-mask system has a self-inflating bag and a fixed expiratory (`fish-lip`) valve. This is derived from the original "Ambu" [™] variety and is usually available in three sizes – for babies (500ml volume bag), children (1000ml) and adults (2000ml). This is the only system which can generate positive pressure for ventilating an apnoeic patient without external oxygen; oxygen comes from inspired air (21%). Other breathing systems have manually controlled valves.

Effective bag-valve-mask ventilation requires the skill to acquire and maintain a facemask seal, to judge inspiratory and expiratory rates, and force of manual ventilation whilst monitoring patient status.

The ability to achieve facemask ventilation varies [9]. Severe facial injury, use of a cervical collar, blood and secretions are common limitations in trauma. Pre-existing anatomical factors may complicate matters. A 5 point (Han) grading system to describe ease of facemask ventilation has been proposed [10].

Grade 0: facemask ventilation not attempted

Grade1: easy, no other equipment or help needed.

Grade2: moderate difficulty, oral or nasal airways needed.

Grade 3: difficult, oral and/or nasal airways needed with a two-handed mask-holding technique, with an assistant compressing the bag.

Grade 4: impossible to achieve gas exchange.

With trained staff Han grade 4 only occurs occur in 1 in 20 000 patients.

For a patient with known or suspected unstable neck injury, movement and manipulation of the obstructed airway is limited to elevation of the mandible. This is the so-called `jaw

thrust', aimed at lifting the `anterior structures' of the mouth (tongue and submental tissue) away from the posterior pharyngeal wall, thus restoring or improving airway patency. Insertion of an oro-pharyngeal airway (Guedel) may be useful in this situation (Han grade 2), but tolerance depends on significant depression of airway reflexes, equivalent to a Glasgow Coma Scale ≤ 8 . Therefore consider providing a more secure airway, *e.g.* tracheal intubation. A nasopharyngeal airway (one or both nostrils) is better tolerated, but avoid if base of skull fracture is possible.

Bag-Valve-Mask ventilation is the "first and fall-back" method of providing gas exchange for a patient with apnoea resulting from "disease or drugs" (injury, illness, poisoning, sedation or anaesthesia). The lungs remain unprotected from any form of aspiration and gas may be forced into the stomach. Cricoid pressure (more accurately force), when applied by a trained assistant, can reduce both of these risks. Cricoid pressure (force) is designed to occlude the oesophageal lumen against the sixth cervical vertebral body (in adults, higher in small children). This is done by pressing with the index finger onto the patient's cricoid cartilage while the thumb and middle finger (of the same hand) prevent the larynx from lateral displacement. The aim is to transfer force posteriorly through the cartilage without deformation of the larynx.

A force of 30 Newtons is advised (about 3kg equivalent). Effective cricoid force can achieve two goals, namely to reduce the risk of gastric dilation during facemask ventilation, especially in children, and reduce the risk of regurgitation of gastric content during facemask ventilation or tracheal intubation. Cricoid force is not intended to manage vomiting. Vomiting is an active reflex process, usually with co-ordinated expulsion of gastric content with glottis closure. Regurgitation, however, is a passive process and aspiration lung injury may occur when airway reflexes are lost during disease or drug therapy (anaesthesia or sedation). In real-life it can be difficult to distiguish vomiting from regurgitation.

There are recognised problems with use of cricoid force. These are difficult facemask ventilation or difficulty passing a tracheal tube, resulting from distortion and compression of the airway. This can happen with misapplied or appropriate force [11].

2. AIRWAY CLEARANCE

Blood, secretions and debris may obstruct the airway, especially with impaired protective reflexes. Careful use of a suction device can clear fluid. Rigid (Yankauer- type) suckers or flexible catheters are used. Care should be taken to avoid further trauma, provoking reflex coughing, retching and vagal responses (bradycardia, bronchospasm) from deep pharyngeal or laryngeal stimulation. Inspection of the oral cavity with a light source may reveal solid debris, which can be removed with forceps, such as the Magill type. Be careful not to push debris into the airway.

Suction through a tracheal tube using soft catheters may be needed to clear blood or secretions. Risks include hypoxia from sucking oxygen from the patient's lungs, airway trauma and parasympathetic reflexes such as bradycardia and bronchospasm.

In severe mid face injury (Le Fort 3 type), the maxillae and nasal pyramid may obstruct the upper airway when supine. A conscious patient will seek to sit up in an effort to open the airway, but an obtunded or unconscious patient is in great danger. The mid face should be elevated to open the airway. Suction and posterior packing of the nasal cavity (with tethered swabs or balloon catheter) may be used to reduce bleeding.

3. TRACHEAL INTUBATION

This procedure is the passage of a suitable tracheal (also called an *endotracheal* tube) through the airway so the tube tip is positioned within the mid trachea.

It is usually the optimal form of airway control for critical patients, proving *patency* for gas exchange, *protecting* the lower airway and lungs from aspiration (using an inflatable cuff) and *permitting* effective positive-pressure ventilation.

The most common approach is oral intubation, passing a cuffed tracheal tube through the oral and pharyngeal cavities, through the glottis (the aperture between the vocal cords) into the larynx, so that the tube tip lies in the mid trachea. A cuff is inflated to provide an airway seal. A pressure seal to 30cm water is the upper safety limit for adults (20cm water for children) [12]. Sustained higher pressures risk impairment of mucosal blood supply, injury, bleeding and longer term problems such as subglottic stenosis. Until recently cuffed tubes were restricted to adult patients, but cuffed paediatric tubes are now accepted, provided that size selection and cuff inflation (preferably with a pressure-measuring manometer) is carefully controlled.

Selection of tube size (diameter) and length is very important. The diameter is traditionally specified as ID (Internal Diameter), measured in mm, even though it is the external diameter of the tube which determines ease of insertion. Tubes are available in 0.5mm increments. For average adult males a tube of 8.5 to 9.0 mm ID is usually acceptable, for females 7.5 to 8.0 mm ID. The tubes must be sufficiently wide to allow easy passage of suction tubes. Excessive force must not be applied during tracheal intubation. A smaller size should always be immediately available. Length of insertion is also important. If placed too far, the tube will enter a main bronchus and only one lung ventilated; the other lung will collapse. For adult males insertion length is 20-23 cm, for females 19-22cm. This should be checked and altered if needed (See Post-procedure checks, below).

Tubes may be cut to desired length which reduce the risks of tube kinking, aids passage of a suction catheter and reduces the work of breathing when a patient is self-ventilating. For a patient with facial burns, however, the tissues can swell markedly and it is wise to leave the tracheal tube uncut.

Tube selection for children depends on the age and size of the child. There is variability, so checking for correct sizing is crucial. Again, alternative sizes must be immediately ready for use.

One set of formulae for selection is [13]: (ID in mm)

Neonate to 3 months	s: 3.0 length 10 cm	
3-9 months:	3.5 length 11 cm	
9-21 months:	4.0 length 12 cm	
>21 months:	(age in years) /4 +4	length = (age in years) /2 +12 cm

Formulae for tube selection correctly calculate appropriate sizes in only 30% cases. Ultrasound assessment at the level of the cricoid cartilage will double success [13].

A variety of tube types are available. For adults, the commonest are the precurved cuffed tubes. Tubes reinforced with spiral wire are less prone to kinking and often used in neurosurgery and head and neck cases, but may be more difficult to suction through. Double lumen tubes are used in thoracic surgery to selectively isolate and collapse one lung. In trauma these can be used with massive bleeding in one lung to prevent the non-injured lung from drowning.

Tracheal intubation requires visualization of the laryngeal inlet with a *laryngoscope*. Traditional laryngoscopes are either straight or curved-bladed and permit visualization of the laryngeal inlet by elevating the epiglottis. For curved blades (Macintosh type) this is achieved *indirectly*. The tip of the laryngoscope blade is inserted into the valleculla followed by antero-inferior traction on the laryngoscope handle sufficient to tension the hyo-epiglottic ligament, raising the epiglottis, exposing the glottis to direct view, a "line of sight". For straight blades (Miller or Magill type), this is achieved *directly*. The blade is positioned posterior to the epiglottis followed by antero-inferior handle traction and the epiglottis is lifted directly. The straight blade technique is harder to learn and results in a greater level of airway stimulation, but with training, is the superior in terms of success. The curved blade method is easier to learn because there is a definite target in placing the blade tip in the vallecula, and nowhere else. For this reason the curved blade method is by far the most popular approach for adult patients.

Both blade methods allow tracheal intubation by achieving two goals:

- 1. A "line of sight" is gained between the intubator and the larynx. The patient's oral, pharyngeal and laryngeal planes are more closely aligned, but this is hindered in the trauma situation because of consideration of neck injury.
- 2. An "airway space" or "working space" is achieved by correct technique, with antero-posterior separation of tissues to allow tube passage through the upper airway and the larnyx. At least 25 successful attempts at intubation "in context" (in the setting of trauma with MILS applied) is necessary for competence. Failure is always a possibility, hence the importance of back-up Plans B, C and D.

All laryngoscopes suffer a major drawback in trauma or critical care: the view is lost with any airway soiling by blood, debris, vomit or secretions. Unless these can be cleared by suction or removal, the device will not aid intubation and other Plan is needed quickly.

Curved blade laryngoscopy is more likely to fail in one of four situations:

- 1. A base of tongue or vallecula lesion prevents successful location of the blade.
- 2. Prominent upper anterior dentition impede blade placement to allow the "line of sight".
- 3. Use of a curved blade requires displacement of the tongue into the mandibular space. If, for anatomical reasons, this volume is small (*e.g.* a short, receding jaw) the tongue cannot be accommodated (it contains blood and cannot be compressed) and is displaced posteriorly This impedes blade progression into the vallecula and prevents acquisition the "line of sight".
- 4. If a patient's epiglottis is big, long or floppy (*e.g.* a baby) it may not be successfully elevated.

For each of these reasons, a straight blade technique using a "paraglossal approach" [14] may be more successful.

The Cormack and Lehane grading scale for the laryngeal view is widely accepted, but only applies to curved blade laryngoscopes:

Grade 1: all the glottis is seen.

Grade 2: the posterior portion of the glottis is seen, the posterior commissure.

Grade 3: only the epiglottis is seen.

Grade 4: only the tongue is seen.

Grade 3 is considered to be "difficult". Grade 4 is usually considered to be "impossible". Application of MILS, and performing the intubation attempt in the neutral position (as opposed to extending the head on the atlanto-axial complex and flexing the lower neck, which is done during elective anaesthesia) usually increases the view by one grade.

Other laryngoscopes are available. A variety of Rigid Indirect laryngoscopes have been introduced [15]. They share a common feature, in that they do not need to form a straight "line of sight" to view the glottis and are marketed for patients with "difficult airways". They allow the intubator to "see around the corner" by using a variety of technologies: rigid fibreoptic bundle (Bullard[™], Upsher[™], Wu[™] laryngoscopes), digital camera ("videolaryngoscopes" such as the McGrath 5[™], AP Advance[™], C Mac[™], AWS[™] etc) or optical prisms and lenses (Airtraq[™]). These devices generally give a good view of the larynx, provided the patient can open their mouth sufficiently, but passage of a tracheal tube may be problematic because they do not align airway axes and create limited "airway space". There is currently little evidence to determine which device is superior for difficult patients [16].

The flexible fibre optic bronchoscope is a versatile device which can be used for tracheal intubation, upper and lower airway inspection and lung toilet. It is however, expensive, delicate, requires intense decontamination and a high degree of skill to be used well. Even when available, it is not routinely used in the trauma setting.

The upper airway, especially the larynx, has a dense nerve supply serving speech, breathing, swallowing, coughing and gagging. Vocal cord closure is termed laryngospasm.

Penetration of the larynx with a tracheal tube requires profound suppression of reflexes. These include the motor reflexes mentioned above and also autonomic responses, both sympathetic (provoking tachycardia, arrhythmia and hypertension) and parasympathetic (provoking bradycardia and bronchospasm). Upper and lower airway secretions increase.

For patients requiring tracheal intubation, the most effective approach is to provide an anaesthetic as part of a rapid sequence induction (RSI). There are usually three parts to the anaesthetic: *unconsciousness*, *immobility* and *reflex suppression*. A hypnotic agent provides unconsciousness, a neuromuscular blocking agent (sometimes called a muscle relaxant or paralyzing drug) provides muscular immobility or relaxation and thirdly a reflex suppressing drug, e.g. an opioid, may be given. An anticholinergic agent (e.g atropine) may be given to reduce secretions or risk of bradycardia.

An obtunded patient successfully intubated without recourse to drugs is usually desperately ill or injured, and the prospects for survival are low.

Giving an RSI anaesthetic is a complex, co-ordinated procedure, best achieved by a trained team, optimally of four people:

- 1) The airway intubator.
- 2) A person to provide cricoid force.
- 3) A person to stabilize the patients's head, providing MILS. (when neck injury is known or suspected)
- 4) A person to supply the required equipment directly to the intubator.

The RSI usually proceeds as follows:

- 1) The team understands the situation.
- 2) The team has a planned airway management strategy.
- 3) Drugs and equipment are readily available, monitors applied and vascular access achieved.
- 4) Drug doses are calculated and equipment sizes estimated.
- 5) The cervical collar is removed and MILS applied.
- 6) Oxygen is administered using a facemask via a bag-valve-mask system for 3 minutes.
- 7) An induction agent is given (*e.g.* sodium pentothal, ketamine).
- 8) Following loss of consciousness, cricoid force is applied. (30 Newton force, equivalent to a weight of about 3 kg)
- 9) A neuromuscular blocking agent (*e.g* suxamethonium) is given. Intravenous opioid or lidocaine may be used to obtund autonomic responses (*e.g.* in head injury). Intravenous anticholinergics (*e.g.* atropine) may also be given.
- 10) Clearance of airway secretions may be needed.
- 11) Oral intubation is achieved. If the view of the glottis is restricted, a tracheal introducer ("bougie") may be inserted and used to guide the placement.
- 12) Two or three attempts are advised. If these fail, resume mask-ventilation immediately.
- 13) The tracheal cuff is inflated, the seal confirmed, the breathing system is connected to the tube and positive pressure ventilation is started.
- 14) Post procedure checks confirm successful placement.
- 15) Cricoid force is removed.
- 16) Post procedure management is started.

There are *four* post procedure checks:

1. Most importantly, the tube must be in the *trachea*. If not, hypoxia will start immediately. Confirmation is achieved *clinically*, with *capnography* (where available) or by compression of the bulb of an oesophageal detector device. The device is attached to the tracheal tube and the bulb compressed. If the tube is placed in the trachea, the bulb will quickly re-inflate. If the tube is in the oesophagus, the bulb will remain deflated.

Clinical confirmation uses the human senses: *Look, Listen, Feel.* Ideally, *look* to see the tube passing through the cords and then look for chest expansion resulting from positive-pressure ventilation. *Listen* (using a stethoscope) on both sides of the chest for breath sounds *and* listen over the epigastrium to exclude

noise resulting from unintended esophageal placement. *Feel* (with your hand) for chest expansion during ventilation.

Capnography provides the best way to confirm tracheal intubation, with detection of expired CO_2 (after four expiratory cycles). Unrecognised oesophageal intubation is catastrophic. **Remember: "When in doubt, take it out!**"

- 2. Confirm that the cuff seal has been achieved by listening to exclude an audible leak during positive-pressure ventilation.
- 3. Confirm that both lungs are effectively ventilated. A tracheal tube inserted too far into the airway will penetrate a main bronchus (usually the right in adults), resulting in one-sided chest expansion and one-sided auscultation with subsequent risk of hypoxia and hypercarbia. The tube should be withdrawn.
- 4. Chest radiography should be performed to provide information about tube position, bony integrity and lung shadows. This is best done during the post-procedure management phase.

Post-Procedure Management includes:

- 1. Secure the tube at the correct depth. The choices include: a tie (avoided in head injury as it can obstruct venous drainage), tape, or (when secretions or bleeding is problematic) fixation to a secure tooth with dental wire.
- 2. Insertion of a bite-block (rolled-up gauze) between the molar teeth to prevent biting onto the tube.
- 3. Reapplication of cervical spine immobilizing devices.
- 4. Gastric drainage *via* an oesophageal tube. For head injury, the wisest route is oro-gastric tube placement.
- 5. Action of drugs given for RSI wear off and therefore effective sedative therapy such as a propofol or benzodiazepine infusion is needed to facilitate further management. Further neuromuscular blockade may be needed.

Failed tracheal intubation:

The priorities are *provision* of oxygenation and *protection* from aspiration. Options include: resort to facemask ventilation (Approach 1), temporary supraglottic airway (Approach 4) or a "Front of Neck" approach (a form of subglottic management, either cricothyroidotomy or tracheostomy – Approach 5). Alternatively, where safety dictates, ventilation may be supported (Approach1) until the patient recovers and wakes up.

4. SUPRAGLOTTIC AIRWAY

Supraglottic Airway Devices (SADs) are midway between facemask ventilation and tracheal intubation in terms of anatomical location, invasiveness and security. All are inserted blindly.

They are very commonly used in elective anaesthesia and difficult airway management, either expected or unexpected [17].

A SAD is designed to form a periglottic seal. This allows for positive pressure ventilation, at pressures less than 20 cm water for early devices.

The original device is the laryngeal mask airway (LMA), now called the `Classic LMA`, and many types are now available, both reusable and disposable. Most SADs are of the `first generation` type, providing airway patency with a low pressure seal. They do not provide protection from regurgitation and aspiration of gastric content, but may provide protection from aspiration of naso -or oro-pharyngeal bleeding (the so-called `umbrella effect`).

"Second generation" SADs have one or more additional features designed to increase efficacy and safety, such as increased pharyngeal seal pressure, a gastric drain tube (to achieve functional separation of the aero-digestive tract) and an integral bite block. The archetypal second generation device is the ProSeal ™ [18].

SADs are best for elective anaesthesia where risk of regurgitation is low. They also have a place in the management of a *failed tracheal intubation* where hypoxia is developing. They are usually placed easily (cricoid force should be removed to allow the tip of the device to position properly) and can rescue and temporize a dangerous situation, permitting other responses, (such as tracheostomy) to be used. The best device to use is the ProSeal[™].

These devices can also be used as an intubating conduit, guiding one of a variety of intubation aids, such as the Aintree Intubating Catheter [™] or tracheal introducer.

In a failed tracheal intubation during an RSI, laryngeal masks can be life-saving, but they are temporary airway devices. They are recommended in guidelines for use in unexpected difficulty and failed tracheal intubation with life-threatening hypoxia [5].

5. SUBGLOTTIC AIRWAY MANAGEMENT (CRICOTHYROIDOTOMY OR TRACHEOSTOMY)

This is the fifth and final approach to airway management, the so-called `Front-of-Neck-Access` or "Emergency Percutaneous Airway" [5,19] which includes either cricothyroidotomy (a temporary approach) or tracheostomy (a definitive airway).

These may be the first choice for airway control, for example in complex maxillofacial injury or as part of a rescue plan when other approaches have failed. Either may be achieved with local or general anaesthesia, and both are more easily performed when a cervical collar is removed and MILS done.

<u>Cricothyroidotomy</u> is done using *narrow* or *wide bore* devices.

A: Narrow bore (<2mm internal diameter) include catheter-over-needle devices, most simply an intravenous cannula, or with a dedicated cannula such as the Ravussin[™] catheter, attached to an aspirating syringe during placement. There are five major considerations:

1. Accurate placement *via* the cricothyroid membrane into the airway lumen is vital. Paratracheal or oesophageal placement of either needle or cannula must be avoided. Careful syringe aspiration during the needle procedure and following placement coupled with confirmation with capnography will avoid this complication.

- 2. Cannulae (especially intravenous types) are prone to kinking.
- 3. The resistance to oxygen flow *via* the cannula is sufficiently high that a dedicated high pressure jet ventilation device (e.g. the Manujet[™] system) is needed. Anaesthetic breathing systems cannot generate sufficient pressure to inflate a patient's lungs.
- 4. Expiration must be achieved through the patient's upper airway.
- 5. There is no protection from aspiration.

Narrow bore cricothyroid cannulae used with jet ventilation is more useful in controlled elective situations.

B: Wide bore devices (>4.0mm internal diameter) usually have greater utility in the setting of the trauma airway. Various devices are available which rely on a dilational step with wire guidance (Melker $^{\text{TM}}$) or without (QuickTrach $^{\text{TM}}$). Some of these tubes have an inflatable cuff, offering better protection form aspiration and aiding ventilation.

A wide bore cricothyroidotomy device or tracheal tube may be placed using a `surgical` approach. One method is the 4 step approach [20]:

- 1) Identification of the membrane.
- 2) Horizontal scalpel stab incision through the skin and cricothyroid membrane.
- 3) Insertion of a tracheal hook (superior or inferiorly) to control access.
- 4) Insertion of a tracheal tube, preferably cuffed. Care must be taken to avoid endobronchial placement.

A wide bore device permits both inspiratory and expiratory flow through the artificial airway. It protects from aspiration when a cuffed airway is inserted and successful ventilation can be achieved with standard breathing systems without recourse to a jet ventilator.

Tracheostomy is an alternative but is somewhat more technically demanding since tracheal access is deeper in the neck than the cricothyroid approach. The isthmus of the thyroid may have to be divided with considerations of haemostasis. Open and percutaneous (Seldinger) techniques are available. In trauma or critical care tracheostomy should only be performed by an experienced operator.

Once inserted, verification of correct placement of either wide bore cricothyroidotomy or tracheostomy is done in the same way as for oral insertion of a tracheal tube.

SUMMARY:

Airway management allows *provision* of gas exchange, *protects* the lungs from aspiration injury and *permits* safe and effective treatments.

Airway control (with due regard for cervical spine integrity) using one or more of five approaches is the first priority for safe and effective management. This is the first step in the systematic, sequential care of the critically ill or injured patient.

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5. CHEST TRAUMA: DIAGNOSIS AND MANAGEMENT.

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INTRODUCTION

"Chest trauma will continue to be a significant cause of acute respiratory distress. It is a major source of morbidity and mortality in otherwise healthy young people. Airway control, oxygenation, drainage of pneumothoraces and hemothoraces and fluid resuscitation are the cornerstones of therapy. With these basic maneuvers, most chest trauma patients can be treated successfully." Carrero, R. and M. Wayne (1989) [1].

These words, written over 20 years ago by Carrero and Wayne bring a grounding sense of calm when confronting the grievously injured chest trauma patient. The combination of clinical foreknowledge, ability to spot changing clinical signs, and even-tempered surgical courage to perform simple but lifesaving procedures can bring about a profound difference in outcome for the chest injured patient - even in resource poor settings.

In this chapter we shall review the basic physiology of breathing, the life-threatening pathophysiology of chest trauma, those conditions that require immediate management during the primary survey, and those conditions managed following secondary survey. Comment will also be made on chest-injury associated conditions the clinician ought to bear in mind.

PHYSIOLOGY OF BREATHING

At its simplest gas exchange in the lungs is composed of three main components:

- a membrane across which gas exchange takes place,
- a bellows mechanism to move gases to and from the membrane,
- and a control mechanism that drives the process while monitoring and making adjustments.

Although slightly more complicated in structure and function we will consider each component in turn. The physiology of breathing may be interfered with at all three levels in the trauma patient.

THE CONTROL MECHANISM

Breathing is automatic and subconscious with a conscious over-ride control mechanism. The automatic control center (central pattern generator) is located in the medulla oblongata of the brain stem and is the primary source of automatic respiratory rhythm determining rate and depth of breathing. At rest the pacemaker cells discharge every 5 seconds giving a resting breathing rhythm of approximately 12 breaths per minute. The medulla oblongata of the brain stem automatic control center has inspiratory and

expiratory neurons and receives input from chemoreceptors (CO_2 , O_2 , pH etc), mechanoreceptors (in the ventilatory tissues), nociceptors, pathways that integrate breathing with other physiologic processes (such as swallowing), and from higher centers with behavioral and volitional activity (e.g. speech and breath-holding). The pacemaker cells also have, amongst other receptors, opioid mu-receptors on their surface.

Although the higher centers have neurons providing input to the automatic control center they are not dependent upon the automatic control center for their effect on breathing. Sensory receptor information from within the lungs is conveyed to the brain in the vagus nerve.

The effective neurologic output of the control mechanism is via the peripheral nerves to:

- the muscles of ventilation: these are the primary muscles of breathing
 - o diaphragm
 - o intercostals
- accessory muscles of ventilation: those affecting patency of the airways:
 - \circ oro-facial
 - o larynx
 - o pharynx
- the accessory muscle of breathing: those called into action when in need
 - o sternocleidomastoid
 - \circ pectorals
 - o abdominal wall muscles, etc.

THE BELLOWS MECHANISM

The bellows mechanism facilitating gas flow is composed of:

- the neurological output from the central control mechanism (peripheral nerves),
- muscles of respiration,
- chest wall bones (e.g. rib cage, sternum, etc) and soft tissues (e.g pleura), and
- conducting airways.

The thoracic vertebrae, rib cage, costal cartilage, and sternum compose the boney thorax and, when acted upon by the muscles of ventilation under the neurologic control of the central control mechanism, the ribs move up-and-out in a 'bucket-handle' manner increasing the intra-thoracic volume. With increased ventilatory requirement the intra-thoracic volume can be increased further by postural changes to straighten out the thoracic kyphosis in time with inspiration.

The co-ordinated movement of the bellows mechanism must move approximately 5-6L of gas (room air)/min for the average healthy adult at rest in an energy efficient way. At rest the diaphragm (innervated by the phrenic nerve with nerve roots C3,4,5 is the principal muscle of respiration (accounting for ~75% of the change in intra-thoracic volume at rest) with additional input from the external intercostal muscles. The chest wall movement and the inherent low pressure gradient in the pleural cavity produces negative intra-thoracic pressure relative to atmospheric pressure and facilitate gas entry to the lungs via the conducting airways. Expiration, at rest, is facilitated by the elastic recoil mechanism of the lung substance and is passive. For ventilation requirements of up to 30-40litre/min increasing the tidal volume (deepening the breath, increasing the cyclical intra-thoracic volume) is usually sufficient in a healthy adult, however, higher ventilatory requirements necessitate increasing the rate of breathing and then recruitment of

accessory muscles. At rest approximately 500ml of gas are moved per breath giving a pulmonary ventilation (or respiratory minute volume of) 6 litre with 12 breaths per minute. The maximal voluntary ventilation is 125-170litre/min.

There are approximately 23 branching points within the airways from the trachea down to The first 16 or 17 divisions constitute the conducting zone the alveolar sacs. (Conducting pathway) and these are the tubes through which gas(es) flow but in which no gas exchange takes place. Their volume constitutes 'dead space' and is ~150ml. The first 6 branch points of the conducting airways account for the majority of the total resistance to airflow in the lung since velocity in peripheral airways is slow. Theseproximal conducting passages are lined by cartilage for support but the terminal bronchioles are <1mm in diameter and are supported by lung substance rather than cartilage in their walls. Bronchioles are thus susceptible to collapse if pressure in the pleural space is greater than in the airway. The distal conducting passages contain proportionately more smooth muscle content and have autonomic innervation and receptors for circulating catecholamines. Activation of local acetyl choline receptors leads to bronchial smooth muscle constriction and stimulation of gland secretion. Activation of beta₂ receptors leads to smooth muscle relaxation and bronchial gland stimulation but bronchial gland stimulation is inhibited by alpha₁ receptor activation.

THE GAS EXCHANGE MEMBRANE

The gas exchange membrane is effectively the alveolar walls of the respiratory bronchioles, alveolar ducts and (predominantly) the terminal alveoli (respiratory zone of the lungs). There are approximately 300 million alveoli in the human lung. Each alveolar wall is effectively composed of alveolar epithelium (type 1 pneumocytes) and pulmonary capillary endothelium, making the diffusion distance for oxygen <0.5mm. The total surface area available for gas exchange in a healthy adult is 50-100m² (70m² for practical considerations). The alveolus size is relatively large compared to the 10 micrometer diameter size of the pulmonary capillaries; this allows over 1000 capillaries to come into contact with an alveolus. The healthy gas exchange membrane is thin at 0.5micrometers minimizing the distance oxygen must diffuse from alveolar lumen to haemoglobin in the 7micrometer sized red blood cells squeezing through the pulmonary capillary bed. In a healthy individual the haemoglobin is fully saturated by the time it is ~25% along a pulmonary capillary.

As a result, there is a tremendous reserve capacity within the ventilatory system to meet increased metabolic demand for oxygen. The chest injured patient, however, not only has increased demands but has impaired ventilatory function exacerbating their condition. Relevant pathophysiology will be discussed with each particular type of injury.

ANATOMIC CONSIDERATIONS

In addition to the functional relationships described above there are some key anatomic points to bear in mind when managing the chest injured patient.

THORACIC INLET

The thoracic inlet is an oblique plane bounded by the first thoracic vertebra, the first ribs and the manubrium sterni. The oesophagus posteriorly and trachea lie in the midline between the body of T1 and the jugular notch of the manubrium. The dome of each lung rises to the inlet on either side of these structures and each is indented by the first rib. The anterior scalene muscles descend to attach to the scalene tubercule of the first rib with the subclavian arteries arching over the first ribs behind the anterior scalene muscles while the subclavian veins pass anterior to the anterior scalene muscles. The phrenic nerves descend in front of anterior scalene muscles and anterior to the dome of the pleura with the internal mammary arteries before accompanying the pericardiophrenic vessels into the superior then middle mediastinum. The vagus nerves descend next to the carotid arteries and behind the jugular veins giving off the right recurrent laryngeal nerve under the brachiocephalic trunk and eventually the left recurrent laryngeal nerve under the arch of the aorta below the thoracic inlet. The thoracic duct rises posteriorly on the left to arch over the left pleural dome and empty into the left subclavian vein as it unites with the left internal jugular vein to form the innominate vein.

This implies that the thoracic inlet is a relatively crowded and fixed space and major shifts of the mediastinum within the chest will lead to compression of the veins draining the head, neck, and upper limbs with resultant decreased venous return and preload.

THE PHRENIC NERVE

Within the chest cavity the phrenic nerves descend with the pericardiophrenic vessels on either side of the heart to penetrate the diaphragm and supply it from below. Since the diaphragm is the main muscle of respiration, it is important to remember the course of the nerve when performing an emergency thoracotomy for penetrating injury to the heart. Dividing the phrenic nerve when incising the pericardium in the process will cause ipsilateral paralysis of the diaphragm, hypoventilation and atelectasis or segmental collapse, with the risk of secondary infection. There is sufficient laxity with the nerve that it may be swept posteriorly when working on the heart from a lateral approach.

INTERCOSTAL NEUROVASCULAR BUNDLE

The intercostal neurovascular bundles run between internal intercostal and the often attenuated transversus muscles outside of parietal pleura. The bundle is oriented with vein uppermost, then artery then intercostal nerve lowermost and all intimate to the undersurface of the rib above. When entering the chest cavity to place a drain or for access to structures demanding surgical attention incise intercostal muscles along their attachment to the upper border of the rib below to minimize injury risk to the bundle and nerve in particular.

EXTENSIONS OF THE PLEURA

As mentioned above, the dome of the pleural over the apices of the lungs rise into the thoracic inlet where fascial thickening of the suprapleural membrane (Sibson's fascia) anchors the pleura to the internal surface of the first rib and transverse process of C7. This prevents ballooning of the pleura up into the neck. Inferiorly the parietal pleura descends to the level of the neck of the 12th rib forming the posterior costophrenic recess. Though the lung does not usually descend down to this level it is important to be aware of this inferior reflection when dealing with a penetrating injury to the kidney. The antero-left pleural reflexion usually occurs before the left border of the sternum so that a

needle may be passed through the 4th and 5th intercostal spaces immediately lateral to the sternum and into the pericardium without crossing the pleura.

THORACIC AORTA

The aorta arises in the middle mediastinum, arches through the superior mediastinum and descends as a relatively fixed structure in the posterior mediastinum. The aortic root and heart have a greater degree of mobility and during sudden deceleration shearing forces occur across the aortic wall at the point where it becomes fixed. The aortic wall just distal to the left subclavian origin is where these forces are usually maximal and this is typically the point of initiation of an aortic dissection or rupture.

PATHOPHYSIOLOGY OF INTERFERENCE WITH BREATHING

As can be appreciated already, the chest injured patient is in a particularly perilous situation for the patient faces not one but two evolving injuries. Firstly there is the injury to the tissues themselves. Secondly there is the effect of hampered ventilation so that oxygen supply to meet the increased metabolic demands after trauma cannot be met. This supply-demand mismatch can result in a negative cycle where ventilatory effort becomes further uncoupled or ineffective due to hypoxia and acidosis, exacerbating all other injuries. Therefore, nowledge of the serious pathophysiologic changes associated with blunt and penetrating chest trauma and their management will be of life saving benefit to the patients.

DIAGNOSIS AND MANAGEMENT OF LIFE THREATENING INJURIES:

As in any patient with trauma, the evaluation and treatment of chest trauma patients follows a certain protocol. These include:

- 1. Primary Survey and resuscitation (ABC of life)
- 2. Secondary survey
- 3. Definitive management

PRIMARY SURVERY (ASSESSMENT)

The purpose of the primary survey is rapid targeted assessment of the airway, breathing, and circulation to identify those injuries that MUST be corrected immediately to prevent rapid death.

Most thoracic injuries end up resulting in compromise of the breathing. The conditions to look for during rapid and systematic primary survey are:

- tension pneumothorax
- open pneumothorax
- sucking chest wound
- flail chest
- massive hemothorax
- cardiac tamponade

The airway (with C-spine control), breathing and circulation are assessed with the patient usually in a supine position and adequate exposure. Control of the examination environment is necessary to prevent undue cooling of the patient and to preserve dignity. Airway assessment and management has been addressed in previous chapters. Breathing and chest examination follows a rapid and clinically fluid progression through inspection, palpation, percussion and auscultation.

SECONDARY SURVEY

During the secondary survery, a more methodical examination of the respiratory system is done. Conditions to think about during the secondary survey with regards to breathing are:

- lung contusion
- cardiac contusion
- rib fractures including the sternum
- blunt aortic injury
- oesophageal injury
- diaphragmatic rupture

The most important findings to look for in patients with chest injury should include:

Inspection:

- signs of cyanosis?
- depth and rate of breathing?
- use of accessory muscles?
- dilated neck veins?
- obvious wounds?
 - penetration points
 - compound fractures
 - abrasions, bruising associated with deceleration injury / blunt trauma
- don't forget the posterior chest
- Paradoxical chest movement?
- Sucking chest wound

Palpation:

- tracheal position is it deviated to one side?
- chest wall deformity?
- normal chest wall excursion?
- asymmetric chest wall movement?
- flail chest segment?
- crepitus from rib fractures?
- Apical heart beat displaced?

Percussion:

- resonant is it normal?
- hyper-resonant is there a pneumothorax?
- dull to percussion is there haemothorax? or collapse? is it too early for dullness from lung contusion or consolidation?

Auscultation:

• Are breath sounds present and normal?

- Are breath sounds present throughout both lung fields?
- Are bowel sounds heard in the chest?

Pulse-oximetry and chest x-ray (CXR) are not essential in the diagnosis and management of chest injury but are adjuncts to your assessment; therefore do not wait for their availability before starting your treatment. Act to treat what you find that is of immediate threat to the patient.

IMMEDIATELY LIFE-THREATENING INJURIES:

TENSION PNEUMOTHORAX

Tension pneumothorax is a clinical diagnosis and requires immediate action, with ipsilateral needle decompression and then a chest drain [2]. Action, not X-Rays, is necessary for immediate survival.

Definition: Tension pneumothorax is a consequence of a flap-valve, one way mechanism in the pleural membrane where the pleural space is in communication with the outside atmosphere or a conducting airway. Air flows in one way only and creates positive pressure (tension) in the pleural space. It is rapidly life-threatening.

Pathophysiology: With each inspiration the negative intra-thoracic pressure becomes progressively positive. Each breath draws air into the pleural cavity which cannot escape. Initially the affected lung collapses, and with increased intra-pleural volume, the mediastinum shifts away from the affected side. This compresses the superior and inferior vena cava. Venous return to the heart drops and cardiac arrest with pulseless electrical arrhythmia (PEA) rapidly occurs [3]. Increasing hypoxia leads to increasing air hunger and tachypnoea, which accelerates the pathological process, a negative vicious cycle.

<u>Diagnosis</u>: The diagnosis of tension pneumothorax is <u>**CLINICAL**</u>. Chest X-ray is <u>**NOT**</u> required and can cause lethal delay.

A patient will present with one or more of:

- History of chest trauma,
- Severe respiratory distress,
- Air hunger,
- Increased JVP or distended neck veins,
- Tracheal Deviation AWAY from the affected side,
- Hyper-resonance to percussion on the affected side,
- Diminshed or absent breath sounds on the affected side,
- PEA arrest.

Signs of respiratory distress includes:

- Tachypnea
- Use of accessory muscles of breathing
- Inte-rcostal and sub costal retroaction
- Flaring of ala nasi
- Decrease in oxygen saturation

Immediate Management = Needle decompression: No further investigations are required. Immediate action is essential. Needle decompression by insertion of a 14 gauge, 5cm long needle in the second intercostals space in the mid-clavicular line should be performed.

Be sure to use a long enough needle. Cadaveric studies indicate that at this site, the pleural cavity can be deeper than perceived.

Once needle decompression has been performed and the pleural space is decompressed, time for definitive management can be obtained, which is insertion of a formal chest drain. Make sure chest drain is put in place no later than 10 minutes.

In Summary:

- 1. confirm the affected side clinically,
- 2. inform the patient,
- 3. antiseptic swab the skin at the 2nd intercostal space in the mid-clavicular line,
- 4. insert a 14 Gauge cannula (usually orange or brown capped) +/- syringe,
- 5. listen for 'hiss' (or 'bubbling' if the syringe barrel is filled with water and the plunger removed),
- 6. protect with gauze swab, tape, and *leave in situ*,
- 7. set up and insert a chest drain.
- 8. Then order a CXR (after all the above are performed)
- 9. Do not forget adequate pain control as subsequent lung expansion depends on adequate breathing effort.

Illustrations can be found at

http://handbook.muh.ie/trauma/Chest/TensionPneumothorax.html

OPEN PNEUMOTHORAX

Definition: A life threatening injury where penetrating trauma opens the pleural space, causing a pneumothorax and a 'sucking' chest wound [5].

Pathophysiology: Penetrating trauma to the chest can open the pleural space. If the communication is **greater than two thirds (2/3) of the diameter of the trachea**, air will preferentially enter the exposed pleural space through the wound on inspiration, leading to the ipsilateral lung to collapse. Chest wall excursion during breathing still generates negative intrathoracic pressure but air moves to-and-fro through the chest wall injury, creating a sucking chest wound. The patient is now dependent upon the contralateral lung for oxygenation but the function of this lung is severely compromised. Minimal air entry occurs as preferential air flow is through the sucking chest wound and progressive mediastinal shift can occur towards the contralateral lung. Again this can cause compression of the inferior vena cava, reduced cardiac return and PEA arrest [6]. If the air is unable to escape from the pleural cavity but still able to enter on inspiration then a tension pneumothorax will develop and the lethal process is accelerated..

Clinical Signs:

- Respiratory Distress
- Tachypnoea and Dyspnoea
- Cyanosis
- Visible chest wound
- Asymmetrical chest expansion
- No tracheal deviation initially, but later can be away from wound

- Hyper-resonant to percussion
- Diminished or absent breath sounds on affected side
- Air movement through the wound; noticed as "bubbling" of blood at the wound site
- PEA arrest

Management:

Immediate management is life saving and consists of:

- Supplemental (100%) oxygen
- Applying a flap-valve dressing
- Immediate Insertion of a chest drain and applying a totally occlusive dressing or suturing of the open wound.

A flap-valve dressing is a temporizing step using an occlusive dressing applied over the wound but only taped down on three sides. With inspiration the dressing occludes the wound but on expiration allows air in the pleural space to escape. After application of the dressing the patient should, if needed, be rolled towards the injured side to relieve pressure on the fully functioning lung. This should be performed with care not to exacerbate an unstable spinal injury.

If there is any concern about a possible tension pneumothorax decompression is performed simultaneously with a large bore needle as previously described.

Once the patient is stabilized, definitive treatment is performed. Under local anaesthetic the wound is explored, debrided and closed. A chest drain is left in-situ.

More can be seen at:

http://www.wonderhowto.com/how-to-treat-sucking-chest-wound-195366/

http://www.trauma.org/archive/thoracic/CHESTopen.html

MASSIVE HAEMOTHORAX

<u>Definition</u>: Accumulation of blood in the pleural cavity caused by bleeding from chest wall, lung parenchyma or major thoracic vessels.

Pathophysiology: The common cause of haemothorax is bleeding from a lacerated lung, followed by intercostal vessels with rib fractures or an internal mammary artery bleeding. This is usually self limiting. Laceration to larger vessels can cause major problems. Major lung vessels can be injured by penetrating objects, including rib fragments during high impact blunt injury. Each adult chest cavity can hold up to 3 litre blood, i.e. the chest cavity can hold their entire circulating volume. Bleeding from injuries to the great vessels leads to haemo-mediastinum and will not enter the pleural space unless there is a concomitant breach of the pleural membrane or injury occurs at the lung hilum. Haemothorax from azygous vein disruption is rare [7].

Haemothorax is a double insult to the patient as there is progressive deterioration of effective breathing and circulation. As circulating volume is lost into the large but fixed volume of the chest cavity, there is less volume for lung expansion. Consequently as the lung collapses hypoxia develops more rapidly as there is ineffective ventilation to

oxygenate the remaining blood in circulation. Circulatory collapse leads to trauma cardiac arrest.

<u>Clinical Signs</u>: Massive haemothorax should be suspected clinically in a patient who has signs of respiratory distress and shock.

Signs of bleeding and haemodynamic instability (e.g. tachycardia, hypotension) normally present before symptoms of respiratory distress.

Chest findings during the primary survey include cyanosis, tachypnoea, tachycardia, tracheal deviation away from the affected side, decreased chest expansion, dullness to percussion, and reduced or absent air entry on the affected side.

Early CXR is a useful adjunct to making the diagnosis but should not delay management in the unstable patient with suspected massive haemothorax. At least 400ml blood has to be lost into the pleural space before blunting of the costo-phrenic angle is seen on an erect CXR.

With blunt trauma one should have a high index of suspicion for injuries that may mimic massive haemothorax, e.g. massive lung contusion, diaphragmatic rupture with intrathoracic abdominal content, and occult tension pneumothorax with small haemothorax.

Management:

Management of massive haemothorax includes:

- 100% oxygen
- insertion of intercostal chest drain
- maintenance of circulating volume

Although insertion of a chest drain is therapeutic for respiratory compromise caused by the massive haemothorax, it will not address the primary problem of ongoing bleeding. With intercostals vessel injury bleeding will usually stop spontaneously, or with low pressure vessels such as lesser pulmonary veins, expansion of the lung will tamponade the bleeding. With major vessel bleeding surgery will be required. Following insertion of a chest drain, emergency thoracotomy is indicated for blood loss of

- >1500ml blood in chest drain at insertion [8],
- >200ml/h for 3 consecutive hours [8], or
- >100 ml/h for > 6 hours

Other indications of an emergency thoracotomy include:

- Massive bubbling from the chest drain which shows a major airway disruption
- Leak of oesophageal content from the chest drain

The patient with a large haemothorax is also likely to have other significant chest injuries such as multiple rib fractures, flail segment, and possibly a tension pneumothorax. Late complications from inadequately drained haemothorax include empyema formation if the clotted blood becomes infected. Prophylactic antibiotics may decrease the incidence of empyema and pneumonia [9].

Further information and images of haemothorax and its management can be seen at:

http://www.thoracicmedicine.org/articles/2011/6/3/images/AnnThoracMed_2011_6_3_14 9_82451_f3.jpg

http://otto.oxfordmedicine.com/content/vol2/issue1/images/large/graphic012004006.jpeg

http://www.trauma.org/archive/thoracic/CHESThaemo.html

http://www.surgical-tutor.org.uk/default-home.htm?core/trauma/chest_trauma.htm~right

CARDIAC TAMPONADE

Definition: A life threatening condition where accumulation of blood (or other fluid) in the pericardial space around the heart restricts cardiac output and rapidly leads to cardiac arrest.

Pathophysiology: Penetrating trauma to the pericardium and heart with subsequent bleeding from the heart usually is the causeThe small hole in the pericardium rapidly seals with clot, but bleeding from the heart continues and fills the pericardial space. The fibro-elastic pericardial sac cannot dilate and the cardiac chambers are compressed, especially the atria, which are prevented from filling, leading to obstructive shock. Cardiac output falls and the patient progresses to cardiac arrest without intervention [2]. As little as 100ml blood can cause tamponade in the adult patient [10].

Diagnosis: A patient with penetrating chest trauma may present with mild cardiovascular instability which quickly worsens, major cardiovascular instability or in cardiac arrest.

Classical clinical signs are Beck's triad of:

- distended neck veins (elevated venous pressure)
- hypotension
- muffled heart sounds.

Other signs are:

- Kussmaul's Sign: Rise in JVP on inspiration.
- Pulsus Paradoxus. An exaggerated fall in blood pressure on inspiration (>10 mmHg in systolic pressure). This can be difficult to elicit and not a reliable sign [11].
- PEA arrest.

On CXR there might be a globular heart shape and ECG may show small complexes with tachycardia. Again these are unreliable signs.

NOTE: Evidence of penetrating trauma to the central chest with hypotension should always raise the suspicion of cardiac tamponade.

Management: Urgent intervention can be life-saving.

Resuscitation should be continued, with 100% oxygen and administration of intravenous fluid or blood products if available. This increases cardiac filling pressure and can temporarily improve the situation. The aim is to maintain cerebral perfusion but not to chase a normal systolic pressure as this will increase the rate and volume of bleeding into the pericardial sac.

Needle pericardiocentesis can be performed by inserting a large bore needle between the xiphisternum and left subcostal margin, aiming at the left shoulder. Withdrawing 50ml of blood can improve the situation. Blood drawn from the pericardium usually does not clot whereas blood drawn from the heart does [12]. This results in temporary improvement and the treating surgeon is able to rush to patient to the operating theatre for an emergency surgery.

Definitive treatment is via sternotomy or thoracotomy which should be done in preference to pericardiocentesis or as soon as possible.. Exposure can be via median sternotomy, a left anterior thoracotomy or 'clam-shell' thoracotomy. The 'bulging' pericardium is identified and incised, avoiding the phrenic nerve. Once this occurs the tamponade is released. Often only a small amount of bleeding from the heart is seen which can be repaired with 4/0 silk or prolene sutures.. Care should be taken to identify the coronary vessels and to check for posterior cardiac wounds.

Complications of management include internal mammary and coronary artery injury, ventricular puncture and aspiration, introduction of infection and precipitation of percarditis, and phrenic nerve injury during surgical approach through the pericardial sac.

Injuries associated with cardiac tamponade include cardiac contusion [13] and coronary artery injury which may have a delayed presentation [14].

Further information and images of cardiac tamponade (including how to perform pericardiocentesis) may be seen at: <u>http://www.nejm.org/doi/full/10.1056/NEJMra022643</u>

OTHER POTENTIALLY LIFE THREATENING INJURIES

FLAIL CHEST

Definition: Flail chest injury occurs when two or more serial and segmental fractures are present in two or more neighbouring ribs with paradoxical movement of the chest wall segment relative to the breathing cycle. Especially in children this can also occur due to disruption at the costochondral junctions, which makes the whole sternum a flail segment.

Pathophysiology: During inspiration the chest wall expands but the flail segment moves inwards due to the sucking effect negative intrathoracic pressure on the flail segment. This limits lung expansion, with ineffective ventilation and hypoxia (ventilation-perfusion mismatch). Significant force is necessary to fracture ribs at multiple sites; therefore this injury is often associated with extensive lung contusion, haemothorax and pneumothorax due to the rib fractures. Underlying injuries are more likely to cause respiratory dysfunction. Severe pain due to multiple fractures leads to shallow breathing, worsening ventilation even further; combined with contusion this often leads to retention of secretions, airway collapse and pneumonia.

<u>Diagnosis</u>: Flail chest is a clinical anatomical diagnosis. It is important to look past the flail segment for underlying pathology [15].

Clinical examination will reveal a patient with tachypnoea, and signs of blunt trauma to the chest wall. The flail segment is identified by its paradoxical movement on spontaneous breathing and is often more obvious to feel than to see (If the patient is intubated this sign disappears with positive pressure ventilation). Palpation may identify crepitus from the broken rib ends and percussion exacerbates pain.

Moderate to severe respiratory distress occur proportional to the severity and extent of underlying injury.

<u>Management</u> is in the form of rib fracture treatment and management of underlying pulmonary contusion [16]. This includes:

- 100% oxygen
- Strong analgesia consider using rib blocks with local anaesthetic
- chest drain(s) for associated pneumothorax or haemothorax
- consider assisted ventilation if there is inadequate ventilation or the patient is tiring. Ventilatory support is more likely with:
 - large flail segment or one involving the sternum,
 - extensive lung contusion.

Further information and images of flail chest injury (including 3D reconstruction) may be seen at:

http://imaging.consult.com/image/topic/dx/Chest?title=Trauma,%20Chest%20Wall%20an d%20Vertebral%20Trauma&image=fig7&locator=&pii=S1933-0332(07)70232-X

http://www.trauma.org/archive/thoracic/CHESTflail.html

http://www.youtube.com/watch?v=snqrcB30ZXQ

PULMONARY CONTUSION

Definition: An injury to lung parenchyma secondary to blunt trauma. Young children have pliable chest walls and can have severe lung contusion without rib fractures.

Pathophysiology: Following blunt trauma, oedema and blood collect in the alveolar space. This causes ventilation/perfusion mismatch which evolves over a period of 24 hours. As the injury evolves, the patient suffers from impaired gas exchange, increased pulmonary vascular resistance and decreased lung compliance [17]. Adult Respiratory Distress Syndrome can occur in conjunction with this injury.

<u>**Clinical Features:**</u> Pulmonary contusion is difficult to diagnose clinically, especially because it doesn't manifest immediately. The presence of rib fractures or flail chest and blunt force trauma should arouse suspicion. Have a high index of suspicion in all patients who were unrestrained during an RTA or who have fallen from a height. The clinical picture is one of a patient with minimal symptoms initially but who gradually developed escalating oxygen requirements and respiratory difficulties as the underlying pathology evolves [18].

Diagnosis: Chest X-ray is useful, though radiographic changes can lag clinical signs. CT gives accurate diagnosis of pulmonary contusion and differentiation from other clinical entities such as atelectasis.

Management:

Supportive management of the patient is required for a period of 3-5 days to allow the contusion to resolve. In general this involves supplemental oxygen if necessary and adequate analgesia and physiotherapy to avoid complications such as pneumonia.

If contusion is severe and ARDS occurs with respiratory failure, further respiratory support will be required, usually with intubation and ventilation.

AORTIC INJURY

Definition: Patients who sustain an aortic transection injury almost always die at the scene of the accident and account for around 15% of trauma related deaths. Only 15% of those who sustain a blunt aortic injury make it to the hospital alive [19] and these patients are likely to have a tear with dissection or pseudoaneurysm formation.

Pathogenesis: During a sudden deceleration injury, such as in a RTA or fall from height, the ascending aorta and aortic arch move within the chest cavity, generating maximal shearing forces between the relatively fixed proximal and the descending thoracic aorta; the majority of tears or transections therefore occur just distal to the left subclavian artery origin [20].

Diagnosis:

Clinical assessment may reveal an interscapular flow murmur in a patient with upper thoracic back pain.

CXR findings suggestive of aortic injury include wide mediastinum (>8cm), indistinct aortic knuckle, and depressed left main bronchus.

A left sided haemothorax that returns arterial blood on chest drain insertion should raise the index of suspicion.

Other diagnostic investigations in the more stable patient include trans-oesophageal Doppler, CT scanning and angiography.

<u>Management</u> involves judicious resuscitation with blood pressure control. Overzealous fluid resuscitation may lead to re-bleeding from the site of aortic injury in the haemodiluted patient. Prompt surgical repair through either endovascular [21] or open approach is necessary; by-pass lowers the risk of post-procedure paraplegia [19].

Further information and images of thoracic aorta injuries may be seen at:

http://www.trauma.org/archive/thoracic/CHESTaorta.html

http://www.surgical-tutor.org.uk/default-home.htm?core/trauma/chest_trauma.htm~right

http://www.east.org/tpg/chap8.pdf

http://www.nejm.org/doi/full/10.1056/NEJMra0706159

OTHER CHEST INJURIES THAT CAN LEAD TO SEVERE COMPLICATIONS IF NOT TREATED OPTIMALLY:

SIMPLE PNEUMOTHORAX

Simple pneumothorax develops following transitory escape of air into the pleural space with partial collapse of the lung. It may occur following either penetrating or blunt trauma

with rib fractures and may be diagnosed clinically (if large enough) or detected incidentally on Chest X-ray (this is one reason why compulsory CXR should be included in the "trauma series" after any serious injury). The patient may be tachypnoeic, have decreased chest expansion on the afflicted side, be hyper-resonant on percussion, and have decreased air entry on auscultation. The patient does not exhibit signs of shock or rapid deterioration as seen in tension pneumothorax or open pneumothorax. A trauma patient with a simple pneumothorax will still require a chest drain but this usually may wait until after the secondary survey. If the patient is transferred or is to have a general anaesthetic a chest drain is essential; a small pneumothorax will rapidly expand and become life threatening with positive pressure ventilation or at lower atmospheric pressure (e.g. in an aeroplane).

RIB FRACTURES

Rib fractures are commonly encountered in thoracic trauma.

Pathophysiology: Rib fractures per se are not problematic but associated pain limits both inspiration and expiration, and prevents effective coughing. The patient is at risk of hypoventilation, retention of secretions, secondary infection and pneumonia, which can have serious consequences.

<u>Clinical Features:</u> The patient will present with pain and or dyspnoea. Always consider a significant underlying injury if there is associated respiratory failure or haemodynamic instability.

Management of simple rib fractures

- Analgesia and targeted physiotherapy to prevent complications.
- Attention to underlying pathology.
- Rib fractures themselves will heal without specific intervention.

Management of pain from rib fractures is essential to prevent hypoventilation and pneumonia. Non-steroidal anti-inflammatory drugs provide excellent analgesia if there are no contra-indications. Paracetamol and opiate drugs could also be utilized if necessary. Occasionally pain control is problematic. Patient Controlled Analgesia (PCA) can be used as an adjunct to therapy, and intercostal/regional anaesthesia can be effective if a sufficiently experienced anaesthetist is present [22]. This is not without associated complications however [23].

Once analgesia is satisfactory, targeted physiotherapy is required to ensure hypoventilation does not occur.

INJURIES ASSOCIATED WITH SPECIFIC RIB AND BONY FRACTURES OF THE CHEST WALL:

Chest wall bony fractures detected on CXR should raise suspicion for associated injuries to neighbouring organs:

1st rib:	lung apices, subclavian vessels
2nd rib:	ascending aorta, superior vena cava
Clavicle:	lung apices, subclavian vessels
Sternum:	myocardial contusion, internal thoracic vessels
10th rib:	diaphragmatic, liver, splenic injury

11th rib:	diaphragmatic, liver, splenic injury
12th rib:	renal injury.

MYOCARDIAL CONTUSION

Cardiac contusion usually occurs due to severe direct blunt trauma to the anterior chest. It is caused by rapid deceleration injury, e.g. against a steering wheel during a car crash. Shearing forces cause bleeding and bruising within the myocardium. This will usually not present with clinical features but with a range of ECG abnormalities once the patient has been stabilised and is monitored in the HDU or ICU. The ECG will almost always return to normal as the bruising settles. No specific treatment is necessary but life threatening ventricular arrhythmias need to be managed as with any other cause.

THORACIC VERTEBRAL FRACTURES

It is important in the patient who has sustained blunt chest trauma that the thoracic vertebrae are not forgotten. A lateral thoracic spine X-ray is helpful in assessing the thoracic spine even when no 'step' deformity or spinous process injury is detected on palpation in the secondary survey. The integrity of each vertebral body should be inspected on the X-ray as well as integrity of the three conceptual columns within the spinal column.

OESOPHAGEAL INJURY

Definition: Oesophageal injury during trauma is rare but under-diagnosed; occult injuries are easily missed during initial assessment.

Pathophysiology: There are two possible mechanisms of oesophageal injury:

- raised luminal pressure against a closed glottis leading to a 'blow out' injury;
- crush injury between the sternum and the thoracic vertebrae with anterior compression injury.

<u>Diagnosis</u>: The patient complains of pain on swallowing. Crepitus or surgical emphysema may be felt in the neck and pneumomediastinum seen on CXR.

Management involves[24]:

- drainage of the chest cavity at the site of the perforation or tear
- delineation of the extent of the injury
- debridement of necrotic tissue
- decortication of soiled pleural space
- defect closure with flap or pedicle buttressing
- diversion? avoid if at all possible.

Life threatening mediastinitis may develop following oesophageal injury and particularly with late diagnosis.

CHEST DRAIN / THORACOSTOMY TUBE:

A chest drain is indicated in the management of a tension pneumothorax, open pneumothorax, simple pneumothorax, haemothorax, and in patients with hemo-pneumo thorax. A chest drain may be placed prophylactically in trauma patients prior to transfer to another institution e.g tertiary care centre, and in patients with rib fractures who require ventilation.

To place a chest drain:

- 1. inform the patient,
- 2. check all equipment required,
- 3. confirm the side requiring the drain,
- 4. prepare the chest wall skin with antiseptic and a sterile field,

5. identify the optimum site for access in the 'triangle of safety' - 4th or 5th intercostal space in the anterior- or mid-axillary line,

6. infiltrate local anaesthetic to skin, subcutaneous tissues, periosteum of upper edge of the rib below, intercostal spaces (avoiding intercostal vessels), and to parietal pleura,

7. aspiration with the infiltration needle at each advancing step, prior to instillation of local anaesthetic, will avoid intravascular injection and confirm entry into the pleural cavity, when air is returned, as well as gauge the depth of the chest wall,

8. incise the skin along the upper border of the rib below,

9. use a curved haemostat / curved forcep to bluntly dissect down to pleura,

10. explore the track with a sterile gloved finger to breach pleura and confirm pleural cavity entry, (think about what lung, diaphragm, liver, intestinal tissues would feel like),

11. occlude the track with a finger during inspiration,

12. Push a large bore (28-32F) chest drain through the incision: either pass it gripped in the curved forcep or by hand into the pleural cavity angling the direction towards the apex to manage a pneumothorax or postero-inferiorly to manage a haemothorax,

13. do not force the drain in but re-explore the track if there is significant resistance,

14. ensure all the fenestrations / holes in the tube are inside the patient,

15. secure the drain and approximate skin around the tube with a heavy suture and apply an occlusive dressing,

16. connect the drain to an underwater seal placed below the level of the patient,

17. re-examine the patient for clinical change,

18. examine what is drained: arterial or venous blood / lymph / intestinal content?,

19. confirm drain orientation, position, and effect with CXR.

Illustrations and guidance for chest drain insertion may be seen in the following links: <u>http://www.nejm.org/doi/full/10.1056/NEJMvcm071974</u>

SUMMARY

Chest trauma can cause immediately life threatening pathology that have to be recognised and managed during primary survey and resuscitation. Often the patient can be kept alive through simple emergency room procedures until help arrives and/or definitive treatment for specific injuries can be instituted. Other serious injuries occur that may not need life-saving treatment within minutes, but still need recognition while the patient is in the emergency room and a definite urgent treatment plan to prevent mortality. Lastly there are injuries that can seem innocuous or are not easily diagnosed, but will have significant morbidity and mortality if missed or treated suboptimally.

All these injuries should be managed appropriately through proper knowledge, a system of rapid primary and secondary assessment, supported by effective resuscitation, rapid emergency procedures and definitive management, and ultimately by repeated practice in simulated and real trauma environments.

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6. HYPOXIA IN SURGICAL PATIENTS

ALEXIS RIDDELL, ANDREW J JACKSON, DAVID R BALL, JACOB S DREYER

INTRODUCTION

This chapter discusses basic physiology of oxygen delivery, pathophysiology and mechanisms of hypoxia, the most common causes of hypoxia in surgical patients and principles of management. The aim is not to provide a detailed overview, but structure to enable early recognition, diagnosis and treatment.

Hypoxia is impaired tissue oxygenation. It is one of the most common post-operative complications but often not recognised because it is not looked for, e.g. post-operative confusion can often be secondary to hypoxia. Patients who are critically ill usually have increased oxygen demands; oxygen delivery is therefore fundamental to managing sick patients.

PHYSIOLOGY OF OXYGEN TRANSPORT

Oxygen Delivery = oxygen content x cardiac output, where

Oxygen content = (Hb x 1.34 x SaO2) + (0.0032 x PaO2). Fully saturated haemoglobin carries 1.34 ml oxygen/gram of Hb, but the constant can vary slightly. The maximum amount of oxygen that can dissolve in blood is 0.0032 ml/dl/mmHg PaO2. At Hb=15 and SaO2=98 blood carries 198ml O2/litre, of which 195 ml is carried as oxygenated haemoblobin.

Cardiac output depends on stroke volume and heart rate (CO = SV x HR); stroke volume is dependent on cardiac preload, contractility and afterload. Heart rate increases early with hypoxia. Peripheral perfusion and tissue oxygen delivery depend on cardiac output and peripheral resistance (BP = CO x PR).

Oxygen delivery therefore depends on:

- A patent and open <u>Airway</u>
- Effective <u>Ventilation</u>
 - Central drive, volume, rate, Functional Residual Capacity (FRC).
- Oxygen availability:
 - Percentage oxygen in inspired air (FIO2), oxygen pressure in the air and alveoli (pAO2), pulmonary capillaries (paO2).
- Oxygen transport:
 - Haemoglobin level (Hb), Cardiac output, Peripheral resistance. Each haemoglobin molecule can bind four oxygen molecules; binding of each molecule facilitates binding of the next until Hb is fully saturated, i.e. the affinity for the 4th oxygen molecule is much higher than for the 1st. This is the biochemical basis for the sigmoid shape of the Oxygen-Haemoglobin dissociation curve.

- Tissue factors:
 - Oxygen release, Diffusion, Utilisation. Oxygen release is enhanced by shifting the oxygen-haemoglobin curve to the right by a lower pH and higher temperature in active tissue (e.g. contracting muscles) and by higher levels of 2,3-DPG (raised by exercise, higher altitude).

PATHOPHYSIOLOGY OF HYPOXIA

THE FOLLOWING FACTORS NEED TO BE MAINTAINED TO PREVENT TISSUE HYPOXIA [1,2]:

- 1. Patent airway
- 2. Effective ventilation
- 3. Effective gas interchange
- 4. Arterial oxygen saturation (SaO2) and pressure (PaO2)
- 5. Effective systemic and capillary circulation
- 6. Haemoglobin concentration and integrity
- 7. Effective oxygen release from Hb
- 8. Unhindered extracellular diffusion
- 9. Normal oxygen use by cells.

PHYSIOLOGICALLY HYPOXIA IS USUALLY CLASSIFIED INTO FOUR GROUPS [1,2]:

(a) Hypoxic hypoxia, when the pressure of oxygen in arterial blood (PaO2) is reduced, accompanied by decreased SaO2 of haemoglobin. This is caused by inefficient gas exchange (when PAO2 would be maintained) or decreased PAO2, e.g. at high altitude or suffocation due to airway obstruction or breathing in a closed space with loss of oxygen.

(b) Anaemic hypoxia, due to decreased oxygen carrying capacity in blood, e.g. due to loss of red blood cells (RBCs), inadequate Hb within RBCs or carbon monoxide (CO) poisoning. Oxygen binding sites on Hb have higher affinity for CO than O2 which prevents oxygenation and patients do not show clinical symptoms and signs of hypoxia. In sickle cell anaemia the O2-Hb dissociation curve shifts to the right so that oxygen is released in the tissues more easily, compensating for a Hb of 6-8 g/l.

(c) Circulatory hypoxia, also known as stagnant or ischemic hypoxia, when too little oxygenated blood is delivered to the tissues. This can be localised, e.g. with acute arterial insufficiency, or general, e.g. with circulatory shock or cardiac failure.

(d) Histotoxic (cytotoxic) hypoxia, which means that oxygen delivery is normal but tissues cannot use O2 due to toxins affecting cellular respiration, e.g. with cyanide poisoning. Methylene blue can be used in cyanide poisoning to bind cyanide molecules but this forms met-haemoglobin (where iron is reduced to Fe3+), which has a much lower affinity for O2, limiting oxygen delivery.

IN CRITICAL CARE THE FOLLOWING MECHANISMS PROVIDE Α PRACTICAL MNEMONIC TO THINK OF POTENTIAL CAUSES OF HYPOXIA:

1. \downarrow **pAO2** and \triangle **FiO2**

Alveolar PO2 can drop significantly at altitude. In practice this is of importance when transferring patients with e.g. chest injuries, acute blood loss, shock or anaemia in unpressurised aircraft at high altitude. This is relevant in regions already at high altitude or crossing mountain ranges e.g. in parts of Ethiopia, the central great lakes states and South Africa.

Patients on ventilators have their FiO2 controlled artificially.

2. ↓V

Decreased ventilation will primarily cause CO2 accumulation. In normal patients this will increase the ventilation rate via chemoreceptors, but patients who are on artificial ventilation or are centrally depressed (e.g. due to opiate overdose) cannot mount this response. Hypoxia occurs late and can rapidly progress to cardiac arrest.

3. ΔV/ΔQ

This means that there is discrepancy between ventilated alveoli and alveolar capillary perfusion, and there are two categories:

(a) Shunt: when alveoli are perfused but not ventilated (e.g. atelectasis, pneumonia), or oxygen diffusion is limited (e.g. in ARDS), right to left shunting is increased, i.e. more non-oxygenated blood reaches the systemic circulation. This is a significant cause of hypoxia in critically ill patients.

(b) Ventilation-perfusion mismatch: when alveoli are ventilated but not perfused, e.g. with pulmonary embolism (PE).

be

The importance of haemoglobin in oxygen carrying capacity is clear. Patients with low Hb have decreased oxygen carrying capacity and in some patients this can critical e.g. those with underlying heart or lung disease or after burns. Ventilated patients will almost always need their Hb to be topped up to >10 g/l before they be weaned from the ventilator.

can

5. LCO

Hypovolaemic, cardiogenic or obstructive circulatory shock or congestive cardiac failure (CCF) can cause significant enough hypoxia to cause rapid death. Patients on artificial ventilation who are in CCF will usually not come off the ventilator unless cardiac function is supported (e.g. with inotropes).

ASSESSMENT OF THE HYPOXICPOST PATIENT

CLINICAL ASSESSMENT

Hypoxia is the inability to effectively oxygenate the tissues and is a threat to life. This may result from pathology of the airway, breathing or circulation. Prompt responses are crucial, allowing accurate diagnosis and effective treatment. A five-step, structured, sequential set of responses to hypoxia is:

1 Review (Primary survery): This *primary assessment is* a rapid, targeted clinical examination of airway, breathing, circulation and disability. This is conducted in correct order, with immediate management of a life threatening problem when discovered.

The most important skills at this stage are the use of the trained human senses to "look, listen and feel", gathering important clinical information, informing diagnosis and acting when necessary. Monitoring devices are useful when available, providing additional information. This information is only helpful to the patient when it is accurate, timely and used to guide effective treatment. Cutaneous pulse oximetry gives real-time data on the oxygenation of haemoglobin (saturation) and peripheral pulse rate. These signs may be lost with severe vasoconstriction, as in severe shock. Arterial blood gas analysis gives information on arterial oxygen tension (which is not "saturation"), carbon dioxide tension and blood acid-base status. Chest radiography and electrocardiography may provide additional data, but may detract from immediate, time critical interventions.

When critical hypoxia is revealed by the primary assessment, the next response, *Resuscitation,* is started without delay. After the initiation of resuscitation, the review phase may continue with the *secondary assessment,* aimed at gathering more information. A patient history is taken with attention to the acute and chronic aspects of the patient's condition, with identification of specific symptoms and risks, such as asthma, smoking, heart disease etc. Review of current and past medication (including missed doses) is necessary. A clinical examination is done to identify signs of organ and system dysfunction.

Chart review is crucial. Changes in vital signs can inform diagnosis and treatment.

There is a further, *tertiary assessment*, but this is done upon completion of patient treatment. This is a review of the clinical process, identifying strengths and weaknesses, aimed at improving future care. This can be done in an educational setting and should be conducted in a supportive, positive way.

2 Resuscitation: This is started when the primary survey shows an immediate threat to life. The resuscitation is also sequential and structured, aimed at restoration and maintenance of oxygen to the tissues, especially the vital organs. The stepwise approach is part of the Basic and Advanced Life support guidelines. Patients with hypoxia need oxygen.

Resuscitation responses to common hypoxic problems are outlined later in this review.

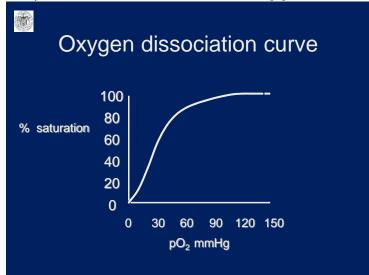
3 Request HELP!. Management of postoperative hypoxia can be complex and demanding. The chance of a successful outcome is increased when skilled help is sought and available to help manage the situation. A team approach requires good clinical leadership, situational awareness with effective task allocation. A key factor is concise, effective communication. An "SBAR" communication format is helpful. A clinical record should be kept: when possible a team member can be given this task.

4. Reassess the situation regularly. The clinical picture will most likely change both with time and treatment and this is only apparent with reassessment. New information may be elicited or become available from other sources, which may alter further management.

5 Resource: your situation. Identify what is needed to improve the chance of a good outcome. This may involve acquisition of drugs, equipment or people to help with any of steps listed above.

PULSE OXIMETRY

Pulse oximetry is a valuable adjunct in the rapid assessment of peripheral oxygenation. It gives *an estimate* of percentage saturation on oxygen binding sites on Hb. It is related to PaO2 through the sigmoid shaped O2-Hb dissociation curve but should not be interpreted as direct substitute for PaO2 [3].



Remember:

Normal arterial blood has a saturation of only 97-98% due to physiologic shunt, but 95%-100% is normal on pulse oximetry for a patient on supplementary oxygen. A value <93% can be a warning and one should ask "Why?". Unless there is a significant shift in the Hb-O2 dissociation curve, a PaO2 >8 kPa with a SaO2 > 90% usually means that the oxygen saturation is still on the plateau part of the curve. With a value of <90% the patient is in serious trouble because the paO2-SaO2 ratio is now on the steep part of the curve and saturation will drop rapidly with a minor decrease in PaO2.

Double check that you distinguish the SaO2 from the pulse rate when looking at the monitor.

Error readings in pulse oximetry can occur due to:

- Low cardiac output
- Vasoconstriction
- SaO2 <70%
- Poor positioning
- Movement
- Hypothermia (often in trauma patients)
- Abnormal Hb (COHb, MetHb)
- Hyperthermic limb
- Dirty probe
- Black, blue or green nail polish
- External light

ARTERIAL BLOOD GAS ANALYSIS (ABGS)

ABG analysis can be useful in the diagnosis and management of critical illness and injury, but waiting for results should not delay immediate management of potential hypoxia. The following account is a traditional interpretation. Another analysis, Stewart's "Strong Ion Difference" approach is an alternative.

The ABG analyser measures:

- Hydrogen ion concentration, reported as either hydrogen ion concentration [H⁺] or pH (-log₁₀[H⁺]). A lower pH value is more acidotic
- Oxygen tension (P_aO₂), reported in kilopascals (kPa) or mmHg.
- Carbon dioxide tension (P_aCO₂) (kPa or mmHg)

Other values such as bicarbonate [HC03⁻] expressed in mmol I^{-1} and Base Excess/Deficit (BE/D), are calculated. Base Deficit is the amount of base that would be needed to correct the pH of the sample to 7.4. Base excess is the amount of acid needed to correct to pH 7.4.

Normal Ranges_(SI units are preferred,) ie not mmHg)):

[H⁺] 40 +/- 4 nmol Γ^1 pH 7.4 +/- 0.04 (pH has no units) P_aO₂ (breathing air, F₁O₂ 0.21) about 13.3 kPa (less with healthy ageing) P_aCO₂ 5.1+/-1 kPa = 40+/- 5 mmHg (remains constant with healthy ageing) [HCO₃] 22+/- 2mmol Γ^1 BD/BE +/- 2

 $[H^+]$ gives the overall acid/base state for the patient. High $[H^+]$ is acidosis (acidaemia), low $[H^+]$ is alkalosis (alkalaemia) These states result from respiratory or metabolic causes, or a mixed pattern.

 P_aO_2 is a measure of arterial oxygen, a balance between oxygen delivery (a function of the cardiorespiratory system) and uptake by the tissues (aerobic metabolism). This varies normally with age and living at altitude, abnormally in cardio-respiratory disease.

The level of P_aCO_2 is a balance between production (cellular aerobic metabolism) and clearance. CO_2 is cleared in two ways. First, by ventilation (acute adaption over seconds) and second, by metabolic compensation (renal excretion) after conversion to HCO_3^- (chronic, over hours and days). [HCO3⁻] level indicates the adaptive responses to acidosis or alkalosis. Low [HCO3⁻] indicates acidosis, high alkalosis.

Four Step Interpretation of ABG

1. What is the $[H^+]$ (or pH)?

Acidosis if above normal, Alkalosis if below normal

2. What is the P_aCO_2 ?

If high, this is hypoventilation. If associated with a high $[H^+]$, it is respiratory acidosis.

If low, this is hyperventilation. If associated with a low $[H^+]$, it is respiratory alkalosis.

3. What is the base deficit or excess?

A base excess indicates metabolic alkalosis. A base deficit indicates metabolic acidosis.

4. What is the P_aO_2 ?

If higher than 13kPa, additional oxygen is being given.

CHEST X-RAY

Chest X-Ray is of no value in diagnosing hypoxia and should not delay immediate treatment, but can help with the diagnosis of specific conditions that cause hypoxia, especially of lung parenchymal disease and pleural or bony abnormalities after trauma (See section below and Chapter 5 on chest trauma).

CAUSES OF HYPOXIA IN POST-OPERATIVE PATIENTS:

PATIENTS AT RISK OF HYPOXIA

- Pre-op hypoxia
 - Smokers, COPD, severe lung or head injured patients
- Reduced FRC
 - Elderly, Obesity, Diabetes, General Anaesthetic
- Surgical pathology
 - Restricted ventilation, SIRS
- Post-op Sedation
- Hypothermia
- Fluid overload

COMMON CAUSES OF POST-OPERATIVE HYPOXIA

- Too early extubation
- Barotrauma
- Pulmonary oedema
- Bronchopneumonia
- Lobar pneumonia
- Pre-existent COPD
- Atelectasis with hypoventilation
- Pulmonary embolism
- ARDS

MECHANISMS OF BECOMING HYPOXIC

Hypoxia can occur via interference of physiological mechanisms of oxygenation at different levels, as discussed above. To keep things practical the causes of post-operative hypoxia are discussed in the following categories:

- 1. Lack of Alveolar Ventilation.
 - Adequate oxygen levels are prevented from entering the alveoli to facilitate gas exchange.

- 2. Lack of Alveolar Perfusion.
 - Inadequate levels of blood are supplied to the lung to facilitate gas exchange.
- 3. Decreased alveolar diffusion.
 - Adequate levels of both blood and oxygen are available in the alveoli and pulmonary circulation but alveolar pathology prevents gas exchange occurring.

LACK OF ALVEOLAR VENTILATION

Alveolar ventilation represents the volume of gas available to the alveolar surface area per unit time. Lack of alveolar ventilation can occur due to numerous mechanisms in the post-operative surgical patient, all of which should be considered during the assessment [4].

UPPER AIRWAY OBSTRUCTION

Airway obstruction must always be excluded as part of ABC assessment of the patient. Immediate clearance of an identifiable upper airway obstruction is necessary if identified [5].

Clinical Features: Features of upper airway obstruction are noted during the initial airway assessment. The airway may be noted to be blocked by a bolus. A patient with reduced consciousness may not be supporting his/her own airway and the soft tissues of the oropharynx impair airway efficiency.

Very rarely anaphylaxis will present post-operatively, with upper airway swelling and obstruction. In such a scenario the patient will be acutely unwell with additional bronchospasm and cardiovascular instability.

Management: Any identifiable obstruction should be immediately cleared if possible. Post-operative patients who become unwell with reduced level of consciousness may not be able to support their own airway effectively. A Guedel airway should then be inserted immediately. Further emergency assessment should be performed and the patient may require intubation and ventilation [6].

If anaphylaxis is suspected adrenaline should be administered immediately along with airway support, supplemental oxygen, intravenous hydrocortisone and intravenous fluid therapy [7].

RESPIRATORY DEPRESSION: OPIATES AND CARBON DIOXIDE NARCOSIS

An often encountered complication in post-operative patients that can present as hypoxia is reduced ventilation secondary to reduced respiratory drive. The commonest causes to consider are impaired consciousness due to drug toxicity (most commonly opiates) and impaired consciousness due to carbon dioxide narcosis.

Opiate toxicity occurs readily in post-operative patients who often have significant analgesic requirements. An opiate 'overdose' need not occur with high drug doses. Patients with renal impairment do not excrete opiates effectively and accumulation can

occur. The elderly are often also susceptible to such effects. In toxic concentrations respiratory depression occurs, with hypoventilation [8].

Carbon Dioxide narcosis occurs in patients with pre-existing Chronic Obstructive Pulmonary Disease (COPD). Respiration in some patients with COPD is dependent upon 'hypoxic drive', rather than hypercapnia [9]. If a patient is given high concentrations of oxygen, their respiratory stimulus is lost and respiratory depression occurs. The patient can quickly develop carbon dioxide retention, respiratory acidosis and reduced consciousness, causing hypoventilation and associated hypoxia.

Clinical features: The patient will present with signs of hypoxia, and reduced level of consciousness (GCS). A reduced respiratory rate will be noticed. In opiate toxicity pinpoint pupils will be seen. In carbon dioxide narcosis a bounding pulse is often noted and a history of COPD. An arterial blood gas sample can be taken to measure carbon dioxide levels.

Management: Initial management is to secure the patients airway and offer respiratory support as necessary. Patients with CO2 narcosis will require non-invasive ventilation (CPAP, BiPAP) if conscious or intubation and ventilation for respiratory support to facilitate 'blowing off' excess CO2.

In patients with suspected opiate toxicity management should be similar but naloxone, an opioid antagonist, should be administered immediately. If the patient recovers quickly always remember that the half life of naloxone is less than morphine, so the effect may wear off before the patient has metabolised enough morphine to prevent recurrent respiratory depression. Further doses may be required [10].

ATELECTASIS AND LOBAR COLLAPSE

Atelectasis is a frequently encountered post-operative complication causing hypoxia. It usually occurs in the first 48 hours following surgery. In abdominal and thoracic surgery the normal mechanisms by which mucus is cleared is impaired by pain, inhibiting deep breathing and coughing. Mucus retention occurs with resorption of alveolar air, leading to alveolar collapse. This normally occurs in the basal lobes. Secondary infection can then occur [11].

Clinical Features: Patients with atelectasis may report dyspnoea and cough with signs of hypoxia, tachypnoea and have reduced bibasal air entry. Normally this is following abdominal or thoracic surgery. There may be an associated fever, though recent evidence questions the link between fever and atelectasis [12].

Management: Management of atelectasis is primarily dependent on adequate physiotherapy and breathing excercise. It also includes assessment of degree of respiratory support required. In general supplemental oxygen therapy will be sufficient, but ventilation is required in extreme cases in a deteriorating patient. An assessment of the patient's analgesic requirements should be performed and this optimised to ensure they can breathe and cough without inhibition. Chest physiotherapy is required to ensure clearance of mucus and secretions. It is also helpful in preventing secondary infection [13]. Difficult cases may require immediate broncoscopic evaluation and treatment. Antibiotic treatment is not initially required unless secondary infection is suspected.

PNEUMOTHORAX

Pneumothorax can occur in post-operative patients either spontaneously or as an iatrogenic complication of a procedure such as insertion of central venous catheter. For detailed assessment and management details see the March 2012 review on chest trauma.

BRONCHOSPASM

Bronchospasm is a sudden constriction of bronchiolar muscle. It is stimulated by histamine release and degranulation of mast cells and basophils. Bronchospasm inhibits air entry and exit into the alveoli. It can occur in post-operative patients with pre-existing pulmonary conditions such as asthma or chronic obstructive pulmonary disease with contributing airway hyper-reactivity [14]. It is also a feature of anaphylaxis.

Clinical Features: The main clinical feature of bronchospasm is wheeze on auscultation of the chest. Often a history of COPD or asthma will be identified.

Management: Nebulised bronchodilators should be administered to the patient. Nebulised salbutamol or ipratropium bromide are highly effective [15].

Very rarely an acute asthma attack will become severe and life-threatening. Under these circumstances immediate input from intensive care is required as the patient may require intubation and ventilation.

Once the patient is stabilised, the patient's prescription chart should be reviewed. Nonsteroidal anti-inflammatory drugs are frequently administered as analgesia postoperatively and may have been the provoking factor for bronchospam. A new prescription of aspirin could also contribute [16]. These should be discontinued.

LACK OF ALVEOLAR PERFUSION (VENTILATION-PERFUSION MISMATCH)

PULMONARY EMBOLUS

Pulmonary embolus causes obstruction to the pulmonary vascular tree by an embolus, usually from a deep vein thrombosis of the pelvic or large leg veins. Inadequate pulmonary perfusion to adequately ventilated areas of the lung occurs, impairing gas exchange and causing hypoxia. Immobility, cancer and surgery are significant risk factors [17].

The embolus is not always thrombus; air embolism can occur following insertion of central venous catheters and rarely fat embolism can occur in patients who have sustained long bone fractures.

Clinical Features: Pulmonary embolus can present in a variety of fashions. Small, subclinical emboli can occur without any symptoms. In general, the larger the embolus and larger the ventilation/perfusion mismatch, the more profound the symptoms.

A massive pulmonary embolus classically presents with a collapsed, haemodynamically unstable patient who may have just visited the toilet (the straining dislodging the distal thrombus). Smaller emboli can present with dyspnoea, pleuritic chest pain and haemoptysis with signs of hypoxia. Other clinically useful signs are [18]:

- Tachycardia
- Tachypnoea
- Signs of DVT
- Low grade fever
- New onset arrhythmia

Investigations of use in identifying pulmonary embolus include an ECG which may demonstrate sinus tachycardia, atrial fibrillation, signs of right heart strain or the classic S1Q3T3 pattern. None of these are specific for pulmonary embolus however [19].

A CT Pulmonary Angiogram or Ventilation Perfusion scan are diagnostic if the patient is stable. In the unstable patient with diagnostic doubt, a portable bedside echocardiogram can be performed. Evidence of right heart strain and signs of increased pulmonary artery pressure are indirect signs of pulmonary embolus [20].

D-dimer testing is not useful in post-operative patients with pulmonary embolus as surgery increases serum levels [21].

If fat embolism is suspected urine can be sent for microscopy, where fat cells can be identified.

Management: Management of pulmonary embolus is dependent on the patient's clinical condition. If the patient is collapsed immediate resuscitation is necessary, with airway support, 100% supplemental oxygen and parenteral fluid therapy. If the patient can be stabilised, or in a non-collapsed patient, investigations to confirm the diagnosis should be performed.

Heparinisation under such circumstances is essential. A continuous infusion of unfractionated heparin is currently recommended for massive pulmonary embolus, while low molecular weight heparin is used in other cases. Adequate physiological support should be provided [22].

Thrombolysis has also been recommended in collapsed patients with massive pulmonary embolus. Absolute contraindications to this are active gastrointestinal or intracranial bleeding. Recent surgery is a relative contraindication. The risk of bleeding must be balanced against the risk of cardiovascular instability caused by pulmonary embolus [22].

Long term management in an established diagnosis of pulmonary embolus is anticoagulation therapy with warfarin for a period of 6 months.

The management of fat embolus is supportive therapy. Anticoagulants and thrombolysis have no role.

DECREASED ALVEOLAR DIFFUSION

<u>PNEUMONIA</u>

Pneumonia is a disorder marked by inflammation of the lungs, most commonly caused by bacteria in post-operative patients. Lung inflammation prevents adequate gas exchange, despite adequate ventilation and perfusion. Progression can lead to segmental bronchial collapse, reducing oxygenation further by preventing alveolar ventilation.

Colonisation of the lung with pathogenic bacteria due to aspiration of contaminated secretions, combined with relative immunosuppression due to surgery make this a common postoperative complication.

Distinction between hospital acquired and nosocomial pneumonia is essential in guiding antimicrobial therapy. Hospital acquired pneumonia is defined as pneumonia which occurs after 48 hours in hospital [23].

Clinical Features: A working diagnosis of postoperative pneumonia can be made in the presence of 3 or more of the following features without any other obvious cause: cough, sputum production, dyspnoea, chest pain, temperature >38 degrees Celsius and tachycardia [24]. One needs to be very careful in interpreting these symptoms as intra abdominal catastrophes such as anastomotic leak, sub diaphragmatic collections and post operative abscess collections manifest the same way and can be misdiagnosed as pneumonia.

Management : Initial management is supportive, with administration of oxygen and intravenous fluid therapy. Physiotherapy is essential to allow the patient to clear secretions from the lung [25].

Antimicrobial treatment is based upon local sensitivities. Typically postoperative pneumonia is poly-microbial, the majority being caused by gram negative aerobes. The most commonly isolated organisms are *Pseudomonas aeruginosa*, *Enterobacter* species, *Klebsiella pneumoniae*, *Acinetobacter* species, *Serratia* and *Citrobacter* species. In the absence of positive culture penicillin and an aminoglycoside should provide adequate cover [23].

The postoperative patient with pneumonia should be frequently reassessed to ensure that no further deterioration occurs. If the patient's condition worsens despite full supportive management and he/she appears to be tiring (especially with worsening tachypnoea), input from intensive care should be sought as the patient may require ventilatory support.

PULMONARY OEDEMA

Pulmonary oedema is accumulation of fluid in the lung parenchyma. It prevents effective diffusion of gas between the alveoli and pulmonary circulation, leading to hypoxia.

Pulmonary oedema can occur readily in postoperative patients. It is generally cardiogenic, as a result of :

- Acute deterioration in cardiac function:
 - Myocardial infarction, acute coronary syndrome or arrhythmia.
- Fluid Overload.
 - Excessive parenteral fluid therapy can cause left ventricular dilatation and cardiac failure.
 - With renal failure this can occur more readily.
 - Patients with pre-existing cardiac disease are more susceptible to this complication.

Clinical Features: The patient may appear to be in respiratory distress with dyspnoea, tachypnoea and signs of hypoxia. They may also have tachycardia, and a narrow pulse pressure. The patient may be hypotensive. An elevated Jugular Venous Pulse may be noted, or an elevated central venous pressure if monitoring is in place. On auscultation of the chest inspiratory crackles will be heard [26].

Fluid charts should be assessed for a discrepancy between urine output and volume of parenteral fluid administered. This may give a clue that the patient's deterioration is due to pulmonary oedema.

In acute deterioration, myocardial infarction should be strongly considered as a possible provoking cause . An ECG is essential, along with cardiac enzymes.

A chest x-ray may demonstrate features of interstitial oedema, Kerley B lines, a batswing appearance and upper zone diversion [27].

Management: Initial management requires supportive therapy, sitting the patient upright and administering 100% oxygen.

When the diagnosis is confidently established, frusemide 40 to 80mg can be given intravenously, and diuresis and clinical response monitored.

Intravenous nitrates can also be given if the systolic blood pressure is greater than 90mmHg. These cause peripheral vasodilation and reduce cardiac pre-load, improving function. An isosorbide dinitrate infusion at 2-10mg/h titrated to blood pressure should be given [28]. Low dose intravenous morphine (given as intermittent 1-2 mg IV boluses) will decrease pulmonary venous pressure and buy time until other therapies take effect.

The patient should then be reassessed. If there is no clinical improvement, a further bolus of frusemide should be given and either non-invasive ventilation (CPAP or BiPAP) or intubation and ventilation considered [29].

<u>ARDS</u>

Adult Respiratory Distress Syndrome (ARDS) is a condition characterised by inflammation of the lung parenchyma causing impaired gas diffusion. It normally presents as a sequel to severe systemic inflammatory response syndrome (SIRS), when release of pro-inflammatory cytokines causes macrophage activation and neutrophil recruitment in the lung. This causes local microvascular disturbance and parenchymal inflammation and oedema [30].

The inflamed lungs become 'stiff', reducing ventilator capacity and compounding the effect on respiratory function [31].

ARDS should not be diagnosed in a post-operative patient who has had uncomplicated elective surgery, but other parenchymal causes of hypoxia should be considered. It is more likely in patients with profound sepsis (who may have had surgery to correct the cause), extensive trauma or massive blood transfusion.

Clinical Features: ARDS occurs within 24 to 48 hours of major injury, sepsis or noninfective SIRS (e.g. pancreatitis). The post-operative patient with ARDS will generally show signs of clinical deterioration with tachycardia, tachypnoea, hypotension and hypoxia or increasing oxygen requirements. ARDS is characterised by [32]:

- Acute onset and rapid deterioration
- Bilateral infiltrates on Chest X-Ray sparing costophrenic angles
- Pulmonary artery wedge pressure <18mmHg (If Swan-Ganz catheter in situ)
- Clinical evidence of left ventricular dysfunction
- A PaO₂: FiO₂ ratio of < 200mmHg (the gradient between inspired oxygen level and that detected in blood)
- The absence of cardiac disease

Management: ARDS causes severe respiratory problems. Whilst supportive management with 100% supplemental oxygen and cardiovascular support may temporise the condition, management with intubation and ventilation is almost inevitable. Mechanical ventilation allows for stabilisation of the patients respiratory condition while the systemic cause of ARDS is treated and reversed. Until the systemic cause is addressed, ARDS is likely to persist [33].

There are little specific treatments for ARDS, although steroids, nitric oxide and surfactant therapy have been investigated in small studies [32].

SUMMARY

Mild to moderate hypoxia is a common surgical complication, often under-diagnosed. Severe life-threatening hypoxia is fortunately rare but needs rapid action to prevent death. A physiological approach guides rapid diagnosis of the pathophysiological cause of hypoxia and of support of oxygen delivery until a specific diagnosis of causitive disease process can be made. Decision for intervention is based mainly on clinical assessment, with discretionary interpretation of chest X-rays, blood gases and pulse oximetry.

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7. SHOCK (WITH SPECIAL REFERENCE TO TRAUMA)

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INTRODUCTION

This is the first of two critical care chapters focusing on "Circulation". This chapter will discuss the definition of shock, different classification systems, basic circulatory physiology and the pathophysiology of shock, with specific focus on hypovolaemic shock. The discussion on management will focus on the treatment of haemorrhagic shock, including discussion of some newer treatment strategies introduced from military medicine.

PHYSIOLOGY

Effective tissue perfusion depends on capillary perfusion pressure, which is dependent on arterial blood pressure; this depends on cardiac output and peripheral resistance (BP = CO x PR). Cardiac output depends on stroke volume and heart rate (CO = SV X HR); stroke volume is determined by preload, contractility and afterload. Preload is delivered by venous return to the heart through the superior and inferior venae cavae, and influenced by the capacity and filling of the large capacitance veins, e.g. in the splanchnic system. The effect of preload on stroke volume is represented through the Starling curve, which indicates the direct correlation between stretching myocardial fibres which stimulates more forceful contraction until a point is reached where the muscle will not contract stronger with further stretching (at this point the venous system is "full"); further filling will now lead to overstretching and decreased contraction.

Contractility is influenced by oxygen and substrate supply to myocytes, and inotropes will move the Starling curve upwards (giving stronger contraction for the same muscle fibre length). Afterload can be influenced by any narrowing of the outflow tract such as aortic stenosis but in healthy patients depends on vascular resistance. Vascular resistance is determined mainly (60%) by the pre-capillary arteriolar bed. Peripheral blood flow is influenced by vessel length, viscosity of the blood and predominantly by blood vessel diameter, as described through Poiseuille's equation [1].

		Δ	=	Change
Blood Flow =	<u>_ΔP r⁴ π</u> η L (8)	Р	=	Pressure
		r	=	Radius of vessel
		π	=	constant (3.14)
		η	=	Viscosity of blood
		L	=	Vessel length

With a systemic mean aorta pressure of 95 mmHg the pressure is still 95 in arteries of 0.3 mm in diameter. The pre-capillary arteriolar bed provides 60% of the peripheral resistance so that, at the arteriolar end of the capillaries, pressure has been reduced to 30-35 mmHg [2,3].

The arterioles' diameter is regulated by a large number of processes and substances [4]: Vasoconstriction is caused by noradrenaline (mainly in the skin, skeletal muscle and splanchnic circulation), adrenaline (skin), alkalosis, thromboxane A2 and serotonin. Vasodilatation is caused by a decline in sympathetic tone, acetylcholine (from the sympathetic system in skeletal muscle), adrenaline in skeletal muscle and the liver, bradykinin with inflammation, histamine from mast cells with IgE stimulation, prostacyclins, increased temperature and nitric oxide; the most potent local vasodilators, however, are hypoxia and increased hydrogen ions.

In essential organs the circulation depends on autoregulation to maintain relatively constant flow with a mean blood pressure of 60-160 mmHg, e.g. the cerebral, coronary and renal circulations. When blood pressure drops the resulting tachycardia and increased contractility increases oxygen demands of the myocardium, which maintains local vasodilatation of the coronary system.

PATHOPHYSIOLOGY AND TYPES OF SHOCK

Shock can be defined as inadequate end-organ perfusion due to circulatory collapse, leading to cellular hypoxia, anaerobic metabolism and cell death.

Traditionally shock is classified according to the <u>etiology</u> of circulatory collapse into Hypovolaemic, Cardiogenic, Neurogenic, Anaphylactic or Septic shock.

A more practical <u>physiological</u> classification taught in critical care is based on the pathophysiological processes that lead to shock, i.e. absolute hypovolaemia (e.g. haemorrhagic shock), relative hypovolaemia or distributive shock, cardiogenic shock, and obstructive shock [5].

HYPOVOLAEMIC SHOCK (TRUE HYPOVOLAEMIA)

The most common cause of true hypovolaemia is haemorrhagic. With hypovolaemia there is a drop in venous return, with an immediate drop in stroke volume; this results in lower blood pressure, less baroreceptor stimulation, lower vagal parasympathetic discharge, allowing sympathetic dominance, leading to tachycardia and higher contractility, shifting the Starling curve upwards.

A number of other compensatory processes are also triggered [6]:

1. Peripheral vasoconstriction, mainly in the skin and viscera, sparing the brain and heart initially.

2. Venoconstriction especially of the splanchnic circulation, pulmonary veins and subcutaneous tissue. Splanchnic venoconstriction can pump a litre of blood into the circulation within a minute.

3. Hyperventilation with increased thoracic pumping.

4. Cerebral agitation increases muscle activity and pumping.

5. Movement of interstitial fluid into capillaries, as described through the Starling equation.

6. Increased secretion of noradrenaline and adrenaline.

7. Increased secretion of vasopressin, renin, angiotensin, aldosterone, erythropoietin and glucocorticosteroids.

8. With moderate haemorrhage the circulating plasma volume is usually restored after 12-72 hours.

DISTRIBUTIVE SHOCK (RELATIVE HYPOVOLAEMIA)

In distributive shock the circulating volume is insufficient due to uncontrolled vasodilatation. These include the traditional septic, anaphylactic and neurogenic shocks. In neurogenic shock this is secondary due to high spinal cord transection and immediate loss of sympathetic vascular tone; in anaphylaxis there is rapid arteriolar and capillary vasodilatation secondary to mainly histamine release secondary to a type 1 hypersensitivity reaction; in septic shock arteriolar vasodilatation is caused by mediators released secondary to the inflammatory response, e.g. bradykinin, histamin, prostaglandins and nitric oxide.

With distributive shock the patient can also develop true hypovolaemia. In neurogenic shock this can be secondary to other injuries and blood loss, or to dehydration due to pre-hospital delay. With anaphylaxis, major trauma and sepsis there is increased capillary permeability secondary to inflammatory mediators which depletes the intravascular volume further. It is therefore not uncommon to have different types of shock occurring simultaneously in the same patient.

NEUROGENIC SHOCK [7]

After high spinal transection above the first thoracic segment, there is a very marked fall in blood pressure due to removal of the influence of the vasomotor centre on neurones of T1 to L2 of the spinal cord [8]. These neurones give rise to pre-ganglionic fibres of the sympathetic nervous system which maintain sympathetic outflow to arterioles for vasoconstriction and normal peripheral resistance and to veins for venoconstriction to prevent venous pooling. With high spinal cord transection this effect is lost and arterioles dilate, causing a fall in peripheral resistance; veins also dilate causing blood to pool with a consequent reduction in venous return.

Heart rate is slow because the vagal supply to the heart is intact but sympathetic supply has been lost. The patient is hypotensive, but there is no tachycardia or vasoconstriction of skin blood vessels. If a patient with a high spinal transection also has sustained blood loss, hypotension will be severe because there is no sympathetic effect to compensate for the hypovolaemia

ANAPHYLACTIC SHOCK [9]

Due to a type 1 hypersensitivity reaction there is massive degranulation of mast cells and basophils with the release of a wide variety of mediators, predominantly histamine, into the general circulation.

The effects of histamine are mediated via the activation of mainly H1 receptors. Histamine causes relaxation of smooth muscle in arterioles and pre-capillary sphincters, resulting in a fall in peripheral resistance. Dilatation of post capillary venules leading to venous pooling.

Prostaglandin E2 and Slow Reacting Substance of Anaphylaxis (SRS-A) increase capillary permeability and histamine increases the permeability of post capillary venules; fluid passes from the intravascular to interstitial fluid resulting in oedema.

The result is massive vasodilatation and a marked fall in blood pressure with circulatory collapse, the passage of fluid into the interstitial space, hoarseness, laryngeal oedema, bronchospasm and respiratory distress and urticaria.

Several antigens might precipitate anaphylaxis such as drugs (e.g. penicillin, anaesthetic drugs), foodstuffs, wasp and bee stings.

Histamine, SRS-A, prostaglandin F2 and thromboxane A2 contract smooth muscle of the bronchial walls, resulting in intense bronchospasm.

SEPTIC SHOCK [10]

Septic shock is caused by Gram-negative bacilli in 70% and staphylococci in up to 25% of cases. Bacteria trigger a local and systemic inflammatory response. This can be worsened by:

- Secretion of exotoxins (e.g. by Streptococci, Staphylococci and Clostridium tetani).
- Release of endotoxins (part of the cell wall lipopolysaccharide) by Gram-negative bacilli on death of the organism.

Fever occurs due to the effects of Interleukin-1 and tumour necrosis factor (TNF α) on the hypothalamic temperature regulation areas, and contributes to vasodilatation.

Arteriolar vasodilatation and a fall in peripheral vascular resistance is predominantly caused by bradykinin, histamine, prostaglandins and nitric oxide (NO). Cardiac output is raised as a result of increased heart rate and increased stroke volume, partially as a compensatory mechanism and partially due to direct stimulation of the myocardium by mediators and raised temperature. This causes a hyperdynamic circulation.

The patient is hypotensive with a narrow pulse pressure and has tachycardia. Their skin is cold, pale, cyanosed and sweating. Tissue perfusion worsens. In the early stages of sepsis, as a consequence of arteriolar dilatation and increased capillary permeability, there is transfer of fluid from capillaries to interstitial space. Blood volume, venous return, cardiac output and blood pressure all fall with time. As a consequence of a low blood pressure sympathetic activity is increased causing vasoconstriction in skin, splanchnic circulation, kidneys and muscle. Myocardial function now becomes reduced, due to a combination of hypoxia, metabolic acidosis, platelet activating factor and tumour necrosis factor. Fluid resuscitation alone may be inadequate to maintain the patient's blood pressure due to vasodilatation. Production of myocardial depressing factor is also believed to play some role in the reduction of myocardial function.

CARDIOGENIC SHOCK

Cardiogenic shock occurs due to failure of myocardial contractility, i.e. Starling's curve becomes flatter. This can be caused by hypoxia of myocytes in acute myocardial ischaemia, arrhythmias e.g. atrial fibrillation, ventricular tachycardia, overstretched myocytes e.g. in cardiac failure and dilated cardiomyopathy, and direct myocardial suppression due to e.g. myocardium depressant factor of acute pancreatitis, or in the late phase of sepsis.

OBSTRUCTIVE SHOCK

Obstructive shock means that the heart cannot pump effectively due to obstruction primarily to inflow of venous return, and occasionally to arterial outflow. The most important causes of obstructive shock in the surgical patient are tension pneumothorax, cardiac tamponade and massive pulmonary embolism.

In tension pneumothorax the mediastinum is shifted to the opposite side, which kinks the venae cavae and obstructs venous return acutely. In cardiac tamponade fluid (usually blood) within the pericardial sac compresses the heart; this firstly stops venous inflow into the atria and can also limit ventricular expansion, decreasing stroke volume. With large pulmonary emboli the outflow of the right heart is restricted (a low pressure pump) which can cause acute cor pulmonale and the inflow into the left atrium is decreased, decreasing left ventricular stroke volume.

HAEMORRHAGIC SHOCK IN TRAUMA

Although trauma as a cause of haemorrhagic shock is discussed here in some detail, similar principles would apply in the assessment and management with any other cause of bleeding in surgery, e.g. bleeding peptic ulcer, aorta or pseudo-aneurysm rupture, lower gastro-intestinal bleeding from Meckel's diverticulum or colonic diverticular disease.

CLINICAL PICTURE

The earliest clinical signs of shock are usually secondary to compensatory mechanisms. Tachycardia is usually the earliest sign of shock; any injured patient who is cool and tachycardic is in shock until proven otherwise. Narrowed pulse pressure indicates significant blood loss. Compensation can prevent systolic pressure drop until loss of 30% of blood volume, but even in young healthy adults further loss of blood can lead to rapid decompensation and threat to life.

Young children and infants especially have very strong compensatory mechanisms. Children will often have tachycardia but no blood pressure drop until very late, and then decompensate rapidly, with severe tachycardia, tachypnoea and cerebral obtundation. Elderly patients can get hypotension early but without tachycardia, and tolerate hypotension poorly due to atherosclerosis, reduced cardiac compliance and medication (beware β -blockers, pacemakers).

Haemorrhagic shock is characterised clinically by the degree of blood loss and the resultant physiological response by the circulatory system. Four classes of shock can be described according to the degree of blood loss [11]:

	1	2	3	4
Blood loss (ml)	<u><</u> 750	750-1500	1500-2000	> 2000
Blood loss (%)	<u><</u> 15%	15-30%	30-40%	>40%
Pulse rate	<100	>100	>120	>140

BP: Syst/Diast pulse pressure	N/N Normal	N/Raised Decreased	Down/Down Decreased	Very low Decreased
Resp rate	14-20	20-30	30-40	>35
Urine output	>35 ml/h	20-30 ml/h	5-15 ml/h	almost 0
Mental state	alert	anxious	aggressive	apathy
Extremities	normal	pale	pale & cool	pale & cold
Complexion	normal	pale	pale	ashen
Initial fluid replacement	crystalloid	crystalloid/ colloid	cryst/colloid + blood	cryst/colloid +urgent blood

The major areas of blood loss following trauma are external losses, bleeding into the chest and abdomen, and blood loss as a result of fractures to the long bones and the pelvis.

CHEST TRAUMA:

Chest trauma is present in 15% of all trauma and accounts for up to a quarter of the mortality in all trauma cases [12]. Mortality secondary to chest trauma can be as high as 77% [13].

The clinical examination of the chest in the primary survey per ATLS principles assists in detecting haemorrhage in the chest. A haemothorax could have one or more of the following signs: reduced air entry to the affected side, asymmetrical inspiration and dullness to percussion. Up to 30% of total blood volume can be accommodated in each side of the chest [14]. The diagnosis can be confirmed by the placement of a chest thoracostomy tube or if the haemothorax is smaller and clinically less apparent may be detected on a plain chest X-ray film.

ABDOMINAL TRAUMA:

Abdominal trauma accounts for 30% of trauma cases, with blood loss most commonly from injury to the liver and spleen (16% of cases) [15].

In penetrating abdominal trauma there are often clinical clues as to the cause of injury, blood loss and the need for surgical intervention. In blunt trauma signs of abdominal bleeding are often subtle and a high index of suspicion is required. Clinical examination can be supplemented with ultrasound scanning (FAST) and selective use of CT scan,

particularly when the patient has a reduced level of consciousness or there are other distracting injuries.

There may be external injuries that can alert the clinician e.g. 30% of patients with abdominal abrasions from seat belts will have internal injury. Rib fractures can suggest liver or splenic injury and left shoulder tip pain can indicate splenic injury.

Abdominal distension may be evident as a result of blood filling the peritoneal cavity but this is a late and unreliable sign because up to 3 litres blood may be present before distension is noted [16]. Retroperitoneal bleeds will only be detected by suspicion of mechanism and radiological studies.

Examination of the perineum can suggest bleeding from pelvic fractures and direct lacerations. Gross or macroscopic haematuria is observed in 65% of patients with renal trauma and can reflect ongoing bleeding of the kidney or urinary tract [17].

PELVIC TRAUMA AND LONG BONE FRACTURE

Pelvic and long bone fractures can result in significant blood loss and reflect high velocity injury and are often associated with other significant injuries [18]. Fracture of the pelvis may hide up to three litres of blood loss without any external signs or visible abnormality of the pelvis. Clinical examination may suggest pelvic instability and radiological confirmation will be necessary. Femoral fractures can cause significant blood loss and open long bone fractures are capable of causing damage to major vessels.

DEFINITIVE DIAGNOSIS

In making definitive diagnoses it is critical to understand the mechanism of injury is very important and this is probably the most effective diagnostic tool [19]. It helps to clarify whether injury is a result of blunt or penetrating trauma, high velocity trauma or a fall from a height.

FAST (Focused Abdominal Sonogram of Trauma) scanning has been utilised with success in the emergency department to assess the presence of fluid or blood within the abdomen. It is most useful for deciding on the need for operative intervention in blunt trauma [20, 20a, 20b].

CT scanning of the patient is a useful adjunct to the resuscitation phase. It has been shown to have a sensitivity and specificity of greater than 90% for detecting organ damage in trauma patients when the patient is haemodynamically stable [21]. It can be used to detect bleeding in the spleen and pelvis that can be managed by interventional radiology as opposed to a surgical operation.

CLINICAL SEQUELAE OF UNTREATED/UNDERTREATED SHOCK

IMMEDIATE

With substantial blood loss, usually 40% or more of circulating blood volume, the injury is life-threatening and without intervention death is imminent (grade 4 shock). Death can

then occur within minutes and major evacuation and pre-hospital matrices have been established to lessen the risk of these potentially preventable deaths. In military medicine catastrophic haemorrhage is viewed as a primary preventable cause of death from trauma [22]. Time is critical; it has long been shown that shortening the time from point of wounding to an operating theatre improves overall survival [23]. Rapid exsanguination can cause death before airway obstruction and hypoxia and therefore a <C>ABC sequence is often followed in military resuscitation in battlefield situations.

LETHAL TRIAD

The major complications of haemorrhagic shock result from end organ hypoperfusion and these can manifest within hours of injury. Such complications can include secondary coagulopathy, hypothermia and metabolic acidosis; if these three occur together it has been described as the lethal triad of trauma:

SECONDARY COAGULOPATHY

As the injury victim continues to bleed they lose blood and coagulation proteins; initial resuscitation with crystalloids further dilutes the remaining coagulation factors. This dilution and depletion, which occurs within hours of injury, cause a secondary coagulopathy. It further worsens bleeding from the injury, propagating the cycle of bleeding. If resuscitation continues but the coagulation cascade proteins are inadequately repleted, bleeding will continue. With tissue hypoperfusion acidosis and hypothermia develop that worsen this secondary coagulopathy.

In trauma victims who have developed early trauma-induced coagulopathy (ETIC), secondary coagulopathy will worsen the effects of ETIC significantly [24]. One hypothesis is that ETIC is caused by endogenous activation of protein C [25].

HYPOTHERMIA

A combination of factors can result in the lowering of core temperature in haemorrhagic shock, e.g. loss of blood itself, widespread vasoconstriction, poorer tissue perfusion with decreased metabolism, exposure and resuscitation with cold fluids.

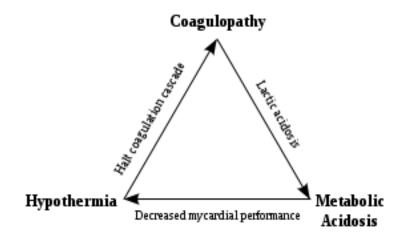
Up to 66% of trauma patients become hypothermic and almost all patients experience a drop in core temperature with initial resuscitation [26, 27]. Hypothermia is associated with an increase in mortality [28].

Hypothermia inhibits the function of clotting factors (because these are enzymes), worsening haemorrhage and acidosis [29]. Hypothermia contributes to adrenergic stimulation and vasoconstriction, worsening hypoperfusion and accelerating the process to multi-organ failure [30].

METABOLIC ACIDOSIS

Metabolic acidosis occurs following haemorrhagic shock as reduced tissue oxygen delivery results in increased anaerobic metabolism and accumulation of lactic acid . Rapid resuscitation with hyperchloraemic solutions such as normal saline and packed red cells, with higher levels of hydrogen ion concentrations, contributes to acidosis after injury [31].

Acidosis contributes to secondary coagulopathy. Hydrogen ions interfere with calcium binding and disrupt clotting factor interactions. An acidic environment inhibits platelet function. Acidosis has a negative inotropic effect on myocardial contractility which further reduces blood flow and worsens acidosis.



These three factors: coagulopathy (both ETIC and secondary), hypothermia and metabolic acidosis have a synergistic effect on each other. Blood loss leads to end-organ hypoperfusion resulting in anaerobic metabolism. This leads to lactic acidosis which has an inhibitory effect on clotting factors and myocardial contractility. This leads to further haemorrhage and hypotension. Blood loss leads to temperature drop which affects coagulation. The whole process is further exacerbated by dilution and cooling due to intravenous fluid replacement. When all three factors are present the mortality risk becomes >50%, even with intervention at this stage.

SYSTEMIC INFLAMMATORY RESPONSE, MULTI-ORGAN FAILURE AND SEPSIS:

Trauma triggers a systemic inflammatory response (SIRS) through a range of mediators that activates endocrine, autonomic nervous and immunological systems.

Some mediators of the systemic inflammatory response seem to be associated with outcome following trauma. A correlation exists between mortality and levels of a DNA protein called High Molecular Group Box 1 (HMGB1) in trauma patients [32]. Patients who develop late complications such as renal failure and acute lung injury following trauma and major surgery have been shown to have significantly higher plasma levels of HMGB1 [33].

Battlefield casualties who survive their initial injuries and reach a field hospital often do not die of their injuries but due to late complications from the systemic response to their injuries. These include acute lung injury, multi-organ failure and nosocomial infection. This is the leading cause of late mortality following trauma [34].

Sepsis is common (>14%) following trauma. Mortality is higher in trauma victims with sepsis compared with those without (23% versus 10% respectively) [35].

MANAGEMENT OF HAEMORRHAGIC SHOCK

INITIAL RESUSCITATION

The initial management of a patient with haemorrhagic shock is centred around restoring the intravascular volume in order to maintain blood flow to vital organs (brain and heart).

During primary assessment of the trauma or other critically ill surgical patient, "C" stands for circulation and follows primary assessment of airway and breathing("A" and "B"); if any signs of shock or hypoperfusion are noted, intravenous access is obtained. Peripheral access with two large bore cannulae in the antecubital fossa preferred (18 gauge catheter or larger). In injuries that cause rapid exsanguination e.g. due to explosions or traumatic amputation, or other causes of major vascular injury, control of haemorrhage might have to take preference over airway and breathing management (<C>ABC).

Fluid replacement with an isotonic crystalloid solution is started, preferably Ringer's lactate/Hartman's solution, or normal saline. According to ATLS protocol an initial 2 litre fluid is given as rapidly as possible. Pressure on the crystalloid bags or hanging the bags high above the patient can increase the rate of flow.

In haemorrhagic shock the main replacement fluid is blood. In trauma patients in particular, there is increasing evidence that crystalloid is only useful to maintain the patient until blood is available for transfusion. In many trauma centres where blood is immediately available, blood transfusions are started preferentially over crystalloid. If blood and blood component products such as fresh frozen plasma are available these can be started before the traditional 2 litres of crystalloid fluid is finished.

There has long been recognition of the advantages of blood product usage over crystalloids because it avoids the disadvantages of large volumes of crystalloid and because of the need for an oxygen carrying solution. Recently a new condition has been described, early trauma-induced coagulopathy (ETIC), which has further pushed the use of blood products early. ETIC is seen in up to 25% of trauma patients and occurs immediately after injury. It is measured as an elevated prothrombin time, usually noted on the first blood draw after trauma, often within an hour of injury. The presence of ETIC is associated with reduced survival (40-50%), independent of other risk factors for death [36]. It appears to have a different pathophysiology than secondary coagulopathy. It is likely that there is overlap between the occurrence of both coagulopathies. As research continues to elucidate the mechanism for the development of ETIC, its relationship to secondary coagulopathy and the lethal triad will become clearer.

The clinical implication of ETIC has been to focus resuscitation on correcting this coagulation defect early before any obvious clinical signs develop. Both civilian and military trauma patients have improved survival when they receive blood products early with fresh frozen plasma in a physiological ratio (close to 1:1), compared to patients who receive less fresh frozen plasma early in resuscitation (1:8) [37]. Massive transfusion protocols that include blood, fresh frozen plasma and cryoprecipitate have been developed and are triggered early upon arrival of a patient with haemorrhagic shock. These protocols have decreased the need for damage control surgery (discussed below).

REASSESSMENT

Whilst resuscitation is being initiated simultaneous clinical assessment should also be performed to assess the source of bleeding and initiate definitive management. During resuscitation the patient should be continuously monitored for response to treatment. A decision about response to fluid resuscitation should be taken not later than 30 minutes after starting. Response to resuscitation should be graded as:

A. Rapid responder:

- Vital signs return to normal;
- Time for further assessment & treatment.
- B. <u>**Transient responder**</u> (responds initially but pressure drops as soon as fluid administration slows):
 - Patient has ongoing blood loss;
 - Needs blood urgently;
 - Early intervention to stop bleeding is probably necessary.
- C. <u>None/minimal responder</u> (resuscitation has no/little effect in improving perfusion):
 - The patient needs blood immediately;
 - Surgical intervention is necessary as part of resuscitation [11].

Ongoing clinical assessment remains the primary method to determine the patient's response and further decision making about ongoing requirements. Looking at any one sign alone can be misleading and it is the overall picture of the patient that is important. This means considering all signs and parameters together, with their trends over time, to ascertain if the resuscitation is improving perfusion of the patient's organs. The physiological signs of heart rate, blood pressure, respiratory rate, urine output and mental status often respond the quickest. Other injuries may mask this response, however, e.g. concomitant brain injury or spinal cord injury. Other markers such as base deficit or lactate levels are therefore often used to follow the response to resuscitation. These markers require laboratory assistance, can be expensive and meaningful change is usually only seen over hours or even days. The values can be affected by comorbidity such as liver, kidney or heart failure, or by consumption of large amounts of alcohol preinjury. Clinicians still lack reliable, valid, inexpensive *direct* measures of organ perfusion and intravascular blood volume that can be used at the bedside in patients with shock. We therefore rely greatly on interpretation of clinical signs and measurement of basic pressure and flow parameters.

CRYSTALLOID VERSUS COLLOID:

Colloids are believed to be beneficial in the absence of blood because of their higher oncotic pressure compared to crystalloids. It is believed that they would stay longer in the intravascular space, improving organ perfusion.

There is a growing body of evidence that colloids are not any more effective at restoring tissue perfusion than crystalloids and may even be harmful. A recent meta-analysis showed increased mortality rates in trauma patients following initial resuscitation with colloids when compared to crystalloids [38]. In critically injured patients in ICU, the SAFE study (the Saline versus Albumin Fluid Evaluation) showed that the use of a 4% albumin solution rather than crystalloid increased the mortality rate for patients with head injury.

Overall this study showed no survival benefit for the use of colloids over crystalloids [39]. A subsequent Cochrane review found a similar result, i.e. that there was no survival benefit for colloids over crystalloids in the management of patients after trauma [40]. Colloids also have disadvantages such as increased cost, side effects such as coagulopathy, allergic reactions and potential for unwanted viral transmission with albumin solutions.

Therefore, the use of crystalloids is preferred over colloids even if blood products are not readily available for hemorrhagic shock. Ringer's lactate is preferred to normal saline as the chloride ion concentration is much less and therefore less likely to cause hyperchloraemic acidosis, which can be seen after administration of large volumes of normal saline.

HAEMORRHAGE CONTROL: TOURNIQUETS AND HAEMOSTATIC AGENTS

Control of external haemorrhage is included in "C" in the primary survey. Several methods of bleeding control have been devised and utilised in both civilian and battlefield settings.

Historically the use of tourniquets has been controversial. Recently the select use of tourniquets has regained popularity in areas with high incidence of extremity trauma, such as with IED explosions. Tourniquets, placed effectively by trained pre-hospital personnel and rapid transport to definitive care has been shown to improve survival and even limb salvage. Placing the tourniquet just above the bleeding vessel or wound and tightening only enough to stop or significantly reduce haemorrhage maximizes the utility while reducing unwanted side effects. Tourniquets are not useful in injuries that result in non-compressible haemorrhage and inappropriate non-standardised use can result in unnecessary loss of limb [41].

A number of haemostatic agents have been developed for application to external bleeding wounds. Novel haemostatic agents such as Quikclot ©, a granular preparation that produces an exothermic reaction on contact with plasma to concentrate clotting factors, and Hemcom ©, a dressing impregnated with a polysaccharide (chitosan) which provides haemostasis by adhering directly to the wound, can be used in a prehospital setting to reduce haemorrhage. These agents have been mainly used in military settings but are expensive, limiting general use.

DAMAGE CONTROL RESUSCITATION

Many patients respond well to initial resuscitation with fluid and blood products and either stop bleeding or have their bleeding stopped and return to a normal physiology with minimal sequelae. In patients who have ongoing bleeding or extensive blood loss, who have developed ETIC or arrived late to definitive care, normalisation of physiological parameters is more difficult even with institution of adequate resuscitation. In patients who received large volumes of crystalloid, whole blood or blood products without fresh frozen plasma this can result in adverse effects that worsen the injury-induced bleeding. The mortality rate of patients who have developed the lethal triad of hypothermia (temperature below 35 degrees Celsius), secondary coagulopathy (abnormal partial thromboplastin time, abnormal prothrombin time and thrombocytopenia) and acidosis (pH less than 7.2) can be 70%. Therefore, the approach with these patients is to perform

damage control surgery (discussed below) and treat the physiological derangements in the ICU setting as soon as possible to prevent complete metabolic failure and death.

The early (upon arrival) use of coagulation products in the form of fresh frozen plasma and/or whole blood with cryoprecipitate, with prevention of hypothermia and early arrest of surgical bleeding, has drastically reduced the incidence of the lethal triad and thereby the need for damage control surgery.

This concept of early blood products with coagulation factors (if available) is referred to as **haemostatic resuscitation**. Haemostatic resuscitation, with permissive hypotension and damage control surgery (both discussed below) are referred to as damage control resuscitation (DCR).

Due to ETIC and the potential for rapid development of secondary coagulopathy, laboratory clotting tests are often too slow for clinical use. Therefore a proactive approach to ETIC and secondary coagulopathy needs to be adopted.

Current US and UK military practice is to resuscitate with a ratio of 1:1 of fresh frozen plasma (FFP) and packed red blood cells (PRBC) [42]. Retrospective analysis of this combination has shown decreased mortality rates when compared to PRBC:FFP given in a ratio of 8:1 [43]. The addition of platelets to this combination in a ratio of 1:1:1 of packed red blood cells, fresh frozen plasma and platelets has also shown improved mortality rates in one study [44].

A further factor that may be beneficial is the addition of fibrinogen as opposed to FFP because it is specifically fibrinogen that is believed to become deficient first [42]. FFP actually has relatively low concentrations of clotting factors and therefore requires significantly more volume to replace. This can worsen dilution rather than improve coagulation.

Permissive hypotension is another component of damage control resuscitation. Hypotensive resuscitation is the avoidance of aggressive fluid resuscitation; the therapeutic aim is to maintain a systolic blood pressure of 90mmHg and prevent tachycardia [45]. In the pre-hospital setting this approach is challenging as non-invasive blood pressure monitoring is often inaccurate. This approach is most useful in penetrating trauma where arterial transection or laceration of an organ could cause more bleeding if the arterial pressure is increased to normal; bleeding is relatively controlled through the preservation of immature clots at a lower systolic pressure. This approach is safe when the patient is transported rapidly to a hospital with definitive surgery capacity, and in one study showed an 8% decrease in mortality in penetrating torso trauma patients [46]. The widespread use of permissive hypotension beyond these indications is still controversial as there have been studies which show no mortality benefit [47].

Another approach to resuscitation, as used in the military is to resuscitate with 250ml boluses of crystalloid until a radial pulse is palpable. The radial pulse is a useful monitor of systolic blood pressure as it is often reliably present above 90mmHg. This measure is not useful should there be no radial pulse available to monitor as can occur with severe double upper limb injury.

DAMAGE CONTROL SURGERY (DCS)

For the trauma patient with severe haemorrhagic shock all complications are worsened by long operating time (e.g. ETIC, secondary coagulopathy, acidosis and hypothermia); an open abdomen or chest only worsens metabolic failure. Surgery adds to the hypothermia, adds to coagulopathy through ongoing bleeding from induced surgical trauma or opening controlled haematomata, and adds to acidosis through anaesthetic drugs which lower systemic resistance. The correction of metabolic derangements is only possible within an intensive care setting. A patient with haemorrhagic shock therefore needs surgery only to control surgical bleeding, preventing or ameliorating the metabolic factors that worsen bleeding.

The concept of damage control surgery was therefore instituted with a focus on haemostasis control only. Definitive repair and restoration of anatomy is not considered as part of this type of surgery and actually increases the risk of mortality if performed during a damage control operation.

The aim is to "turn off the tap" and prevent the lethal triad [48]. Once haemorrhage has been stopped, physiological normalisation can occur more easily within the ICU; once stabilized the patient can then go back to theatre for definitive repair and restoration of anatomy.

The main techniques in DCS include ligation of bleeding vessels, packing bleeding viscera and shunting of major vessels; open bowel ends are stapled or tied off for later re-anastomosis; gastric, biliary or pancreatico-duodenal injuries are drained only. Formal closure of the abdominal wall is not performed to reduce operating time and prevent compartment syndrome [49].

Duration of damage control surgery should be limited to the least possible time period, preferably <1 hour. Active significant bleeding has to be contained, however; intraoperative packing is a common and effective approach to rapidly control massive haemorrhage.

Damage control surgery is most effective in improving survival when utilized early. In rapidly bleeding patients the decision to go to theatre should be taken early and decisively by the most senior surgeon available. Once the lethal triad has become established it is often irreversible and survival is unlikely.

Moore et al (1998) has suggested criteria on which damage control surgery should be considered [50]: pH <7.1, systolic blood pressure <90mmHg, core temperature <34°C and Injury Severity Score >25. These physiological factors are now considered a late stage of the lethal triad and many surgeons would recommend DCS much earlier to prevent deterioration to these parameters. Good clinical judgement by a senior surgeon to operate and an early decision to stop the operation after reasonable haemorrhage control provide best outcomes.

There are no randomised controlled trials to support the efficacy of damage control surgery but retrospective analysis has shown reduced mortality rate in military [51] and civilian trauma patients.

SUMMARY

Shock is decreased tissue perfusion due to circulatory collapse, leading to cellular hypoxia, anaerobic metabolism and cell death. Haemorrhage is the usual cause of shock in trauma patients but different types of shock can occur simultaneously in the same patient. If resuscitation is delayed the lethal triad of coagulopathy, hypothermia and acidosis will probably develop. The best fluid for resuscitation is blood, with rapid supplementation with clotting factors and platelets if available. Early surgery to stop bleeding is often necessary and can be life-saving.

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8. FLUID THERAPY; RENAL AND CARDIAC DYSFUNCTION

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INTRODUCTION

This is a second chapter addressing "Circulation". Although most surgeons are quite confident in managing the initial phases of haemorrhagic shock, most find it difficult to deal with renal or cardiac complications. These are usually handed over to the anaesthetist or medical specialist to manage, but if one understands the basic pathophysiology of when these systems go wrong, it is possible to solve many problems through a logical physiological approach. This is what this article is about.

Both the heart and the kidneys depend on a minimum perfusion pressure and circulatory volume to function, i.e. the vascular system must be "full"; for the heart this is described as "preload" and for the kidneys as "glomerular filtration pressure". Both organs have inherent cellular mechanisms delivering "contractility" in the heart and "exchange mechanisms" in the kidney; in both organs this inherent function is dependent on oxygen and substrate delivery, can be stimulated by drugs (e.g. inotropes for the heart and diuretics for the kidneys, but such drugs will only work effectively if the preload/perfusion pressure is effective). Function of both organs can also be damaged by sepsis and drugs. Lastly both organ systems are dependent on "afterload", dependent on peripheral vascular resistance for the heart and affected by outflow obstruction in the kidney (e.g. in abdominal compartment syndrome).

Effective fluid replacement therapy is essential in maintaining both cardiovascular and renal function in the critically ill patient. For this reason fluid therapy is discussed here. This article therefore has three distinct parts: fluid therapy, renal dysfunction and cardiac complications in critical care, but there is significant overlap in pathophysiology and the physiological basis of management.

PART 1: FLUID THERAPY

INTRODUCTION

Approximately 60% of total body mass of an adult consists of water. This water mainly resides within the intracellular compartment (2/3) and is separated from extracellular water (1/3), which comprises interstitial and intravascular space [1].

The normal daily requirements for fluid and electrolytes for a healthy adult are:

Water	1.5 ml/kg/hour
Sodium	1–1.5 mmol/kg
Potassium	1 mmol/kg
Chloride	1.5 mmol/kg

For most patients fluid losses are replaced during surgery and oral intake of fluid is rapidly resumed after surgery. However, for some procedures (e.g. gastrointestinal surgery), the preoperative deficit and losses during surgery vary widely and may not be adequately replaced. Inadequate fluid replacement leads to reduced cardiac output and oxygen delivery to injured tissues, which is associated with increased postoperative complications. Fluid restriction may reduce tissue oedema but also increases the incidence of acute kidney injury. Avoidance of peri-operative hypovolaemia remains an essential requirement and pre-operative intravascular optimisation improves outcome [2]. On the other hand excessive fluid administration has adverse effects including acidosis, coagulation defects and oedema of both lungs and peripheral tissues. Excessive fluid infusion leading to sodium, chloride and water overload is now recognized as a major cause of postoperative morbidity and a contributory factor to length of hospital stay, organ failure and mortality [2]. A UK study in 1997 showed that postoperative patients were frequently in positive fluid balance of 7 litres or more, with a positive sodium load of 700 mmol in the first few postoperative days [3].

FLUID SOLUTIONS

Intravenous salt solutions were first used in the 1830s for the treatment of fluid loss due to cholera and intravenous saline was administered to surgical patients as early as the nineteenth century [4,5]. Commonly used fluids are divided into 2 broad categories:

- a. <u>Crystalloid solutions</u>: These are low molecular weight salts or sugars which dissolve completely in water and pass freely between the intravascular and interstitial compartments e.g., Ringer Lactate, Darrow's Solution, Dextrose solutions, Normal saline (0.9%) etc..
- b. <u>Colloid solutions</u>: These contain larger molecular weight substances that do not dissolve completely and, depending on their molecular size, structure and the permeability of the capillaries of the patient, remain for a longer period in the vascular compartment than crystalloid solutions. Colloids in common use are dextrans, gelatins, albumin and hydroxyethyl starches. All have a significant sodium and chloride content. Because of different formulations and widely differing properties of colloids care must be taken to ensure that sufficient water is given to avoid hyperoncotic states which may lead to acute kidney injury.

Several times more crystalloid than colloid is required to achieve the same degree of vascular filling. As crystalloid solutions move rapidly into the interstitial compartment, a side effect of crystalloid resuscitation is more interstitial oedema than in colloid treated patients. It is important to distinguish between fluid and electrolytes required for normal existence (daily maintenance) and for resuscitation or replacement of abnormal losses.

With the exception of plain dextrose solutions, almost all intravenous solutions contain sodium and chloride. Some fluids like Hartmann's solution and Ringer;s lactate have near physiological concentrations of electrolytes (Hartmann's: Na 131 mmol/l and Cl 111 mmol/l) while the so called "normal" saline has supranormal concentrations (154 mmol/l Na and 154 mmol/l Cl). In comparison with more physiological solutions such as Hartmann's, even healthy subjects find it difficult to excrete solutions with high chloride content such as in 0.9% saline. For the surgical patient it is more difficult to excrete a salt and water load and to maintain normal serum osmolarity for several reasons:

- 1. The stress response to the injury or surgery causes anti-diuresis and transient oliguria mediated by activation of the renin-angiotensin system (RAS). Water and salt are therefore retained even in the presence of overload [6,7].
- 2. Following surgery, even when the serum osmolarity is reduced by administration of hypotonic fluid, the ability to excrete free water is limited [6,8]. As the capacity of the kidney to concentrate urine is impaired, excess free water infusion risks dilutional hyponatraemia.
- 3. If saline (0.9%) is infused, chloride overload accompanies sodium overload. This hyperchloraemia causes renal vasoconstriction and reduced glomerular filtration [7,9], further compromising the ability of the kidney to excrete sodium and water.

In the peri-operative phase 2-3 times the normal volume of urine is required to excrete a sodium and chloride load. Although major concerns have been expressed in the context of paediatric practice about the risks of dilutional hyponatraemia, especially with hypotonic solutions, it seems prudent that intravenous fluids be used with care and knowledge, rather than denounce any particular solution entirely and risk over-infusion of sodium.

In the absence of complications, oliguria (<0.5 ml/kg/hr) occurring soon after operation is usually a normal physiological response. A reduced urine output is commonly interpreted as indicating hypovolaemia and prompts infusion of yet more sodium-containing fluids. This not only expands the blood volume (often unnecessarily) but also over-expands the interstitial fluid volume, causing oedema, weight gain and haemodilution while also resulting in reduced serum albumin concentration [10]. The response to injury impairs the patient's ability to excrete the additional saline load, making interstitial oedema worse, compromising organ function and increasing the risk of morbidity and mortality. The key question is whether or not the oliguric patient has significant intravascular hypovolaemia which needs treatment. This can usually be decided on clinical grounds, but in more severe cases, and particularly intra-operatively, it may necessitate more invasive monitoring. The clinical signs reflecting intravascular volume include capillary refill, jugular venous pressure, and the trends in heart rate and blood pressure. Urine output should be interpreted in the light of these clinical signs, bearing in mind the normal short term physiological effects of surgery on urine output.

STRUCTURED APPROACH TO PERI-OPERATIVE FLUID MANAGEMENT: PRE-OPERATIVE

PRE-OPERATIVE FASTING:

Starvation and fluid restriction should be considered as separate issues. Free administration of clear fluids where possible up to two hours prior to an anaesthetic leads to lower gastric content volumes and prevents the need for significant "fluid catch-up" [11]. The Holliday & Segar formula (4ml/kg for the first 10 kg of body weight, 2ml/kg for a further 10 kg and 1 ml/kg for the remaining weight) is commonly used to calculate hourly maintenance requirement. When crystalloid replacement is indicated, balanced salt solutions e.g. Ringer's lactate or Hartmann's solution should replace 0.9% saline because of the risk of inducing hyperchloraemic acidosis, except in cases of hypochloraemia e.g. from vomiting or gastric drainage. Solutions such as 4%/0.18% dextrose/saline and 5% dextrose are important sources of free water for maintenance, but should be used with caution as excessive amounts may cause dangerous hyponatraemia, especially in children and the elderly. These solutions are not appropriate for resuscitation or replacement therapy except in conditions of significant free water deficit e.g. diabetes insipidus. Excessive losses from gastric aspiration/vomiting should be treated pre-operatively with an appropriate crystalloid solution which includes an appropriate potassium supplement. Losses from diarrhoea, small bowel obstruction or ileus should be replaced volume for volume with Hartmann's or Ringer's lactate type solutions.

Although currently logistically difficult in many centres, pre- or intra-operative hypovolaemia should be diagnosed by flow-based measurements (e.g. oesophageal Doppler) whenever possible.

BOWEL PREPARATION:

Weight loss of up to 2 kg, largely caused by fluid depletion, is not uncommon. Routine use of preoperative mechanical bowel preparation may complicate intra- and postoperative management of fluid and electrolyte balance. Its use should therefore be avoided whenever possible. Where mechanical bowel preparation is used, fluid and electrolyte derangements commonly occur and should be corrected by simultaneous intravenous fluid therapy with Hartmann's or Ringer-Lactate type solutions.

INTRAOPERATIVE

Traditional teaching has been to correct for the following:

- a. Insensible respiratory losses: 0.5ml/kg/hr with crystalloids.
- b. Hourly maintenance requirement as dictated by Holliday & Segar formula
- c. Third space losses: Unmeasured losses have traditionally been thought to amount to as much as 10 ml/kg/h when the body cavity is open. This loss of sero-sanguinous fluid from wounds and pleural and peritoneal cavities has long been over-estimated but recent research into third space losses indicate that such losses are no more than 1 ml/kg/hr intra-operatively [12]. Replacement of these fluids should be with a mixture of Hartmann's solution and a colloid.

d. Continuing losses from intravascular volume due to uncontrolled bleeding: this can be replaced either by blood or colloid or crystalloid and the choice of agent should be guided by the amount of blood loss and haematocrit level. A 3:1 ratio of crystalloid to blood/colloid is usually quoted.

POSTOPERATIVE

For maintenance requirements adult patients should receive sodium 50-100 mmol/day and potassium 40-80 mmol/day in 1.5-2.5 litres of water by oral, enteral or parenteral route (or a combination of routes). Additional amounts should only be given to correct deficit or continuing losses. Fluid prescription should be guided by clinical examination, fluid balance charts, and regular weighing when possible. Clinical context should also be taken into account, especially in critical care, as this will provide an important indication of whether hypovolaemia is possible or likely. When direct flow measurements are not possible, hypovolaemia can be diagnosed clinically on the basis of heart rate, capillary refill and venous (JVP) pressure, with acid-base and lactate measurements. A low urine output in the immediate post surgical period can be misleading and needs to be interpreted in the context of the patient's cardiovascular parameters. Hypovolaemia due to blood loss should be treated with either a balanced crystalloid solution or a suitable colloid until packed red cells are available (see "Management of Shock", Critical Care article no 5, May 2012). Hypovolaemia due to severe inflammation such as infection, peritonitis, pancreatitis or burns should firstly be treated with a balanced crystalloid. Care must be exercised to administer sufficient balanced crystalloid and colloid to normalize haemodynamic parameters and yet minimise overload. The ability of critically ill patients to excrete excess sodium and water is compromised, placing them at risk of severe interstitial oedema. The administration of large volumes of colloid without sufficient free water (e.g. 5% dextrose) increases risks of hyperoncotic states and renal impairment. When the diagnosis of hypovolaemia is in doubt and the venous pressure not raised, the response to a bolus infusion of 200 ml of a suitable colloid or crystalloid should be tested. This clinical response may be monitored by measurement of the pulse rate, capillary refill, CVP/JVP and blood pressure before and 15 minutes after receiving the infusion. This procedure should be repeated until there is no further improvement in the clinical parameters (indicating peak of Starling's curve). Patients undergoing non-elective major abdominal or orthopaedic surgery should receive intravenous fluid to achieve an optimal value of stroke volume during and for the first eight hours after surgery. In patients who are normovolaemic and haemodynamically stable, a return to oral fluid administration should be achieved as soon as possible. In patients requiring continuing IV maintenance fluids, avoidance of sodium rich fluids and use of the minimum effective volume will help patient to achieve a neutral sodium and fluid balance over the perioperative period. When this has been achieved the IV fluid volume and content should be adjusted to that required for daily maintenance and replacement of any on-going additional losses.

CONCLUSION

The goal of peri-operative fluid therapy is to ensure adequate blood flow in vital and traumatised tissues and to enable effective wound healing while avoiding collateral damage due to interstitial oedema. Adequate and timely replacement of extracellular fluid deficit can be done with the following protocol:

1. The extracellular deficit after usual fasting is minimal.

- 2. The basal fluid losses via insensible perspiration is 0.5 ml/kg/hr, increasing to 1 ml/kg/hr for major abdominal surgery.
- 3. Contrary to traditional thinking, so-called third space losses are negligible.
- 4. Replacement of plasma losses is ideally done with iso-oncotic colloids, assuming the vascular barrier to be intact and acknowledging that colloidal effects are context sensitive. This timely replacement of losses should be supplemented by a goal directed approach.

PART 2: RENAL DYSFUNCTION

PHYSIOLOGY: FUNCTIONS OF THE KIDNEY

These can be summarised as:

- 1. Fluid, electrolyte and hydrogen ion homeostasis.
- 2. Excretion of water soluble waste products of metabolism.
- 3. Excretion of water soluble drugs.
- 4. Endocrine functions renin-angiotensin, erythropoietin, vitamin D.

The chief function of the kidneys is to regulate the composition of extracellular fluid within tight limits by filtering the plasma. The kidney does this through the processes of glomerular filtration, tubular re-absorption and tubular secretion to produce urine which contains excess water, salts and waste products. It also has important hormonal functions [13,14,15].

Each kidney has over a million nephrons, the functional unit of the kidney. It is made up of the glomerulus (fine capillaries supplied by an afferent arteriole and drained by an efferent arteriole) and associated Bowmans capsule, which in turn drains into the proximal convoluted tubule, loop of Henle, distal convoluted tubule and collecting ducts.

Glomerular filtration occurs where a plasma-like fluid (ultrafiltrate) transfers from blood in the glomerulus into Bowman's capsule, minus larger molecules such as albumin. This process is driven by the net filtration pressure, which mostly relies on the difference in hydrostatic pressure. Blood flow through the kidney is fairly constant, despite physiological changes in the systemic blood pressure, due to local autoregulation(1). Autoregulation fails when the systolic blood pressure drops too low (mean <60 mmHg) or with cellular damage e.g. with sepsis.

Around two thirds of the ultrafiltrate (by volume) is re-absorbed in the proximal convoluted tubule. Glucose and amino-acids are absorbed by a carrier-mediated process, due to sodium re-absorption across a gradient. Some organic anions and cations are also actively secreted here [13,16].

The loop of Henle's function is to dilute the urine by actively transporting sodium, potassium and chloride ions from the tubules of its ascending limb without the concurrent movement of water (the ascending limb is impermeable to water). This also increases the osmolality of the interstitial fluid in the renal medulla, allowing for regulation of water re-absorption under the influence of antidiuretic hormone (ADH) in the collecting ducts. Calcium, magnesium and other cations are also reabsorbed here [13].

In the distal convoluted tubule and proximal collecting ducts, principal cells reabsorb sodium and water but secrete potassium. This stage is regulated by the renin-

angiotensin system. Renin is secreted by the granular cells of the afferent arteriole in response to stimuli e.g. sympathetic stimulation, catecholamines or decreased sodium re-absorption across the macula densa [14,15]. Renin converts angiotensinogen to angiotensin I, which in turn converts to angiotensin II under the influence of angiotensin coverting enzyme (mainly found in lung capillaries). Angiotensin II stimulates the adrenal cortex to release aldosterone, which promotes sodium re-absorption as well as potassium secretion. Intercalated cells in the distal tubule/ proximal collecting ducts regulate acid-base balance by secreting hydrogen ions, and reabsorbing bicarbonate. Calcium re-absorption here is promoted by parathyroid hormone, which also inhibits phosphate re-absorption [13,14,15].

PATHOPHYSIOLOGY OF OLIGURIA

PATHOPHYSIOLOGY OF THE EFFECT OF HYPOPERFUSION/ HYPOTENSION ON THE KIDNEYS:

Hypoperfusion of the kidneys can occur secondary to hypovolaemic, cardiogenic or septic shock. Occlusion of the major renal vessels occurs extremely rarely [17,18].

Hypoperfusion leads to a decrease in pressure in the afferent arteriole. Autoregulatory mechanisms can act to maintain glomerular filtration – for example Angiotensin II causes vasoconstriction of the efferent arteriole. Drugs such as ACE-inhibitors interfere with this mechanism and can exacerbate the effect of hypoperfusion on the kidneys [17,14,13].

With sufficient drop in pressure, there is a decrease in glomerular filtration leading to oliguria or even anuria. This situation is known as 'pre-renal' acute kidney injury (or pre-renal failure), and the urine is characterised by [17,19]:

- low sodium concentrations (<20 mmol/l)
- high osmolality (urine:plasma osmolality > 1.5:1)
- high urea and creatinine concentrations (urine:plasma urea concentration > 10:1).

This decrease in filtration of the plasma can lead to hyperkalaemia and potentially fatal cardiac arrhythmias.

Pre-renal failure will resolve rapidly following correction of the underperfused state of the kidneys.

Prolonged or severe hypoperfusion will lead to Acute Tubular Necrosis (ATN), a form of 'renal' acute kidney injury. The principal mechanism is ischaemic damage to the cells of the renal tubules [17,21,15]. The full length of the tubules can be affected but it is usually most marked in the distal convoluted tubules because the renal medulla naturally has poorer blood supply than the cortex; histological features include flattened, vacuolated epithelial cells, and thinning of the brush border. Often there is no actual 'necrosis' seen pathologically and the name is hence misleading. Unlike pre-renal failure the urine will be dilute (osmolality similar to plasma) and brown granular casts (which include Tamm-Horsfall protein) are often seen [19,21]. Casts can lead to luminal obstruction of the tubules [20].

Renal function will not improve rapidly with return of normal perfusion in ATN, but can fully recover with supportive therapy.

FIVE GOLDEN RULES OF ACUTE RENAL FAILURE PHYSIOLOGY IN THE CRITICALLY ILL SURGICAL PATIENT:

- 1. The kidneys can't function without adequate perfusion.
- 2. Renal perfusion depends on an adequate blood pressure.
- 3. A surgical patient with a poor urine output usually requires more fluid.
- 4. Absolute anuria is usually due to urinary tract obstruction.
- 5. Poor urine output in a surgical patient is NOT due to frusemide deficiency.

MANAGEMENT OF OLIGURIA IN SURGICAL CRITICAL CARE

This can be approached by asking a series of questions, like in an algorithm or checklist:

A. Is the problem pre-renal, renal or post-renal?

- 1) Pre-renal: is there hypotension, hypovolaemia, unrecognised blood loss, dehydration?
- 2) Renal: is there a potential cause of acute tubular necrosis (ATN) e.g. shock, sepsis, trauma, NSAID's, myoglobinuria, aminoglycosides/cephalosporins.
- Post-renal: Is the outflow blocked (acute urinary retention or a bladder stone, or a blocked catheter) or is the outflow pressure too high (abdominal compartment syndrome).
- B. What pathophysiological mechanism is causing the oliguria? Could it be:
 - 1) Inadequate renal perfusion (e.g. hypotension due to insufficient fluid resuscitation, cardiac failure or peripheral vasodilatation from sepsis).
 - 2) The metabolic stress response that causes increased ADH and aldosterone secretion, with resultant sodium retention and decreased water excretion.
 - 3) Abnormal re-distribution or losses of fluid e.g. unrecognised bleeding into a body cavity or major fracture site, capillary leak due to the inflammatory response, ileus with fluid sequestration in the gut, diarrhoea secondary to *C. difficile*, high ileostomy output or fistulae.
 - 4) Inadequate fluid replacement e.g. unrecognised poor oral intake, problems with venous access (especially at night).
- C. What do I need to think of when assessing the patient?
 - 1) In what risk category is this patient? High risk for renal dysfunction in:
 - major emergency / elective surgery;
 - minor surgery in high risk patients (chronic kidney disease, on certain drugs, poor cardiac function).
 - 2) Check fluid balance: inflow-outflow balance chart; hourly urine volumes; urine colour; extrarenal losses (diarrhoea, fistula, ascites, ileus).
 - 3) Check the perfusion: pulse rate and volume, blood pressure, peripheral perfusion (warm vs cold/clammy), cerebral function, Central Venous Pressure.

CHECKLIST:

- 1. Is the patient anuric? If yes, check catheter and for retention.
- 2. Is the patient oliguric (<0.5 ml/kg/h for 2-3 hours): do full assessment; Is this "physiological" (i.e. in immediate post-operative period in a patient with a well-filled vascular system) or is it pathological (i.e. associated with signs of hypoperfusion of other organ systems e.g. skin, brain).
- 3. Is patient hypovolaemic? Give fluid bolus (250 ml colloid or 500 ml crystalloid over 1-2 hours, depending on whether hypotensive or not).
- 4. Is patient hypotensive (mean arterial BP<80 mmHg)? Treat hypotension with intravenous fluids, inotropes or vasopressors depending on cardiovascular assessment findings (Is the problem with preload, contractility or afterload?).
- 5. Is patient still oliguric? Check potassium, for acidosis, systemic sepsis, CVP. If all these are normal and patient well perfused, give appropriate diuretic e.g. frusemide in regulated dosage schedule.
- 6. Restart or replace previous renal medication.
- 7. Still oliguric? Ask intensivist or renal physician for help.

PART 3: CARDIAC COMPLICATIONS IN SURGICAL PATIENTS:

This section specifically discusses the most common and serious cardiac complications after non-cardiac surgery. These complications all affect cardiac contractility, i.e. make the heart a less effective pump. (Causes of and complications from decreased preload have been discussed in the article on Shock, May 2012. Afterload abnormalities have been touched on in the article on Shock as well, and will be further discussed in a future article on "SIRS and Surgical Sepsis").

When the pump fails it triggers a cascade of complications. On the efferent side this leads to poor tissue perfusion and hypoxia, leading to other organs failing e.g. pre-renal type renal failure, gut ischaemia with release of inflammatory mediators, poorer liver function which compromises immunity. On the afferent side the blood pools; although clinical signs are often more obvious due to right ventricular inefficiency (increased JVP), it is failure of the left ventricle that can become rapidly life-threatening through pulmonary oedema and poorer gas-exchange, worsening hypoxia through shunting (See the article on "Hypoxia", April 2012, for more information).

PREOPERATIVE RISK ASSESSMENT

Cardiac comorbidity is increasingly common in patients undergoing both elective and emergency surgery for several reasons that include increasing age, changing lifestyles, dietary and other environmental factors. Thorough preoperative assessment is useful in the identification of patients at increased risk of perioperative cardiac complications. This in turn allows for informed decision-making regarding the risks and benefits of proposed surgery, optimising perioperative care to minimise risks as well as appropriate allocation of finite postoperative surgical critical care resources.

Simple risk scoring systems such as the cardiac specific Lee's Revised Cardiac Risk Index or the more generic ASA Physical Status Classification System are useful in identifying high risk patients and must be routinely used in the preoperative assessment of all patients undergoing surgery. Old age, obesity, ischemic heart disease, diabetes mellitus, peripheral vascular disease and smoking are all associated with increased incidence of postoperative cardiac complications. There is increasing evidence that perioperative beta-blockade, statins and aspirin reduce cardiac complications including atrial fibrillation and myocardial infarction. Patients on cardiac medication before surgery should be allowed to restart them as soon as is feasible. In patients with significant cardiac disease, preoperative evaluation by the anaesthetist and/or cardiologist may allow optimisation of medical therapy to reduce perioperative risks.

POSTOPERATIVE CARDIAC COMPLICATIONS:

ATRIAL FIBRILLATION (AF)

Atrial fibrillation (AF) is the most common tachyarrhythmia in patients undergoing cardiac surgery, non-cardiac thoracic surgery as well as major abdominal surgery.

PREOPERATIVE RISK FACTORS:

Comorbidity including old age, ischemic heart disease, pre-existing atrial fibrillation, obesity, chronic obstructive pulmonary disease and endocrine disorders especially hyperthyroidism, diabetes and pheochromocytoma are associated with an increased risk of postoperative atrial fibrillation.

INTRAOPERATIVE AND POSTOPERATIVE PRECIPITATING FACTORS:

- Major thoracic or abdominal surgery
- Myocardial ischemia/infarction
- Hypotension
- Hypoxia
- Anaemia
- Sepsis of any source, especially pneumonia or mediastinitis
- Pulmonary embolism
- Electrolyte abnormalities including hypokalaemia, hypocalcaemia and hypomagnesaemia

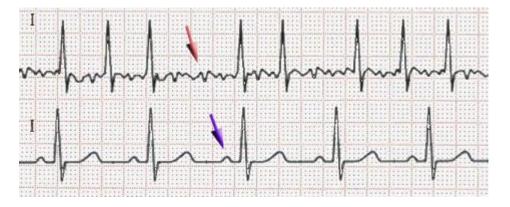
DIAGNOSIS

Atrial fibrillation is diagnosed by the presence of an irregularly irregular pulse and characteristic ECG findings. Patients are often asymptomatic but may complain of palpitations, chest pain, breathlessness or dizziness. Atrial fibrillation with a rapid ventricular response may present with life-threatening hemodynamic instability. In some patients, especially when atrial fibrillation has been present for more than 48 hours, the

first presenting symptom may be the result of an embolic event such as stroke or mesenteric ischemia.

All patients suspected to be in atrial fibrillation must undergo a complete systematic examination adhering to fundamental critical care principles (ABC approach). Blood tests including full blood count, serum urea, serum creatinine and serum electrolytes must be checked. Elevated cardiac enzymes may be the result of rate-related ischemia or infarction.

An ECG must be performed to confirm the diagnosis as well as to look for changes suggestive of myocardial ischemia or infarction. ECG changes are characteristic with irregular QRS complexes and absent P waves which are replaced by irregular F waves (see below, upper trace shows atrial fibrillation, lower trace shows sinus rhythm. Upper arrow: absent P wave: lower arrow, P wave present)



TREATMENT

Management of atrial fibrillation is aimed at the treatment of the tachyarrythmia itself as well as the prevention of the associated thromboembolic complications, i.e. stroke or TIAs. Precipitating or aggravating factors must be sought and corrected. When AF suddenly occurs in a surgical patient one must always ask "Why?", and address this question through systematic assessment and appropriate investigations to look for the precipitating factors mentioned above. One has to specifically look for causes of hypoxia, for sources of sepsis and for electrolyte and acid-base disturbances. Immediate management includes administration of oxygen, correction of electrolyte abnormalities and anaemia and treatment of sepsis if present.

Patients with new-onset atrial fibrillation of less than 48 hours duration and no haemodynamic compromise should undergo pharmacological cardioversion with either Flecainide or Amiodarone, if available. Amiodarone is the drug of choice for patients with structural heart disease while Flecainide can be used in patients without structural heart disease. In patients with AF and cardiac failure amiodarone can worsen the cardiac failure because it has a negative inotropic effect; in that situation digoxin is often a better first choice drug as it is positively inotropic. Digoxin will usually not reverse the AF immediately but will slow down the heart rate and improve contractility; when the reason for acute onset of AF is treated (e.g. electrolyte abnormality, chest infection) the heart often returns to sinus rhythm.

When atrial fibrillation is associated with life-threatening haemodynamic instability, emergency electrical cardioversion is required. Where facilities for electrical cardioversion are not available, chemical cardioversion with amiodarone or flecainide

must be initiated. Alternatively the patient can be digitalised for rate control and to make the pump more efficient; this can overcome the haemodynamic instability even if the patient is not cardioverted.

Magnesium sulphate infusion (10 mmol in 100mls of saline infused over 30 minutes or 20 mmol in 100 mls of saline infused over 4 hours) may be used to attempt cardioversion where other drugs are unavailable or in addition to amiodarone.

In the stable patient where cardioversion has not been achieved after 48 hours, focus must be turned to rate control and thromboprophylaxis. A beta-blocker such as bisoprolol or a rate-limiting calcium channel blocker such as verapamil can be used to control ventricular rate. If these are not available digoxin can be used.

Thromboprophylaxis using low molecular weight heparin must be initiated in all patients with atrial fibrillation of more than 48 hours duration to minimise the risk of thromboembolic complications such as stroke or mesenteric ischemia. Long-term anticoagulation with warfarin must be considered in patients being discharged from hospital with persistent AF.

SUPRAVENTRICULAR TACHYCARDIAS (SVT)

OVERVIEW

Sustained and paroxysmal SVT refers to any arrhythmia that originates in the atria or the atrioventricular node. The most common type of SVT is atrial fibrillation and its management is described above. Some of the other forms of SVT include atrial tachycardia, multifocal atrial tachycardia, atrial flutter and atrioventricular nodal reentrant tachycardia. Diagnosis is established by ECG.

MANAGEMENT

The initial management of SVT in the haemodynamically stable patient involves the use of vagal manoeuvres such as the Valsalva manoeuvre or carotid sinus massage to prolong AV nodal conduction thus terminating the SVT. Carotid sinus massage should be avoided in elderly patients who may have atherosclerotic disease of the carotid arteries and are at risk of stroke. Haemodynamically unstable patients should undergo immediate electrical cardioversion where facilities exist. Intravenous adenosine, amiodarone and calcium channel blockers (e.g. verapamil) are less suitable alternatives for chemical cardioversion. Any precipitating or aggravating factors such as myocardial ischaemia, hypoxia, anaemia, electrolyte abnormalities should be corrected promptly. All patients with paroxysmal/sustained SVT should be referred to a cardiologist or physician for long term medical treatment.

MYOCARDIAL INFARCTION

OVERVIEW

Myocardial ischaemia and infarction are most likely during the intra-operative and immediate postoperative period when the patient is under maximal physiological stress. Symptoms may be atypical or absent. Diagnosis is established by characteristic ECG

changes and/or elevated cardiac enzymes. Postoperative myocardial infarction is sometimes but often not preceded by ST segment depression which would have indicated myocardial ischaemia.

MANAGEMENT

Initial management is similar to management of myocardial infarction in the nonsurgical patient and involves administering supplemental oxygen, sublingual glyceryl trinitrate and adequate analgesia/anxiolysis if appropriate. The use of high dose aspirin must be balanced against the risk of bleeding. Low molecular weight heparin or unfractionated heparin may preferentially be used to provide better control of anticoagulation. Precipitating causes such as anaemia, hypoxia, hypotension, electrolyte abnormalities and tachyarrhythmias must be corrected. Haemoglobin should be maintained above 9 g/dl in patients known to have ischaemic heart disease and elderly patients. Oxygen saturations should be maintained over 95%.

Peri-operative beta blockade and aspirin reduce the incidence of postoperative myocardial infarction. Patients who are on beta-blockers should continue these until surgery and these should be restarted as soon as possible after surgery.

CARDIAC FAILURE AND PULMONARY OEDEMA

OVERVIEW

Postoperative cardiac failure is a major cause of mortality in patients undergoing noncardiac surgery. Patients may present with breathlessness, desaturation in spite of increasing oxygen requirements, tachycardia and hypotension. Patients in cardiogenic shock due to left ventricular dysfunction will present with pulmonary oedema, with or without hypotension and cool peripheries.

Thorough pre-operative evaluation will help identify patients at high risk of postoperative cardiac failure. Patients with pre-existing cardiac disease or elderly patients are susceptible to fluid overload as a result of aggressive fluid resuscitation or due to fluid accumulation over a period of days. Judicious use of fluids with central venous pressure monitoring both during surgery as well in the postoperative period will help avoid overload in patients with pre-existing cardiac failure or ischaemic heart disease.

INVESTIGATION

When a patient is suspected to be in acute cardiac failure after surgery, evaluation and initial resuscitation should follow fundamental critical care principles (ABC approach). ECG may show evidence of myocardial ischaemia/infarction, cardiomegaly or arrhythmias. CXR may show cardiomegaly, pulmonary oedema or pleural effusions. Pulmonary oedema in surgical patients may be due to acute lung injury (ALI) or acute respiratory distress syndrome (ARDS). Pleural effusions may be caused secondary to intra-abdominal inflammatory processes. It is therefore important to differentiate cardiogenic shock from haemorrhagic or septic shock and this may not be straightforward in all patients. Surgical patients will often have a combination of factors including hypovolaemia, sepsis and cardiac dysfunction contributing to the shock.

MANAGEMENT

Treatment of postoperative cardiac failure is aimed at the treatment of the cause of the failure as well as the consequence. Supplemental oxygen, through a face mask or by non-invasive positive pressure ventilation (NIV), must be administered in patients who desaturate. Precipitating causes such as atrial fibrillation or myocardial infarction must be treated promptly. Surgical patients in cardiac failure often have a combination of fluid overload and cardiac dysfunction.

Pulmonary oedema is treated by sitting the patient upright, administering supplemental oxygen and the use of a loop diuretic such as furosemide (frusemide). Glyceryl trinitrate (sublingual or intravenous in severe cases) is effective at reducing preload rapidly in patients with severe cardiogenic pulmonary edema who are not fluid overloaded or don't respond completely to diuretics. Where facilities permit, dobutamine or adrenaline may be used to improve cardiac contractility in patients with cardiogenic shock. Morphine should not be routinely used in the treatment of pulmonary oedema as the associated risks of reduction in cardiac output and respiratory depression outweigh any potential benefits.

LONG-TERM MANAGEMENT

Long term survival is reduced in patients who develop serious cardiac complications after non-cardiac surgery. They should be referred to a cardiologist or a physician for ongoing cardiac care including optimisation of medical therapy such as beta-blockers, ACE inhibitors, anti-arrhythmics and diuretics. Patients must be advised regarding the importance of long-term medical therapy as well as any lifestyle changes required. They must also be advised to inform other health care professionals involved in their future care regarding their cardiac disease so that appropriate precautions are taken.

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9. DISABILITY; CONFUSION IN SURGICAL PATIENTS

JACOB S DREYER

DISABILITY

Lots of things can disable patients and affect their cognitive ability, emotions, behaviour, mobility and/or other psychomotor skills. Some are conditions that are obvious, such as head or spinal injuries, or well understood from physiology, such as causes of central hypoxia caused by shock, inhalation burns or severe sepsis. In some other situations it is more difficult to quantify disability or assess its effect on critically ill patients, e.g. with severe pain or after anaesthesia or sedation. All these topics are discussed in following chapters. Patients who go through critical illness, their families and the staff who care for them can all become functionally disabled due to the psychological effects of severe trauma or serious surgical illness, especially as critically ill patients often have a "rollercoaster ride" of improvements and setbacks during their hospitalisation. This aspect of critical care is discussed under the section on Surgical Humanities.

CONFUSION

One of the most frightening scenarios that a junior doctor can be called for is for a patient who has become confused, pulled out his catheter or IV line, is threatening to leave the ward and acting aggressively towards nursing staff. This almost always happens at night when there is a minimum number of nursing staff on duty and senior doctors are either at home or not easily available. The following mnemonic "DIMTOP", gives a simple approach to thinking through such a situation in a structured way. (As you "dim" your car's headlights, so your "top" gets "dimmed" when confused).

The two conditions that can cause death or serious brain damage if not managed immediately are hypoxia and hypoglycaemia. So, if a nurse contacts you to tell you that a patient is confused or has pulled out his lines, reply "Please give the patient oxygen and check the blood glucose. I am on my way". When you arrive in the ward where the patient is, ask "What is the saturation? What is the blood glucose?" as you walk in to see the patient. If the patient is not hypoxic and not hypoglycaemic, go through the ABC's of primary assessment and resuscitation, and then think through DIMTOP to consider all causes of confusion.

"D"= DIABETES OR DRUGS

- 1. Diabetes mellitus:
 - a. Hypoglycaemia
 - b. Diabetic keto-acidosis
- 2. Drugs:
- a. That you had given e.g. Midazolam, diazepam, anaesthesia
- b. Heroin, Cannabis, Glue/petrol sniffing etc
- c. Too much opiate pain relief
- d. Alcohol or alcohol withdrawal

"I" = INFECTION

- 1. Meningitis
- 2. Encephalitis
- 3. Systemic Sepsis
- 4. Malaria
- 5. Rare events e.g. Kala azar, Tetanus, Rabies

"M" = METABOLIC

- 1. Electrolyte disturbances
 - a. Hyponatraemia (In malnutrition body protein gets broken down for energy; for every 1g protein released 4g free water becomes available. Inappropriate ADH secretion can contribute. The answer is **not** to give sodium [e.g. saline] but treat the underlying cause).
 - b. Hypernatraemia (Rarer, but potentially more serious. Can occur due to severe dehydration or Diabetes Insipidus. Too rapid correction [e.g. with IV dextrose] can cause fatal brain swelling).
 - c. Hypomagnesaemia (common in ICU patients).
- 2. Eclampsia: convulsions are treated with IV magnesium sulphate.
- 3. High steroids e.g. in acute ulcerative colitis, exacerbation of COPD
- 4. Hypothyroidism
- 5. Uraemia

"T" = TOXINS, TUMOUR OR TRAUMA

- 1. Toxins
 - a. Industrial toxins e.g. industries with poor environmental control
 - b. Smoke inhalation
- 2. Tumour
 - a. Primary brain tumours (Benign, Malignant, Abscess, Echinococcus cyst)
 - b. Metastases
 - c. HIV related primary CNS lymphoma
- 3. Trauma
 - a. Head injury
 - b. Central effects of polytrauma due to e.g. hypotension, hypoxia

"O" = **LACK OF**OXYGEN

- 1. Give Oxygen to all confused patients while assessment is undertaken.
- 2. Think of ALL possible causes of hypoxia (see chapter 6).

"P" = PSYCHIATRIC

(See chapter 20E).

- 1. Delirium (common in ICU)
- 2. Disorientation (common in the elderly)
- 3. Dementia
- 4. Psychosis

Although DIMTOP does not provide a complete list of causes of confusion, it provides a structure of thinking of the most common causes that a surgical trainee or other junior doctor has to consider when suddenly confronted with a confused patient.

10. TRAUMA NEUROSCIENCES

BAREND A. DREYER, PETER A.R. ARMSTRONG

INTRODUCTION

Traumatic brain and spinal injury is the world's leading cause of death and morbidity in those under the age of 45, and is often the result of high-energy road traffic collisions, or sporting injuries. Approximately 1 million cases of traumatic brain injury occur in the UK annually, and a third of such patients admitted to hospital with GCS \leq 13 will die. For those presenting with GCS<8, survivability falls to 50% [1-4].

Death following head trauma assumes a trimodal distribution [1]. First phase death occurs at the time of, or within seconds of, catastrophic injury. Such injuries will rarely be treatable, and morbidity can only be reduced through accident prevention or modification (such as enforcing seat belts) 1,4]. The majority of deaths, however, fall into the second phase, when death occurs in the minutes or hours following injury. It is during this period that serious life threatening injuries are identified and managed, and clinicians endeavour to prevent and treat "secondary' brain injury. Death in the third phase occurs days, weeks or months after the original injury. Such deaths may be the result of infection, or multi-organ failure and unrelated to the specific brain or cord injury; suboptimal resuscitation and subsequent secondary injury could play a significant role in the pathological processes leading to death.

Of patients admitted with traumatic head injury, 5% will have an associated cord injury, most commonly at cervical spine level [4,7]. Usually high energy transfer causes these injuries, hence they are more common in young males, but significant cord injuries are also seen in the older population, in whom osteoporosis and arthritic changes within the spine are more common, and predispose them to injury with seemingly minor trauma or falls. The mortality rate for patients with combined head and spinal injury is approximately 14%, but for those who survive the long-term morbidity is much harder to quantify.

ANATOMY [2,3]

The central nervous system consists of the brain and spinal cord. The brain is protected from physical injury by the skull bones, forming a rigid box and the spinal cord by the vertebrae, forming the human spine. The brain and spinal cord are surrounded and bathed by cerebro-spinal fluid within the subarachnoid space, which helps to cushion the brain during impact and supplies the CNS with essential nutrients. The brain's arterial supply is from the common carotid arteries, dividing into the external and internal carotids, as well as the vertebral arteries. These join to form the circle of Willis enabling communication of arteries, providing back-up circulation should the blood supply become compromised. From the circle of Willis arises the anterior, middle and posterior cerebral arteries which all supply specific areas of the brain; between these named cerebral

arteries there is no functional collateral circulation. The spinal cord's blood supply comes from the anterior and two posterior vertebral arteries, running in the subarachnoid space and forming anastomoses. Venous drainage of the CNS is via the dural venous sinuses exiting through the jugular foramen into the internal jugular vein. As the spinal cord travels down it gives off various spinal nerves and receives many connections and eventually terminates at the level of L1-2 after which only the cauda equina remains.

PHYSIOLOGY [2,5,6]

Brain tissue is metabolically very active using 20% of the body's oxygen and receiving 15% of cardiac output. It is therefore very sensitive to changes in the supply of nutrients and for this reason there are various autoregulatory mechanisms allowing it to function in a range of conditions. For example, if blood pressure drops this is sensed by the brain and results in cerebral arterial dilation allowing adequate perfusion. With an increase in blood pressure the reverse happens and the cerebral vessels will constrict. This allows the brain to maintain an adequate supply for cerebral function between mean arterial blood pressures of 50-150 mmHg. It is only in extreme blood loss that the brain will not be able to maintain adequate blood flow. The brain is also very sensitive to oxygen and carbon dioxide concentrations, sensed by chemoreceptors within the carotid bodies located at the bifurcation of the carotid arteries. In response to this feedback the brain tightly regulates cardiac and pulmonary function maintaining oxygen and carbon dioxide levels within an appropriate range as well as changing the diameter of cerebral blood vessels to allow a greater or lesser volume of blood. The brain and spinal cord receive nutrients from the cerebrospinal fluid (CSF), which is continuously produced by the choroid plexus in the ventricles and reabsorbed by the arachnoid villi into the bloodstream, carrying away waste products. CSF flows around the brain and spinal cord; if a blockage occurs within the system it may lead to an obstructive hydrocephalus and raising intracranial pressure (ICP). ICP is maintained within a tight range of approximately 7-15mmHg in an adult. The brain is contained in a rigid skull so any changes in the volume of CSF, amount of brain tissue and volume of circulating blood flow may cause the ICP to increase beyond the normal range. The skull can only hold a finite volume therefore, as ICP increases, blood and CSF is squeezed out resulting in neuronal ischaemia. It may also force out brain tissue through the foramen magnum. This is termed "coning" and can lead to respiratory depression and eventually death.

PATHOPHYSIOLOGY

In traumatic brain and spinal injury, a primary injury refers to the immediate damage occurring at time of impact [1,4]. Whilst often preventable, such injuries are rarely treatable, and there is little by way of medical intervention or management. Secondary injury describes the subsequent pathological processes following a primary insult that may cause further brain and spinal cord damage. It is the prevention and management of secondary injury where we concentrate medical efforts. Many causes and types of secondary brain injury exist, but hypoxia and brain/cord ischaemia due to hypotension or hypovolaemia are the most common [9,14-16].

Following injury, neuronal breakdown occurs and cell contents are released activating an inflammatory response causing cyctoxic cellular oedema. As well as damaging neurons through direct compression, such cellular swelling raises intracranial pressure, ultimately reducing cerebral perfusion. Following trauma a rise in the levels of circulating catecholamines increase brain metabolism. Impaired perfusion and metabolism deplete cellular ATP stores, resulting in lactic acidosis, aggravating cell damage, increasing membrane permeability and worsening cell oedema. Membrane pump dysfunction facilitates calcium influx leading to cell death. Brain and cord vasogenic oedema may also occur with the release of mediators increasing intracranial pressure. This response to injury becomes a vicious circle, and factors activating or augmenting the mechanism require early and aggressive management. Specific factors associated with secondary injury and worsening response include:

- Hypoxia
- Ischaemia
- Cerebral vasospasm
- Hypoglycaemia
- Acidosis
- Coagulopathy
- Raised intracranial pressure [4,9,11].

By understanding this pathophysiology of brain and cord injury, it becomes obvious why patients require aggressive management. To adequately treat such patients, however, it is equally important to understand the relationship between cerebral, arterial and intracranial pressures. Cerebral perfusion pressure (CPP) can be calculated using the formula:

CPP = MAP - ICP

(where CPP = Cerebral Perfusion Pressure, MAP = Mean Arterial Pressure, ICP = Intracranial pressure).

Normal CPP is approximately 60-70mmHg, and normal ICP approximately 7-15mmHg. ICP > 20mmHg is considered abnormal, and severity of brain injury increases significantly with ICP >40mmHg.

In their doctrine, Monro and Kellie described the relationship between ICP and intracranial volume. Volume increases may occur due to bleeding, or space occupying lesions such as tumours or oedema as described above. Monro and Kellie described the skull as a non-expansile "closed box" [1,4]. During periods of non-injury, or initial periods following injury, increased intracranial volume is tolerated and compensated by reducing venous blood volume and displacing CSF from ventricles into the spinal canal. Once saturated, no further compensation can occur, and any further volume increases attempt to squeeze the brain from the cranial cavity through the tentorium cerebelli or foramen magnum. As well as directly damaging brain tissue, this exponential rise in ICP reduces CPP causing further cell damage.

Maintenance of an adequate CPP and ICP reduction is therefore of paramount importance in patients with head injuries [6]. The uninjured brain autoregulates CPP and

Cerebral Blood Flow (CBF) whilst MAP is in the region of 50-150mmHg [1]. Unfortunately this protective mechanism becomes impaired in injury. A head injured patient with an increased ICP will reduce CPP, exacerbating a secondary injury. Similarly, hypotensive patients with multiple injuries will compromise CPP due to falling MAP and rising ICP.

MECHANISM AND TYPES OF INJURY

Brain and spinal cord injuries often result from accidents with high energy transfer e.g. road traffic collisions and falls from heights. Specific patient groups are prone to significant injury through seemingly minor or repeated injury, however. A good understanding of anatomy and physiology, combined with an appreciation of the mechanism of injury and a thorough assessment, will allow the clinician to anticipate the nature and type of injuries expected. It would be reasonable to expect an unrestrained car driver partially ejected from the vehicle following rapid deceleration to have a significant head and cervical spine injury. Equally, an elderly, anticoagulated patient with mobility issues falling forward with lower energy, but striking his head on a wall, may sustain an intracranial injury and associated cervical spine injury through neck hyperextension.

Broadly speaking injuries can be classified according to their location and nature. Severity of traumatic brain injuries can be graded according to Glasgow Coma Score, with GCS 13-15 considered mild, 9-12 moderate, and <8 severe [1,2,7]. Where indicated, CT examination may demonstrate specific focal intracranial lesions:

EXTRADURAL HAEMATOMAS

This refers to bleeding and haematoma collection within the skull but outside the dura mater. Classically the result of tearing the middle meningeal artery, such injuries may be accompanied by temporal bone fracture. This type of injury is associated with the so-called "Talk and Die" scenario. A patient falls sustaining a head injury, but quickly recovers, as there is no underlying brain injury. Due to rapid accumulation of blood and subsequent rising ICP, the patient succumbs to a secondary brain injury from brain compression, shift and attempted displacement. Extradural haematomas are thought to account for 0.5% of all head injuries. Whilst frequently arterial, they can also result from torn venous sinuses. Neurosurgical intervention is required, and burr hole evacuation may be life saving in rural situations where neurosurgical skills are unavailable [2,4].

SUBDURAL HAEMATOMAS [2,4]

This much more common injury refers to bleeding between the dura and arachnoid mater, and usually results from tearing of bridging veins running between the surface of the brain and draining sinuses. Subdural haematomas are reported in up to 30% of severe head injuries. Subdural bleeding may be associated with severe underlying brain injury, with poor outcome. Certain patient groups are susceptible to subdural injury. These include:

• Patients prone to recurrent falls e.g. the elderly;

- Patients with atrophied brain matter and stretched bridging veins e.g. elderly and chronic alcoholics;
- Patients with compromised coagulation e.g. haemophiliacs, patients taking antiplatelet agents or anticoagulants e.g. Heparin and Warfarin.

Subdural haematomas may be acute or chronic. Acute subdural collections occur rapidly following injury and result in a mass effect to a large region of brain. Such injuries may require craniotomy to remove clotted blood and reduce a mass effect. Chronic subdural bleeds are more commonly seen in elderly or alcoholic patients where recurrent falls and stretched bridging veins predispose them to significant injury with seemingly minor trauma. The presentation of patients with chronic subdural collections is less dramatic, often several days after injury with subtle signs such as weakness, confusion or personality changes.

SUBARACHNOID HAEMORRHAGE

Often a spontaneous injury, subarachnoid blood can be seen on CT scan following significant head injury. Bleeding results from traumatic disruption to subarachnoid vessels. Management is normally conservative.

CONTUSIONS AND INTRACEREBRAL HAEMATOMA

Contusions and intracerebral haematomas describe little more than bruising to the brain. Whilst these terms are often used interchangeably, they can be separate entities with initial contusions becoming more organised over time forming distinct haematomas. Patients may have minimal clinical signs, although localised swelling around such injuries can be dramatic, peaking around 48 hours post insult and causing a significant rise in ICP. Neurosurgery is unnecessary to treat the haematoma, but aggressive medical and surgical management may be necessary to reduce ICP [2,4].

Whilst such distinct intracranial lesions will be evident following CT scan, less distinct diffuse injuries may also occur. CT findings in such situations are often minimal, and the extent of the diffuse injury is only identified through clinical examination and observation. Diffuse injuries describe a spectrum of conditions ranging from milder concussion to severe diffuse axonal injury in which axons are irreversibly stripped and damaged. Due to the difficult and protracted nature of these injuries, management is often undertaken in a tertiary neurosurgical centre.

SPINAL CORD INJURIES [1,2,3]

Spinal cord injury, like traumatic brain injury (TBI), may be primary or secondary, the former describing the physical cord damage including compression, tearing or transection. Cord in the cervical spine is most commonly injured. Causes of secondary brain injury can also cause secondary cord injury, especially hypoxia, ischaemia, haemorrhage and inflammation. Following injury, damaged cord cells form a Zone of Critical Ischaemia, which becomes larger with progressive hypoxia and ischaemia. The

aim of medical treatment is therefore avoidance and management of this secondary injury.

SPINAL VERSUS NEUROGENIC SHOCK

These terms often confuse non-neurological specialists and are regularly used wrongly. Both types of shock may occur following spinal cord injury.

Spinal shock, otherwise considered as a cord neuropraxia, is the <u>temporary</u> "shut down" of cord activity in response to injury, manifesting as paralysis, areflexia and sensory loss. While a patient has spinal shock, it is impossible to determine whether their underlying cord injury is partial or complete. Recovery usually occurs within 24-48 hours, and is the time when the reflex arc most distal to the level of injury has returned e.g. bulbocavernosus reflex.

Neurogenic shock describes the loss of sympathetic outflow following damage to the thoracic sympathetic tracts, associated with cord injury above the level of T6, with higher injuries having a more profound effect. Patients with neurogenic shock are hypotensive with warm peripheries due to the loss of peripheral vascular tone and resistance and reduced venous return from the subsequent pooling of blood in the peripheries. Bradycardia results from reduced sympathetic input and unopposed parasympathetic vagal activity. In the polytrauma patient, warm peripheries and bradycardia help clinicians differentiate neurogenic shock from other types of shock. Neurogenic shock is initially managed with controlled intravenous fluids and, if needed, vasopressors for peripheral vasoconstriction. Failure to adequately treat hypotension will result in further cord ischaemia and worsening secondary injury.

COMPLETE VERSUS INCOMPLETE CORD INJURY

Complete spinal cord injury, as the name suggests, is a complete injury across the cord resulting in the absence of motor or sensory function below the level of injury. In particular there is <u>no sacral sparing</u>. With incomplete cord injuries, partial sensory or motor function remains below the level of injury with sacral sparing. Sacral sparing occurs because the dorsal columns and lateral corticospinal tracts that supply sacral structures are more resistant to primary and secondary injury, and it is confirmed by the presence of normal perianal sensation, anal tone and flexor activity to the big toe.

The level of neurological injury is described as the most distal level at which normal bilateral motor function and sensation is preserved. Due to cord and spine anatomy, the neurological level may not correlate exactly with the level of cord injury. More proximal injuries have more profound clinical signs e.g. complete high cervical cord injuries will result in quadriplegia and often needs ventilatory support due to loss of phrenic nerve and diaphragmatic function.

Incomplete spinal cord injuries are described through defined syndromes. To understanf these injuries it is essential to have a good understanding of cord function and anatomy.

CENTRAL CORD SYNDROME

Typically occurring in elderly patients with a history of cervical spondylosis or arthritis, this is the most common incomplete cord injury. Resulting from neck hyperextension, the central cord is damaged, affecting upper limbs more than lower limbs. Proximal limb function is often preserved with distal limbs affected to a greater extent.

ANTERIOR CORD SYNDROME

Injury to the anterior spinal artery, usually due to retropulsion of bone fragment, compromises the anterior two thirds of the cord. The dorsal columns are preserved resulting in largely motor signs with continuing fine touch and proprioception. Motor recovery is poor.

POSTERIOR CORD SYNDROME

Rare, this results from injury to the posterior spinal arteries, compromising the dorsal columns and subsequent loss of proprioception.

BROWN-SEQUARD SYNDROME (BSS)

Otherwise known as hemisection of the cord, BSS typically occurs following penetrating injury. Patients present with a mixed neurological picture including ipsilateral motor, proprioception and fine touch loss, and loss of contralateral pain and temperature sensation.

CAUDA EQUINA SYNDROME (CES)

This lower motor neurone syndrome occurs from compression of the cauda equina usually due to disc protrusion. Although CES has the best prognosis of all spinal cord injuries, it is potentially devastating if missed. Often occurring in patients with pre-existing chronic low back pain and relatively minor injury, presenting symptoms may include worsening back pain, unilateral or bilateral sciatic nerve signs, saddle anaesthesia, lower limb weakness \pm rectal or urinary dysfunction. Suspicion of CES warrants further investigation and, once confirmed, is considered a neurosurgical emergency, requiring urgent decompressive surgery.

RESUSCITATION AND INITIAL MANAGEMENT

Patients with a traumatic brain or spinal cord injury require aggressive, high quality treatment to minimise secondary injury [11]. Such management begins as early as possible in the patient journey, ideally in the prehospital arena and might include airway protection, ventilatory and cardiovascular support. Suboptimal management at this early stage will worsen morbidity and mortality. Prehospital care is an evolving medical specialty, with dedicated prehospital teams with specific interest, skills and expertise,

enabling them to manage such patients through the provision of advanced critical care away from the normal clinical environment.

Resuscitation in any critically ill or injured patient should follow a robust, A,B,C system as taught through Advanced Trauma Life Support [1]. An initial Primary Survey following a structured system will identify immediate life threatening injuries requiring treatment. The primary survey is followed by a more thorough head to toe examination or Secondary Survey. A structured approach for undertaking a primary survey is outlined as follows [1]:

A – AIRWAY WITH CERVICAL SPINE CONTROL

During initial assessment, <u>always</u> assume that cervical spine injury exists.

Obstructed or compromised airways should be opened in an attempt to relieve obstruction, avoiding unnecessary neck movement. An untreated obstructed airway will rapidly result in hypoxic brain injury. Early intubation with a cuffed endotracheal tube and securing it with tape is considered the gold standard. Lack of appropriately trained personnel or hostile prehospital conditions may prevent this. In situations where patients are semi-conscious, drug assisted intubation may be required. Where skills or personnel are unavailable, use of airway adjuncts including nasopharyngeal (NPA) and oropharyngeal airways (OPA) may be indicated. Concerns exist regarding the use of NPAs in patients with suspected basal skull fracture. In such situations, hypoxia due to airway obstruction will be more detrimental to patient outcome, and a gentle NPA insertion technique should be used.

Use of a hard cervical collar to immobilise the neck and prevent further injury is indicated. Complete neck immobilisation involves the use of sand bags and tape, however in the very young, scared or agitated, this may prove difficult. Clinical judgement should determine whether complete immobilisation or collar-only immobilisation is used. If resources are available, Manual In-line Stabilisation (MILS) may be an appropriate alternative. This involves a person gently holding either side of the head and neck, preventing movement.

B – BREATHING WITH VENTILATORY SUPPORT

Hypoxia and hypercapnia can both cause secondary brain and cord injury. Both can occur following injury or as a result of reduced conscious level, and ventilation may be indicated. Whilst additional oxygen may be sufficient to prevent hypoxia, the aim of ventilation should be to achieve normocapnia in the intubated patient. Hyperventilation, whilst once advocated as a method of reducing ICP by reducing cerebral blood flow, results in potent vasoconstriction and hypoxia through hypoperfusion and is <u>no longer</u> recommended.

C – CIRCULATION WITH HAEMORRHAGE CONTROL

Significant head injury impairs cerebral autoregulation, therefore an adequate MAP is essential to maintain CPP and prevent secondary injury. Patients with multiple injuries

may have an elevated ICP due to their head injury, but also be hypotensive from other injuries resulting in significant cerebral perfusion compromise (CPP = MAP-ICP). Maintaining MAP and reducing ICP are essential [2-4,9].

The first step in maintaining MAP is haemorrhage control. This may be achieved by applying pressure over sites of compressible haemorrhage, splinting long bone fractures or use of pelvic binders, but may require surgical intervention for damage control. Volume replacement with crystalloid or blood products may be indicated, but can be controversial. Hypotension is associated with poorer neurological outcome following traumatic brain injury, with doubling of mortality. While this is obviously undesirable, volume replacement can actually have a paradoxical effect. Whilst restoring normovolaemia, blood pressure and perfusion, fluid replacement may disrupt newly formed and immature clots, dilute clotting factors, and exacerbating further bleeding from non-compressible sites. As a rule of thumb, conscious, talking patients have adequate brain perfusion and do not require fluid replacement. In unconscious patients a systolic blood pressure of 100mmHg is desirable, maintaining MAP whilst minimising the risk of further bleeding [1,9,10].

TYPES OF FLUID:

This is a further area of controversy. Isotonic saline has been the mainstay of treatment, however, use of hypertonic saline has increased in popularity, particularly as an alternative to mannitol in patients with signs of raised ICP. Current evidence suggests that hypertonic saline has a benefit in head injured patients presenting with GCS <8. Early use of blood products is indicated in patients with multiple injuries [12].

D – DISABILITY

Establishing the extent of brain or spinal cord injury requires thorough assessment. The primary survey concentrates on assessing conscious level, pupil response and limb weakness. Using GCS is preferable in providing specific information, but use of the AVPU score may be of benefit to junior staff, paediatric patients or during rapid assessment (AVPU means the patient is assessed as either being <u>A</u>lert, responding to <u>V</u>oice, responding to <u>P</u>ain or is <u>U</u>nresponsive). It should be remembered that conscious level may be affected for reasons other than head injury e.g. alcohol or drug intoxication, hypoxia, hypoglycaemia and hypovolaemia.

Pupil response can sometimes be difficult to record, however, it is important to record size and light response as a baseline. Considered a lateralising sign, alongside limb movement, examination of the pupils may provide information as to where in the brain pathology exists. Dilated, non-reactive pupils may be indicative of an expanding intracranial haematoma stretching the occulomotor nerve as it passes along the edge of the Tentorium Cerebelli. Up to 25% of the uninjured population may have asymmetrical but reactive pupils. This can be considered normal in patients who are GCS 15 with normally reacting pupils.

SPECIFIC HEAD INJURY SIGNS INDICATIVE OF MORE SIGNIFICANT INJURY [4]:

CSF Rhinorrhoea – clear nasal drip due to an anterior base of skull fracture.

CSF Otorrhoea – clear or bloody drip from ear due to a base of skull fracture.

Bilateral peri-orbital bruising (panda or raccoon eyes) – indicative of a base of skull fracture.

Subconjunctival haemorrhage without a posterior limit – result of blood tracking from the orbital cavity

Battle's Sign = late sign. Bruising over Mastoid process

E – EXPOSURE

The final stage of the primary survey is to expose the patient looking for signs of injury. This should be done in an efficient and timely manner, respecting patient dignity and beliefs and anticipating consequences of exposure such as hypothermia. Many specialist teams advocate the term "Skin to Stretcher", reflecting the importance of full exposure to facilitate a complete examination. During this stage, a blood sugar and temperature should be recorded.

SPECIFIC MANAGEMENT GOALS [12-16]

Following completion of the primary survey, immediate life threatening injuries should have been identified and treated. A patient remaining on a spinal board must be log rolled and removed from the board **as soon as possible**. During this stage, the back, spine and peri-anal sensation and tone is examined. Although the board has been removed, cervical spine immobilisation must be maintained. Patients with spinal injuries should be removed from the board as a matter of urgency due to the high risk of skin breakdown and pressure sores.

Maintenance of CPP is essential. Ensuring an adequate MAP and lowering ICP reduces the likelihood of secondary injury [9]. Specialist centres may have the ability to directly measure ICP but this will be unavailable in the remote and rural environment. Less technical methods of lowering ICP include:

- 1. Head up position Patients should be nursed in a 30-degree head up position. In cases of suspected spinal injury this can be achieved by angling the patients' bed to a head up position.
- Correctly fitted cervical collar Overly tight collars (and tube ties) may constrict jugular veins, resulting in elevated ICP. Once sedated and ventilated, many clinicians choose to loosen the collar completely, but leaving in situ, and only retightening before transfers. To avoid neck constriction, it is preferable to secure endotracheal tubes with tape.

- 3. Sedation, paralysis, intubation and ventilation patients with traumatic brain injury may be agitated. Adequate analgesia and sedation, reduces the metabolic rate, ischaemia and ICP. Most anaesthetic agents depress the cardiovascular system resulting in a drop in blood pressure. It is therefore important to strike a balance between sedation and hypotension to prevent a worsening secondary injury. Fluids and inotropes may be indicated to ensure an adequate MAP (ideally 80mmHg).
- 4. Controlled ventilation aim to maintain oxygenation and normocapnia (PaO₂ >13.5 kPa, PaCO₂ 4.5-5.0 kPa). Hyperventilation to a PaCO₂ of 4.0 kPa may result in vasospasm and cerebral ischaemia, and conversely hypercapnia will cause vasodilatation, increasing cerebral blood flow and potentially increasing ICP.
- 5. Gentle handing during the transfer stage rapid acceleration, deceleration and aggressive turning should be avoided. Whether on a hospital trolley or in an ambulance, such manoeuvres cause fluid and pressure shifts within the body and brain worsening secondary injury.
- 6. Normoglycaemia adequate glucose is essential for cellular metabolism. Hypoglycaemia can result in tissue acidosis causing further cell damage.
- 7. Cooling Evidence now supports the active cooling of patients following cardiac arrest, where it is considered to have a neuroprotective effect. Mild hypothermia to about 35 degrees is thought to have a similar neuroprotective effect in head injury management as well as reducing ICP.Profound hypothermia whilst being neuroprotective causes cardiac dysrhythmias, therefore should be avoided [4].
- 8. Hypertonic Saline versus Mannitol Mannitol is an osmotic diuretic and has been the mainstay of treatment for patients with TBI, used for raised ICP with signs of impending brain herniation. Available as a 20% solution and typically administered as an intravenous bolus of 0.5 -1g/kg, it is used to "buy time" whilst awaiting definitive neurosurgical intervention. Mannitol has two distinct mechanisms of action. Initially, it expands intravascular volume, reduces viscosity, increasing cerebral blood flow and oxygen delivery. After approximately 15-30 minutes, an osmotic gradient is established across an intact blood brain barrier drawing fluid from neurons, reducing intracranial volume and pressure. Mannitol is not, however, a panacea for head injury management. It may contribute to blood brain barrier breakdown, and may paradoxically worsen cerebral oedema after repeated administration. Mannitol also induces a large diuresis. This may cause a significant fall in MAP reducing CPP.

Hypertonic saline works in a similar manner to mannitol, initially reducing viscosity and improving cerebral perfusion, and drawing extracellular fluid into the vascular system across an osmotic gradient. It reduces brain water and increases plasma volume. Hypertonic saline does not cause diuresis and therefore is of particular advantage in the hypotensive head injured patient. Considered the fluid of choice in many centres, it is available as a 3% solution, and can be administered at 6ml/kg up to a maximum of 500ml.

 Steroids – In 2004, a well-organised, multi-centre randomised control trial (CRASH Trial) compared the use of steroids and placebo in the acute management of patients with TBI. Terminated early due to a statistically significant rise in two-week mortality, the study recommends that steroids <u>should</u> <u>not</u> be used in the treatment of acute traumatic head injury [8].

SPINAL INJURY MANAGEMENT [13,15,16]

Patients with spinal injuries require very careful assessment and observation [13]. High cervical cord injuries are associated with diaphragm paralysis and respiratory failure. Unrecognised neurogenic shock may result in pulmonary oedema, due to overadministration of intravenous fluids in an attempt to optimise blood pressure. Significant non-spinal injuries may be less evident due to loss of pain sensation. Whether requiring transfer or not, patients with cord injury should be managed on an appropriate, comfortable and supportive mattress. Good nursing care is essential in the prevention and management of pressure sore areas. Subsequent transfers should not be undertaken on a hard spinal board. Where possible a Vacuum Mattress should be utilised. This provides spinal support, warmth and comfort, and is the gold standard for cord-injured patients undergoing transfer.

It is essential that non-spinal injuries be appropriately managed. Chest drain insertion, pelvic binding or long bone splintage may be required. Patients with upper spinal injuries run the risk of tiring and developing respiratory failure. Supplemental oxygen is essential and the availability of clinical expertise to provide supported ventilation is highly recommended. Gastric stasis may occur with a high risk of regurgitation and aspiration in the supine patient. NG tube insertion is advised. Urinary catheterisation is necessary as bladder sensation and control may be lost. As well as avoiding bladder over distension, this will provide the clinician with valuable information concerning fluid balance.

SAFE TRANSFER [15-18]

Unless conveyed directly to a tertiary neurosurgical or spinal injury centre, most patients with moderate-severe traumatic brain injury or spinal injury will require a secondary transfer for definitive management. Such transfers arise more frequently in remote and rural areas where resources and expertise may be limited. Transfers may also be intrahospital with patients moving to/from CT, operating theatres and wards etc. The period during which a patient is being transferred is considered a period of enhanced patient risk during which complications may occur. Many complications can be anticipated and avoided through appropriate preparation and stabilisation; therefore, rather than adhoc co-ordination and dealing with problems as they arise, clinical staff should be encouraged to use a reliable, systematic approach to transfer preparation. The principles of safe transfer ensures that the **Right** patients are taken by the **Right** people, at the **Right** time, to the **Right** place, using the **Right** transport, and receiving the **Right** care throughout. To adequately prepare for a safe transfer the following mnemonic may be useful – **ESCAPE:** Evaluate the situation – Is transfer needed and, if so, how quickly?

Surveillance – Survey the situation. Who is the team leader? What tasks need completion pre-transfer? Who is doing them?

Communicate – Talk to the receiving ward, department or hospital e.g. ensure they have the necessary resources available.

Agree – Agree with the receiving team that transfer is required, and they are accepting the patient.

Prepare – Prepare the patient – ensure the patient is adequately resuscitated and stabilised. Lines, catheters, drains and airways are secure and accessible. Prepare any drugs and equipment needed e.g. spare batteries for monitors if no method of charging en route. Prepare yourself – have you got appropriate clothing and protective equipment, food, money, contact numbers, mobile phone or radio.

Exit – All transfers required a "final push" to leave a clinical area as a matter of urgency. Ensure that transport has been organised, with adequate monitoring available and the correct route to the receiving department or hospital has been identified.

Whilst not covering all aspects of transfer, the ESCAPE mnemonic provides a structure around which transfer templates can be built. Whilst no system can prevent all complications, use of a pre-defined template will allow clinicians to address key areas in a timely and structured manner, providing opportunity to consider, manage and avoid many adverse incidents.

SUMMARY

Traumatic brain and spinal injury is the main cause of death in those under 45. Whilst difficult to quantify, the morbidity associated with such injuries are significant. Anticipation of and prevention of secondary injury has the most important effect on improving quality of life and outcome following head or spinal injury. This article summarised current management goals for patients with brain or cord injury.

It is reasonable to assume that patients with significant injuries warrant quality treatment and monitoring. It must be agreed then that quality treatment and monitoring be continued at all times, even during periods of patient transfer. Safe transfer is the result of careful planning and co-ordination. Many hospitals will have established transfer proformas and checklists to facilitate timely transfers. A template is provided that can be adapted to reflect local arrangements. Experienced clinical staff capable of providing ongoing critical care should accompany patients. The most senior team members available should take responsibility for patients with serious brain and spinal injury.

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11. SURGICAL SEPSIS

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INTRODUCTION

Sepsis and SIRS are widely used terms, often used interchangeably, but wrongly so. Surgical infection with organ dysfunction is one of the major causes of surgical mortality and morbidity, the others being major/severe trauma and major elective surgery. Surgical patients with sepsis can either present with a primary source e.g. due to a perforated viscus (typhoid) or bacterial overgrowth (cholangitis), or with infection secondary to major trauma or as a complication after surgery (pneumonia, wound infection).. The mortality of major surgical sepsis is about 50% and therefore aggressive resuscitation, supportive therapy, diagnosis and treatment are essential components of effective therapy. Early recognition of patient deterioration and initiation of therapy such as oxygen, intravenous fluids and antibiotics can often make major differences in patient outcomes.

The purpose of this article is to discuss the different definitions that exist in the sepsis literature, hopefully helping to clear up some confusion in terminology, describe the pathological processes of the systemic inflammatory response and sepsis, and how these can lead to multiple organ failure, an all too familiar event in sepsis, and how to recognise and treat sepsis early and efficiently. In critical care the focus is on early recognition of deterioration, effective resuscitation and organ support while a definite diagnosis is reached and specific therapy instituted, whether medical, interventional or surgical. Specific therapies for specific sources of infection will not be discussed, but some used as examples of treatment. This article will also provide simple algorithms of thinking for finding the source of surgical infection and for systematic patient management.

Different literature sources are quoted in this paper but it is very valuable for all surgical trainees to look at the Surviving Sepsis Campaign website and read their guidelines on managing sepsis [1]. This not only gives valuable advice on structured management, but also summarises the evidence and makes recommendations on controversial therapies such as steroid use and blood glucose control in sepsis.

DEFINITIONS

"Sepsis" is a term that is used very loosely in critical care, and probably means different things to anaesthetists, surgeons and physicians. In clinical practice this can apply to a wide range of infected and non-infected illnesses, from mild post-operative pyrexia to life-threatening multi-organ failure. Various consensus documents have been produced to define sepsis and related syndromes and it helps to keep things simple [2, 3, 4]:

The Systemic Inflammatory Response Syndrome **(SIRS)** can be defined as: <u>Two</u> or more of:

- Temperature <u>></u>38 °C or <u><</u>36 °C
- Heart Rate <u>>90 beats/min</u>

- Respiratory Rate >20 breaths/min
- White Blood Cell count ≥12000 or ≤4000 per mm², or >10% immature neutrophils.

It means that a severe inflammatory response can produce signs that are usually associated with infection but without an infective trigger. Severe pancreatitis, burns and major trauma are probably some examples of such a process.

Sepsis = SIRS plus definite infection. It can sometimes be difficult to pinpoint the source of infection. However, it would be wise to remember that patients may present with severe signs of sepsis and are resuscitated and given intravenous antibiotics, without clarity whether they have SIRS from an infective or non-infective source.].

Severe Sepsis = SIRS + Infection + Organ dysfunction or Tissue hypoperfusion. Examples include signs of acidosis due to hypoperfusion (low pH, raised lactate), mild hypoxia, oliguria or depressed consciousness. Organ dysfunction should be suspected in any critically ill patient who seems breathless, has poor perfusion, confusion, poor urine output or abnormal coagulation [5]. Essential however is that the patient responds to initial resuscitation with oxygen and intravenous fluids and the organ dysfunction is reversed early.

Septic Shock = SIRS + Severe sepsis + Refractory Hypotension. Organ dysfunction does not respond to initial fluid resuscitation.

PATHOPHYSIOLOGY OF SIRS

The systemic inflammatory response syndrome (SIRS) was first defined by the Society of Critical Care Medicine in 1992 [6]. It is characterized by activation of multiple pathways including cytokine release, complement cascade, acute phase protein and cellular activation of leucocytes and vascular endothelium. It represents the clinical response to non-specific insult of either infectious or non-infectious origin [7]. Left unchecked, SIRS progresses to cause end-organ damage and multiple organ dysfunction syndrome.

Triggers of SIRS are wide ranging and reflect a number of both infective and non-infective conditions:

- Infection: The list of infections that can trigger SIRS is long and distinguished. Key points to remember is that seemingly benign infection can trigger the pathway in patients with other comorbidities and relatively poor physiological status.
- Inflammation: Sterile inflammatory conditions such as pancreatitis.
- Trauma: including major injuries, burns and electrocution.
- Surgery
- Haemorrhage: as a consequence of trauma, or other causes such as a gastrointestinal bleed
- Myocardial infarction
- Profound dehydration
- Drug Reactions: theophylline, or drugs of abuse such as cocaine
- Haematological malignancy

- Auto-immune conditions such as vasculitits
- Any other cause of shock: cardiogenic, anaphylactic or hypovolemic

Once activated, the pathophysiology of SIRS is generally defined through three stages.

Stage 1: Follows the initial insult (this may be related to infection, trauma or inflammation). Cytokines are released at the site of infection or injury with the goal of promoting a localized inflammatory response to promote repair or eradicate infection.

Stage 2: Small quantities of locally released cytokines are leaked systemically. This leads to leucocyte and platelet recruitment. The goal of this stage is to ensure homeostasis.

Stage 3: If homeostasis is not restored, a significant systemic reaction occurs which is destructive rather than protective. Systemic release of cytokines and pro-inflammatory mediators, as well as activation of the reticulo-endothelial system, leads to extensive loss of macrovascular and microvascular integrity. This causes end-organ damage [6].

Left unchecked, this initially protective process becomes destructive, leading to multiple organ dysfunction syndrome (MODS) and life-threatening clinical deterioration.

This differentiation of the steps of SIRS allows one to analyse the pathophysiology in more detail.

STAGE 1: INITIATION OF LOCALISED INFLAMMATORY RESPONSE

The method of initiation of the localized inflammatory response depends on the aetiology of the insult. In trauma, ischaemia and sterile conditions such as pancreatitis, direct trauma to the tissue causes inflammatory cytokine release. With infection, the response is better studied and more complex. It is thought that pathogen-associated molecular pathogens (PAMP's), activate the inflammatory response via activation of Toll-Like Receptors (TLR's). Toll- Like Receptors recognize PAMP's, with each member of the TLR family having a high affinity for specific PAMP's, for example lipopolysaccharide with TLR4 [8]. Binding of PAMP's to TLR as part of an infective process initiates local cytokine release.

The cytokines commonly associated with SIRS are TNF- α , the interleukin family (mainly IL-1, IL-6 and IL-8) and macrophage inflammatory protein 1- α [9]. Local cytokine release helps direct the inflammatory response, but systemic release promotes progression to a generalized inflammatory response.

STAGE 2: SYSTEMIC CYTOKINE RELEASE

Systemic cytokine release in small quantities has benefits in terms of maintaining homeostasis. TNF- α and IL-1 released systemically are responsible for the release of stress hormones (noradrenaline, vasopressin and activation of the renin-angiotensin system) as well as producing fever. IL-6 is responsible for systemic recruitment of acute-phase reactants such as C-reactive protein (CRP) and procalcitonin [10]. Complement activation and activation of the coagulation cascade occur. Interestingly, infection promotes more TNF- α release than trauma, a reason why fever is more apparent in infection [11].

Again, in the early stages of infection or trauma, these changes can be beneficial to the patient in terms of homeostasis but, if unchecked, progression to stage 3 occurs, where an unbalanced pro-inflammatory state progresses to cause multiple organ dysfunction.

STAGE 3: DESTRUCTIVE SYSTEMIC INFLAMMATORY RESPONSE

Whilst initially protective, the systemic inflammatory response becomes amplified and destructive to the patient's physiology. The circulating cytokines promote activation of the coagulation cascade, activation of the complement cascade, activation of prostaglandins, platelet activating factor and leukotrienes. These pathways in turn provoke further inflammatory processes which progress to causing multiple organ damage and dysfunction.

Activation of the coagulation cascade occurs as TNF and IL-1 promote vascular endothelium to express tissue factor. Tissue factor in turn promotes activation of the coagulation cascade, with release of prothrombin and platelet activating factor [12]. This leads to microvascular thrombosis, which impairs oxygen exchange and nutrient flow at a local level; this effect is widespread contributing to multiple end-organ failure.

Systemic complement cascade activation also occurs in tandem with other processes. C3a and C5a activation leads to systemic release of further cytokines and causes vasodilation and increased vascular permeability [13]. This causes vascular endothelial damage which further contributes to organ dysfunction.

Further systemic responses of this pro-inflammatory state, led by both cytokines and complement, include enhanced nitric oxide release, release of platelet activating factor and recruitment of leucocytes.

Nitric oxide release, again an initially protective response, leads to mitochondrial dysfunction and cellular hypoxia when in excess [14]. This cellular hypoxia impairs organ capability and further contributes to eventual organ dysfunction.

Platelet activating factor, activated in response to cytokines, helps to guide the cellular component of SIRS. Platelet aggregation is stimulated, which further contributes to microvascular thrombosis which precipitates organ damage. Both activated platelets (via P-selectin [15]) and platelet activating factor stimulate leucocyte recruitment [16]. Leucocyte recruitment and adherence to the already inflamed vascular endothelium further increases permeability and subsequent organ dysfunction [17]. Furthermore activation of cellular pathways of the inflammatory response amplifies the already exaggerated and deleterious response.

At cellular level cell hypoxia begins to occur which results in organ dysfunction. Even the cellular response to this hypoxia contributes to further propagation of the deleterious response. In response to tissue hypoxia (also seen as an initiating factor in SIRS secondary to ischaemia-reperfusion injury) Hypoxia-Inducible Factor-1 is produced. This is an important cell signalling molecule which, via activation of Mitogen Associated Protein (MAP)-kinases, leads to further increases in endothelial nitric oxide production and enhanced Nuclear Factor kappa beta activity. Both these responses again serve to enhance the inflammatory response seen in SIRS [18].

In SIRS, as in other cascades, the pathways do not act completely unopposed. The compensatory anti-inflammatory response system (CARS) is also activated in an attempt to reverse this overwhelming inflammation. IL-4 and IL-10 are cytokines that directly

reduce the production of TNF- α , IL-6 and IL-8. Antagonists to these cytokines are produced that act by direct inactivation or by receptor blockade [19]. In a patient with a minor inflammatory or infective insult, CARS is sufficient to restore normal physiology, but with more serious insults or patients with significant co-morbidity, this antiinflammatory effect is minor in comparison to the overwhelming pro-inflammatory state and cannot redress the balance.

MULTIPLE ORGAN DYSFUNCTION SYNDROME (MODS)

These underlying pathophysiological changes explain how, once initiated, the pathway of SIRS can progress to MODS. MODS is defined as the development of potentially reversible physiological derangement involving two or more organ systems not involved in the diagnosis causing initial pathology [20]. The pathophysiological changes initiated by SIRS lead to characteristic histological changes in organs, with evidence of oedema, inflammation, tissue ischaemia or necrosis with variable degrees of fibrosis or repair. These changes are responsible for dysfunction and explain how this occurs in each organ system.

MAJOR ORGAN SYSTEMS AFFECTED IN MODS AND SPECIFIC MANAGEMENT

Management of MODS is essentially supportive, with directed therapy to treat the initiating event that triggered SIRS, if possible. This may take the form of surgery (for example treatment of a gastrointestinal perforation), antibiotic therapy for infection or arrest of haemorrhage in trauma. Specific treatments for such conditions are covered in other review articles.

CARDIOVASCULAR SYSTEM

Cardiovascular instability occurs readily with the advancement of SIRS to MODS, with subsequent instability contributing further to deleterious effects of SIRS on other organs. The hallmarks of cardiovascular derangement are [21]:

- Reduced vascular tone, mediated by the enhanced nitric oxide release in SIRS; this contributes to the development of hypotension as the effective space required to be filled by the intravascular volume is increased.
- Increased capillary permeability, contributing to local organ oedema.
- Alterations in blood flow to specific organs e.g. kidney and gut.
- Myocardial dysfunction compounds problems further by causing hypotension that is refractory to fluid resuscitation and limiting flow to other critical organs.

Management of cardiovascular failure is supportive but can present complex challenges to the physician. Essential management is to ensure adequate intravascular volume through parenteral fluid therapy. This can be difficult to achieve, particularly if the patient is experiencing third space fluid losses. Insertion of a central venous catheter to allow invasive measurement of central venous pressure and an arterial line for more accurate, continuous measurement of blood pressure and mean arterial pressure allows parenteral therapy to be better directed and helps prevent complications from 'fluid overload', where the already labouring cardiac system function is further compromised.

If adequate blood pressure cannot be maintained with parenteral fluid therapy alone, inotropic and vasopressor support may be required. These work by stimulating the autonomic nervous system. Vasopressors (such as noradrenaline) act by inducing peripheral vasoconstriction, with the objective of reversing the profound peripheral vasodilatation. Inotropes act to improve the efficacy of cardiac contraction.

PULMONARY SYSTEM

The hallmark of respiratory failure in association with MODS is impaired gas exchange, with the patient often requiring ventilator support to maintain adequate oxygenation. The process is similar to that seen in Adult Respiratory Distress Syndrome (ARDS) [see Surgery in Africa (SiA) review on hypoxia, April 2012 for more detail]. SIRS-induced changes cause microvascular disturbance, inflammation and oedema of the lung parenchyma with localized leukocyte recruitment [22]. The inflamed lungs, with impaired localized gas exchange become 'stiff', reducing ventilatory capacity and hastening the deterioration in respiratory function [23].

Diagnosis and management is similar to that of ARDS, i.e. essentially supportive. Ventilatory support allows for adequate oxygenation until underlying SIRS processes can be treated or reversed. Respiratory failure is likely to progress or persist until this occurs, and will not spontaneously resolve.

RENAL SYSTEM

Renal failure in SIRS progressing to MODS occurs as a combination of the pathophysiological mechanisms described, having direct effects on renal perfusion and renal cells. Cardiovascular instability and hypotension further compounds the problem, as renal tubules are very vulnerable to ischaemia as a result of hypoperfusion. Furthermore nephrotoxic drugs can contribute to renal dysfunction.

Renal failure manifests clinically as oliguria, often identified with a concurrent increase in serum creatinine and potassium. Initial management is supportive, with the aim of maintaining adequate renal perfusion through cardiovascular support. Nephrotoxic agents should be discontinued. Renal failure can be refractory to these measures and progress. Serum potassium rises as it is not being effectively excreted and reach dangerous levels. Fluid overload can occur as a consequence of inefficient excretion failure which further compromises the efficacy of the cardiac system. Under these circumstances Renal Replacement Therapy (dialysis or haemofiltration) becomes necessary.

GASTROINTESTINAL FAILURE

Gastrointestinal failure occurs as a combination of the described pathophysiological changes, disrupting microvascular flow; hypotension reduces global intestinal perfusion and affects changes in gut bacterial flora.

Loss of integrity of the intestinal mucosa allows for translocation of bacteria and endotoxin into the portal venous system and the liver which further promote the inflammatory response and contribute to global patient deterioration. Loss of intestinal function presents as intestinal failure with paralytic ileus and malabsorption. Difficulties in maintaining nutrition are encountered and parenteral nutrition may be required.

HEPATIC FAILURE

Hepatic failure in MODS generally presents as hyperbilirubinaemia with evidence of cholestasis (without associated biliary obstruction). Accompanying these changes acute phase protein synthesis is effected. CRP typically rises while albumin synthesis falls. This reduction in albumin has further impact on cardiovascular support – lower serum protein impairs efficacy of capillary osmotic exchange, promoting further difficulties in maintaining intravascular volume.

If profound hypotension is present with SIRS, hepatic failure can be compounded by development of 'shock liver', which is demonstrated by derangement of transaminases.

ENDOCRINE FAILURE

The best characterized endocrine dysfunction associated with SIRS/MODS is hyperglycemia and relative insulin resistance. When detected, normoglycemia is maintained through use of a sliding scale, but one must guard against overzealous control of blood glucose [24, 25].

NEUROLOGICAL FAILURE

Neurological failure is readily identified in the critically unwell patient with SIRS as patients often have a reduced level of consciousness, as measured by the Glasgow Coma Scale. When the level of consciousness is impaired to such a degree that airway maintenance is compromised or respiratory effort is impaired, intubation and ventilation will be required.

PRINCIPLES OF MANAGEMENT

A. RESUSCITATION:

The aim of resuscitation is to restore **oxygenation** and **perfusion** as rapidly as possible. The treatment initiated in the ward can have a profound effect on outcome. The first member of the team who recognises signs of sepsis should therefore initiate the following steps of resuscitative treatment and not wait for the patient to be seen by a senior surgeon or transfer to ICU/HDU:

1. Airway: septic patients can have depressed consciousness; make sure that they can maintain their airway or initiate appropriate support.

2. Oxygen: in septic patients oxygen delivery to cells is compromised. Give highflow oxygen even if saturation is >90% on monitor. 3. Intravenous fluid resuscitation: These patients need large amount of restorative IV fluids. Minimum challenge is 1000 ml crystalloid or 300-500 ml colloid in first 30 minutes (unless patient has seriously limited cardiac contractility). Septic patients are always more fluid deficient than initially estimated. 4. If already apparent that patient is septic, give broad spectrum antibiotics after taking appropriate specimens for microbiology culture

B. FULL PATIENT ASSESSMENT:

Assess the patient systematically by talking to the patient, asking family or guardians and nursing staff for extra information, checking previous notes, patient charts and results of investigations yourself, followed by thorough systematic clinical examination, remembering to look at drains and all sites where the skin had been breached (wounds, lines, cannula and epidural sites). Think of potential sites of infection, as discussed below. [See principles of assessment in critical care, SiA review December 2011].

When assessing a patient with potential sepsis, ask the following specific five questions:

- 1. Are there signs of SIRS?
- 2. Is there evidence of infection?
- 3. Is there evidence of organ dysfunction?
- 4. If sepsis, where is it coming from?
- 5. How to deal with the source of sepsis quickly and efficiently?

If there are clear clinical signs of SIRS the patient needs aggressive resuscitation, as discussed above. If there is evidence of infection, blood and other specimens for culture (e.g. sputum, urine, wound swabs, CVP catheters, CSF, as appropriate) have to be taken and appropriate antibiotic therapy started immediately. In case of organ dysfunction the patient needs to be moved to a higher level of care (ICU or surgical HDU) rapidly and/or other specialists have to be consulted (e.g. anaesthetist/intensivist, microbiologist, chest physician). To find the source of sepsis do the simplest investigation that gives an answer the quickest. Remember that getting a CT scan will not cure the patient and patients should not be allowed to deteriorate physiologically while waiting for test results. It is often better to do a second laparotomy on clinical suspicion than to wait hours for a CT or other test results.

If the patient deteriorates at any point during systematic assessment and waiting for investigation or treatment, go back to primary assessment of ABCDE and review resuscitative measures.

C. SPECIFIC DIAGNOSIS (SOURCE OF SEPSIS)

As part of the clinical assessment of patients with sepsis one should think of five potential sites of infection. These are described in somewhat chronological order:

1. LUNGS:

A very high temperature within 12-24 hours of general anaesthetic is usually due to atelectasis. This has a typical appearance on Chest X-ray of fine horizontal lines, usually in the lower lobes, and is due to small airway collapse. The treatment consists of physiotherapy and proper pain relief so that the patient can breathe deeply and cough; it helps to sit the patient up. Antibiotics are not required. Pneumonia can follow if atelectasis is not dealt with or can happen spontaneously; it is more common if patients are not mobilised quickly enough and helped to breathe deeply and cough efficiently

after general anaesthesia and major abdominal surgery. It is also more likely to happen in patients who are malnourished or emaciated, or immune compromised for any other reason. Pneumonia usually develops after 2-3 days and should be regarded as a hospital acquired infection; antibiotics should be selected according to organisms usually found in hospital bacteriological cultures, not as found in community acquired pneumonia.

2. URINE:

Any instrumentation or other breaching of the urinary tract increase the risk of urine infection. Symptoms can vary from mild dysuria and low-grade fever to very high temperatures and systemic signs of sepsis. Urine infection typically presents a few days after catheter removal or bladder instrumentation. If urine testing strips or culture is not readily available it can be very valuable to do microscopy and gram staining. If a patient arrives with catheter in situ the causing organisms would usually be community based but if infection develops after hospital catheterisation one should assume this is due to hospital acquired organisms.

3. WOUND:

This is an obvious site to look for infection and usually shows between 3 and 7 days after surgery, depending on the virulence of the infecting organism. With clean operations (breast surgery, orthopaedic surgery) infection is usually due to skin organisms (Staphylococcus, Streptococcus), with mixed infections (e.g. in necrotising fasciitis) and multi-resistant organisms (e.g. MRSA) of specific concern. With operations in contaminated or septic surgical fields (e.g. with intra-abdominal sepsis or colonic resection) wound infection can be caused by lower gastro-intestinal organisms (e.g. E. coli, pseudomonas species). Patients who are chronically ill, immune compromised or have received various antibiotics before hospitalisation, can have unusual bacteriological or fungal causes of infection.

When thinking of wound infection one should always assess all sites where the skin had been breached e.g. for peripheral or central venous cannulation, epidural catheters, aggressive shaving techniques, skin pricks by traditional healers, tattooists or acupuncture. I recall a specific patient who developed MRSA meningitis through an epidural site; he presented with acute nocturnal confusion but it was not until daytime that somebody noticed the red tender swelling on his back.

4. "INSIDE":

Sepsis related to deep sites of infection usually present later than wound infections would. Examples are an anastomotic leak after colorectal anastomosis which typically present after 5-10 days, puerperal sepsis due to retained products after caesarean section and septic arthritis or osteomyelits after seemingly clean orthopaedic surgery.

Patients in this category are usually very unwell, have profound SIRS and develop organ dysfunction and failure rapidly. Aggressive resuscitation, organ support, investigations and treatment is necessary. Most patients will need further surgery or radiological intervention (e.g. drainage of an abscess) urgently. Surgery is often of a salvaging nature e.g. a Hartman's resection for a leaking rectal anastomosis.

5. "OTHER" OR LATE CAUSES:

These are patients who develop low grade or swinging fever, usually 10-14 days postsurgery, or have mild signs of infection that never really settle, or respond to antibiotics temporarily. Causes include deep vein thrombosis that can cause low grade fever 14 days after operation, infective endocarditis in patients who had bacteraemia during their immediate post-operative course (e.g. from a virulent wound infection or intra-abdominal sepsis) or after cardiac instrumentation (pacemaker wires, pulmonary catheter) or *Clostridium difficile* overgrowth in the GI tract which is more often after using certain antibiotics [27].

D. SELECTION OF ANTIBIOTICS

GENERAL PRINCIPLES [28, 29]

What antimicrobial agent to choose in any particular clinical situation can often be a difficult decision to make. Antibiotic guidelines are used in many healthcare situations to offer appropriate choices for agents for common clinical conditions in a particular hospital, region or country. Antibiotic guidelines are not easily transferable between hospitals, because each hospital may have different problems with various species of bacteria, bacterial resistance, supply of particular antibiotic agents, and ability to measure therapeutic levels or monitor side-effect profiles. Every hospital or clinical environment should consider what particular antibiotic agents are most suitable for their particular requirements, and issue guidance that includes the clinical condition, choice of agent, dose, method of administration, minimum duration of treatment, and under what circumstances can intravenous therapy be converted to an oral alternative. A penicillin (or beta-lactam) allergy alternative may also be appropriate.

There are some principles of antimicrobial treatment that should be considered before starting any antibiotic treatment:

- The advice given in any antibiotic policy is based on the information available at the time of writing. It should be interpreted by the prescriber in the light of professional judgement and clinical assessment.
- Prescribe an antibiotic only when there is likely to be a clear clinical benefit.
- Use simple generic antibiotics first whenever possible.
- The use of expensive antibiotics (e.g. quinolones and cephalosporins) is inappropriate when standard less expensive antibiotics remain effective.
- Avoid widespread use of topical antibiotics (especially those also available as systemic preparations).
- In **pregnancy avoid** tetracyclines, aminoglycosides, quinolones, high dose metronidazole. Short-term use of trimethoprim (theoretical risk in first trimester in patients with poor diet, as folate antagonist) or nitrofurantoin (at term, theoretical risk of neonatal haemolysis) is unlikely to cause problems to the foetus.
- Gentamicin therapy requires monitoring. Once Daily Gentamicin Dosing is more clinically effective and less toxic.

- If renal function is impaired, antibiotic dose adjustments may be necessary.
- If the patient is penicillin allergic, review the nature of the allergy. If allergy is minor (eg rash), it is safe to use cephalosporins (cross-over sensitivity is 10%). If patient has had previous anaphylaxis, do not use any of the β-lactam antibiotics (including piperacillin/tazobactam and carbapenems).
- As broad spectrum antibiotics (e.g. cephalosporins) are implicated in *Clostridium difficile* infection, an alternative antibiotic should be recommended where possible.
- Review intravenous antibiotics daily. Consider switching to oral therapy when the patient is clinically improved and the following criteria are satisfied: temperature is resolving; patient can tolerate oral therapy; suitable oral alternative is available.

SEPSIS: WHAT ANTIBIOTIC TO USE? [30, 31]

There are a number of factors to consider when determining what antibiotic is appropriate, for a particular infection, in any patient. The first factor involves the clinical origin of sepsis, and the likely micro-organisms which may then be causing the sepsis; therefore, if a chest infection is diagnosed in the community setting, then it is likely that the micro-organism is *Streptococcus pneumoniae*. If a lower urinary tract infection is diagnosed as the source of sepsis, the likely organism is *Escherichia coli*. The appropriate antibiotic would be an agent that had a spectrum of cover that included the most likely pathogen, however, other pathogenic organisms can also cause chest or urinary sepsis, and these organisms may need to be covered with a broader spectrum agent. In chest infection, *Streptococcus pneumoniae* may be the most common cause of community acquired pneumonia, and penicillin may be sufficient for treatment, but *Haemophilus influenzae* can also be implicated, and this organism would be resistant to penicillin. So for chest infection amoxicillin or ampicillin may be more appropriate.

Abdominal sepsis would be highly likely to be due to gram negative bowel organisms ("coliforms") with the possibility of anaerobes also being present. Appropriate treatment of abdominal sepsis would therefore include an agent with broad gram negative activity, such as an aminoglycoside, and an agent with anaerobic activity, such as metronidazole. Wound infections and other skin and soft tissue infection, the likely pathogen would depend on the cause and nature of the wound, with *Staphylococcus aureus* and *Streptococcus pyogenes* often implicated.

Resistance of certain micro-organisms to antimicrobial agents is an area of global concern. In some parts of the world, organisms such as *meticillin resistant Staphylococcus aureus* (MRSA), vancomycin resistant enterococci (VRE), and multidrug resistant gram negative organisms including carbapenemase resistance, have become a major clinical problem. Penicillin resistant *Streptococcus pneumoniae* can vary from 4% in the UK, 44% in Spain, and 80% in parts of southern Africa. The local rates of bacterial resistance will drive the choice of agent in a particular area, which over time will drive the resistance rates in a cyclical manner. Appropriate choice of antimicrobial agent, short duration of therapy when possible, and surveillance of bacterial resistance will help prevent rapid progress of antimicrobial resistance.

Some antimicrobial agents have different pharmacodynamics and tissue penetration abilities. For example, some agents do not penetrate the blood-brain barrier well, whilst other do not attain good serum, tissue, sputum or urine concentrations, and would not be

useful for treating infection in sites of poor penetration. Nitrofurantoin does not attain good serum levels, but does accumulate within urine; gentamicin attains very high serum levels but does not penetrate into CSF well.

Cost and availability of antimicrobial agents will be a major factor in choice. A course of broad spectrum carbapenems (e.g. meropenem) can be several thousand times more expensive than basic penicillins, aminoglycosides or metronidazole. A good antimicrobial guideline, locally produced, and based on common pathogens, costs and available antimicrobial agents, is an essential requirement for any healthcare facility.

E. PHYSIOLOGICAL ORGAN SUPPORT

Global management of the patient with SIRS/MODS is aimed at optimising physiological parameters through parenteral fluid and inotropic support of cardiovascular dysfunction, oxygen therapy and ventilator support of respiratory dysfunction and targeted support of other organs as necessary. Patients with severe SIRS and MODS need to be managed in an intensive care unit with regular assessment and invasive monitoring to allow for optimisation of treatment and to detect early deterioration, facilitating further rapid interventions as necessary.

The goal of treatment, in conjunction with this physiological optimisation, is to reverse and treat the underlying/provoking cause of SIRS, depending on the primary condition.

F. SURGICAL MANAGEMENT

Specific surgical management are not discussed in this paper, but the essential principle is that the right procedure should be performed at the right time on the right patient. Decision making with regards to the timing of surgery is critical and involves a teambased approach with input from both intensive care specialists and senior surgeons. Surgical management should be performed when patients are in the optimal physiological condition, but not delayed to allow the provoking cause to become irreversibly detrimental to the patient. Such decision making takes both skill and experience and should be done by the most senior specialists available, with low thresholds to ask for second opinions.

G. MANAGEMENT ADJUNCTS IN SIRS

STEROID THERAPY

While initial research demonstrated worsened outcomes in patients with SIRS treated with high dose steroids, low doses of hydrocortisone (200mg for 5 days), has been shown to improve survival and reversal of shock in inotrope-dependent patients and should be considered [2].

TNF-A INHIBITORS

Considering the central role that $TNF-\alpha$ plays in the SIRS pathophysiological pathway, research has been directed into benefit of inhibition with etanarcept or monoclonal antibodies. Small studies have shown a small but significant benefit, though routine use is not currently recommended [32].

TLR ANTAGONISTS

In sepsis, activation of inflammatory pathways via TLR activation is thought to play a central role. Inhibition of TLR4 with Eritoran (E5564) is currently undergoing analysis in the small ACCESS trial [33], which is yet to report. Earlier trials demonstrated small but not-statistically significant differences in mortality, though they were really only aimed at assessing the safety of such agents [34].

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12: CAUSES OF SIRS IN SURGERY

12A. ACUTE PANCREATITIS

FRANCIS ROBERTSON

INTRODUCTION

Acute pancreatitis is characterized by inappropriate activation of trypsinogen to trypsin within the acinar cells of the pancreas, resulting in inflammation and destruction of the gland [1]. Acute pancreatitis has a prevalence of 22.4-40 per 100000 in the UK and has been demonstrated to be increasing in prevalence for unknown reasons [2,3].

In the overwhelming majority of patients the condition is mild and self-limiting. Around 20% of patients will progress to develop severe acute pancreatitis, characterized by the Systemic Inflammatory Response Syndrome (SIRS) and resulting Multi-Organ Failure (MOF). The presence of MOF lasting for longer than 48 hours increases the predicted mortality rate from around 1% to 35% [4,5]. This group of patients requires prompt identification and aggressive supportive management. Current trends have gone towards less invasive and delayed interventions [6].

AETIOLOGY:

Common causes of acute pancreatitis can remembered with the pneumonic **GET SMASHED**:

- G gallstones
- E ethanol (alcohol)
- T trauma (especially blunt abdominal trauma)
- S steroids
- M mumps
- A autoimmune disorders
- S scorpion bites
- H hyper and hypo (metabolic disturbances)
- E ERCP
- D drugs

PATHOPHYSIOLOGY: THE INFLAMMATORY RESPONSE:

The inflammatory response is the body's defence mechanism against invading pathogens and has two distinct arms: the innate and the acquired response.

Invading bacteria and cells infected by a virus present antigens on their surface membrane. These antigen presenting cells bind with T cell receptors on the immune cells and are recognised as both foreign and a threat. An immune response is triggered to isolate and destroy infection. This involves neutrophils, T and B cells and a variety of interferons and cytokines.

Acute pancreatitis is a form of sterile inflammation. This phenomenon can be seen in other physiological conditions such as ischaemia reperfusion injuries during transplantation and several research groups are investigating the underlying mechanisms and possible preventative strategies to reduce this response. Damage to the pancreatic parenchyema as a result of the early activation of trypsinogen results in cellular necrosis. The direct release of intracellular molecules and the resulting stress on surrounding cells results in the local release of danger associated molecular patterns (DAMPs) and intracellular glycoproteins as self-antigens. Up-regulation of ICAMS (intracellular adhesion molecules) on neighbouring endothelial cells and the release of inflammatory cytokines (TNF- α , IFN- γ , IL-1 and IL-6) results in the recruitment of neutrophils and other leucocytes from the blood stream [7]. In the overwhelming majority of patients this allows the resolution of the cellular insult and removal of the necrotic cellular debris.

In a proportion of patients, this process becomes uncontrolled and destructive resulting in distant end organ damage. For a more detailed description of the patho-physiology of this process please see Chapter 11 (Surgical Sepsis).

The most commonly affected systems are the pulmonary, cardiac and renal systems.

MANAGEMENT:

All patients require thorough assessment. The Atlanta classification (2012) requires **two** of the following to be present to allow a diagnosis of pancreatitis:

- 1. a history consistent with acute pancreatitis;
- 2. a serum amylase\lipase level of greater than 3 times the upper limit of normal;
- 3. radiological evidence of acute pancreatitis [8].

Once a diagnosis of acute pancreatitis has been reached, all patients require to be risk stratified. Several scoring systems exist – the authors favour the Glasgow (Imrie) scoring system. A Ranson or APACE II score can also be calculated. These scores were designed to predict a severe attack and remain useful as they highlight the existence of end organ dysfunction. A single initial value of C-reactive protein (CRP) >200 also indicates a severe inflammatory response.

The overwhelming majority of patients who progress to develop MOF will have evidence of end-organ dysfunction on admission or very early on in their admission. Few patients who are admitted with no evidence of end organ dysfunction will deteriorate further. Around half of patients who ultimately succumb to acute pancreatitis will die within 7 days but a significant proportion will die within 72 hours of admission emphasising the need for prompt diagnosis and early supportive care.

Early identification of patients exhibiting signs of end organ dysfunction allows prompt relatively non-invasive organ support to be initiated.

On admission all patients should be started on high flow humidified oxygen via a non-rebreathing mask at 10 litres/minute.

Adequate venous access should be obtained – allowing both venous blood tests (including full blood count and urea and electrolytes) to be taken and intra-venous fluid resuscitation to be commenced.

An arterial blood gas should be taken to check the acid base status and oxygenation levels. A pO_2 level of less than 8kPa on room air indicates respiratory failure.

A urinary catheter should be inserted to further monitor perfusion and guide fluid resuscitation. One should aim for a urine output of greater than 0.5ml/kg/hr.

Antibiotics are not indicated unless superimposed infection is proven.

Patients should be started on adequate analgesia. An intravenous bolus of an opiate such as morphine should be given followed by a standard regimen. Some patients may benefit from Patient Controlled Analgesia.

The presence of end organ dysfunction or a high severity score indicates a severe attack and patients should be monitored closely in a high dependency unit with regular observations of their vital signs allowing a trend to be charted.

Deteriorating patients may require invasive organ support in the Intensive Care Unit.

Several studies have investigated the role for immuno-modulation in these patients but results are discouraging [9]. The mainstay of management remains supportive care.

NUTRITION:

Acute pancreatitis is a produces a catabolic state and early enteral nutrition should be instigated. Several studies have shown the benefit of the enteral route over the parenteral route and nasojejunal feeding can be safely used in these patients [10].

IMAGING:

All patients should undergo an abdominal ultrasound early on their admission to check for evidence of cholelithiasis [11]. There is no longer a role for early ERCP unless there is a clinical concern of ascending cholangitis. An active policy of early laparoscopic cholecystectomy should be pursued for patients with gallstones who present at first episode with mild to moderate pancreatitis.

In patients that fail to settle after 7 - 10 days, a CT abdomen and pelvis with intravenous contrast should be performed to look for pancreatic necrosis and intra-abdominal collections. The management of pancreatic necrosis and the associated collection is a rapidly evolving field and often requires specialist and radiological support.

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12B. SURGICAL INFECTIONS [1,2]

STUART C WHITELAW

INTRODUCTION

Bacteria are unicellular organisms which have the ability to synthesise their own proteins and nucleic acids. They reproduce autonomously but do not possess a nucleus like mammalian cells, and all their DNA is packaged within a single chromosome. The outer membrane component of Gram-positive bacteria (peptidoglycan) and the outer membrane component of Gram-negative bacteria (endotoxin) play an important role in sepsis as they initiate the septic cascade. A further property of bacteria is their prolific division rate. Bacteria frequently undergo mutation to gain a selective growth advantage over their competitors and this can lead to the acquisition of genes encoding antibiotic resistance.

There are literally thousands of species of bacteria, but four main groups account for the majority of sepsis seen in everyday surgical practice. These are *Staphylococcus*, *Streptococcus*, gram negative intestinal bacilli (coliforms) and anaerobes.

STAPHYLOCOCCUS SPECIES

Staphylococcus species are gram-positive cocci (round organisms) that commonly colonise the nostrils and skin. Under the microscope they form grape like clusters and this is because the bacterium divides along two axes during reproduction. Traditionally, staphylococcus species have been divided into two groups according to their ability to produce coagulase, an enzyme that converts fibrinogen to fibrin in human and rabbit plasma, causing it to clot. *Staphylococcus aureus* is coagulase positive and most other staphylococcus species are coagulase negative. The microbiology laboratory will use slide coagulase, tube coagulase, and DNase production tests to confirm *S. aureus* identification.

Although Staphylococcus species are principally aerobic organisms and can grow on virtually any culture medium, they are able to tolerate an anaerobic atmosphere (i.e. they are facultative anaerobes). On blood agar, shiny convex colonies approximately 3mm in diameter will appear within 24 hours when Staphylococcus species are present. *S. aureus* colonies are golden yellow in colour and *S. epidermidis* is creamy/white. The genus has over 40 species, but only a few are pathogenic in man, and their pathogenicity is due to invasive virulence characteristics and toxin production. All Staphylococcus species produce the enzyme catalase, which converts hydrogen peroxide to water and oxygen, and the catalase test helps distinguish staphylococci from other common bacteria such as Streptococcus species and coliforms. It is sometimes forgotten that staphylococci are "hardy" bacteria and can survive on floor and ward surfaces, and in dust for long periods of time. However, they can be destroyed by alcohol

hand gel, and good hand hygiene remains a most effective weapon against staphylococcal infection.

S. aureus is the main cause of staphylococcal infection and it mainly affects the skin (abscesses, boils, carbuncles, eyelid stye), nails (paronychia), wounds and intravenous cannulae. *S. epidermidis*, our most common skin commensal, is significantly less pathogenic, but it can cause sepsis in those who are immunocompromised, have central venous cannulae or implanted prosthetic material. Staphylococci are particularly dangerous when they gain access to the bloodstream as Staphylococcus septicaemia has the potential to cause pneumonia, osteomyelitis, septic arthritis, infective endocarditis and "metastatic" abscesses. They can also rarely produce toxins that cause Toxic Shock Syndrome. Most Staphylococcus species produce an enzyme called **penicillinase** and this is why flucloxacillin (a penicillinase-resistant penicillin) is the mainstay of treatment of infections with *S. aureus*, which are not caused by MRSA (meticillin resistant *S. aureus*).

MRSA is any strain of *S. aureus* that, through the process of natural selection, has developed resistance to beta-lactam antibiotics. These include all penicillins (including meticillin) and cephalosporins. The evolution of such resistance does not cause the organism to be intrinsically more virulent, but resistance makes MRSA infection more difficult to eradicate with standard antibiotics, and thus more dangerous. MRSA can be identified using selective culture media or molecular diagnostics. Patient screening upon hospital admission prevents the co-habitation of MRSA carriers and non carriers and has been widely adopted in the UK and other European countries.

STAPHYLOCOCCAL SKIN INFECTIONS

The first stage in the development of any abscess involves a circumscribed necrosis of tissue at the site of bacterial entry, rapidly followed by a diffuse infiltration of polymorphs and an inflammatory exudate. The S. aureus subsequently kill many of these migrating polymorphs, and the combination of dead polymorphs, bacteria, liquefying necrotic tissue, lipids and nucleic acids is known as pus. A localised collection of pus is known as an abscess and pus from an abscess will track along a path of least resistance until the first free surface is reached. At this point the abscess will usually either discharge spontaneously or get drained surgically. If this does not happen, and infection persists, a state of chronicity develops, in which an inflammatory response and attempts at healing will proceed at approximately the same time. A thick wall of fibrous tissue will encase the abscess and pus will either discharge externally through a sinus or a hollow viscus, or become completely sequestered in the tissues, causing chronic ill health.

It is important to note that in patients on chemotherapy a leucocytosis may not occur, and consequently pus may only appear as the bone marrow recovers from chemotherapeutic suppression.

In diabetic patients deep tracking sepsis of the sole of the foot may be much more extensive than is superficially apparent. The feet of such patients should be X-rayed to identify osteomyelitis, and treatment may require extensive drainage, debridement,

amputation of one or more toes, or forefoot amputation to bring the sepsis under control. In very severe cases, below- knee amputation may even be indicated.

CARBUNCLE

A carbuncle is a complex, loculated and ill defined abscess involving subcutaneous fat that discharges on to the skin through multiple sinuses. It usually commences as a boil which spreads causing the subcutaneous tissue to become honeycombed by small abscesses separated by trabeculae of fibrous tissue. The affected skin is swollen, brawny and painful. Carbuncles usually affect the neck or shoulders and are more common in diabetic patients.

TREATMENT

The treatment of all skin abscesses is surgical drainage. Antibiotics may be also be required if there is co-existing cellulitis, but antibiotics alone will rarely permanently eradicate the sepsis. Following incision, a sample of pus should be taken for culture and antibiotic sensitivity. All granulation tissue and fibrin should be removed and the wound irrigated with a hydrogen peroxide solution. Thereafter, the wound should be packed with an alginate or iodine soaked gauze pack, and the pack replaced daily, until the cavity is small and healing by secondary intention is almost complete. There is limited evidence that incision, drainage and primary suture may produce a satisfactory healing in some cases, without increased risk of recurrent sepsis, but this is potentially risky, especially in a tropical environment, and incision and packing is a "tried & tested" reliable form of treatment. Very occasionally an "old –fashioned" magnesium sulphate poultice may be sufficient to encourage spontaneous discharge.

IMPETIGO

Facial sepsis, e.g. impetigo, can be particularly dangerous. The facial vein communicates with the cavernous venous sinus via the ophthalmic vein and the pterygoid venous plexus, and this provides a route for sepsis to spread from the face to the brain causing a potentially fatal cavernous venous thrombosis. For this reason patients should be discouraged from "squeezing" septic pustules and antibiotics prescribed at earliest opportunity.

TOXIC SHOCK SYNDROME

This is a serious and life-threatening condition caused by infection with a specific toxin producingS. aureus. The toxin in question is known as TSST1, and it acts as a "super-antigen", triggering significant T-lymphocyte helper cell activation and massive cytokine release. A potential source of TSS is a neglected intra-vaginal tampon but it occurs most often as wound associated TSS.

Toxic shock syndrome has an abrupt onset with high fever, headache, sore throat, myalgia, vomiting and the development of a generalised blanching erythematous rash. It can rapidly progress to multi-organ failure in a matter of hours.

Treatment consists of aggressive fluid resuscitation, and administration of intravenous flucloxacillin or vancomycin plus clindamycin. The tampon should of course be removed. Severe cases may require treatment with immunoglobulin.

STREPTOCOCCAL SPECIES

Streptococci are Gram-positive spherical organisms which are usually found as commensals in the gut (Enterococcus) and nasopharynx. Under the microscope they appear as chains, but this is only conspicuous when the organisms are cultured in fluid media. The vast majority of these bacteria are aerobes or facultative anaerobes but a few strains are obligatory anaerobes, meaning they will only thrive in an anaerobic environment. Most streptococci are oxidase and catalase negative. There are over 50 species in this genus and they can be classified according to the haemolysis they produce on blood agar and serotyping.

BETA HAEMOLYTIC STREPTOCOCCI

STREPTOCOCCUS PYOGENES (LANCEFIELD GROUP A)

The typical lesion produced by this bacterium is a spreading cellulitis affecting the dermis and subcutaneous fat. The spreading nature of streptococcal infection has been attributed to the production of the enzymes **streptokinase** which dissolve fibrin, and **hyaluronidase** which liquefies the mucinous ground substance of connective tissue. Abscesses occur much later than in staphylococcal infections and the pus is watery, pinkish or blood-stained and usually odourless.

<u>Cellulitis</u> is characterised by the presence of a painful, tender, red swollen area which increases in size over a few days, and spreads by lymphatics with the production of red linear streaks of lymphangitis and lymphadenitis. This may be followed by septicaemia. The patient feels generally unwell with nausea and a headache. The legs and face are the most common sites of infection although any part of the body may be affected. Predisposing factors include an insect or animal bite, obesity, lymphoedema and intravenous drug abuse.

<u>Erysipelas</u> begins as a rapidly spreading, erythematous, oedematous cutaneous infection, usually involving the face. In contrast to cellulitis, the involved area usually has a sharp, well demarcated border, but gross areas of suppuration are uncommon.

Strep pyogenes (sometimes called "Group A Strep") is also a common cause of tonsillitis, pharyngitis, otitis media and sinusitis. It is also the organism responsible for scarlet fever, and can lead to life threatening infections of the floor of the mouth (Ludwig's angina) and retropharyngeal space. Historically, streptococcal puerperal infection killed thousands of women before the introduction of sulphonamides, but today is very uncommon.

Strep pyogenes infections may be followed by acute rheumatic fever leading to chronic heart valve damage, acute glomerulonephritis and erythema nodosum.

STREPTOCOCCUS LANCEFIELD GROUP B (STREP. AGALACTICAE)

Strep. agalacticae is a common member of the vaginal flora and may cause endometritis, urinary sepsis and bacteraemia. It is a leading cause of neonatal sepsis (pneumonia and meningitis) and may cause sepsis in immunosuppressed patients suffering from diabetes..

ALPHA HAEMOLYTIC STREPTOCOCCI

Strep viridians is not a single defined species but is the collective term given to alpha haemolytic streptococci. They are a normal commensal of the mouth and throat and an important cause of dental caries and infective endocarditis.

NON HAEMOLYTIC STREPTOCOCCI

From a conceptual point of view, these bacteria can be considered under the anaerobe category (see below).

STREP PNEUMONIAE

Strep. pneumoniae, also known as the "**pneumococcus**", is an aerobic gram positive, non-motile, non-sporing organism. Morphologically the bacterium has an oval shape and it is arranged in pairs with the long axis in line with each other. On blood agar culture medium the colonies have a greenish pigmentation and superficially resemble chequers used in a game of draughts. Unlike other streptococci, the pneumococcus is lysed by bile salts very rapidly. Perhaps the most important feature of the pneumococcus is its capsule. Sub classification and type specificity depends on the polysaccharide component within the capsule and it is this substance that is related to its virulence.

The most important pneumococcal infection is classical lobar pneumonia. Other conditions include otitis media, suppurative sinusitis (both of which can lead to meningitis), purulent conjunctivitis and primary peritonitis in cirrhotic patients who have ascites.

A polyvalent pneumococcal vaccine derived from the capsules of the most prevalent types (Pneumovax®) is available for treating those at increase risk of pneumococcal sepsis (post-splenectomy, sickle cell disease, nephrotic syndrome, and children under 5 years).

GRAM NEGATIVE INTESTINAL BACILLI

The nomenclature of these bacteria is constantly under review, but the majority belong to the family Enterobacteriaceae. This family in turn can be subclassified into the genera including Escherichia, Klebsiella, Enterobacter, Proteus, Yersinia, Salmonella and Shigella. Gram negative aerobic organisms commonly found in the lower gastrointestinal tract are commonly referred to as "coliforms". The Pseudomonas genus does not belong to the family Enterobacteriaceae, but is an important group in its own right, with *Pseudomonas aeruginosa* probably being the best known example.

The Enterobacteriaceae and Pseudomonas bacteria are gram negative non-spore forming bacilli that grow well at normal body temperature in an aerobic and facultative anaerobic environment. With the exception of Klebsiella (which also differs by having a capsule), they are all very motile.

On blood agar, the Enterobacteriaceae tend to from grey, shiny colonies. On nutrient agar, Pseudomonas tends to produce bluish- green colonies due to the production of the pigments fluoroscin and pyocyanin. Pseudomonas also has a characteristic foul smelling odour.

The classical test for *E. coli* is the lactose fermentation test performed on McConkey's selective medium. *E coli* colonies have a pink appearance while those of Salmonella, Shigella, Proteus and Pseudomonas remain colourless.

E.coli is always present in the bowel, Proteus is present in the bowel of a third of patients and about 10% of patients harbour Pseudomonas in their faeces.

The pathogenicity of the gram negative intestinal bacteria is due to the presence of endotoxin in their cell membranes which has the ability to trigger the cascade responsible for the systemic inflammatory syndrome (SIRS), which can culminate in septic shock and multi-organ failure. They can also readily invade and multiply in the blood stream. Some Enterobacteria also produce exotoxins, with perhaps the best example being *E. coli 0157* which can produce life-threatening enterocolitis and renal failure due to a verotoxin.

E.coli and other coliforms are the main organisms responsible for acute appendicitis or abscess, acute diverticulitis and its sequelae, and generalise peritonitis due to the perforation of a hollow viscus. It is also commonly cultured in cases of acute cholecystitis (presumably bacteria reach gallbladder via the portal vein), and is a common cause of urinary tract sepsis. These bacteria are also common causes of nosocomial pneumonia on the Intensive Care Unit.

Proteus organisms are a common cause of urinary sepsis. They break down urea to ammonia and this renders the urine alkaline, favouring the formation of phosphate calculi in the bladder. They also commonly cause wound infection and can infect chronic leg ulcers.

Pseudomonas aeruginosa commonly affects burns injuries and abdominal wounds and produces a foul smelling bluish-green pus. It also commonly affects the urinary tract and can cause chronic otitis media and otitis externa.

The Klebsiella group share the general pathogencity of *E. coli* and other coliforms but additionally one species, *Klebsiella pneumoniae*, can produce a particularly virulent bronchopneumonia. This is often associated with the production of red currant jelly sputum.

PERIANAL SEPSIS

Patients with an acute perianal abscess usually present 2-3 days after the onset of symptoms with pain and a palpable tender lump adjacent to the anal margin but with little in the way of systemic upset. Those with ischiorectal sepsis tend to present later with more vague tenderness and a fever, because more pus is able to accumulate in the relatively loose connective tissue of the ischiorectal fossa. In this situation the surgeon may only be elicit tenderness on palpation rather than identify a tender lump *per se*. A submucosal rectal abscess, which is relatively rare, may only be diagnosed at digital rectal examination as a tender bulge. In this situation the examination itself may rupture the abscess with immediate relief of symptoms.

As with any abscess, the most effective treatment of perianal sepsis is incision and drainage. It is the author's opinion that this should be accomplished via a cruciate incision with the removal of a small disc of skin. This will ensure adequate drainage, while the abscess cavity "fills" with granulation tissue prior to re-epithelialisation. If a simple linear incision is employed instead, there is an increased risk that the skin edges will stick together and heal over a cavity containing live bacteria, with recurrence of sepsis. In the case of an ischiorectal abscess, exploratory needle aspiration under anaesthetic may confirm the diagnosis. Perianal sepsis is commonly associated with a fistula-in-ano, and if identified at the initial drainage operation, it is some surgeons practice to perform a primary fistulotomy. This reduces the likelihood of recurrent sepsis but carries an increased risk of damaging the sphincter complex and rendering the patient incontinent. It is therefore the author's practice to simply drain and pack the abscess cavity in the first instance and evaluate any co-existing fistula at a later date. If the culture of pus grows skin related bacteria, it is highly unlikely that the patient has a fistula. If the culture grows coliform bacteria it is highly probable that the patient has a fistula.

Finally, it should be borne in mind that acute perianal sepsis may be the presenting feature of Crohn's disease, diabetes or less commonly a locally advanced carcinoma of the anal canal or lower third of the rectum. It can also be associated with syphilis infection and compromised immunity.

ANAEROBES

Anaerobic infections are caused by three main groups of organisms: Clostridia, Bacteroides and non-haemolytic streptococci.

CLOSTRIDIA

Clostridia are gram positive, spore forming anaerobic bacilli, which are responsible for gas gangrene, tetanus, botulism and pseudomembranous colitis. Gas gangrene is caused by a mixture of *C. perfringens, C. novyi, C. septicum, C. sporogenes*, and *C. histolyticum*. Tetanus is caused by a single species *C. tetani*. Botulism is a rare and potentially fatal type of food poisoning caused by *C. botulinum* toxin and Pseudomembranous Colitis is caused by *C. difficile*.

The most characteristic feature of Clostridia bacteria is the spore, which is produced by the bacterium in response to adverse environmental conditions. Clostridia are frequently found in the intestines of humans and animals and as they are excreted in faeces, the spores become widely dispersed in soil. It is this ability to from spores that renders them particularly resistant to heat and desiccation.

All Clostridia are highly motile and capsulate with the exception of *C. perfringens* which is non-motile and capsulate. They all grow on blood agar in an anaerobic atmosphere, and the classical microbiological test for diagnosing C. perfringens is Nagler's reaction, although they are now identified by rapid biochemical profile tests.

Biochemically Clostridia can be subdivided into Saccharolytic and Proteolytic groups.

The Saccharolytic groups ferment lactose and glues producing lactic acid, hydrogen and carbon dioxide.

The Proteolytic group breakdown producing foul- smelling gases like hydrogen sulphide and ammonia.

GAS GANGRENE

Gas Gangrene evolves as a series of steps.

- 1) A wound becomes contaminated with a mixture of the saccharolytic bacteria, *C. perfringens, C. septicum* and *C. novyi*.
- 2) These bacteria germinate from their spore state and release powerful exotoxins, which causes rapidly progressive muscle necrosis.
- 3) Muscle carbohydrate is then fermented by these bacteria releasing lactic acid, hydrogen and carbon dioxide. This is the origin of the gas in gas gangrene and at this point of the illness the wound is odourless.
- 4) The Clostridia also release hyaluronidase and collagenase which breaks down the connective tissue of the muscle, leading to more necrosis.
- 5) The capillaries supplying the muscle become "leaky", and this leads to a compartment syndrome, rendering the muscle ischaemic which favours spread and growth of the clostridia. The site of infection becomes tense, oedematous and crepitant.
- 6) This stage is rapidly followed by putrefaction as necrotic tissue is destroyed by the proteolytic Clostridia also present in the original contaminant. These bacteria thrive on and decompose dead muscle which becomes greenish-black in colour. Hydrogen sulphide and ammonia is released at this stage and it is this that gives the muscle its foul-smelling odour.
- 7) The whole process is accompanied by the features of systemic sepsis and haemolysis and untreated multi-organ failure and death is inevitable.

The key to preventing gas gangrene is thorough debridement of ALL contaminated wounds. If there is doubt about residual contamination following debridement, the safest policy is always to leave a wound open. The wound can then be treated at a later date by delayed primary closure, grafting, a flap or just by leaving it to heal by secondary intention. Failing this, the key to improving survival from gas gangrene is early

recognition and aggressive surgical and high dose antibiotic therapy. Intravenous penicillin should be administered, high flow oxygen provided and fluid resuscitation commenced without delay. From an operative point of view, all dead tissue should be excised until only healthy, bleeding, well oxygenated muscle remains and the wounds adequately dressed. The patient should then be transferred to ITU for full supportive care. Further debridement may be required and the wounds should be inspected on a regular basis.

PSEUDOMEMBRANOUS COLITIS

This condition is caused by *C. difficile* and usually presents with watery diarrhoea, severe colitis, toxic megacolon, and/or systemic sepsis. It can occur after even one dose of almost any antibiotics, although the cephalosporin group, clindamycin, ciprofloxacin and co-amoxiclav are those most commonly implicated. Diagnosis depends on clinical awareness, high index of suspicion, the identification of C. difficile toxin in the stool. Treatment consists of fluid resuscitation and the administration of Metronidazole (oral or IV) or Vancomycin (oral only), and close surveillance to check that the patient does not develop toxic megacolon, diagnosable on a plain supine abdominal X ray. If the a patient develops severe colitis that does not respond to five days conservative management, or if the patient develops toxic megacolon, total colectomy with formation of an end ileostomy should be performed earlier rather than later, as delayed surgery is associated with a high mortality.

NECROTISING FASCIITIS (NF)

Necrotising fasciitis is a rapidly evolving severe skin and soft tissue infection characterised by spreading erythema, oedema, bullae and necrosis. Type 1 infection is caused by a mixed infection with gram negative bacteria and many types of anaerobes and is usually seen in post-operative, diabetic or immunocompromised patients, while Type 2 is usually due to infection of an insignificant skin breach with Group A or other streptococci. In contrast to cellulitis, the skin signs are often minimal relative to the intensity of the patient's pain. Although the diagnosis may be made by imaging, its rapid spread means that surgical inspection of involved muscle groups is urgently required to facilitate treatment and limit tissue damage. The patient should be fully resuscitated with intravenous fluids, oxygen and broad spectrum high dose antibiotics, the boundaries of the erythema marked with a pen, catheterised and taken to theatre without delay. In theatre, all dead tissue should be removed and the wounds dressed. Regular inspection of the wound should be performed as further debridement is almost always necessary. When NF involves the scrotum and perineum, it is sometimes known as Fournier's gangrene.

WOUND SEPSIS

Wound infection can be caused by any organism that contaminates a wound either from the skin (e.g. *Staph aureus*) or from the gastro-intestinal (GI) tract (e.g. *E. coli*) or any infected organ (e.g. urinary tract, liver abscess). It remains the most common

complication after surgery. Most patients have longer hospital stay but are not in danger, although wound infection can lead to the serious complications of necrotising fasciitis or wound dehiscence. The incidence depends on whether the surgical wound would be classified as **clean** e.g. non-traumatic wound that does not enter a hollow viscus such as GI or urinary tract (infection rate <1%), **clean-contaminated** e.g. where the respiratory, GI or urinary tract is entered but with minimum spillage (infection rate 1-2%), or **contaminated** e.g. traumatic wound from a dirty source, gross spillage of GI content or infected bile or urine, or a serious break in aseptic technique (infection rate 10% and more) [3].

The risk of wound infection can be decreased by [3]:

1. Pre-operative screening for high risk bacteria e.g. MRSA (now routine practice in the UK) and identification of high-risk patients (e.g. diabetics, on steroids, post-splenectomy, advanced cancer or congenital or acquired immuno-suppressive disease).

2. Proper aseptic technique:

- a. Proper hand washing or decontamination technique.
- b. Protective equipment (gloves, gown, mask, visors or glasses).
- c. Skin preparation (limit shaving to minimum area; shave in theatre immediately pre-operatively; antiseptic skin cleaning before draping).
- d. Sterile instruments: all instruments should go through a process of decontamination, physical cleaning, disinfection and sterilization.

3. Antibiotic prophylaxis: this should occur early enough to achieve high tissue concentrations before the skin incision (30-60 minutes pre-incision as per WHO Safe Surgery Checklist), must cover expected pathogens that could affect that operative site but not increase the risk of resistance or hospital-acquired infections (e.g. *Clostridium difficile*) significantly. It usually consists of a single dose but a second dose should be considered if the procedure takes very long or has high blood loss with replacement transfusion.

4. Safe surgical technique with gentle handling of tissues, sharp dissection, not leaving devascularised or crushed tissue behind, and short cut ends of ties and sutures.

Post-operatively the wound should be inspected regularly and the wound edges palpated for unusual tenderness. Local signs of wound infection include erythema, tenderness, swelling, cellulitis, a sero-sanguinousor purulent discharge [4]. It classically occurs 5-7 days post-operatively, but much sooner in the tropics. If there are signs of wound infection some skin sutures or clips should be removed for proper drainage, and cleaned regularly with saline; the wound can heal by secondary intention. If there is a possibility of necrotising fasciitis the wound must be widely opened and debrided early under general anaesthesia. Antibiotics is not necessary for localised infection but must be given for cellulitis, and for systemic signs of infection such as pyrexia or elevated neutrophils. If there are signs of systemic sepsis the patient is treated aggressively according to the principles of sepsis management [Chapter 11].

INTRA-ABDOMINAL SEPSIS

This remains the second most common cause of death in the intensive care unit. Trauma aside, intra-abdominal sepsis is usually due to inflammation of an organ (e.g. acute cholecystitis, appendicitis, diverticulitis), perforation of an organ (e.g. perforated duodenal ulcer or sigmoid diverticulum) or ischaemia (e.g. due to a strangulated bowel loop due to volvulus, internal hernia or adhesions; or mesenteric thrombosis or embolism). Irrespective of the cause there are some basic surgical principles that can be applied to most cases of intra-abdominal sepsis:

- Type of incision: if the source of sepsis is unknown, a midline vertical incision is incision of choice. This allows full exploration of the abdomen and effective saline lavage of entire peritoneal cavity, reducing the chances of subphrenic or pelvic abscess formation.
- Source Control: it is important to identify and gain control of source of sepsis at the earliest stage possible. This will help reduce further contamination while a preliminary peritoneal toilet is performed.
- 3) Post-bowel resection decision: In cases where resection has been performed the decision has to be taken as to whether to anastomose the bowel, exteriorise the bowel or plan a second look laporotomy. This decision is subjective depending on haemodynamic stability of patient, general health and co-morbidity of patient, degree of contamination, site of resection and surgeon's experience. The golden rule is when in doubt, do not anastomose.
- 4) Thorough peritoneal lavage: this will dilute contaminating bacterial count and hopefully tip the balance in favour of peritoneal macrophages and other natural defences with respect to killing bacteria.
- 5) Drain usage: there is limited evidence to support the use of drains, but it would seem intuitively sensible to drain infected fluid from the peritoneal cavity when there is a risk of a localised collection that could lead to an abscess. It is the author' preference to use a tube drain, which used to be from Latex but now more often is made from silastic.
- 6) Closure of abdomen: if the abdominal wound closes without tension the author believes that this should be attempted. If bowel distension or oedematous tissue makes this difficult, or if the patient has potential to develop abdominal compartment syndrome then consideration should be given to leaving the wound open, packing or treating it with some form of vacuum assisted drainage. There are several commercially available vacuum assisted drainage kits on the market, but an improvised kit can be assembled from basic equipment which can be used to equivalent effect. Similarly, if the surgeon is contemplating a second look or more laparotomies, it may be preferable to leave the midline wound open. Delayed primary closure rather than prolonged assisted open dressing is usually possible in these circumstances.

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12C. STOMA COMPLICATIONS

HAYTHAM ABUDEEB, ALISTAIR BROWN, HAZEL KEARNEY

INTRODUCTION

Stoma formation is an essential part of gastrointestinal surgery which is used for decompression, lavage, diversion or exteriorisation. Although significant advances have been made to stoma formation surgery, unfortunately almost 25 % of patients with either ileostomy or colostomy will have stoma related complications [1]. Stoma complications can be minor or they can have major impact on quality of life and even trigger Systemic Inflammatory Response Syndrome (SIRS) or Multiple Organ Failure (MOF). Some are immediate complications and others can present even years after initial surgery. The most common immediate post-operative complications of stoma formation, electrolyte and metabolic derangements, bleeding, ischaemia and necrosis and stoma retraction. Delayed complications include parastomal hernia, prolapse, stenosis, obstruction and ulceration. Ileostomy and emergency stoma formation are especially associated with high complication rates [2].

SKIN IRRITATION

This is common complication mainly associated with ileostomies, hence spouting of ileostomies is required. Irritation can be a minor complication that can be treated by stoma care nurses and the use of skin protector products and appliances, but can be costly and time consuming. It can indicate more serious complications are to follow, such as mucocutaneous separation or stoma retraction and eventually infection and abscess formation. Mucocutaneous separation is identified as the detachment of the stoma from the skin edges leading to separation and formation of usually superficial cavities within the subcutaneous tissues which can be a focus to wound infection and abscess formation. It is more common with patients who are malnourished, diabetic or on steroids. It can be treated conservatively with packing of the wounds and use of certain skin agents, such as Orahesive paste to fill the cavity and prevent further faecal contamination of the area, but if the cause of the separation is tension on the stoma this can progress to stoma retraction, leading to spillage of faecal material into the depth of the abdominal wall and more serious infection which will require surgical re-fashioning of stoma as these infections can be difficult to treat. Stoma retraction, on the other hand, can be caused by mucocutaneous separation which is more common in obese patients with a thick abdominal wall or short mesentery and inadequate stoma length which can lead to stoma ischaemia and necrosis. A convexity stoma product can help to prevent seepage under the flange.

STOMA NECROSIS

This results from impaired blood supply to the stoma which can be evident within 24-48 hours of surgery. Blood supply to the stoma can be compromised by local or systemic causes. Local factors include constricting sutures, inadequate stoma wound formation

with abdominal wall constriction, mesenteric tension or inadequate mesenteric trimming during surgery. Systemic factors are mainly causes of global mesenteric ischaemia caused by atherosclerosis or emboli of mesenteric vessels. Regardless of the cause, ischaemia and necrosis of stoma is a major complication as it could lead to systemic inflammatory response and sepsis by translocation of toxins and eventually could lead to multiple organ failure. The degree of vascular compromise will dictate management. Conservative treatment of watchful waiting can be attempted in superficial ischaemia of the mucosa only; this will usually slough off or can be removed with debridement. It can lead to mucocutaneous separation or stenosis. An ischaemic stoma could be the tip of an iceberg of a concealed long ischaemic bowel segment which would require prompt treatment with bowel resection and refashioning of the stoma. A simple way to distinguish between superficial and deep ischaemia is to inspect the stoma with a proctoscope; if pink mucosa is visible within 1-2 cm the ischaemia is usually limited to superficial mucosa and will slough off, with no systemic signs of sepsis and satisfactory stoma function.

ELECTROLYTE AND METABOLIC DERANGEMENT

These are associated particularly with proximal stomas such as jejunostomy or high ileostomy. Increased stoma output has a great impact on patient electrolyte and fluid balance as some stomas can produce over 2000 ml/day causing dehydration, hypomagnesaemia and malnutrition [3]. The most vulnerable patients affected are those who already have a reduced physiological reserve (e.g. elderly patients). Careful fluid and electrolyte maintenance is required for these patients to prevent further complications such as renal failure and cardiac arrhythmias. Nutrition is difficult because the proximal stoma creates short gut syndrome, with insufficient length to absorb enough nutrients, and they may well require parenteral nutrition which has its own set of potential problems. Support from a stoma nurse and dietician is invaluable to help manage fluid intake, types of fluid to drink and to eat small volume regular meals with nutritional supplementation. These patients should all go on a proton pump inhibitor, avoid caffeine and spicy or concentrated foods, and might even need octreotide to decrease stoma output. Over time the shortened small bowel will adapt but the challenge is to manage the patient metabolically for weeks or months. Patients might need long-term TPN, preferably at home but this is a labour-intensive nurse-led service. If the patient has sufficient bowel remaining distal to the stoma, it might be necessary to plan early reanastomosis of the gut after weeks to months of hospital-based intensive fluid therapy and parenteral nutrition; an especially difficult problem in these patients is to prevent super-infection with hospital acquired resistant bacteria such as MRSA.

BLEEDING

Bleeding from the stoma is a common occurrence due to good bowel blood supply, in fact oozing of the stoma edges is a reassuring sign to surgeons that their stoma is not compromised from a blood supply point of view, and usually this is seen as minor bleed from the edge of stoma or small haematoma which will resolve. Some patients might experience more profuse bleeding, especially if they are on anticoagulants and antiplatelets agents like aspirin, clopidogrel and warfarin. These can be treated with correction of coagulation and blood transfusion if required; sometimes an extra stitch has to be placed at the stoma-skin interface but this can usually done under local anaesthesia. Intraluminal bleeding is more challenging to manage as the source of the

bleeding cannot be seen and endoscopy might be needed with therapeutic endoscopic treatment of injection, clips or argon-beam photocoagulation. Surgical intervention with further resection may be required. Bleeding can occur late and is usually associated with granuloma tissue formation around the edge of the stoma. These appear as small growths and can bleed profusely when the patient is cleaning the stoma. Simple treatment with a silver nitrate stick will cauterise the area and smooth the growths.

INFECTION

Surgical site infection is the third most common hospital acquired infection and account of 14-16% of these infections [4]. Wound infections can be as high as 20% in elective colorectal surgery and this can be higher in emergency settings and with stoma formation. It is a potentially morbid and costly complication as even minor wound infections require courses of antibiotics, dressings, incisions and drainages or debridement. Stoma formation increases the risk of wound infection due to faecal contamination, hence the closure of abdominal wound prior to opening bowel for fixation. As mentioned, mucocutaneous separation and stoma retraction can predispose to severe infection and abscess formation. It is important to remove a new stoma's appliance daily is to check for signs of infection and that any skin changes are marked and reported. Gross faecal contamination as well as antibiotic resistance are further factors that can lead to severe sepsis or septic shock and therefore prompt treatment for even simple wound infection is required. Synergistic infections leading to conditions such as necrotising fasciitis can develop and early intervention is required if this is suspected.

LONG TERM COMPLICATIONS

These are mainly stoma prolapse, parastomal hernia and stenosis.

Stoma prolapse occurs more with looped stomas and more with colostomies, although prolapsing end-ileostomies can be difficult to manage, especially in people who are at risk of short bowel syndrome. Prolapse causes difficulty with appliance management and can lead to stoma oedema, obstruction and even ischaemia. Difficulty in treatment of stoma prolapse is the fact that although it can often be reduced successfully, most of the time it recurs and the only definitive treatment is surgical repair. In an emergency situation covering the prolapsed with sugar or dextrose soaks will help reduce the oedema present and make the stoma more manageable and allow the bowel to be reduced.

Parastomal hernia has an incidence of 20-30% [1] and again can only be treated surgical but surgical repair can be challenging as most surgical techniques for repair of parastomal hernia have a high recurrence rate of up to 50% [2]. Some new techniques such as lateral repair and laparoscopic double mesh repair have shown some promising results. For many patients the fitting of hernia support belts can be helpful, especially for manual workers.

Stomal stenosis can be caused by several factors such as excessive tension, necrosis, retraction, mucocutaneous separation and recurrence of disease. Mild stenosis can be treated conservatively with low residue diet and stool softener with increasing liquid diet, while severe stenosis would require dilatation, which the patient could be taught to do themselves. In severe stenosis surgical revision of the stoma is necessary.

Ulceration is a late complication which can be due for various reasons. Pyoderma gangrenosum is one ulcer that is distinguished by an irregular shape, with a blue tinge. These ulcers are painful to touch and patients have difficulty keeping their appliance on. Mild cases respond to topical steroids such as Locoid scalp lotion or Haelan tape, which absorb well and allow the appliance to stick. Severe cases may require systemic steroids such as prednisolone, biopsies of the area and referral to a dermatologist. Cyclosporin has also been used in severe cases, but the patients renal function needs to be closely monitored and regular follow up is paramount as monitoring of the ulcer is important. Nicorandil is a vasodilator used to control severe angina and widely used in patients with coronary heart disease but can cause peri-stomal ulceration, usually with higher doses of 40-60mg/day [5]. Treatment is the same as for pyoderma and response is slow. Consultation with a cardiologist is necessary, as stopping the Nicorandil or dose-reduction may be necessary.

SUMMARY

This chapter does not discuss all stoma complications in detail, as it is merely a summary of the most common complications. Complication of stoma formation and subsequent wound infections can have a serious implication which can result in extensive inflammatory response and lead to multiple organ failure. The knowledge of these complications and treatment could prevent such serious end results and therefore recognition of symptoms and signs and early intervention are of great importance.

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12D. MUSCULO-SKELETAL CAUSES

ANDY TANAGHO

(I) COMPARTMENT SYNDROME

INTRODUCTION

Compartment syndrome is 'a condition in which the circulation and function of tissues within a closed space are compromised by an increased pressure within that space'. The muscles and nerves of the extremity are enclosed in osteofascial compartments and are therefore susceptible to this condition. It is a surgical emergency which if not recognised and treated early can lead to ischemic contractures, neurological deficit, amputation, renal failure and even death. Compartment syndrome is most commonly seen following trauma, but may occur after ischemic reperfusion injuries, burns and positioning during surgery. Fractures of the tibial shaft and the forearm account for 58% of compartment syndromes.[1]

PATHOPHYSIOLOGY

Three theories have been proposed to explain the development of tissue ischemia:

(1) The increased compartmental pressure may lead to arterial spasm.

(2) When tissue pressure rises or arteriolar pressure drops this reduces the transmural arteriolar pressure difference to maintain patency and arterioles close.

(3) If tissue pressure rises then the veins will collapse and venous pressure will rise until it exceeds tissue pressure. This reduces the arteriovenous gradient and as a result reduces tissue blood flow.[2]

When muscles become anoxic histamine-like substances are released and these increase endothelial permeability. Transudation of plasma occurs and this increases the pressure within the compartment. It is only in the late stages of compartment syndrome that arterial flow into the compartment is compromised. Neural tissues demonstrate functional abnormalities (parasthesia and hyperesthesia) within 30 min of the onset of ischemia, and irreversible functional loss after 12 h. Muscle shows functional changes after 2–4 h and irreversible changes beginning at 4–12 h.[3]

DIAGNOSIS

CLINICAL

Clinical diagnosis is made on a combination of physical signs and symptoms. These include **pain out of proportion to the stimulus**, **pain on passive stretch of the affected muscle compartment**, altered sensation, muscle weakness and tenderness over the muscle compartment.[4] All compartments in the injured limb segment must be

individually assessed for active power and for pain on passive stretch. In the tibial segment this includes evaluating anterior, superficial posterior, deep posterior, and peroneal compartments. In the forearm segment this includes evaluating volar flexors, dorsal extensors, and the superficial group on the radial side. Careful objective recording of findings and repeated examination by the same clinician are important in patients at risk of compartment syndrome. The classical signs of compartment syndrome are pain, pallor, parasthesia, paralysis and pulselessness (The 5 p's). However by the time all these symptoms have developed (especially pulselessness) the limb will be non-viable. A patient with pain out of proportion to the injury, pain on passive stretch of the muscle, and weak active muscle contraction who has not responded promptly to division of circumferential dressings and slight elevation of the limb has a clinical diagnosis of compartment syndrome and should be treated operatively.

INTRACOMPARTMENTAL PRESSURES (ICPS)

If on clinical examination an obvious compartment syndrome is present pressure measurement is not necessary. However it can be a useful adjunct in the diagnosis of compartment syndrome in unconscious patients, in those with equivocal clinical findings, or when the diagnosis is entertained intraoperatively with an anaesthetised patient. There is inadequate perfusion when the pressure within a closed compartment rises to within 10–30mmHg of a patient's diastolic blood pressure. The diastolic pressure minus the ICP is called the delta pressure. The most commonly used delta pressure is 30mmHg or less. Kits have been developed to measure ICPs or an arterial line pressure transducer can be used however many hospitals do not have such equipment readily available. Pressure measurements are usually higher closer to the fracture, and may vary up and down with time, so clinical diagnosis remains the gold standard.[5]

TREATMENT

A high index of suspicion is required and early decompression of all at risk compartments is the treatment of choice. Removal of all dressing down to skin and slight elevation of the limb should be performed promptly if early signs of compartment syndrome are present. If signs and symptoms do not resolve promptly this should be followed by **open extensive fasciotomies** with decompression of all muscle compartments in the limb. In the tibia the four compartment fasciotomy through two incisions is preferred [6]. In the forearm a volar Henry incision allows access to the superficial and deep volar muscles as well as the radial 'mobile wad', and this may be supplemented with a dorsal incision and decompression if clinically indicated. The hand and the foot have multiple small compartments for which a variety of approaches have been described. [7]

In patients whom the diagnosis is being considered and in those in whom resuscitation is proceeding the following steps should be performed:

- (1) Ensure the patient is normotensive;
- (2) Remove any circumferential bandages;
- (3) Maintain the limb at or slightly above heart level;
- (4) Give supplemental oxygen.[6]

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(II) FAT EMBOLISM SYNDROME

INTRODUCTION

Fat Embolism Syndrome (FES) is 'a condition in which fat globules are demonstrated within the lung parenchyma or peripheral microcirculation'. It manifests clinically as acute respiratory insufficiency.[1]

CAUSES [2]

FES is most common after skeletal injury, and is most likely to occur in patients with multiple long bone and pelvic fractures. Other causes include acute pancreatitis and burns.

PATHOPHYSIOLOGY [2]

Two theories have been proposed:

- □ **Mechanical theory:** Increased intramedullary pressure after injury forces marrow into injured venous sinusoids leading to obstruction of the pulmonary and systemic vasculature.
- Biochemical theory: Hydrolysis of triglyceride emboli by pneumocyte lipase together with excessive mobilization of free fatty acids from peripheral adipose tissue by the catecholamines results in toxic pulmonary concentration of these acids. The biochemical theory helps to explain non-traumatic forms of FES.

DIAGNOSIS [3]

Various criteria were proposed by different authors such as Gurd and Wilson (**Table 1**) [4].

CLINICAL FEATURES

Classic presentation - asymptomatic interval for about 12-72 hours followed by triad:

- Pulmonary changes Earliest manifestations.
 - Dyspnoea, tachypnoea and cyanosis
 - \circ $\,$ Respiratory failure $\,$ 10% of cases $\,$
- Cerebral changes Due to cerebral edema.
 - Acute confusion, convulsions and coma
 - Dermatological changes Petechial rash due to occlusion of dermal capillaries.
 - Appears within 36 hours and disappears within a week
 - Distributed to the upper anterior portion of the body conjunctivae, chest, neck, axilla and upper arm. It is theorized to be due to fat particles floating in the aortic arch and embolizing through the carotids and subclavians

Other features:

- Retinal Signs: retinal haemorrhage, and presence of fat droplets in the vessels
- Renal Signs: transient oliguria, lipuria, and haematuria

LABORATORY STUDIES

- Thrombocytopenia, anemia and hypofibrinogenemia.
- Decreased hematocrit is attributed to intra-alveolar hemorrhage.
- Cytological examination of urine, blood, CSF and sputum may detect fat globules.
- ECG findings may show right heart strain or ischemia.

IMAGING STUDIES

- **Chest radiography**: Diffuse bilateral pulmonary infiltrates (snow storm appearance).
- Head CT: May reveal diffuse white-matter petechial hemorrhages

TREATMENT [5]

No specific drug therapy for FES is currently recommended. Treatment is essentially preventive (early stabilization of long bone fractures in adults) and supportive (cardiovascular and respiratory resuscitation). Maintenance of intravascular volume (albumin binds to fatty acids) and adequate analgesia are important.

LEGEND

 Table 1: Gurd and Wilson's diagnostic criteria for FES.

Table 1

Major criteria (one essential for diagnosis)	Petechial rash Respiratory insufficiency Cerebral involvement
Minor criteria (four essential for diagnosis)	HR >120 beat per minute Temp > 39.4°C Retinal signs - fat or petechiae Jaundice Renal signs - anuria or oliguria
Laboratory findings (one essential for diagnosis)	Thrombocytopenia Anaemia High ESR Fat macroglobulinemia

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INTRODUCTION

Rhabdomyolysis (RM) is the "dissolution of sarcolemma of muscle and the release of potentially toxic intracellular components into the systemic circulation and the attendant consequences".[1]

CAUSES

A prerequisite for the development of this disease process is muscle injury. There are various causes of RM: vascular interruption, ischemia-reperfusion, crush injury (crush syndrome), improper patient positioning, seizures, extreme exercise, electrical injury and infection.[2]

PATHOPHYSIOLOGY [1,2]

As the ischemic time lengthens irreversible muscle damage occurs allowing the release of toxic metabolic by-products:

- Cell membranes are damaged leading to leakage of its contents (e.g. potassium, myoglobin, and hydrogen), depletion of intracellular ATP (due to oxidative phosphorylation malfunction) and vulnerability to oxygen free radicles.
- Intracellular hypocalcaemia (due to Ca⁺⁺-ATPase malfunction) leads to the activation of intracellular autolytic enzymes (proteases and lipases).
- Release of myoglobin (MG) leads to myoglobinemia. MG contains iron which subsequently becomes an electron donor leading to the formation of *free radicals*. MG also has the potential to release *vasoactive agents* such as platelet activating factor and endothelins that may lead to renal arteriolar vasoconstriction, thus worsening renal function. A high concentration of MG in the renal tubules leads to the formation of tubular casts and resultant tubular obstruction and myoglobinuric Acute Renal Failure (ARF). The incidence of ARF in RM is 10-30%.
- Reperfusion-induced injury: Reestablishment of blood flow after prolonged ischemia aggravates the tissue damage, either by causing additional injury (mediated by oxygen free radicles, leukocytes, leukotrienes and inflammatory mediators) or by unmasking injury sustained during the ischemic period (influx of MB, potassium and phosphorus into the circulation).

DIAGNOSIS [3]

CLINICAL FEATURES:

- A high index of suspicion is necessary to allow prompt recognition and treatment to avoid the development of ARF and need for hemodialysis.
- Patients present with signs of the underlying cause, muscle pain and shock. With worsening renal function patients develop oliguria and classic "tea colored urine".

LABORATORY STUDIES:

- Elevation of serum CPK (its level has been seen to correlate with the development of ARF): Creatine phosphate (CP) is found in striated muscle. CPK catalyzes the regeneration of ATP from the combination of CP with ADP. In RM, muscle cells die and release this enzyme into the bloodstream.
- Urine is found to be dipstick "positive" for blood despite the absence of erythrocytes on microscopic examination due to myoglobinuria.
- Increasing blood urea nitrogen (BUN) and creatinine,
- Other findings include: hypocalcaemia, hyperkalemia (potential for cardiac toxicity), hyperuricemia, hyperphosphatemia, lactic acidosis, and disseminated intravascular coagulation (DIC) from thromboplastin release.

TREATMENT [4]

- The cornerstone of treatment is aggressive volume resuscitation (maintain a urinary output of >100 mL/hour) and correction of electrolyte imbalance (hyperkalemia, hypocalcaemia and acidosis).
- Bicarbonate use increases MG solubility and induces solute dieresis.
- Mannitol is an *osmotic diuretic*. It is a *volume expander*, *reduces blood viscosity*, and acts as a *renal vasodilator*. Perhaps more importantly, it has been found to be an *oxygen free radical scavenger*.
- Another key element in the treatment and prevention of renal failure is the avoidance of other iatrogenic renal insults such as the use of nephrotoxic antibiotics, IV contrast medium, ACE inhibitors, NSAIDS and so forth.

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12E: BITES, STINGS AND OTHER INJURIES BY ANIMALS

12E(IA). SNAKEBITE SYNDROMES AND MANAGEMENT

STEPHAN B DREYER, JACOB S DREYER

INTRODUCTION

Snake bite is a significant public health problem in rural areas of many parts of the world [1]. Venomous snakes are found worldwide, except for a few islands and the frozen environments. Snake bite most commonly affects those living in the tropical and sub-tropical areas of Africa, Asia, the Americas and Oceania. The morbidity and mortality resulting from bites are significant. Huge variation in management, coupled with many patients' traditional cultural beliefs and lack of resources contribute to a huge disease burden from snake bites [2]. The World Health Organisation (WHO) recently recognised snake bite as a neglected tropical disease and this has led to a global snake bite initiative to improve clinical outcome following snake bites [3].

The aim of this paper is to review current literature on the incidence, pathophysiology and management of snake bite. The aim is to help clinicians to a better understanding of the management of bites, especially when in situations with minimal resources and lack of anti-venom, which is where most snake bites occur. This review discusses a safe approach to clinical management in a field with limited evidence. A treatment checklist and guide to use of anti-venom are included to facilitate rapid decision making in stressful clinical situations.

Surgeons in rural hospitals in low and middle income countries are often involved in the management of snake bite patients due to the nature of tissue damage caused by venom or wrong primary management, or because surgeons might be amongst the more senior staff available to help manage critically ill patients in such district hospitals. Most surgeons are outside their comfort zone, however, when they have to manage a snake bite patient, and this paper attempts to provide a structured approach to management.

BURDEN OF DISEASE

Snake bite has recently been recognised by the World Health Organisation as a neglected tropical disease [1]. An exact estimation of the incidence of snake bite has not yet been achieved and remains an epidemiological challenge [2, 4]. Estimates vary greatly and no accurate morbidity and mortality data exist.

Swaroop and Grabb first attempted to quantify the global burden of snakebite but admitted that their data was flawed [5]. Their study suggested that the global annual mortality from snakebite is between 30 000 and 40 000. This was calculated mostly from hospital data and the authors recognised the gross inaccuracy from these results since most bites go unreported or take place in regions where data is not accurately collected [5].

More recent attempts to determine the annual global deaths from snake bite vary between 20 000 and 125 000 [1, 6, 7]. Estimates are that the number of bites may be around 5 million per year with more than 2.5 million envenomings [6, 7]. The highest incidences appear to be in Latin America, sub-Saharan Africa, South and South-East Asia [6, 7]. Interestingly, mortality rates were less in Latin American countries than Africa and Asia with similar incidences of bites [7]. The reason for this is unclear, but has been suggested to be due to increased availability and better developed local anti-venom, or better local guidelines on management of bites [7].

There remains very little evidence detailing the extent of morbidity, long term disability and major psychological impact from snakebite. This is of particular importance since many victims are agricultural workers and a return to work will likely provide significant psychological stress. Disability may also hamper the victim's functional ability to work.

Some studies suggest permanent disfigurement or disability in 18-19% of victims [8, 9]. This is mostly due to local tissue necrosis resulting in debridement, amputation or permanent scarring. Hypoxic brain injury secondary to neurotoxic bites or haemorrhagic complications from envenoming are also causes of long term disability [2]. Significant renal injury can lead to dependence on dialysis following envenoming and is common after bites from Russell's viper in South Asia [10]. Permanent disability and disfigurement is of particular concern to the majority of snakebite victims, since most bites occur in regions with poor access to healthcare or income support such as Sub-Saharan Africa and South East Asia [7].

PATHOPHYSIOLOGY

Bites occur most commonly on the lower extremity as a result of accidentally stepping close to the snake [11]. This is particularly so in low and middle income countries where victims use rural footpaths, often at night. In regions where it is customary to sleep on the ground or on low beds, bites occur at night as cold blooded snakes search for a warm environment. There has been growing reports of exotic venomous snake bites in the Western world due to increasing numbers being kept as pets. Here victims are often bitten on the upper extremity when attempting to handle the snake, often while intoxicated [11].

Most venomous bites occur from species with anteriorly located fangs, such as the *Viperidae* and *Elapidae* species. Envenoming from posterior fanged snakes is rare, yet can be highly dangerous, as with bites from species such as the boomslang (*Dispholidus typus*).

Snake venoms are complex collections of peptides, enzymes and other toxins that vary greatly even amongst sub-species [2, 11]. This allows the venom to induce several systemic responses in potential prey. The most clinically significant toxins are those that cause tissue necrosis and adversely affect the neurological, cardiovascular and coagulation systems [2].

Snake venoms contain multiple compounds that cause systemic effects. These vary from neurotoxic pre- and post synaptic blockers, to cytotoxic compounds such as Phospholipase A2 that cause severe local necrosis [2, 12, 13]. The toxicology of snake venom is complex and there remains great heterogeneity amongst species, making development of anti-venom difficult and challenging [14].

Probably the most common clinical effect of snake bite is tissue necrosis that can cause extensive soft tissue destruction. Envenoming by a wide range of species, particularly the *Viperidae* such as the puff-adder and rattlesnake species are responsible for tissue necrosis through cytotoxic compounds. Cell lysis, increased vascular permeability and thrombosis within the micro-circulation lead to cell death, severe local inflammation and ischaemia [2, 12]. The systemic inflammatory response syndrome is triggered to varying degree and can result in severe local and systemic sepsis. Debridement is often required [2, 11, 12]. Compartment syndrome and the requirement for fasciotomy are not as common as previously thought, and can be prevented by good medical management [15]. Snake bite induced nephropathy is a common sequel to cytotoxic envenoming leading to acute renal failure [12]. Rhabdomyolysis, cardio-vascular compromise, changes within the micro-circulation and coagulopathy all contribute to nephropathy [12]. Pathological changes that can be seen in the kidney include acute tubular necrosis, glomerulonephritis and vasculitis, producing a range of clinical manifestations [12].

Snake venom is thought to cause neurotoxicity exclusively by affecting the peripheral nervous system with almost no penetrance into the central nervous system [2, 16]. Toxicology is complex, affecting both pre- and post-synaptic receptors. The clinical effects vary greatly, with the most feared that of respiratory depression and neurogenic shock [2]. In certain species such as the black mamba (*Dendroaspis polylepis*), symptoms of neurotoxicity start with metallic taste, ptosis and gradual bulbar paralysis [2, 15]. These patients carry a high risk of death and should be treated with great urgency (see management section).

Patients with significant envenoming can have profound cardio-vascular compromise leading to a variety of clinical manifestations with multi-factorial causes. Increased vascular permeability and dilatation is thought to be implicated, and may be due to the release of cytokines such as bradykinin [2, 17]. Cardiogenic shock is seen in severe bites secondary to cardiac specific myotoxic compounds and venom induced conduction defects. This can be further complicated by ischaemia secondary to coronary artery thrombosis secondary to coagulopathy [18].

Snake bite induced coagulopathy is a complex and diverse clinical problem. It is responsible for a large proportion of snake bite mortality and can be lethal due to complex pathophysiology which is often only reversed with anti-venom [13, 15]. Venom heterogeneity results in disruption of the coagulation pathway at various stages. A range of haemostatic disturbances can be seen due to vessel damage due to cytokines and trauma, reduced coagulability, disseminated intravascular coagulation and the development of pro-thrombotic states [13]. Disintegrins, lectins and phospholipases are examples of substances that are thought to inhibit haemostasis [2, 13]. In some species snake venom contain pro-coagulant factors, such as factor V, X, XIII and pro-thrombin activators resulting in a pro-thrombotic state [2, 13]. Platelet aggregation can be either inhibited or induced depending on the venom sub-type. Laboratory results of patients are often dramatically deranged without correlating clinical manifestation [13]. It is important that snake bite coagulopathy is managed differently to the more common causes of deranged clotting, as usual treatments can be ineffective and dangerous (see management section).

MANAGEMENT

The management of venomous snake bite remains a challenge for even the experienced clinician. Lack of emergency transport and rural location of most bites result in patients often presenting late after the clinical effects of envenoming is well established [2, 19]. The cultural beliefs of many rural populations further exacerbate the problem with traditional healers often attempting to manage the bite using traditional methods [2, 8]. Poor education amongst rural populations and healthcare professionals alike result in poor first aid measures that often worsen the effects of envenoming [8, 19]. Some studies in Africa have suggested that late presentation is not associated with worse outcome [4, 8]. These conclusions can be challenged: with neurotoxic bites late presentation can result in respiratory failure and hypoxic death while haemotoxic envenoming can lead to fatal coagulopathy if untreated.

A major obstacle in snake bite treatment is the correct identification of the responsible snake. Snake bite species vary greatly from one geographic region to another, even within countries. This makes developing a national or regional treatment strategy problematic. In 40% of cases the patient does not identify the snake and mistaking for a different species is common [15, 20]. Even expert herpetologists can misidentify the snake, resulting in inappropriate treatment with anti-venom [2, 20]. Attempting to kill or capture the snake that caused the bite further endangers the individual attempting this, as well as being detrimental to the local eco-system. Capturing the snake responsible for identification should therefore be discouraged.

The difficulties facing clinicians treating snake bite is further exacerbated by the lack of availability of anti-venom and modern medical equipment. Most bites occur in the rural tropics and sub-tropics in low and middle income countries where access to health care is difficult and resources are limited [7]. Clinicians often face treating patients with advanced stages of envenoming without anti-venom. A systematic approach to managing the clinical syndromes resulting from snake bite is an effective and safe strategy for clinicians even with limited resources [15, 20].

FIRST AID

Suggestions for initial treatment of snake bite vary greatly [2, 15]. Most important are to avoid the use of a tourniquet and transport the patient to medical care as soon as possible [2, 15]. Attempts to clean or incise the wound and to suck out any venom are ineffective and should be discouraged [15]. The Sutherland technique of pressure immobilisation involves compression bandaging of the affected limb along a splinted support [21]. This has been widely taught to reduce venom transport but there is no evidence that this is indeed successful [10, 15, 22, 23]. It may be effective in treatment of bites in which the venom is mainly transported via the lymphatics [15]. Direct pressure pad application, on the other hand, has been shown to reduce venom uptake in experimental settings, although the evidence for the clinical benefit of this technique is limited [10, 23]. Educating health care professionals and first aiders in these techniques is fraud with difficulty and inaccuracy; patients are more likely to be harmed by over-tight bandaging resulting in a tourniquet effect [15, 23]. Tourniquets should be discouraged for use in immediate care except for bites with neurotoxic venom (e.g. mamba species) that are confidently identified; tourniquets should be removed within 90 minutes of application [15, 24, 25]. Tourniquet use as a first aid measure is associated with increased hospital stay and worse outcome [8]. The ischaemic effects of tourniquet use can greatly

increase the tissue damage resulting from cytotoxic envenoming which accounts for 90% of bites in Africa [15].

All patients that suffer venomous snake bite should be resuscitated as per Advanced Trauma Life Support (ATLS[®]) guidelines [26]. The most rapid threat to life is with neurotoxic bites in which respiratory depression secondary to muscle paralysis is a frequent cause of mortality [2, 15]. The airway must be secured while ensuring adequate oxygenation. Patients may become hypotensive due to direct neurotoxicity, cardiogenic shock, bleeding or sepsis. Shock must be urgently treated with IV fluid therapy with appropriate monitoring. Avoiding hypoglycaemia and hypothermia are important resuscitative adjunct measures prior to definitive treatment. All patients should receive tetanus vaccination.

SYNDROMIC MANAGEMENT

The shortage of anti-venom globally, particularly in the rural tropics, provides a major challenge to snakebite management. Management of the specific clinical syndrome caused by envenoming can be effective, whether anti-venom is available or not [15, 20]. As described previously, snake venom produces different clinical syndromes depending on the venom constituents and varies greatly [14, 15].

LOCAL NECROSIS / PAINFUL PROGRESSIVE SWELLING

Bites from *Viperidae* (e.g. puff-adder, diamondback rattlesnake) and some *Elapidae* (e.g. kraits, cape cobra) species are associated with severe cytotoxic effects [14, 15]. This is the most common presentation associated with snake bite in many parts of the world, particularly Africa. The cytotoxic effects of the venom progress rapidly and may be severe in patients presenting late [14]. Due to local tissue necrosis and the chemical nature of cytotoxic venom, the administration of anti-venom can be fairly ineffective once tissue damage has occurred [14]. Clinical management of these bites can be very effective dealt with in a systematic fashion [15].

Patients presenting with progressive tissue necrosis should be resuscitated as stated above. It is worth keeping in mind that some snakes such as the African spitting cobras can have neuro- and cytotoxic venom and progressive paralysis is a greater initial threat to life [15].

The affected limb should be elevated and patients should receive adequate analgesia. Fluid resuscitation is an important aspect of management. The cytotoxic effects of the venom can cause fluid loss and patients are at risk of acute kidney injury from processes causing myoglobinuria [14].

The affected limb should be monitored closely for tissue necrosis. If debridement is required, it is recommended that this is performed 5-7 days after the bite [15]. This allows adequate demarcation margins to develop and can avoid unnecessary returns to the operating theatre in an unstable patient. Anti-biotic therapy is only indicated if signs of sepsis are present.

Complications of cytotoxic envenoming include compartment syndrome, rhabdomyolysis, myoglobinuria and acute renal failure. Compartment syndrome is uncommon and should be managed with fasciotomy, if required on clinical grounds [15]. Femoral vessel entrapment by the inguinal ligament can occur rarely, resulting in an ischaemic lower

limb [27]. Carpal tunnel syndrome from bites to the upper limb usually recovers with elevation and analgesia [15].

Not all patients suffering from cytotoxic bites require anti-venom. The indications include compartment syndrome or serious associated complications such as coagulopathy or adult respiratory distress syndrome (Appendix 1). This is required in less than 10% of cytotoxic bites [15].

PROGRESSIVE PARALYSIS

Neurotoxic envenoming can cause rapid deterioration and death. This is commonly caused by *Elapidae* such as the black mamba in southern Africa and cobra species [14, 15, 20]. Some patients may have minor local tissue damage or they may have severe necrosis and associated coagulopathy.

In neurotoxic envenoming the application of an arterial tourniquet is indicated whilst awaiting hospital transfer, as the initial risk to life is much greater from neurotoxicity than tissue necrosis [15]. Initial management of neurotoxic envenoming is appropriate resuscitation with primary attention to Airway and Breathing. This is crucial in order to prevent respiratory failure secondary to bulbar and respiratory paralysis [15]. Patients with severe envenoming will require intubation and full respiratory support and this should not be delayed if indicated during primary survey. Muscle-relaxants should be avoided, unless absolutely required for initial intubation [15].

These patients require anti-venom in almost all cases. Lack of information regarding the snake responsible should not delay anti-venom administration if clinical signs and symptoms are highly suggestive of neurotoxicity. This includes difficulty in swallowing, peri-oral paraesthesiae, metallic taste, excessive salivation and respiratory failure. If patients are supporting their own respiratory function but a rapid onset generalised weakness occur, then anti-venom administration is required to prevent respiratory complications [15]. Early intubation should be considered as this allows respiratory support prior to inevitable respiratory failure. Unlike in cytotoxic envenoming, anti-venom is very successful in reversing synaptic neurotoxicity [14, 24]. If patients are ventilated and anti-venom administrated then recovery can be excellent, unless the venom also had significant cytotoxic or coagulopathic effects.

COAGULOPATHY

Coagulopathy can be the primary venomous effect of some bites, or in conjunction with neurotoxic or cytotoxic venom [13]. The coagulopathic effects vary greatly depending on the venom and the haematological interference it produces. It is worth remembering that even if the coagulopathic effects of venom can produce extremely abnormal laboratory results, these do not always transpire into clinical morbidity or mortality [13].

Most snake bite coagulopathy result in haemorrhagic tendency, but can rarely result in pro-thrombotic events and overall is a major source of snakebite mortality globally, causing as many as 50% of deaths [13].

As discussed previously, underlying mechanisms of coagulopathy vary greatly. Unlike other more common clinical causes of coagulopathy, those resulting from snake bite are not successfully treated using standard treatment strategies [13, 15]. The only successful treatment is administration of anti-venom [13, 15]. The indications for anti-venom include persistent bleeding from minor skin wounds, clinical evidence of intra-cranial

haemorrhage, systemic bleeding or significantly deranged laboratory measurements of coagulation [15]. Patients may require repeated administration of anti-venom depending on clinical response. Blood coagulation profiles should be rechecked six hours after administration of anti-venom and, if still abnormal, a repeat dose is indicated [2].

The clinician should keep in mind that coagulopathy is often associated with concurrent cyto- or neurotoxic envenoming. These patients should be resuscitated and managed as required for **all** the clinical sequelae of the bite.

ANTI-VENOM

Anti-venom was first developed by Calmette in the late 19th century [2, 14]. Immunoglobulins are extracted and purified, usually from animal serum after previous immunization to that specific venom. Anti-venom can be mono- or polyspecific, depending on whether it is effective against a single or multiple species' venom. Polyvalent anti-venom is usually created geographically to cater specifically for the most common bites in that particular region. The large variability in inter- and intra-species venom constitution makes development of anti-venom challenging. This is compounded by the requirement of having venom from all the particular species available to manufacturing companies. Economical and distribution difficulties result in anti-venom being unavailable to large populations that are at particular risk of snake bite.

Anti-venom reactions are common, with more than 10% of patients developing a reaction [2]. These vary from early Type I hypersensitivity reactions to late serum sickness type reactions [2]. Hypersensitivity is due to the use of animal serum and patients with previous exposure to animal serum are at particular risk [2]. The use of preadministration sensitivity testing is inaccurate, wastes time in patients that are critically ill and should therefore be avoided [2]. Anti-venom should always be administered, resources permitting, with suitable monitoring and resuscitation equipment available. Intra-muscular adrenaline is the treatment of choice in patients with immediate reactions. Corticosteroids and anti-histamines are indicated as in other causes of anaphylaxis. Patients who receive anti-venom must be monitored for at least 2 hours post-administration.

The lack of anti-venom availability and the risks of its administration must always be considered by the clinician treating a patient with snakebite. The majority of bite victims can be managed safely and successfully without anti-venom. Administration must, however, not be delayed in cases in which anti-venom is indicated (see Appendix 1). Clinicians must familiarise themselves with regional anti-venom availability and whom to contact to obtain these in case of a venomous bite.

CONCLUSIONS

Snake bite is a huge public health concern, mostly affecting those in rural areas in low and middle income countries with poor access to healthcare. This is further complicated by a lack of availability of anti-venom, and no good quality evidence base on how to manage bites most effectively. This paper helps to provide clinicians who might have to treat snake bite patients with information on the identification and management of the syndromic sequelae of snake bites, with or without the availability of anti-venom. It is essential that the evidence base for effective snake bite treatment is expanded in order to reduce the devastating public health impact of this neglected tropical disease.

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12E(IB) APPX1: INDICATIONS FOR ANTI-VENOM

Always use anti-venom with appropriate medical staff and monitoring available. Treat reactions appropriately and ensure adrenaline, corticosteroid and anti-histamine are available prior to administration

AIRWAY/BREATHING

- Swelling affecting airway
- Bulbar paralysis affecting breathing / swallowing
- Respiratory distress (ARDS) after cytotoxic bite

CIRCULATION

- All confirmed envenoming from species with haematoxic venom e.g. boomslang
- Systemic bleeding
- Signs of intra-cerebral bleeding
- Significant deranged clotting measurements eg APTT/PT, TEG
- Shock not responsive to fluid therapy
- Cardiac arrhythmias

DISABILITY

- Triad of Pins and needles, profuse sweating and excessive salivation with metallic taste [suggest severe neurotoxic envenomation]
- Evidence of severe/progressive neurotoxicity (low threshold in species known for neurotoxicity such as black mamba)
- Seizures / reduced conscious level / severe headache [suggesting intracerebral haemorrhage]
- Severe local swelling
 - More than ½ of limb within 24 hours
 - Significant swelling involving digits
 - Rapid extension within few hours
 - Compartment syndrome / vessel entrapment

REPEATING ANTI-VENOM

- Continued bleeding 1-2 hours after initiating anti-venom
- Deteriorating neurological function after 1-2hours
- Continued coagulopathy as per laboratory measurements after 6 hours

12 E(IC): APPX 2: CHECKLIST

(Use in conjunction with Appendix 1: Indications for anti-venom)

AIRWAY

AIRWAT	_				
Maintain Airway – assess for possible swelling / neurological compromise.					
Ensure adequate oxygenation					
Indication for anti-venom Yes No					
BREATHING					
 Assess respiratory rate – if signs of respiratory depression suspect neurotoxic bite [consider anti-venom] 					
Complete respiratory exam and chest x-ray if available – assess for evidence of ARDS					
 Is intubation + ventilation required? If yes, anti-venom is indicated unless strong contra-indication 					
Indication for anti-venom Yes No					
CIRCULATION	_				
 Determine heart rate / rhythm, blood pressure, signs of peripheral perfusion. Any evidence of shock? Yes → attempt to correct with fluid challenge No improvement → consider antivenom Treat cardiac arrhythmias appropriately, consider Atropine in bradycardic patients 					
 Any evidence of bleeding? Examine puncture sites Assess for systemic haemorrhage Asses for intra-cerebral haemorrhage 					
 Measure coagulation profile, full blood count, blood biochemistry: Take care in suspected cases of coagulopathy – avoid arterial puncture / central venous access if possible Severe coagulopathy → consider antivenom 					
Indication for anti-venom Yes No					

- Determine GCS / AVPU score to be repeated every 15 mins to assess deterioration in neurological state.
- Is there any evidence of significant neurotoxic envenoming?
 - Triad of pins n needles, perfuse sweating, excessive salivation with metallic taste
 - Severe/progressive neurological compromise
 - Bulbar palsy
 - If yes → anti-venom is indicated, continue with supportive care
- Is there evidence of intra-cerebral haemorrhage?
 - Convulsions
 - Severe headache
 - Depressed conscious level
 - If yes → consider anti-venom & CT head if available, continue with supportive care
- Assess limb that was bitten: Any signs of
 - Vessel entrapment (commonly at inguinal ligament)?
 - Compartment syndrome?
 - Severe/rapidly progressive swelling?
 - Swelling involving hands/digits?

Indication for anti-venom	Yes No	
	EXPOSURE	

- Remove any limb tourniquets!
- Ensure patient is warm
- Complete Secondary Survey and exclude other injuries

ATTEMPT TO MAKE SYNDROMIC DIAGNOSIS

1. PROGRESSIVE SWELLING / CYTOTOXIC ENVENOMING?

- Elevate Limb
- IV fluid therapy [use with caution carefully monitor for fluid overload and ARDS]
- Analgesia [use with caution if concurrent neurotoxicity suspected]
- Consider surgery if compartment syndrome or vessel entrapment develops [ensure no coagulopathy present prior to any surgical treatment]

2. PROGRESSIVE WEAKNESS / NEUROTOXICITY?

- Anti-venom is indicated in significant envenoming
- Ensure supportive treatment
- Intensive care unit (if available) with respiratory / haemodynamic support

3. BLEEDING / COAGULOPATHY?

- Anti-venom almost always required
- Avoid invasive procedures / monitoring
- Repeat coagulation profile every 6 hours until resolution
- Consider transfusion of blood products [if available] including use of platelets, fresh frozen plasma and clotting factors

12E(II). ARTHROPODS

NICHOLAS CY KONG, JACOB S DREYER.

Two main groups of medically important arthropods relevant to critical care will be discussed in this section, namely scorpions and spiders. A third group, Hymenoptra (bees, wasps and hornets) are covered briefly as well.

BURDEN OF DISEASE

Arthropoda form the largest phylum in the Animal Kingdom, with over 2 million species [1]. Bites from large vertebrates often cause more serious and dramatic traumatic injuries but arthropod bites and stings pose different challenges. Because of their small size arthropods depend to large extent on chemical injury, with the additional risk of being vectors for bacteria, viruses or parasites; because of large population numbers they probably inflict a significant large number of injuries [2].

The true burden of disease of arthropod bites is poorly known and there is scarce literature on its global prevalence [1, 3]. Chippaux et al reports an estimated burden of 1.25 million scorpion stings annually contributing to 0.27% of total deaths worldwide [3].

There are 30,000 species of spiders, all known to be venomous except the family *Uloboridae*. There is to date no reported global prevalence of spider bites due to many reasons, including the diagnostic challenge of identifying a spider bite [1].

Globally most arthropod envenomations are caused by species belonging to the Order Hymenoptra; these include bees, wasps and hornets [2].

PATHOPHYSIOLOGY

Arthropod envenomation by spiders, scorpions and hymenoptra result in a variety of host clinical effects and responses ranging from overt anaphylaxis requiring prompt emergency management to respiratory dysfunction and neurotoxicity to just the local inflammation at the sting site [1,2,3]. These effects depend on the incriminating species (See Table 1). The main pathophysiological pathways in arthropod envenomation are toxicological and immunological.

Arthropod/ Species	Pathophysiology	Clinical Effects/ Presentation
<u>Main Spiders</u> Widow Spiders (Latrodectus)	Venom contains α- latrotoxin which enhances neurotransmitter release from pre- synaptic terminals at neuromuscular junctions, and parasympathetic and sympathetic systems	Little local effect. May not complain of spider bite. Generalised pain. Severe Pain and Spasm of large muscle groups. Muscle twitch. Abdominal rigidity and cramps. Dyspnoea from chest wall tightness. Occasionally fasciculations. Hypertension, Diaphoresis, tachycardia, headache, nausea and vomiting. Symptoms peak in hours and resolve in 1 or two days. Death unusual.
Brown Recluse Spiders (Loxosceles)	Several enzymes present in venom, main one being sphingomyelinase D, which causes dermonecrosis and haemolysis	Necrotic Arachnidism. Local effect ranges from mild inflammation to overt necrosis with ulcer formation. Patient often unaware of incriminating bite. A blister forms which proceeds to a darkening lesion of impending necrosis with subsequent eschar formation. Challenging as other diseases result in a similar presentation: herpes zoster and vasculitis. Systemic features include headache, fever, malaise, arthralgia and a maculopapular rash. Disseminated Intravascular coagulation may develop and must be investigated for.
<u>Scorpions</u>	Neurotoxins which target ion channels and exciteable tissues	Local Cytotoxic Effects (and its spread): Local irritation which progresses to necrosing ulcer depending on venom dose. Lymphangitis may occur from local spread Systemic Neurotoxic Effects: Hyperstimulation of parasympathetic, sympathetic and peripheral nervous system: respiratory distress, bradycardia, hypertension, bronchospasm Cranial nerve manifestations: Ptosis, dysphagia. The paediatric age group develops a more severe manifestation than adults.
<u>Hymenoptra</u>	Highly allergenic peptides and enzymes	Local pain and IgE mediated Anaphylaxis and Anaphylactic Shock

Table 1: Pathophysiology and Clinical Presentation of A Few Medically Important Arthropods (1-3) (Sourced from Norris et al, Chippaux et al, Petrizevich and Diaz)

EMERGENCY AND DEFINITIVE MANAGEMENT

RECOGNITION OF THE CRITICALLY UNWELL AND STABILISATION

The first tenet in emergency management of the patient with an arthropod bite or sting is the recognition of the acutely unwell patient for whom there may be a risk of death or acute destabilisation.

This most often presents as anaphylaxis with respect to hymenoptra stings and venom induced systemic effects of neurotoxicity or cardiorespiratory distress with regards to scorpion and spider bites [1-4].

These patients need to be first stabilised and managed and observed in an intensive care setting with vital function monitoring before definitive management is sought or becomes available.

DEFINITIVE CARE

Patients who suffer from mild arthropod bites and stings can be managed by cleaning the wound, applying cold compresses and tetanus prophylaxis or update as required [1-4]. Wound analgesia is indicated. In some cases, elevation of the affected site may be considered [2]. Stingers from hymenoptra are removed [2].

A major challenge to definitive management of more unwell patients remains first recognizing the sting as that arising from an arthropod and identifying the arthropod [1, 2].

Table 2: Definitive management of severe arthropod stings (Sourced from Norris et al, Chippaux et al, and Diaz)

Arthropod/ Species	Definitive Management of More Severe Stings
<u>Main Spiders</u> Widow Spiders (Latrodectus)	Controversial. Antivenom only for serious cases due to risk of anaphylactoid reactions- may not be available in local setting. Narcotics and benzodiazepines can be used to relieve pain.
Brown Recluse Spiders (Loxosceles)	Wounds with no necrosis within 72 hours heal well with no additional therapy than local wound care. 100 mg/day Dapsone use is somewhat beneficial but is contraindicated in glucose-6-phosphate deficiency. Early surgical debridement of necrotic ulcer is disfiguring and heightens the risk of pyoderma gangrenosum. Once eschar formation is defined, surgical excision can be performed. More extensive wounds are widely excised with split-thickness skin grafting.

Scorpions	Antivenom is definitive management and effective but there may be availability issues. Supportive care of vital organ systems for systemic envenomation, with intensive care.
<u>Hymenoptra</u>	Oral prednisone (1 mg/ kg/ day) for non anaphylactic reactions. Immunotherapy for recurrent anaphylaxis available but not widely available. Patients with a known history of Hymenoptra stings anaphylactic reaction may need to carry along with them injectable epinephrine at all times.

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12E(III).INJURY BY LARGE ANIMALS

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BURDEN OF DISEASE

In general, wild animal attacks causing human injury are uncommon. Much of the evidence base regarding these attacks come from individual case studies or small case series reflecting the rarity of these events. In the Western Hemisphere infrequent incidents usually occur in zoos, circuses, or from handling of "exotic" pets. Attacks on humans in the wild remain more common in Africa, Asia and South America, where significant populations of wild animals still exist. The exact incidence of animal attacks worldwide is impossible to determine due to significant underreporting in rural populations and local variations of indigenous species. Despite this, injuries sustained through the actions of large animals can be severe and it is estimated that thousands of deaths are caused annually both on land and in water [1]. Anecdotally, in many remote areas, the incidence of attacks on humans seems to be rising. This is likely the result of shrinking wild animal habitats, human encroachment and loss of natural prey or food, forcing these animals to hunt outside their protected areas [1]. Attacks usually occur because animals either feel threatened or because victims are identified as edible prey. The severity of injury from such attacks is dependent both on the intentions of the animal and the skill of the target to avoid harm. It is difficult to determine if attacks on children are as common as on adults, but clearly children are more vulnerable and often have more severe injuries due to their inexperience, smaller size and inability to fend off predators [1].

The patterns of injury from wild animal attacks are distinct from other causes of trauma. Combinations of avulsion, laceration and sheering penetrating injuries with accompanying high impact blunt force trauma, crush and fall injuries can be identified. Large animals may strike with their head and extremities, bite with powerful jaws or crush victims with their body weight. Attacks can result in a range of major injuries to the skin, muscle, nerves, blood vessels, tendons, joints and in more severe cases cause fixed organ injury, intracranial or intraperitoneal bleeding, spinal injuries, airway compromise and death. In addition to potential disfiguring and devascularising tissue injuries, wounds contaminated with a variety of pathogens can be a major concern leading to delayed localised and life threatening systemic infections. To add extra complexity, many animal attacks occur in remote wilderness areas, hostile environments and water where there can be substantial delays in notification, rescue and access to treatment [2]. Even after a brief attack, optimum management can be extremely challenging due to multiple mechanisms of injury, the victim's physical and mental exhaustion and the lack of immediate expertise, equipment and facilities. Therefore, much higher physical and psychological complication rates can be expected after these attacks when compared to similar injuries caused by other means.

Despite differences in animal behaviour, size, primary weapons (i.e. teeth, claws, horns, strength, speed) and attack strategies, the general principles of trauma management for these patients must be adhered to. Rapid and thorough assessment of the casualty's

airway with cervical spine control, breathing and circulation, appropriate resuscitation, relocation to a suitable facility and definitive management of their injuries should always be the priority. This review explores some of the current literature on wild large animal attacks and present different mechanisms of injury from some of the wild's most potent attackers. Some knowledge of common injury patterns and possible complications may help the primary carer to identify and manage these patients quickly and effectively in time-limited challenging situations.

TRAUMATIC INJURY PATTERNS

The intentions of the animal towards the victims will dictate the ferocity and persistence of any attack. It is likely that a true predator will mount a premeditated strike to vulnerable areas of their prey seeking a swift kill, whereas those who are trying to defend themselves (their young and territory) will engage in an attack only until the perceived threat has been neutralised. In this way it is possible to predict patterns of human injury after identifying the animal species, likely behaviour and main armamentarium of the attacker. Wild animals can therefore be best categorised by their predatory nature; either as carnivores, herbivores or omnivores.

CARNIVORES

The most commonly referenced carnivores are the big cat family. This informal term refers to the five members of the Panthera family: lions, tigers, jaguars, leopards and snow leopards, although it can also be applied to most other large wild cats. Their attack strategies usually focus on targeting the head and neck of their prey, often using an element of surprise by approaching suddenly from the rear. Often a large cat will place their teeth between the prey's vertebrae into the spinal cord, shake they prey violently with its teeth tightly clenched causing cervical spine hyperflexion and extension injuries [3]. With their primary weapons, large powerful jaws and long canine teeth, they also use their sharp claws to tear through soft tissue. The result of these attacks can be fractures of the cervical spinal vertebrae, spinal cord injury, soft tissue loss and suffocation [3]. Due to their immense power, speed and ferocity the majority of large cat attacks reported in the literature are fatal. Predatory wolves also have a similar attack strategy dependent on their motivation. Many attacks from predatory cats and wolves are directed at children. The small size of children and their excited movements mimic those of small prey, stimulating animals to attack [4]. The two most dangerous wolf attack scenarios are those by rabid wolves and predatory wolf. Wolves develop the "furious" phase of rabies to a very high degree, which, coupled with their size and strength, makes rabid wolves the most dangerous of all rabid animals. During "determined" predatory attacks, the victims may be repeatedly bitten on the head and face and dragged off to be consumed away from the attack site. In India the term "child lifting" describes another strategy by predatory wolves in which they silently enter a hut targeting small children [5].

Other dangerous predators include those from the order Crocodilia which include true crocodiles, alligators, caimans and gharial. These predators have a unique method of capturing and killing their prey. They grip their victims with their massive strong jaws; drag them under water in a spinning manner (death roll) to disorientate their prey. Death

usually results from the crushing injuries, haemorrhage or as a result of drowning [2]. Other mechanisms of death from crocodile attacks include transection of the torso or decapitation. For those in deeper waters shark attacks can cause severe injury and fatalities, although these are actually very rare. The three main species involved in fatal attacks around the world are the great white shark, tiger shark and the bullshark [6]. When shark attacks occur, it is often with impressive efficiency demonstrating their extreme agility, speed and power underwater. The direct traumatic effect of a shark attack is dependent upon the severity and nature of the attack, as well as the size and species of the shark. Like predatory mammals, sharks also apply attack strategies dependent on their motivation. The tentative bites associated with exploratory 'hit and run attacks' usually occur in isolation, and on extremities. Wounds are principally incisions, with a minimal crushing component [6]. If the shark is truly exerting premeditated hunting behaviour, they may employ either "sneak attacks" or "bump and bite" strategies. With the bump and bite, sharks encircle their victims, bumping them prior to attacking [6]. Sharks have specialized scales which give their skin the texture of coarse sandpaper. When bumped at speed, these abrasions can leave long deep grooves in the skin and damage underlying tissue. In more serious concerted attacks shark bites cause substantial tissue loss and extremity amputation. The spike-like teeth of the lower jaws fix their prey while the serrated upper teeth saw through flesh. The cutting action is aided by head shaking or rolling movements by the shark. Their bites have been demonstrated to exert as much as 18 tons per square inch at the tips of the teeth, capable of biting through surfboards, small boats, torsos and limbs. Death is usually due to haemorrhagic shock and drowning, combined with lack of on-scene resuscitation [6].

HERBIVORES

Dangerous herbivores are usually large and can be aggressive especially when protecting territory and young. These can include buffalo or bison, large primates such as baboons and gorillas, rhinoceros, elephants and hippopotamus (baboons and chimpanzees are omnivores, like humans, but their attack behaviour is like those of herbivores because they do not see humans as food). Bison, bulls, rhinoceros and wild boars, being horned and tusked animals employ similar strategies of attack. Two mechanisms of injury can be observed. The first is by direct goring by the horn known as "hooking," resulting in deep puncture wounds [7]. When a buffalo or other horned animal charges, the head is lowered with the horns leading. The horns are thus at the level of a human adult's thighs, buttocks, perineum, or lower abdomen. These powerful animals charge unexpectedly and at the moment of goring impact, the animal extends its head, with the horn tracing an upward arc, tossing the victim into the air [7]. Deep penetrating punctures, evisceration and avulsion injuries from goring are common results from these attacks. The second mechanism of injury is high speed blunt force trauma occurring when victims are either butted by the animal's head directly or are the consequence of rapid deceleration after being tossed into the air. These large animals may also cause multiple injuries by trampling on their victims once on ground or crushing with the horns or powerful head against the ground. Similar injuries can occur with cattle. This is especially dangerous if caught under a stampede from a charging herd. Multiple sites and types of fractures, abrasions, and contusions may be seen. Large primates such as baboons and gorillas can weigh up to 200Kgs and have the strength of a number of adult men. They are usually peaceful vegetarians, but the adult male can attack intruders that may pose a threat to their family. Armed with enormous club-like arms, very long sharp canines, and the ability to run over twice as fast as humans, an angry gorilla is extremely dangerous. Injuries are usually caused by bites or multiple blunt injuries caused by punching, gouging, dragging and tossing of victims. Elephants again are usually placid creatures but sudden unprovoked attacks have been well described by lonely tuskers due especially to the aggressive behaviour of elephants in musth [8]. In an attempt to kill they usually grab the victim with their trunks and then attempt to crush them either with their trunks or by putting them under foot. Sometimes they have been known to throw their victims from a high lifting position to the ground followed by purposeful trampling [8]. Elephant bulls can use their tusk as lethal weapons against other elephants or large animals but rarely use them to injure humans, probably because they lack the precision needed to target vital organs. Of all the large herbivores in Africa, the hippopotamus is one of the most dangerous; it kills more people every year than lions, leopards and crocodiles [9]. Extremely territorial, the hippopotamus, particularly the male, can weigh three tons or more, and has been known to attack both in water (capsizing boats and kayaks) and on land. They can run incredibly fast despite their bulky appearance and are fearless. The hippopotamus has the largest and mightiest jaws along with the longest canines of any mammal. So powerful are its jaws that a hippopotamus has been known to have the ability to bite an adult crocodile in half [9]. Even one landed strike by a hippopotamus can be lethal.

Horses, donkeys, mules and camels can all kick and bite, but camels are able to withdraw their lips and cheeks quite far laterally which means they can bite with the side of their mouths. Because they have big crushing molars they can avulse a chunk of flesh in the process. Camel bites are more common in the rutting season. Most injuries occur to the upper limbs, often with accompanying fractures, but also to the head and face; mortality has been reported due to injuries to the spinal cord and carotid artery [10, 11].

OMNIVORES

Omnivores have a varied diet of meat and vegetation and therefore are more opportunistic rather than predatory hunters. Their intentions are usually not to attack humans as prey but if encountered are no less deadly. Bears are the largest of the omnivores. To add to their menace, they have an ability to seem even larger by standing erect on their hind legs whilst using their significant strength and clawed forepaws as attack weapons. When attacking humans, they use their superior height to great advantage knocking victims over with their paws followed by one or two bites to arms or legs, finishing with a potentially lethal snap to the head [12]. Characteristic lacerations in bear attacks are usually inflicted on the upper half of the body by forepaw attacks and bites to the scalp, neck and face causing extensive bleeding and fractures. Other omnivores such as wild boar have been described along with other horned animals previously. Despite their smaller size compared to most of the animals described, their ferocity and determination must not be underestimated. Following an initial assault the agile boar, if not stopped, will turn and attack repeatedly with their long tusk-like teeth leaving victims with multiple injuries [13]. When adult humans run away the head of a

chasing boar or feral pig is usually at the level of the victims lower body so that bites from behind often affect the gluteal and anal regions, which can make surgical management very challenging.

INFECTIVE INJURY

Wild animal trauma is distinguished by the combination of open penetrating injury and significant contamination from teeth, claws, horns and the surrounding environment. All these potential sources can cause severe sepsis although the highest risk is from animal bites, occurring in approximately 10-30% of cases [14]. The rate of infection varies depending on the location of the wound. Hand injuries have a higher risk of infection due to poor vascularisation, whereas bites in the head and neck region have a lower risk of infection, in part due to an abundant blood supply [14]. The age of the wound is also an important factor in the development of infection. The longer the wounds remain untreated, the more likely they are to become infected. The bacteriology of animal wounds can vary depending on the method of injury and the species of animal. The most common organisms are Pasteurella species, Staphylococcus aureus, Streptococcus viridans, Escherischia coli, diptheroids and anaerobic bacteria (Fusobacterium nucleatum, Prevotella, Clostridium, Porphyromonas species and Peptostreptococcus) [15]. In addition to these micro-organisms, based on differing evolutionary processes and diet, each animal may harbour micro-organisms specific to their own species. These pathogens, although commonly colonised in those particular animals, in general may be rarely seen and therefore a knowledge of the likely attacker can inform the most appropriate use of anti-microbial treatment. Awareness of the potential for infectious complications is paramount, including the risks for chronic deep infection, meningitis, necrotising facilitis, osteomyelitis, septic arthritis, abscess formation, or even septic embolisation resulting in distant organ infection. Healthcare providers must also be aware of the potential for animal wounds to harbour and host a number of important zootoxic organisms which can be transferred, including rabies virus, Mycobacterium tuberculosis complex, Yersinia pestis, and Francisella tularensis [15]. Universal precautions must always be used when treating these patients. Large animal bites also mandate the consideration of rabies and tetanus prophylaxis. Both are feared potential complication from animal bites. Rabies is a viral zoonosis that can lead to fatal encephalitis and tetanus a bacterial sequelae causing severe muscle spasms that can also lead to death.

Pasteurella multocida, a nonmotile, gram-negative bacteria that can cause wound infection upon inoculation is the most common bacteria found in infected bite patients [16]. It forms part of the normal flora in the nasopharynx or gastrointestinal tract in many wild animals including bears, wild boar, wolves and most notably the big cat family. Serious P. multocida infection has been reported as a septic complication within the first 24 hours of both big cat bites and scratches [14]. Less commonly, Neisseria weaveri, Moraxella species and Bartonella henselae (causing cat scratch disease) has been reported after big cat attacks. Wound infections from large mammal attacks result in a median of five bacterial species per culture, which underscores the importance of obtaining pre-antibiotic wound cultures. For victims of goring injuries from horns or tusks,

infection with multiple soil pathogens, including Bacillus species and fungi must also be considered.

Attacks in aquatic environments bring another dimension to wound contamination. A number of aerobic and anaerobic bacteria and fungal species have been cultured from cocodilia wounds. The organisms found in crocodiles' and alligators' mouths are typically Gram-negative species such as Salmonella and Aeromonas hydrophilia and anaerobic species include Clostridia [2]. Other micro-organisms described from crocodiles attacks include Vibrio vulnificus, Citrobacter species, Burkholderia pseudomallei, Pantoea Bacteroides melaninogenicus, Serratia fonticola. Pseudomonas agglomerans. areogenosa and Proteus vulgaris [17]. Overwhelming infection is also typical of other reptile bites, e.g. the Komodo Dragon of Malaysia is a predatory lizard that is known to attack and feed on man. Cultures grown of saliva and plasma have yielded both Grampositive and Gram-negative bacteria, with Escherichia coli being the most common bacterium [18]. Considering the varied diet of the shark, it is not surprising that many shark bites become infected. In addition, contrary to the concept that the sea is a sterile environment for microorganisms, there is an array of atypical bacteria very capable of infecting human tissue. Most commonly described are the Vibrio and Aeromonas species which can cause fulminant sepsis in a matter of hours [6].

MANAGEMENT

PRE-HOSPITAL CARE

At the scene, emergency medical services and appropriate authorities should be notified immediately to address injuries promptly and secure the area safely. Treatment has to begin immediately and outcome is dependent on adequate pre-hospital care [6]. Victims are often in a state of extreme exhaustion, often hypothermic and if in water are near-drowned. Early determination of the level of care that is required is crucial, so that expeditious and appropriate evacuation and transport can be arranged. The extent of on-scene treatment will depend on the rapidity of evacuation to an appropriate medical facility: the earlier the victim receives definitive stabilisation and treatment, the better the outcome.

At the scene the victim should not be moved if at all possible, owing to the high potential for spinal cord injury. However, if the victim and others remain at risk from the animal, movement to as a safe area only as far as is necessary to commence immediate resuscitation is advisable. Priorities are the same as in any other severe trauma situation. These are fundamentally to secure the airway, regulate breathing, control haemorrhage, institute resuscitation and re-warm, as per ATLS principles. Immobilisation of the if head, neck and back are paramount if spinal injury is suspected. Care providers should wear gloves because of potential exposure of rabid animal saliva in and around wounds [3]. Control of bleeding with direct pressure is advocated but tourniquet application above arterial pressure can be used to controlling massive bleeding. If available, a large bore intravenous line can be started and (preferably warm) fluid resuscitation administered. Early reduction and splintage of large wounds and suspected fractures will limit haemostasis and pain [3]. Patients are often young and can

compensate for their assault as long as bleeding is stopped and resuscitation is started quickly.

If significant transport delay is anticipated, wounds should be cleansed and vigorously irrigated at the scene to reduce bacterial and viral contamination. Available water, preferably boiled, is adequate for wound irrigation, and regular soap possesses some bactericidal and virucidal activity. Foreign material should be gently removed with a soft, clean cloth or sterile gauze. After cleansing, wounds should be covered with a clean, dry cloth [3]. If appendages or limbs have been amputated, and if retrieving them does not pose a risk to rescuers, they should be collected for transport with the victim to facilitate any possible reimplantation. Unfortunately many of these limbs or appendages will not be salvageable. Prior notification to the receiving hospital is recommended, and the use of a specialised trauma retrieval team to collect the patient should be considered [3].

HOSPITAL CARE

Once in hospital a thorough secondary survey of all injuries can be undertaken, documented and if possible photographed. Resuscitation and stabilisation of the patient must be continued in parallel to the evaluation of all injuries. Although bite or claw marks may initially seem superficial, they may overlie areas of extensive tissue injury, open fractures, neurovascular trauma, airway injury, and intracranial and spinal cord penetration. Investigation may require plain X-rays, computed tomography (CT), or advanced imaging as available. Computed tomographic angiography (CTA) is particularly useful for evaluating potential carotid or vertebral artery injuries in the head and neck and major injuries involving the chest, abdomen and extremities. Management of significant injuries may require activation of the major haemorrhage protocol (including blood, platelets and fresh frozen plasma) and rapid operative management, with damage control surgery, amputations and consideration of reimplantation of salvageable limbs when the patient has been stabilised [20]. Later sequelae may include major infections and neuromuscular, vascular, a massive systemic inflammatory response and renal compromise [6]. Varying areas of tissue loss, degloving injury, and crush may be present. Compartment syndrome must be considered in all cases of significant crush injuries to the extremities. Because of the increased likelihood of wound contamination, retained foreign body, deep tissue and musculoskeletal involvement and devitalised/necrotic tissue, all wounds should be explored carefully. This is best carried out in the operating theatre under safe general anaesthetic. Wound cultures should be taken and then the wounds can be appropriately debrided, cleaned meticulously and irrigated vigorously. This can be done with large amounts of sterile saline using highpressure syringe irrigation or employing a bacteriocidal-virucidal agent (e.g. 1% povidone-iodine solution), if available [3].

Surgical debridement of facial wounds should be kept to a minimum, particularly in landmark areas such as the vermilion border of the lips, the nasolabial fold and the eyebrows [12]. The decision to close superficial wounds should be made on the basis of cosmesis, function, and infection risk factors, as is the case with other animal bites. As bite wounds are essentially contaminated, meticulous debridement and delayed closure may be one choice. Supporters of this method claim that as long as the repair is performed before the wound reaches the proliferative phase of healing, cosmetic results

are indistinguishable from those of primary repair. There is accumulating evidence, however, that in certain bite wounds, primary closure yields the best cosmetic result without significantly increasing wound infection rate [19]. Bite wounds under consideration for primary repair should be further assessed on the basis of time elapsed since the bite; in general, suturing of non-facial bite wounds older than 6-12 hours is not recommended. Significant delays beyond 6-12 hours in seeking medical attention increase the likelihood of infection. For uncomplicated bite wounds presenting beyond the "golden 24h period", primary closure is controversial. In these cases delayed closure is a time honoured practice [19]. This implies a waiting period of 4-5 days before definitive wound closure, during which time the wound is kept open, usually with moist gauze dressings providing drainage, while oedema is allowed to subside. Reimplantation, delayed primary closure, and grafting decisions will be made when more severe wounds are carefully evaluated under optimal conditions. The presence of overt infection normally precludes primary closure of the wound. Options include secondary healing with subsequent revision surgery, delayed closure, or primary closure with insertion of a drain. For any complex wound, if in doubt expertise should be sought from a plastic/reconstructive surgeon.

TREATMENT OF INFECTION

Because polymicrobial infections have been reported, treatment with broad-spectrum antibiotics such as a beta-lactam/beta-lactamase inhibitor combination (e.g. amoxicillinclavulanate, known as co-amoxyclav) is considered standard of care for all large animal bites [3]. For patients with a penicillin allergy a third-generation or extended action cephalosporin plus Clindamycin, Trimethaprim-sulfamethoxazole, or carbapenems or a combination of a Fluoroquinolone and Clindamycin may be utilised. Note that first generation cephalosporins are not recommended as sole treatment owing to variable Pasteurella species susceptibilities and the possibility of gram negative concurrent infection. Due to a multitude of variable factors, pre-antibiotic cultures should be taken and discussion with a microbiologist is advisable. Standard rabies prophylaxis after exposure, consisting of vigorous wound cleansing and administration of human rabies immunoglobulin and the initial dose of 4 doses of human diploid cell vaccine (with additional doses administered on days 3, 7, and 14) should be instigated. If available, a virucidal agent (e.g. povidine-iodine solution) should be used to irrigate the wounds. If anatomically possible, the full calculated dose of immune globulin should be infiltrated in and around all wounds. If that is not possible, any remaining volume should be administered intramuscularly at an anatomic site distant from that of rabies vaccine administration [3]. Tetanus immunization should be assured to be up to date. Tetanus/diphtheria/pertussis (Tdap) or diphtheria/tetanus (dT) vaccine should be administered to all patients who last received a vaccination more than 5 years previously. Tetanus immunoglobulin is added in those with significant bite wounds if the tetanus immunisation history is unknown or if fewer than 3 doses of tetanus vaccine were administered previously [19].

POST-OPERATIVE CARE

Close follow-up of patients with these injuries is necessary with an emphasis on postoperative vigilance for signs or symptoms of wound infection, which should prompt immediate re-exploration of wounds for further debridement and drainage [20]. The severity of wounds inflicted by large animals frequently requires a multidisciplinary approach with extensive reconstructive plastic, maxillofacial and orthopaedic surgery, intensive care, microbiology, dietetics, neurologic rehabilitation, physical and occupational therapy. If the site of injury involves the head and neck, early speech language pathology assessment should be considered. This is usually required for many months to years to ensure optimum results. Public health and animal management must also be notified.

PSYCHOLOGICAL SUPPORT

The psychological effects of the attack on the patient should also not be underestimated. The victim of an attack of this magnitude is at risk of developing post traumatic stress disorder (PTSD), which should be addressed proactively by the medical team [3]. In addition to classic PTSD symptoms such as flashbacks, dissociative reaction, affective dysregulation and nightmares reliving the event, the sudden appearance and rapidity and severity of injury inflicted by these large predators produce intense ensuing feelings of helplessness, powerlessness, threat to life, loss of body image and function in surviving persons, and survivor guilt in family and community members [3]. Symptoms may persist for months or years but can improve with appropriate counselling. Critical incident stress debriefing and counselling may also provide significant assistance to friends and family or healthcare providers that may have witnessed the attack or the severity of inflicted injuries.

SUMMARY

Injuries from large wild animals are rare but underreported; injuries are usually severe and often fatal. The motivation of the animal, the knowledge and skills of the victim and care providers are major factors in determining the outcome of these attacks. If severe injuries are sustained, swift and appropriate management of these patients using ATLS protocols are imperative for survival. Careful evaluation throughout the patient's admission and timely treatment using a multidisciplinary approach are key to achieving optimum outcomes. As with all things, prevention is better than cure. Minimising human encroachments into animal habitats, practicing strict vigilance and knowledge of dangerous animal behaviour can reduce human-animal conflicts. Sensible precautionary measures by humans visiting animal habitats and strict adherence to rules and mantras of experienced trained guides will significantly lessen the risk of sustaining such injuries.

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13. BURNS: EMERGENCY CARE

SUZANNE THOMSON, MASSEY BEVERIDGE.

INTRODUCTION

Globally, burn injury is characterized by considerable variability: the frequency of burns varies 15–fold from country to country, severity ranges from trivial to lethal and the correct treatment depends, more so than for most other types of injury, on medical resources available. This article provides an overview of burn injury and the different methods that may be employed in the emergency treatment of burn patients. There is no single formula, rather a range of options that may be adapted to both the hospital environment and patients' needs. The most important question to answer is "What can I do for this patient in this environment?" Much of the initial management of a burn depends on when and how it will be grafted.

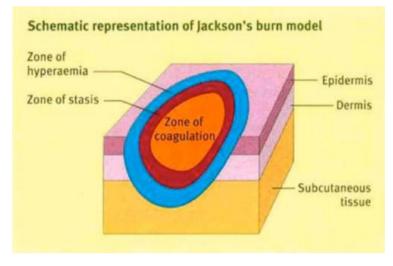
Accurate data on global burn injury is difficult to come by. Burn injury includes inhalation, flame, scald, contact, electrical and chemical injury but World Health Organization (WHO) global estimates are only available for fire-related injury (flame burns plus inhalation injury). Scalds account for about a third of total burn injuries, while electrical, contact and chemical burns comprise a much smaller proportion. Some generalizations can be made regarding global burn injury, however:

- Globally, fire related injuries cause 195 000 322 000 deaths per year [1].
- Every year nearly 11 million people suffer burns severe enough to require medical attention [2].
- 95% of fatal burns occur in low and middle income countries [3].
- Half of global fire-related burn deaths occur in the WHO SEAR Region and 70% of these are of women (mostly in India, Pakistan and Bangladesh) [4].
- Over half of the DALYs lost globally to burns are among children aged 0-14 years
 [3] (DALY = Disability adjusted life year; 1 DALY = 1 year lost due to disability).
- Children in Africa, particularly toddlers, have very high rates of burn injury, sometimes comprising more than half of all burn admissions [4,5].
- Non-fatal burns causing lifelong disability are more common than burn fatalities.

Burn mortality depends on the patient's age (infants and the elderly fare worse), the extent of the burn, the presence of inhalation injury as well as concomitant medical disease and trauma. Historically, the three principle developments that have allowed people with larger burns to survive were the development of topical antibiotics, early excision and grafting of burns and sophisticated intensive (ICU) care.

PATHOPHYSIOLOGY AND THE METABOLIC BURN

Thermal injury causes pain and release of inflammatory mediators that increase capillary permeability and cause tissue swelling. During the first day following full thickness burn injury three concentric areas of pathology are present. The central zone of coagulation is composed of dead cells and is white or charred in appearance. This is the area which has sustained the maximum heat contact, surrounded by the zone of stasis (initially red and may blanch but its blood supply is precarious) and the outermost zone of hyperaemia (intact circulation, definitely blanches on pressure and generally starts to heal by day 7 following injury). Good burns management aims at preventing transformation of the zone of stasis into the dead zone of coagulation. Release of histamine, prostaglandins, bradykinin and free radicals result in increased permeability of epithelial basement membrane and burn oedema, causing swelling and increasing the zone of coagulation.



[http://www.vicburns.org.au/burns-assessment/burns-pathophysiology.html]

In burns involving less than 15% of total body surface (TBSA) the swelling remains localized, but in burns larger than about 25% TBSA, localized capillary permeability becomes generalized and the inflammatory response becomes systemic (Systemic Immune Response Syndrome [SIRS]). These are called *metabolic* burns. Previously healthy patients with localized burns (<15% TBSA) usually are able to maintain oral fluid intake sufficient to prevent severe dehydration and renal failure and, if given appropriate analgesia and dressings, are very likely to survive. Pre-existing medical or nutritional problems, smoke inhalation and extremes of age (<5 years or >60) lower this threshold. Burns above 15% TBSA generally require fluid resuscitation and patients with burns >30% TBSA need attention to their airway as the systemic inflammation can cause enough laryngeal oedema to threaten its patency. Without fluid resuscitation the massive capillary leak causes sludging of blood in all organs and causes renal failure, coma, heart failure, respiratory failure and intestinal ischemia. Without ICU care and the ability to graft the large areas of skin involved mortality rises sharply after 30% TBSA and few patients will survive burns larger than 40% TBSA. Smoke inhalation increases initial fluid resuscitation requirements and causes a delayed chemical pneumonitis that peaks at about the third post-burn day. Lung protective ventilatory strategies (permissive

hypercapnia) and sophisticated ICU care may be required to manage this. Smoke inhalation significantly increases mortality.

In patients who survive the initial burn injury and resuscitation, burn wound infection is the next challenge. Meticulous attention to sterility and occlusive dressings with topical antibiotics that penetrate the wound may delay or avert infection of the burn wound, but early excision of the dead burned tissue is the single most effective way to reduce the burden of infection and should, where possible, be undertaken in the first 3-5 days post-injury. Burn excision, however, does cause considerable blood loss and leaves large wounds that require coverage with skin grafts. Excision and grafting a large burn can be technically demanding and is costly in terms of blood use, operating time and dressing material.

Adult Respiratory Distress syndrome (ARDS), ventilator acquired pneumonia and acute renal failure are all common complications in severely burned patients that require sophisticated ICU management.

BURN DEPTH AND HEALING

Burn depth is alternately described as first, second and third degree or superficial, partial and full thickness. The terminology can be used interchangeable once it is understood. Depth is affected by the large variation of skin thickness around the body, between individuals and depending on age.

A **first degree** or **superficial burn** is red and quite painful, but does not blister, like sunburn in a fair-skinned individual. No specific treatment is necessary and it will invariably heal without leaving scars. First degree burns are NOT counted when estimating TBSA.

Second degree or partial thickness burns may go on to heal or may require grafting. They cause large blisters that lift a variable amount of epidermis off the underlying dermis. Small blisters may be left intact, but large floppy blisters, those filled with blood or pus and those that interfere with joint movement, dressings or wound assessment should be debrided. Partial thickness burns can be either superficial or deep and one should avoid prognostication until the wound has been examined on the third day. Scald burns in particular are notorious for being deeper than they appeared initially. Second degree burns are exquisitely painful because of the exposed nerve endings. Thin-walled blisters suggest more superficial injury than those with thick walls. Examine the base of the wound to assess its potential for healing. If it is glistening, pink and blanches to light touch it is superficial and likely to heal, but if all you see at the base of the wound is dull white dermis with fixed cherry red staining it is likely deeper. Test sensation with a wisp of gauze, the less sensation felt the deeper the burn. The admixture of coagulated serum and silver sulfadiazine can create a fairly adherent gel layer on a wound that one should not mistake for true burn eschar. Wipe away at it to see if it comes off. Finally, in hairy individuals, tug gently with forceps at the remaining hairs in the burn bed. If they resist the wound is not that deep, but if they pull out easily it is deeper.

Partial thickness burns heal by two mechanisms, re-epithelialisation and contraction (Figure 1). If sufficient epithelial cells remain in the wound, often protected deep in hair follicles and sweat glands, they will grow out and become confluent, healing the wound by re-epithelialisation (Figure 2). Areas where sufficient epithelial cells do not remain will take much longer to heal and will do so by contraction. This takes much longer (months)

and yields a much worse result. In children, where the force of skin contraction is opposed by the force of normal longitudinal bone growth, the leverage created by the two forces can give extremely disabling wound contractures, bending digits or joints up to 180 degrees out of normal position (Figure 3). If healing occurs by re-epithelialisation, the wound is usually closed by 21 days; if not, contraction will be the dominant force in the wound and a poor result can be predicted. The rule of thumb therefore is that partial thickness burns that look like they will re-epithelialise in 21 days or less should be left alone, while those that will take longer to heal will have a better result if grafted. This can be a difficult decision to take.

Third degree or full-thickness burns involve the entire thickness of the skin. At the time of initial presentation the skin is waxy and somewhat hard, like leather. It is predominantly insensate and the wound does not cause much pain although surrounding partial thickness areas may be very painful. Sheets of epidermis will slide off with gentle transverse digital pressure and a gentle 'hacking' action with the examining fingernail will lift off the outer layers of skin. The underlying dermis may be dull and white or the skin may not separate at all but feels like old dried leather. This is called "eschar". The burns may be charred but don't mistake soot for underlying burn, wipe it away to see.

Full thickness burns do not stretch like normal skin and if circumferential will cut off the circulation to a swelling limb like a tourniquet, so consider the need for escharotomy. Full thickness burned skin will eventually separate from the underlying viable tissue through a slow bacterial and enzymatic process and if small may heal by contraction, but if large will become a chronic wound with thickened scar at its edge.

So called **fourth degree burns** involve underlying muscle, bone and connective tissue and may need early amputation.

When examining older burns look for the promise of little white pearls of epithelium which are a sign of healing, and red raspberries of granulating fat which imply the wound is not re-epithelialising. "Healthy granulations" in an old burn wound do not mean it is healing, but that it needs grafting.

BURN MECHANISMS

Flame Burns are the most common (and lethal) of burn types. The etiology is usually clear from the history (and odour) and it is important to ask if the burn occurred in an enclosed space as this suggests concomitant smoke inhalation, especially if the victim was found unconscious. The victim's clothing often catches fire; even the combustion of a light cotton shirt releases enough energy in close proximity to the skin to cause full thickness burns. Flame burns typically show charring and the patient is covered in soot, but not always, especially if a clean burning fuel like alcohol was the accelerant. Flames lick up over the body and have feathered edges.

Scald burns caused by hot water, steam or oil are the next most common and tend to drip down the body. Oil splatters. Immersion burns have a clear red demarcation line and sharp edges. If seen in the first few hours they can look much less deep than three days later. Never discharge a scald burn without making arrangements to see it again in three days. Super-heated steam can cause a true thermal burn of the tracheal mucosa. Water at 49 degrees C takes 10 minutes to cause a full thickness burn but at 54 degrees can cause a full thickness burn in 30 seconds [6].

Contact burns are usually small and particularly deep. The worst occur when a person falls into a fire or is unconscious or trapped against a hot surface. Even relatively low temperatures can cause burns when the exposure time is long enough e.g. an unconscious road traffic injury patient lying on a hot road. Molten metal causes very deep burns.

Electrical burns fall into two very distinct categories: electrical conduction burns caused by the conduction of high voltage electricity through the body and electrical flash burns which occur when a short-circuit causes a flash of extremely intense energy.

With **Electrical Conduction burns** a small cutaneous wound may conceal massive internal tissue damage and myonecrosis. Normal household current seldom causes major conduction burns which usually involve high voltage (>1000V) current. There may be discrete entry and exit wounds but in really high voltage injuries it may be difficult to distinguish the entry and exit points because the victim is enveloped in a cloud of electricity The current may seek the path of least resistance through the body and travel along blood vessels and nerves, or the body may act as a mass conductor. Bone has the greatest electrical resistance and so retains the most heat after the conduction incident and can literally cook the limb from the inside out. Current may arc between limb and torso causing a secondary flash burn. If conscious, the patient may give a history of a "can't let go" phenomenon. Cardiac arrhythmias are common and the jolt of the shock may cause fractures and cervical spine injury. Those who survive may note strange transient neurological symptoms months later.

Electrical flash burns are pure thermal burns, typically involving the face and hands that occur when an incautious electrician sticks his tool in a connection box and causes a short circuit arc and flash. Victims are usually men. The resulting flash burns his face and hands and may set his clothing on fire. Look for a speckled appearance and corneal injury. If severe this kind of burn can cause enough swelling to jeopardize the airway and prophylactic intubation should be considered before an emergency surgical airway is necessary (Figure 4).

Chemical burns are relatively uncommon but can be devastating. Remember to protect yourself and your staff before touching a patient with a chemical burn: gowns, gloves, masks and eye-protection are essential. Brush away dry chemicals like sodium hydroxide, remove patient's clothing and get them into a shower as fast as possible. The burns should be irrigated until they are no longer painful, perhaps as long as an hour.

Concentrated sulphuric acid is readily available in many places as it is used in car and motorcycle batteries. Sadly it has become a means of inflicting horrific injury when thrown in a person's face. The burns are invariably full-thickness and blindness commonly results. After the acid is washed away the burns have a benign grey appearance that gives no clue to the disfiguring wounds that will appear later after the eschar separates. Concentrated acid causes coagulation necrosis whereas concentrated alkali causes liquefaction necrosis and may burrow deep into tissue. The classic example is a worker shovelling NaOH crystals that get in his boot which then dissolve in the sweat. Both acid and alkali are extremely harmful to the eyes and these too must be irrigated with copious amounts of water.

Hydrofluoric acid is used to etch glass and clean refrigeration equipment. It has tremendous avidity for calcium and causes great pain without much visible burn. As it leaches into the deeper tissues it can cause profound hypocalcaemia, cardiac arrhythmias and death. Injection of calcium gluconate into the tissue may help relieve

pain but serum calcium levels should be monitored closely and intravenous calcium gluconate given until these return to normal.

White phosphorus ignites on contact with air and is used in 'star shells' and tracer rounds. It burns deep into the tissue and adheres to fat. In such a situation, protect your staff, remove patient's clothing and any particles of burning phosphorus, placing them in a bowl of water, and cover the wounds with dripping wet towels.

"When surgical treatment is available, the idea is to identify and remove the remaining phosphorus particles. The wet wound can be irrigated with a neutralizing agent. A freshly prepared solution of 1 % copper sulphate combines with the phosphorus to form black copper sulphide, which impedes violent oxidation and identifies the particles. The black particles can then be removed with forceps and placed in a basin of water. The solution must be very dilute, the palest blue colour, since its absorption can cause haemolysis and acute renal failure. If used, the copper sulphate solution must be washed away immediately. Or, if copper sulphate solution is not available, the operating theatre lights may be put out; any remaining particles will glow with phosphorescence in the dark and can be carefully picked out with forceps and placed in a basin of water" [7].

BURNS PRESENTING LATE

In low and middle income counties (LMIC) patients often present some time after the injury occurred. If they present within the first 24 hours after burn injury it is reasonable to try to get the calculated volume of fluid into them; after the first 24 hours fluid should be given to correct dehydration and clinical assessment of renal function is important. If they present after the third day without renal failure it is likely that they have compensated by taking oral fluids. Often they present weeks or months later with established burn wound infection, and malnutrition. Try to control infection by debriding necrotic slough and improve nutritional status before attempting to graft the wounds.

SPECIAL BURNS PROBLEMS

There are some typical patterns of burn injury that the clinician should recognize. Burn assaults and homicide are not uncommon:

Massive Burns. Individuals with lethal burn injuries burns should be admitted for comfort measures and should be allowed fluids to relieve thirst and adequate opioid analgesia while the family can gather and say their farewells. If there is doubt about outcome a trial of fluid resuscitation usually gives a fair idea of the direction in which the patient is headed. What constitutes a massive burn varies from place to place but the determination depends on a number of factors. The basic predictors of mortality are age, burn size and inhalation injury. Small children and particularly the elderly have much less reserve to cope with a severe burn. The availability of advanced burn care has a big impact on survival in major burns. If the chances of meaningful recovery are slim it is good to be frank about this with both patient and family from the outset. (See later article on "Care of the dying patient in critical care".

Dowry burnings in South Asian communities are common and very difficult to prove but massive burns of young married women living with their in-laws should arouse suspicion,

particularly if the women was not wearing jewellery at the time and if no one else was injured trying to rescue her or put out the fire [8].

Acid assault is a particularly vicious way to harm an individual and patterns vary considerably around the world. Organizations such as the Acid Survivors Trust International with branches in Bangladesh, Cambodia, Uganda, Pakistan and Nepal work hard to support the victims of such assaults and to bring assailants to justice [9].

In children with **epilepsy** seizures can be brought on by watching the flickering of an open fire. During the seizure the victim falls into the fire and is severely burned.

Tar burns are pretty common and can be treated as routine once the tar is removed. There are special creams containing polysorbates (e.g. Tween 80 $^{\text{TM}}$) that are safe and effective but so is petrolatum jelly (Vaseline $^{\text{TM}}$) or Polysporin $^{\text{TM}}$ though it takes a bit longer. Under no circumstances use liquid solvents such as kerosene or gasoline.

Diabetic neuropathy leaves the feet numb and they feel cold. Patients then immerse their feet in hot water or warm them over a toaster. This can cause very deep burns and will often result in amputation.

Friction, such as that resulting from being dragged for a long distance on the back can cause full thickness dermal injury similar to a burn and should be treated the same way.

INHALATION INJURY

The term "inhalation injury" is commonly used to describe respiratory problems associated with burns but actually divides into three distinct entities.

Carbon monoxide poisoning should be suspected in anyone burned in an enclosed space, especially if they were found unconscious at the scene. Treatment with 100% oxygen, preferably delivered through an endotracheal tube, should be initiated as soon as the problem is suspected and before the carboxy-haemoglobin level is available from the lab and should be continued until the laboratory confirms normal levels. The ruddy or cherry red colour skin colour often associated with carbon monoxide poisoning is a post-mortem finding more helpful to the forensic pathologist than to the clinician. Carbon monoxide toxicity causes a range of symptoms from confusion and headache to lethargy, convulsions and death.

Cyanide poisoning is most commonly seen following inhalation of the by-products of burning plastics. It binds readily to the cytochrome system of mitochondria resulting in reduced consciousness, neurological symptoms and convulsions.

Laryngeal oedema causing stridor and acute airway obstruction does not necessarily imply the inhalation of smoke or hot gasses and may be found in scald burns and electrical flash burns affecting the lower face, neck and upper chest. It should be suspected in every burn > 30% TBSA, even those not affecting the neck, and is best managed by early prophylactic endotracheal intubation. Do not wait for the patient to develop stridor. Place the largest possible endotracheal tube (ideally #8) to allow for easy bronchoscopic access, and leave the tube long. If the tube is cut close to the lips it will be too short as the patient's face swells with fluid resuscitation (Figure 5). Swelling will get worse as soon as fluid resuscitation begins and once the patient is stridorous it is much more difficult to perform the intubation. Rapid sequence intubation in burn patients with laryngeal oedema is avoided by experienced anaesthetists and the best approach is

with topical anaesthesia, gentle sedation and optimal visualization. Fibreoptic intubation may be helpful. Under no circumstance should one poke blindly with a tube as this will only cause the friable mucosa to bleed and make the problem more difficult. These are cases for the most experienced intubator available. Have the difficult intubation cart close at hand and an operator capable of creating an emergency cricothyroid airway standing close at hand. This is not the time for formal tracheostomy. Try to get the largest possible tube in the cricothyroidotomy (a #8 armoured tube in an adult) to facilitate bronchoscopic access. In most cases it is not necessary to convert this to a formal tracheostomy. In facial burns it may be helpful to secure the endotracheal tube to the teeth with heavy sutures or wires to avoid ties crossing the burned skin of the face. In situations where mechanical ventilation is not possible and airway protection is required tracheostomy is a reasonable alternative. This decision should be taken early and all patients should be intubated prior to their tracheostomy. Tracheostomy in burn patients is technically demanding as the trachea will be found to lie very deep within the burned neck and standard tubes may not be long enough. Percutaneous tracheostomy should therefore not be attempted in acute burns; the tissue may be too swollen for the tube to lie safely within the trachea.

True **smoke inhalation** injury is found in individuals who were involved in a fire in an enclosed space. Sometimes they will have no burns, and a purely respiratory presentation. A history of being found unconscious at the scene is strongly suggestive and a careful inspection should be made for soot in the nose and mouth, singed nasal hairs and soot on the face. Early intubation is recommended and bronchoscopy can be performed through the endotracheal tube as soon as it is secured. That will help identify soot in the airways and inflammation of the endobronchial mucosa but these findings do not really predict the severity of the inhalation injury. Smoke and hot gasses inhaled during a fire cause a chemical pneumonitis that peaks on about the third post-burn day. Smoke inhalation injury demands fluid resuscitation in excess of the calculated requirements and this is often the first indicator of the severity of the inhalation injury. Fluid should not be withheld. There is no role for prophylactic antibiotics as that will only help select more resistant strains of bacteria. Lung-protective ventilation strategies are prudent and good pulmonary toilet is essential. Inhaled bronchodilators, heparin and N-acetyl-cystein may help.

BURN MANAGEMENT

FIRST AID

First aid can greatly affect the outcome following burn injury. School children should learn to "stop, drop and roll" if their clothing catches fire, to cover a burning pot of oil, rather than try to carry it outside and to cool a burn for 20 minutes with clean water.

Rescuers should remove the victim from danger, taking care not to jeopardize themselves. Cool the burn with cold running water or wet clean towels (they don't have to be sterile). After 20 minutes remove the cold wet towels and wrap the patient in dry sheets or blankets to prevent hypothermia. In chemical injury rescuers should always protect themselves before removing the patient's affected clothing, brushing off dry chemicals and showering or immersing the patient in cool water. In case of an electrical conduction injury turn off the power source or push it away with a non-conducting pole. If there is any suggestion of carbon monoxide poisoning or smoke inhalation, give oxygen in as high a concentration as possible. Care should be taken not to miss underlying

traumatic injury or medical conditions. If transport to definitive care will be delayed start intravenous (IV) fluids. Field dressings are not necessary but the patient will be more comfortable if the burns are covered in surgical towels or a clean dry sheet.

OUTPATIENT BURNS MANAGEMENT

Many burns can be treated on a outpatient basis. The American Burn Association has published criteria for admission to a burn centre that are widely applicable [10]. Burn injuries that should be referred to a burn centre include:

- 1. Partial-thickness burns of greater than 10% of the total body surface area.
- 2. Burns that involve the face, hands, feet, genitalia, perineum, or major joints.
- 3. Third-degree burns in any age group.
- 4. Electrical burns, including lightning injury.
- 5. Chemical burns.
- 6. Inhalation injury.
- 7. Burn injury in patients with pre-existing medical disorders that could complicate management, prolong recovery, or affect mortality.
- 8. Any patients with burns and concomitant trauma (such as fractures) in which the burn injury poses the greatest risk of morbidity or mortality. In such cases, if the trauma poses the greater immediate risk, the patient's condition may be stabilized initially in a trauma centre before transfer to a burns unit. Physician judgment will be necessary in such situations and should be in concert with the regional medical control plan and triage protocols.
- 9. Burned children in hospitals without qualified personnel or equipment for the care of children.
- 10. Burn injury in patients who will require special social, emotional, or rehabilitative intervention.

Small partial thickness burns are readily treated on an outpatient basis. The key points in outpatient burn management are to evaluate the depth and extent of the wound, provide tetanus prophylaxis and analgesia, dress the wound properly and ensure there are mechanisms in play for daily dressings and re-examination three days later. Elevation of the affected part will significantly reduce pain and swelling. Modest blisters are best left intact and covered with a dry dressing as there is no point covering a blister with topical antibiotics. A sterile occlusive dressing is advised.

RESUSCITATION OF A METABOLIC BURN

Ideally, initial assessment and resuscitation of a major burn should take place in a room designated for the purpose. It should be large, warm, clean and well lit, equipped with ceiling mounted shower hoses, heat lamps and a steel stretcher that can be covered with plastic sheeting for washing the patient on. Best practice is to have a diathermy machine for performing escharotomies, a ventilator, the difficult intubation cart, IV infusion pumps, fluid warmers, flexible bronchoscope, digital camera and monitoring equipment available; dressing supplies should be close at hand.

The burn team leader should be a surgeon or intensivist experienced in burn evaluation and care and the team should comprise at least two burn nurses. Senior anaesthesia personnel should be available. On arrival the Burn Team Leader should assess the patient with a primary and secondary survey looking both at the burns and for associated traumatic injuries with an ABCDE approach.

The airway should be assessed for oedema and evidence of soot inhalation, early endotracheal intubation is required if there are any concerns. If the patient is maintaining their own airway oxygen should be administered. Once the patient is safely intubated, flexible bronchoscopy can be performed. Cardiac and oxygen saturation monitoring should be attached. Peripheral or central IV access should be secured and an arterial catheter placed and connected to the monitor. Blood should be drawn for haematology, blood chemistry, carboxyhaemoglobin, group and screen, and arterial blood gasses. A morphine infusion is started and midazolam may be added once the airway is secured. The eyes should be inspected for corneal damage with fluorscein dye and an ophthalmoscope before the lids swell shut. A nasogastric feeding tube should be placed to decompress the stomach and a bladder catheter to monitor urine output. A portable chest X-ray may be delayed until after the endotracheal tube, lines and feeding tube are placed. If the patient's tetanus status is uncertain, tetanus toxoid should be given. In some circumstances tetanus immune globulin may be indicated.

Burn wounds are carefully assessed and photographed and an accurate burn diagram is completed; this can be facilitated by using a Lund and Browder chart. In an adult the surface area of the palm (including the fingers) is around 1% TBSA and is helpful in assessing small burns. Fluid requirements are calculated according to the Parkland formula (2-4 ml/kg/%TBSA). Half of the calculated fluid requirement should be delivered in the eight hours from the time of injury and the other half over the next 16 hours. This is only a guide and urine output should be used to evaluate adequacy of fluid resuscitation. The patient is washed with antiseptic soap and warm water with care being taken not to let the patient become hypothermic.

The patient should be assessed for circumferential burns. Limbs will swell progressively with fluid resuscitation; if there are clinical concerns that the circulation will be impaired with resuscitation escharotomy should be performed immediately, rather than risk losing a limb. Escharotomy incisions are placed in the mid-medial and mid-lateral axes of limbs and should extend to the subcutaneous fat. They are best made with a diathermy blade and should not extend onto unburned skin; they may be "T'd" at each end to reduce limb constriction. Breathing rate and effort should be examined and if there is full thickness burn restricting chest wall movement escharotomy should be considered. Typically thoracic escharotomy is required only in very large burns and usually not till after fluid resuscitation has begun. Even though full thickness burn is mostly insensate, the edges of the wound may be exquisitely tender and it is appropriate to use sedation e.g. fentanyl, midazolam and/or ketamine.

Most people clench their fists when being burned so that the dorsal skin of the hand is often badly burned while the palmer surface may be spared. If you need to perform escharotomy of the hand extend the axial incisions from the forearm onto the dorsum of the hand. Each burned finger can be released with a single incision joining the apices of the flexor creases situated on the least used side of the finger (Figure 6).

Once this is accomplished the burns should be wrapped in occlusive dressings with topical antibiotics and the patient transferred to the room where he will be cared for.

Routine orders for a burn admission may include provision for the patient's diet, activity, vitals, ventilation, IV fluids, investigations, drugs, drains, dressings and DVT prophylaxis. **DAVIDddd** is a convenient mnemonic to use:

DIET:

Start enteral feeds at 20ml per hour and increase gradually until goal rate is established.

Some children can take their fluid resuscitation via a feeding tube. Use oral rehydration solution.

Estimate nutritional requirements. A major burn requires twice the patient's usual calorie intake and 2g/kg of protein daily in an adult and 3g/kg daily in a child.

Adequate resuscitation, early enteral feeding and gastric acid suppression are the best ways to avoid the complication of haemorrhagic gastritis.

Parenteral feeding (TPN) carries the risks of line sepsis and gut-bacterial translocation and should be considered only in most extenuating circumstances.

ACTIVITY:

- Activity is permitted.
- Positioning: Elevate Head of Bed, Elevate burned limbs with pillows or foam wedges
- Physiotherapy
- Splints

VITALS & VENTILATORS:

Monitor ECG, arterial line, CVP, SaO2, hourly urine output, ventilator settings, oxygen.

IV FLUIDS:

Parkland Formula (Box I).

Use Ringer's Lactate. Normal saline adds a hyperchloremic acid load. Do not use hypotonic fluids. Small children will need dextrose in their fluids. (Dexrose 5% in 0.9 % Saline)

Adjust IV fluids if urine output <0.5 ml/kg/h. If urine sodium concentration is low the patient most likely needs more fluid; if it is high and the patient appears well-filled, but is not passing urine, a small dose of furosemide may help. Beware of the perils of over-resuscitation including abdominal compartment syndrome.

Colloid or plasma, if safe and available, may be used in the second 24 hours.

Burn patients typically loose calcium, magnesium and phosphorus during their ICU stay and these levels need to be monitored regularly and judiciously replaced.

Investigations: Routine blood analysis (arterial blood gases, haemoglobin, leukocytes, platelets, electrolytes, creatinine, clotting, liver function) and chest X-ray.

DRUGS:

- Analgesia and sedation: Provide background analgesia with regular paracetemol + NSAID + narcotic + sedative and specify procedural sedation with short acting agents for dressing changes.
- Give tetanus toxoid if not up to date, and tetanus immune globulin when indicated.
- Prophylactic antibiotics are not generally advised in fresh burns.
- Bronchodilators may be needed.
- H2 blocker or proton pump inhibitor.
- Glycaemic control.
- Heparin for DVT prophylaxis.
- The patient's routine medications.

DRAINS

Flush the feeding tube regularly and crush medications thoroughly. Check residual stomach volume until feeds are being well absorbed.

Connect the urinary catheter to a bag that measures hourly urine output.

DRESSINGS

- Write down dressing orders.
- Eye care: Artificial tears, lacrimal lubricant gel or antibiotic eye ointment to eyes as indicated.
- Face care .

DVT RISK

Assess the risk of DVT and order heparin accordingly.

The burn team leader should now update the family and write a detailed and comprehensive admission note.

The ICU care of a large metabolic burn can be challenging and resource intensive but extraordinary results can be achieved in the right settings. The US National Institutes of General Medical Sciences funds a large, multicentre Host Response to Injury Study and has published its Standard Operating Procedures for the clinical management of burn patients for ten issues in ICU management: resuscitation, prevention of hyperglycaemia, wound management, perioperative antibiotic prophylaxis, nutrition and enteral feeding, management of central venous catheters and sepsis, acute lung injury, ventilator associated pneumonia, diagnosis of inhalation injury and prevention of venous thrombo-embolism [11].

BURNS DRESSINGS

There is a huge variety of dressing materials marketed for burns, many of which work nicely. The choice of which to use is a balance between efficacy, availability and cost so

we will attempt to provide some principles to help in making the choice. The perineum, hands, face, and scalp require special mention.

A dressing team of nurses expert in burn care is at the very core of burn care and the careful attention paid to the wound is probably more important that the material used to dress it. Prevention of cross infection is paramount which is why the routine daily immersion of burn patients in shared water baths (tubbing) is avoided. Either the dressing team visits each patient in their room to do dressings or the patients come to a central dressing room. Each system has merit, but regardless of the system chosen, meticulous attention to the prevention of cross infection is mandatory. Gowns, gloves and masks should be used by the dressing team and changed between each patient.

Burn dressings have three main functions and like all dressings have three layers. The functions are to reduce pain, maintain a moist healing environment and to prevent or control infection. Changing dressings can be particularly uncomfortable, especially for children and particularly when the dressing adheres to the wound, so a non-adherent material should be selected. When dressings do adhere, it should be soaked off with saline or water. Especially where healing by re-epithelialisation is anticipated it is important to maintain a moist environment so that fragile young epithelial cells do not dessicate or get pulled off at dressing change. It will also be more comfortable than a wound that is allowed to dry out and crack. Finally, in large deep burns that are prone to invasive wound infection it is helpful to have antibiotic in the dressing to discourage bacterial growth.

All good dressings have three layers: the contact layer, the absorbent layer and the holding layer. The simple domestic band-aid is a perfect example: there is a thin non-adherent contact layer, an absorbent pad to soak up exudate and a sticky plastic layer to hold it on. Generally speaking in burns the contact layer should be non-adhering and if antibiotics are to be used they should be in contact with the wound surface. An exception is the classic wet to dry dressing where moist saline gauze is allowed to dry on the surface of a contaminated granulating wound bed and changed frequently affording mechanical debridement of the wound surface. This is painful but can be quite effective for cleaning an old granulating wound prior to skin grafting. The thickness of the absorbing layer depends on how much exudate the wound produces and should be adequate to contain the amount of exudate that accumulates between dressings – about 2cm thick. The holding layer is usually made of rolled bandage material that is elastic enough to conform to the body's irregularities, it should extend enough beyond the borders of the burn to avert slippage and wound exposure and should be solid enough not to slip or creep or dig-in at the edges.

Infection can convert a partial thickness burn to full thickness skin loss, so it is the deeper partial thickness burn that will benefit most from the use of antibiotics in the dressing. Superficial partial thickness burns are less likely to get infected and will usually heal pretty much regardless of the dressings applied. Full thickness burns are prone to invasive wound infection but this is best treated by excision and grafting. In this context antibiotic dressings are best used to delay the onset of infection while awaiting excision and grafting. When grafting is not possible they become the mainstay of treatment.

TYPES OF DRESSINGS

Silversulfadiazine (SSD) / gauze. SSD is a soothing white cream, has very broad antibiotic activity and some ability to penetrate the surface of the burn wound. Prepare

first a pile of large gauze pads, moistened in saline and apply the SSD cream to the gauze with a gloved hand or tongue depressor. This saves SSD and distributes it evenly on the contact layer of the dressing. Make as big a pile ready as will be needed to complete the dressing, and have at least as much dry gauze (+/- ABD pads) available for the absorbing layer and some rolled bandages ready to hold it on. Only when all is ready should the patient's old dressing be taken down, the wounds gently inspected, cleansed and any loose tissue removed. Then the new dressing is ready to go on and the wounds are left uncovered for the least possible time. SSD dressings should be changed daily. They are best for large areas of partial and full-thickness burn and may be used while waiting to excise and graft the burns

In large burns where facilities do not permit early excision and grafting, SSD dressings may be used and the painful the daily cycle of *éplouchage*, or plucking of the burns and SSD dressings may continue 6-8 weeks or until there is no remaining eschar and reepithelization of partial thickness areas has stalled. At this point the granulating areas that require grafting are usually smaller than what would have been grafted had the wound been excised primarily - but they still need grafting.

Silver Nitrate solution (0.5%) is an inexpensive alternative but oxidizes turning everything black.

Acticoat $^{\text{TM}}$ is microcrystalline silver that when activated by moisture saturates the wound with bactericidal silver. It can be left on for 4-5 days at a time and so reduces the nursing costs of dressing changes. It is expensive, however.

Antibiotic ointment (containing polymyxin and bacitracin) combines well with openmeshed paraffin gauze as the first layer of a burn dressing. It is very good for superficial partial thickness burns of smaller areas. It is also used in face burns without a covering bandage.

Biologic dressings include human cadaveric skin (allograft), human amnion, and porcine skin (Xenograft).

Fresh human cadaveric skin is a superb biologic dressing but concerns with the potential for disease transmission mean that frozen processed human allograft skin is more commonly used. It is placed on the wound just like a skin graft, and for the first week or two behaves like one, sealing the wound. Epithelial cells growing beneath (if they are sufficient) will lift the allograft off the wound bed, or if there are no epithelial cells beneath, the collagen on the underside of the allograft will become incorporated in the wound and in an immune competent individual, the allo-epithelial cells will melt away. After a light scraping the surface can take a skin graft once a donor site is available. Allograft serves as a temporary wound cover and may be used in a variety of circumstances: to cover excised wounds while waiting to re-harvest a donor site, to cover widely expanded skin grafts, to protect deep face burns while they heal, or as rescue for a failed skin graft.

Allograft may be available from regional Burn Centres and can be obtained commercially. Surgeons should satisfy themselves that allograft is obtained from an ethical and reliable source. Many burn units start tissue banks so they can maintain their own stock of allograft.

Porcine xenograft is less expensive than allograft, but does not adhere as well. It is used widely in China to cover widely expanded 'postage stamp' grafts and is very effective in that context. Cultural sensitivities may preclude it use in some places.

Engineered dressings such as transcyte[™] and Integra[™] can be very effective but tend to be costly and tricky to use successfully, particularly Integra[™]. Cultured autograft skin and spray-on skin cells have long been considered an attractive solution but have yet to see widespread clinical uptake.

Bland dressings may be used on non-infected burns, and where antibiotic containing dressing materials are in short supply may be the mainstay of burn dressings. A simple occlusive dressing with Paraffin gauze will provide comfort, prevent or delay bacterial contamination and provide a moist healing environment.

Honey dressings for burns were first described in ancient Sanskrit texts and remain a practical alternative to medicated dressings for un-infected burns. There is evidence that honey has antibacterial properties, contains proteolytic enzymes and promotes epithelial growth. It may be superior to SSD [12]. There has even been a Cochrane review of Honey dressings in burns that concluded that honey may improve healing times in superficial partial thickness burns compared with conventional dressings [13].

Honey is conventionally mixed with ghee or clarified butter. If this is not available, edible or mineral oil may be substituted. Warm and mix two parts honey and one part oil then pour over gauze sheets in a shallow pan, cover and cool and store in the dark. The sheets may then be lifted off one at a time to make dressings. Home-made paraffin gauze can be prepared in similar fashion.

There are a variety of materials used for burn dressings including povidone iodine, mercurochrome, gentian violet, tea, boiled potato skins, toothpaste, fish sauce, papaya and banana leaves. While there is little evidence to support their use there is also little evidence they cause any great harm. In some circumstances it may be helpful to work with local materials if there are no better alternatives at hand. However the practice of covering wounds with frankly noxious materials such as water buffalo dung should be discouraged.

Burns to the **perineum**, typically scalds, present a challenge in preventing their becoming contaminated with faeces. In severe cases faecal diversion by means of a temporary loop ileostomy or colostomy may be considered.

Hand burns are common and lead to severe disability if not treated well. A plastic bag or large fitting surgical glove filled with silver sulfadiazine is a good way of dressing a hand while maintaining the ability to keep the joints moving. Splint the hand in the safety position and remove the splint to range the joints, particularly the MCPs, as often as the patient will tolerate it (Figure 7).

Face burns are difficult to cover with occlusive dressings. To perform face care apply warm wet saline dressings for about 20 minutes to loosen dried wound exudate, lift off the gauze, clean the face and apply antibiotic ointment or SSD to raw areas. Repeat three or four times daily and combine with eye care. Facial hair should be clipped at the outset and then new beard growth should be shaved every two days to prevent build-up of exudate, infection and conversion to deeper skin loss. Likewise burned areas of scalp should be clipped and shaved periodically to prevent build up of difficult to remove accretions of scabbed infection. Ongoing hair growth implies the burn is partial thickness and with time will heal. Deeper face burns with eschar may occasionally go to theatre for a good scraping and debridment, often in association with another operation. If Transcyte or other engineered dressings are available, the face is the place to use them. For deeper facial burns allograft or xenograft gives wounds the optimum chance to heal

beneath. Burns to the eyelids may mandate early full thickness skin grafting to prevent blindness. Blepharoplasty, suturing together of the eyelids to protect vision is seldom effective as the sutures pull out, further damaging the edge of the lid.

The Exposure Method for treating large burns is sometimes the only option and should be understood. The conventional wisdom is that exposure done well is better than occlusive dressings done poorly. This method is best described in *Primary Surgery Vol II.*

NUTRITION IN BURNS

A large burn carries a nutritional stress factor of 2.1 meaning that the patient needs a little more than twice his daily calorie intake until the wounds heal. Children require 3g/kg protein daily and adults 2g/kg. An egg contains about 15g of protein so the traditional "3 egg" diet will be adequate for children up to 15 kg body weight. The great majority of burn patients cannot eat enough to meet their metabolic requirements and so supplemental feeding via a nasogastric tube is necessary. Commercial feeding solutions are available and expensive but homemade solutions can be just as good <u>(Appendix I)</u> [14]. In some situations, where early excision and grafting are impossible, good nutritional management (and dressings) may be the most that can be done for the patient.

OUTCOMES IN BURN CARE

Burn outcomes vary widely. Innumerable small superficial burns will go on to heal without medical intervention, whereas untreated small deep burns, especially in children, may lead to contractures, lifelong disability and visible disfigurement that may prevent the individual from being accepted in school, work or marriage. Large deep burns consume vast amounts of hospital resources and may still result in death or profound disability [15,16]. In situations where the availability of ICU space, operating time and burn care skills are limited more suffering will be averted by focusing scarce resources on small disabling burns than on large potentially lethal burns. The excision and grafting of small potentially disabling burns and the release of burn scar contractures is well within the capacity of small hospitals with skilled surgeons, whereas the ICU management of large burns and massive skin grafting may consume resources better spent elsewhere. As local skills and confidence grow surgeons are able to take on aggressive management of selected larger burns. An interesting recent study from South Africa has shown that early excision and grafting of burns both reduces length of stay and hospital costs in moderate sized burns [17].

<u>Appendix I</u> [14]

ANNEX 15. A Nutrition in major burns: calculating nutritional requirements Calorie requirements = Basal energy expenditure x stress factor x activity factor The basal energy expenditure is calculated as follows: $[66 + (14 \times \text{weight in kg}) + (5 \times \text{height in cm}) - (6.8 \times \text{age in years})]$ The stress factor for minor procedures is 1.3; for skeletal trauma, 1.35; for major sepsis 1.6; and for major burns 2.1. The activity factor is 1.2 for those in bed and 1.3 for those who are mobilizing. Women require about 4 % less than men for equal body size and age. Example For a 25-year-old male weighing 60 kg and 170 cm tall, in bed with a major burn, the calorie requirements = $[66 + (14 \times 60) + (5 \times 170) - (6.8 \times 25)] \times 2.1 \times 1.2 = 3,997$ kcal/day Protein, alucose and fat requirements Daily protein requirement for acute burns is 2 g/kg in adults and 3 g/kg in children. Protein provides about 4 kcal/g (120 g and 480 kcal in the example given above). Daily glucose requirement is about 6 g/kg/day in burns. Glucose provides 4 kcal/g (360 g and 1,440 kcal in the example). The difference between the calculated energy requirement (3,997 kcal) and that provided by protein and glucose should be made up with fat. Daily fat requirement = 3,997 kcal - 480 - 1,440 = 2,077 Kcal Each gram of fat provides 9 kcal, therefore, 2.077 Å \ddot{A} 9 = 231 g of fat The larger the volume and the higher the fat concentration the more likely the patient is to develop diarrhoea. In an adult with a major burn, 3 litres of feeds per day is a reasonable target; therefore for the patient in the example, a "cocktail" containing 40 g of protein, 120 g of glucose and 80 g of fat per litre should be prepared. Making a high-energy enteral feeding solution for burn patients Ingredients Glucose Protein Fat kcal Skimmed milk powder 110 g (244 ml) 44 g 40 g 385 Edible oil 80 g (80 ml) 80 g 720 Sugar 50 g (50 ml) 50 g 200 1 Banana (15 mEq potassium) 25 g 110 Add: Salt 3 q Calcium containing antacid 3 tablets Multivitamin tablet 1 daily Ferrous sulphate + folic acid tablets Codeine 30 – 60 mg per litre provides analgesia and reduces diarrhoea Eggs contain 15 g of protein each: beware of salmonellosis from raw eggs Supplement tube feeds with cooked eggs fed by mouth when possible Boiled and filtered water to make 1,000 ml of solution Total 1,415 kcal per litre Make a paste of milk powder with a little water; add sugar, salt, crushed tablets and oil. Slowly add more water while mixing well; add mashed banana and mix thoroughly (using a blender if possible). Filter the mix through a gauze compress and refrigerate. Irrigate the feeding tube regularly with water to keep it from blocking. Use within 24 hours

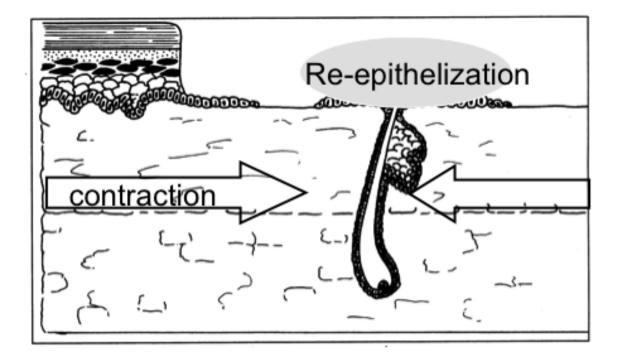


Figure 2 Donor site healing by re-epithelialization

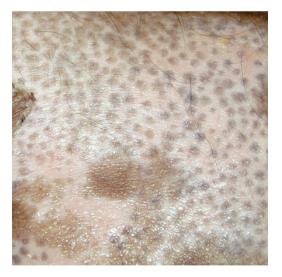


Figure 3. An old neglected burn of the arm in a teenage girl. Note how the combination of burn scar contracture and normal longitudinal bone growth have distorted the wrist anatomy.



Figure 4. Emergency cricothyroidotomy following electrical flash burn. Note the spackled appearance of the skin.



Figure 5. Leave the endotracheal tube long to allow for facial swelling.



Figure 6. Incision placement for escharotomy of the hand

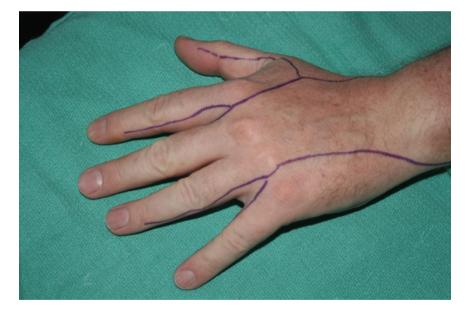
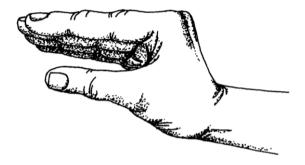
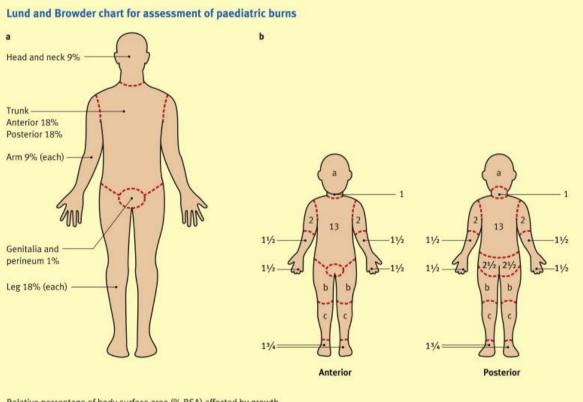


Figure 7 The "safe" position for splinting a burned hand.



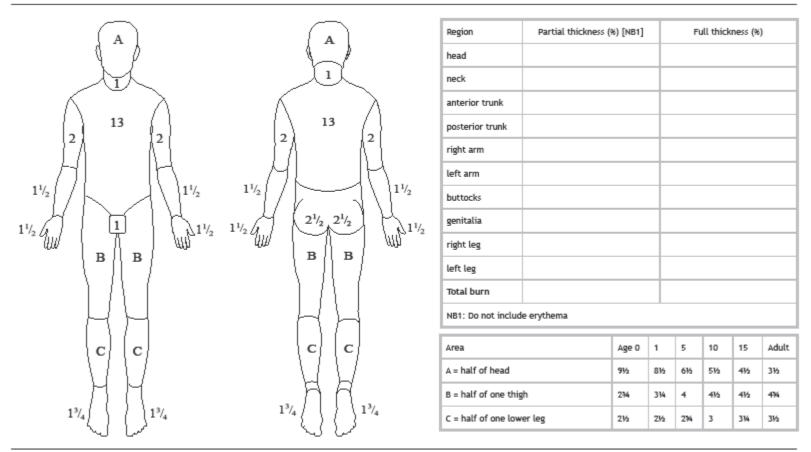


Relative percentage of body surface area (% BSA) affected by growth

Body part	Age (years)				
	0	1	5	10	15
$a = \frac{1}{2}$ of head	91/2	81/2	61/2	51/2	41/2
b = ½ of one thigh	23/4	31/4	4	41/4	41/2
c = 1/2 of one lower leg	21/2	21/2	23/4	3	31/4

Therapeutic Guidelines

Lund and Browder chart for calculating the percentage of total body surface area burnt (Fig 14.19)



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Likewise the fire injury death rate in India in 2004 is given as 13.3/100,000 in the Global Burden of Disease, 2009 Disease & Country Estimates

http://www.who.int/entity/healthinfo/global burden disease/gbddeathdalycountryestimates2004.xl s and as 8.3/100,000 for 2000 in Peden M, McGee K, Sharma G. *The injury chart book: a graphical overview of the global burden of injuries*. Geneva, World Health Organization, 2002. http://whglibdoc.who.int/publications/924156220x.pdf

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14. OBSTETRIC OBSTACLES

FRIEDA E DREYER, PAUL MENSAH

OVERVIEW

In district general hospitals surgeons will often be asked to help look after pregnant patients who are critically ill. In many parts of the world general surgeons will be responsible for Caesarean sections, and then need to know something about the pathology that necessitates such surgery. Surgical pathology can present at any time during pregnancy and pregnant patients can be involved in road traffic accidents. Sometimes a general surgeon is simply the doctor on site with most experience of managing critical illness. It is therefore important to know how problems in airway management, breathing, circulation and sepsis can be different in pregnancy and labour.

ABCS OF OBSTETRICS

INTRODUCTION

Physiological and anatomical alterations develop in many organ systems during the course of pregnancy and delivery. The earlier changes are due to the increasing levels of pregnancy hormones, particularly progesterone and oestrogen, and the metabolic changes brought on by the foeto-placental unit. Later changes are anatomical and mechanical in nature, due to the increase in size of the uterus. These changes bring new challenges to the obstetric team and require a modified approach to the critically ill obstetric patient compared to the general population.

The anatomical and functional changes affect all organ systems as summarised in Table 1. These changes in turn affect mechanisms of trauma and resuscitation, as discussed in more detail later.

Table 1:	PHYSIOLOGICAL CHANGE	EFFECT
RESPIRATORY	 ↑Respiratory rate ↑Oxygen consumption (by 20%) ↑Tidal volume ↑Minute ventilation ↑Laryngeal angle Pharyngeal and nasal oedema ↓Functional residual capacity (by 25%) ↓Arterial PaCO₂ ↓Serum bicarbonate 	 ↓Buffering capacity ↓Tolerance to hypoxia ↓Buffering capacity Compensated respiratory alkalosis Difficult/failed intubation Difficult/failed intubation ↓Buffering capacity ↓Buffering capacity Respiratory alkalosis
CARDIOVASCULAR	 ↑Plasma volume (by 40-50%) ↑Erythrocyte volume (by20%) ↑Cardiac output (by 40%) ↑Heart rate ↑Clotting factors (VII, VIII, IX,, 	Dilutional anaemia ↑Circulation CPR demands Hypercoagulable state
	 ↑Dextrorotation of heart Oestrogen effect on myocardial receptors 	↑ECG left axis deviation Supraventricular arrhythmias
	Aortocaval compression	Supine hypotension and ↓cardiac output by 30%
	↓Arterial BP	Susceptible to cardiovascular insult
	↓Systemic vascular resistance	↓BP and sequesters blood during CPR
	↓Colloid oncotic pressure	Susceptible to third space losses
	↓Pulmonary capillary wedge pressure	Susceptible to pulmonary oedema
	↓Uterine perfusion in response to haemorrhage	↑Fetal risk during haemorrhage
GASTROINTESTINAL	↓Motility ↓Gastric emptying Compartmentalisation ↓Gastroesophageal sphincter tone	↑Risk of aspiration ↑Risk of injury with upper abdominal trauma
BREAST	↓Chest wall compliance secondary to breast hypertrophy	↑CPR compression force

GENITOURINARY	↓Bladder emptying	↑Risk of infection
	↑Ureteral dilatation (especially on right)	Physiological abnormality of radiographic studies

AIRWAY PROBLEMS IN PREGNANCY

Anatomical and physiological changes in pregnancy place the patient at increased risk of airway management problems. Failure to manage the airway appropriately in a gravid patient potentially threatens not one life, but two, as maternal complications are the leading cause of foetal insult and death. This risk can be minimised by understanding these anatomical and physiological changes, and taking the correct precautions and preparation in airway management.

The average patient gains about 12kg in bodyweight during her pregnancy. About a third of this weight gain is from the foetus, placenta and amniotic fluid. The other two thirds are down to uterine hypertrophy, increased blood volume and interstitial fluid, breast enlargement and body fat stores in preparation for breastfeeding. This leads to an overall increase in body mass index (BMI) which has previously been shown to increase the risk of difficult intubation.

The elevated progesterone levels in pregnancy lead to generalised oedema, including of airway structures. Mucosal oedema of the tongue, larynx, pharynx and trachea can impair visualisation during laryngoscopy and further complicate intubation. The anaesthetist's view can be compromised further by bleeding from the airway during difficult intubation secondary to a more fragile airway mucosa.

The supine position can complicate anaesthetics. Large breasts can fall against the neck and make handling of the patient's airway more difficult during laryngoscopy. The enlarging uterus causes displacement of the intra-abdominal organs and ultimately the diaphragm. The displacement of the diaphragm lowers the residual volume, expiratory reserve volume, and the functional residual capacity by up to 70% in the supine position. Ultimately this leads to hypoxia more quickly due to the increased oxygen demand of the pregnant patient. It is therefore important to pre-oxygenate the patient with 100% oxygen prior to rapid sequence intubation. If the patient has to remain in the supine position the uterus should be manually displaced to the left lateral position by creating a wedge under the right hip or by tilting the theatre table to at least 15 - 30°. This also relieves aortocaval compression which helps to improve venous return to the heart.

Changes in the gastrointestinal tract can add to airway problems in pregnancy. The reduced tone of the lower oesophageal sphincter and delay in gastric emptying increases the chance of passive regurgitation and aspiration of gastric content during intubation. These risks can be minimised at time of anaesthesia by maintaining cricoid pressure and using rapid sequence intubation, rather than bag-mask ventilation. Bag-mask ventilation further increases the risk of gastric insufflation due to the lowered gastro-oesophageal sphincter tone, but oxygenation by any method always takes priority when intubation is difficult.

Every obstetric airway should therefore be considered a difficult airway. By anticipating difficult anatomy and rapid de-saturation, complications can be avoided or identified early, and managed appropriately.

BREATHING PROBLEMS IN PREGNANCY

Of all the physiological changes in pregnancy, altered respiratory function may be one of the most obvious to the mother.

Physiological dyspnoea during pregnancy is quite common and affects about 60% of mothers on exertion and 20% at rest. It can be explained by the physiological changes previously discussed. It is important to distinguish between this physiological dyspnoea, and breathlessness caused by disorders complicating pregnancy. The presence of other symptoms and signs of cardiopulmonary disease indicates a possible pathological underlying disorder. Some of these disorders are discussed in more detail:

AMNIOTIC FLUID EMBOLISM

Amniotic fluid embolism (AFE) is a rare (1:8000-80000 births) obstetric emergency with a high mortality rate of 10-80%.

It is associated with labour and delivery but also with uterine trauma, uterine manipulation or the early postpartum period. Amniotic fluid, fetal squamous cells, hair or other debris enter the maternal circulation, obstruct pulmonary vessels and lead to pulmonary hypertension. Women often present with severe dyspnoea, hypoxemia and cardio-respiratory collapse. Less commonly they may present with haemorrhage and disseminated intravascular coagulation or fetal compromise.

AFE is often a diagnosis of exclusion based on clinical presentation. Other possible causes of cardio-respiratory collapse e.g. pulmonary thromboembolism, septic shock, placental abruption, tension pneumothorax and myocardial ischaemia should be considered. Diagnosis is only confirmed at autopsy with the finding of fetal squamous cells in the maternal pulmonary circulation.

Treatment is often supportive and involves routine resuscitation with prompt oxygenation, ventilation and inotropic support.

PULMONARY OEDEMA

Another uncommon event in pregnancy is pulmonary oedema. It's most common causes include the use of tocolytic agents, underlying cardiac disease, fluid overload and preeclampsia. The patient is further predisposed to the development of pulmonary oedema by the physiological changes of pregnancy and can be further precipitated by tachycardia and increased cardiac output during labour.

The standard approach to treatment includes fluid restriction, oxygenation, diuresis and vasodilator therapy.

PULMONARY EMBOLISM

Venous thromboembolism (VTE) is 20% more common in the pregnant patient compared to non-pregnant women of a similar age.

Obesity (BMI>30) remains the most important risk for VTE. Other risk factors include advanced maternal age, haemorrhage >1litre, sepsis, pre-eclampsia, multiparity, previous VTE, thrombophilia, smoking and delivery by caesarean section.

The signs and symptoms of pulmonary embolism (PE) may be difficult to interpret in the healthy pregnant patient due to the physiological changes that lead to tachypnoea and dyspnoea. Women are at increased risk of PE from the first trimester until the end of puerperium, and therefore there should be a low threshold for investigation for any woman who present with chest symptoms for the first time in pregnancy.

Arterial blood gases will show a decreased PaO₂ and chest X-ray and electrocardiogram may help support the diagnosis. The first objective test should be compression ultrasonography of the lower limbs. If leg studies are negative then proceed to ventilation-perfusion lung scanning. Pulmonary angiography may be necessary if clinical suspicion remains high.

Treatment with low molecular weight heparin (LMWH) should be started immediately if VTE is suspected and can be continued until diagnosis excluded. LMWH is thought to be safe in pregnancy and does not cross the placenta. Warfarin should be avoided during pregnancy. It is teratogenic, especially during the first trimester and increases the risk of haemorrhage and placental abruption.

Guidance regarding the use of LMWH can be found on the Royal College of Obstetricians and Gynaecologists website.

ACUTE RESPIRATORY DISTRESS SYNDROME

Obstetric patients are at an increased risk of developing acute respiratory distress syndrome (ARDS). AFE, chorioamnionitis, trophoblastic embolism and placental abruption can all lead to acute lung injury. Other pulmonary insults leading to ARDS include gastric aspiration, pneumonia, air embolism and massive haemorrhage. The lowered albumin levels in pregnancy and reduced oncotic pressure lowers the critical pulmonary pressure at which pulmonary oedema develops.

CIRCULATION PROBLEMS IN PREGNANCY

Pregnancy is associated with an increase in blood volume. A loss of 10-15% of blood volume (500-1000ml) is usually well tolerated as young fit women are able to compensate and maintain their basic cardiovascular parameters until about 30-40% of the blood volume is lost. Table 2 summarises the changes in these parameters in relation to blood loss.

Blood Loss	Heart Rate	Systolic BP	Tissue Perfusion
10-15% (500-750 ml)	Increased	Normal	Postural hypotension
15-30% (750-1500ml)	Increased +	Normal	Peripheral vasoconstriction
30-40% (1.5-2 litre)	Increased ++	70-80 mmHg	Pallor, oliguria, confusion, restlessness
40%+ (>2 litre)	Increased +++	<60 mmHg	Collapse, anuria, dyspnoea

Table 2:

Pulse rate (as opposed to blood pressure) is the most accurate parameter to assess the degree of blood loss as the young pregnant woman can remain normotensive until a significant amount of blood loss has occurred; the degree of haemodynamic instability may be out of proportion the visually estimated blood loss. The degree of concealed blood loss may not be immediately obvious in cases such as concealed abruption and uterine scar rupture.

Blood loss greater than 1000ml can lead to acute hypovolaemia and shock, disseminated intravascular coagulopathy, hypoxia and multiorgan failure.

MASSIVE OBSTETRIC HAEMORRHAGE

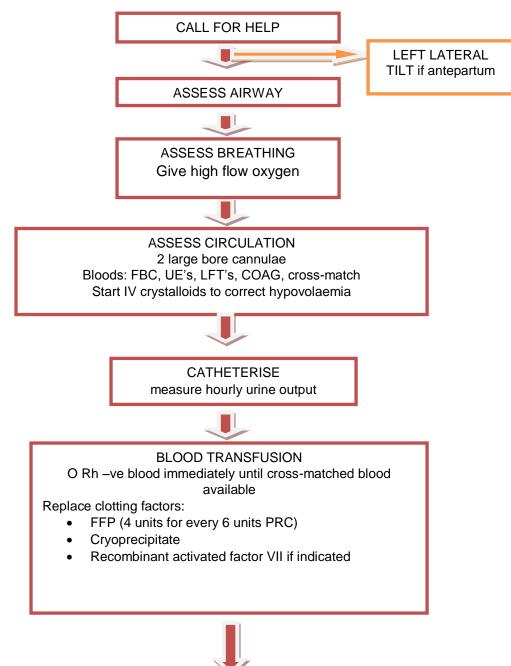
Massive obstetric haemorrhage refers to a loss of 30-40% (2000ml) blood volume. Coagulopathy secondary to hypovolaemia will worsen blood loss, or coagulopathy can be the primary cause of severe blood loss and hypovolaemia.

The exact management will depend on the cause of bleeding (Table 3), but basic resuscitation measures should be followed (see Figure 1).

Table 3:

Antepartum	Intrapartum	Postpartum
Placental abruption Placenta previa Severe chorioamnionitis, sepsis Severe pre-eclampsia and hepatic rupture	Uterine rupture AFE Placenta accreta Ruptured vasa previa Surgical complications – uterine incision extensions, tears	Primary Postpartum Haemorrhage Atonic uterus Genital tract trauma Coagulopathy Retained products of conception Secondary Postpartum Haemorrhage Infection/ retained products of conception Gestational trophoblastic disease

Figure 1 = ALS Algorithm in Pregnancy:



MEDICAL MANAGEMENT

- 500mcg Ergometrine IV/IM (take care in hypertensive patients)
- 40 IU Oxytocin infusion
- 800-1000mcg Misoprostol PR
- 250mcg Carboprost in thigh or directly into myometrium (15minute intervals up to 4 doses)
- All these should be accompanied by attempts to manually help contract the uterus e.g. rubbing up contractions/ bimanual compression

SURGICAL MANAGEMENT - Examine Under Anaesthesia

- Empty uterus (foetus, placenta, clots, retained products of conception)
- Repair genital tract injuries (tears/ episiotomy)
- Bakri/ Rusch balloon
- Laparotomy and B-lynch suture
- Uterine artery or internal artery ligation
- Total or subtotal hysterectomy

MODIFICATIONS OF CPR IN PREGNANCY

There are a number of reasons why the processes of cardiopulmonary resuscitation are more difficult to perform and can be less effective in the pregnant patient. This may be the case from about 20 weeks gestation and will become more marked as the mother approaches term. Most of these reasons have briefly been discussed earlier in this chapter. The basic or advanced life support algorithms should be followed with some modifications as summarised in Table 4.

Table 4:

ACTION	RATIONALE	
BASIC LIFE SUPPORT Manual uterine displacement, or 30° left lateral tilt ↑Chest wall compression force Cricoids pressure if assistance available	↓Aortocaval compression ↓Chest wall compliance with breast hypertrophy and diaphragmatic elevation ↓Gastric aspiration	
ADVANCED CARDIAC LIFE SUPPORT Early tracheal intubation, use short laryngoscope handle and smaller endotracheal tube Perimortem caesarean section at 4 minutes* Consider other causes e.g. magnesium toxicity Consider left wide paddle/ adhesive pad/ breast displacement	breast hypertrophy, diaphragmatic elevation ↓Aortocaval compression Tocolytic therapy Dextrorotation of heart, breast hypertrophy	
NO CHANGE Defibrillation regimen Pharmacologic therapy	Early return of effective maternal circulation	

<u>*Perimortem caesarean section</u> should be considered as a resuscitative procedure for cardiac arrest in a near-term pregnancy. Delivery of the fetus will relieve aortocaval compression and significantly improve the chance of maternal resuscitation by reducing maternal oxygen consumption, increase venous return, make ventilation easier and allow CPR in the supine position.

Evidence from literature review of maternal and foetal physiology suggests that the caesarean should begin within four minutes of cardiac arrest and delivery accomplished by five minutes, as pregnant women develop anoxic damage by 6 minutes following cardiac arrest. CPR should continue throughout caesarean section and afterwards as this increases chances of successful maternal and neonatal outcome.

Perimortem caesarean section should not be delayed while trying to listen for foetal heart sounds or ultrasound to check gestation. A uterine fundus four centimetres above the pubic symphysis may indicate a singleton pregnancy of gestation from 23 weeks. Personnel with the appropriate skill and equipment (surgical knife and forceps should be sufficient) should perform the procedure, and a sterile field and transfer to theatre is not thought to improve survival. Once resuscitation is successful and the mother gains cardiac output, appropriate sedation/ anaesthetic and transfer to theatre for completion of procedure is indicated.

SEPSIS IN PREGNANCY

Sepsis in pregnancy is still the leading cause of direct maternal deaths in the UK, according to the most recent (2006-2008) triennial report of maternal deaths from the UK Centre for Maternal and Child Enquiries (CMACE). Infection and the complications of sepsis are among the most common causes of severe maternal morbidity and mortality worldwide.

The course of sepsis is often insidious and healthcare professional should be aware that pregnant women with serious illness may appear deceptively well before suddenly collapsing, often with little or no warning. Once sepsis is established, it may quickly become irreversible and deteriorate into septic shock, disseminated intravascular coagulation and multi-organ failure.

The most recent CMACE report emphasises early recognition of the following signs and symptoms of maternal sepsis as the altered physiology of pregnancy may obscure the early signs of shock, complicating early diagnosis and treatment:

- Pyrexia
- Hypothermia
- Persistent tachycardia >100 beats per minute
- Tachypnoea (respiratory rate >20 breaths/minute)
- Leucopoenia (WCC <4x10⁹/l)
- Diarrhoea and/or vomiting
- Lower abdominal pain
- Abnormal or absent fetal heart beat
- Offensive vaginal discharge

If sepsis is suspected, regular frequent observations should be made using Modified Early Obstetric Warning Scoring (MEOWS) charts, to aid early recognition and treatment of women who have or who are developing a critical condition.

Sepsis in pregnancy is generally considered under the description of 'puerperal sepsis' and occurs at any time between the rupture of membranes or labour, and the 42nd day postpartum. This generalised term includes genito-urinary, extra-genital and incidental infections:

- Genito-urinary: endometritis; retained products of conception; surgical sites e.g. caesarean section scar, episiotomy site; urinary tract infections, peritonitis.
- Extra-genital (but still related to the birth process): mastitis/breast abscess, thrombophlebitis
- Incidental: respiratory tract infection, malaria, enteric fever.

Infection must be expected or actively ruled out when a pregnant or recently delivered woman becomes pyrexial or develops persistent abdominal pain or bleeding. Blood culture is a key investigation and should be obtained prior to treatment, but antibiotic treatment should not be delayed by waiting for microbiology results. Administration of intravenous broad spectrum antibiotics is recommended within one hour of suspicion of severe sepsis. Other microbiology investigations should be guided by the suspected focus of infection and may include high vaginal swabs, mid stream specimen of urine, throat swabs and cerebrospinal fluid. Radiological investigations such as chest X-ray or ultrasound scan may guide diagnosis.

The most common organisms identified in pregnant women dying from sepsis are Lancefield group A beta-haemolytic *Streptococcus* and *E.coli*. Other common infections may be due to a mix of Gram-positive and Gram-negative organisms, especially in chorioamnionitis. Urinary sepsis, preterm premature rupture of membranes and cervical cerclage is often complicated by coliform infections. Anaerobic infections include *Clostridium perfringens*, *Peptostreptococcus* and *Bacteroides species*.

Prompt treatment with high dose broad spectrum antibiotics is required if genital tract sepsis is suspected. In addition to antimicrobial treatment, the focus of infection should be sought and removed early e.g. deliveries of the fetus if chorioamnionitis is suspected, evacuation of retained products of conception or laparotomy.

Choosing the most appropriate antibiotics may be difficult and early involvement of a microbiologist should be considered. Broad spectrum antibiotics that may be used when the woman is not critically ill include co-amoxiclav plus metronidazole, or cefuroxine plus metronidazole. In cases of penicillin or cephalosporin allergy one can use clarithromycin or clindamycin plus gentamicin. In severe sepsis or septic shock, use tazobactum or ciprofloxacin plus gentamicin. When there is a risk of MRSA add teicoplanin or linezolid.

If the mother is critically ill the fetus should be monitored continuously with cardiotocography. Delivery of the baby should be considered if it is beneficial to the mother, or if the cardiotocograph shows changes that suggest foetal compromise. If preterm delivery is anticipated, cautious consideration should be given to the use of corticosteroids to aid foetal lung maturity. Epidural and spinal anaesthesia should be avoided in cases of severe sepsis and general anaesthetic will be required for delivery by caesarean section.

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15. PAEDIATRIC PITFALLS

DAVID R BALL

Surgeons in district hospitals will generally look after children who present with surgical conditions within their areas of expertise. In principle the priorities of resuscitation, assessment and organ support are the same in children as in adults, but the surgeon has to remember certain physical, physiological and psychological differences that can offer "pitfalls" in management of critically ill paediatric patients. In certain situations critical care management will be very specific e.g. with burn injuries. This will be discussed in the next critical care review article. Management of specific paediatric surgical pathology is also not discussed here; readers are referred to a complete paediatric surgical textbook for such information e.g. **Paediatric Surgery: a Comprehensive Text for Africa** (available online from <u>www.global-help.org</u>).

INTRODUCTION

The management of the paediatric patient follows the same principles as in the adult, using a structured, systematic approach. There are, however, important differences that, if not managed, will result in *"paediatric pitfalls"*.

For this article, we describe:

- A *newborn*: a baby at time of birth;
- A *neonate*: as less than one month of age;
- An *infant*: between one month and one year of age;
- A *small child*: between one year and 8 years; and
- A *large child*: over 8 years.

When the term "**young**" is used in this article, this refers to an infant, neonate and small child.

The development of a neonate from birth through childhood to adulthood is a process of growth and change to maturity. This is manifest in many ways. One practical way to consider these changes is a division into *physical, physiological* and *psychological* change, all of which can influence effective delivery of clinical care.

PHYSICAL CONSIDERATIONS

Size and shape and anatomy vary greatly with age. In health there is progressive increase in size and weight with age.

A general consequence of managing the smaller paediatric patient is that equipment needs to be matched to the size of the patient.

It is important to know the patient's weight because fluid regimes, drug doses and defibrillation energy (in cardiac arrest, 4 Joules/kg) are usually based on this. Fluid therapy for burn injury is, however, based on patient body surface area (BSA). Wherever possible, weight should be measured, bearing in mind that this value, if taken for an ill child, may not reflect healthy weight. A healthy full term newborn weighs about

3.5kg, at one year about 10 kg and, with health, increase with age. One formula to calculate weight is:

 $2 \times (age in years + 4) = body weight in kg.$

This formula applies to those over one year and under 12 years.

Smaller, younger patients have a higher surface are to volume ratio. This is 2.5 times more for a neonate compared to an adult. Therefore, transfer of heat is relatively higher (to or from patient, dependent on the ambient heat gradient). Evaporative losses also increase. The neutral thermal environment (the temperature at which there is minimal heat loss) is 34°C for the premature baby, 32°C for the term newborn and 28°C for an adult.

Shape varies with age. A neonate has a larger head to body size. A consequence of this is that scalp wounds in infants and small children can result in hypovolaemia, which is unusual in adults. For a newborn the surface area of the head is about 10% of total body area, falling to less than 5% in adulthood. This is matched by a fall in the head length to body length ratio from 1:3 to 1:7 by adulthood. These changes in body proportion with age are important in measuring burn area.

Compared to the adult there are relevant anatomical differences which can alter clinical management. These differences are most apparent for the newborn, infant and small child. This is particularly important for **airway management**, since a variety of complicating factors may interact to hinder effective, time-sensitive treatment. These factors are generally simple to manage as long as these are always considered. These can be summarised as follows, from peripheral to central, with the clinical implications in *italics*:

- larger occiput; supine positioning results in neck flexion, airway obstruction more likely and direct laryngoscopy more difficult. Padding under the shoulders may help.
- infants are edentulous, mask ventilation may be impaired.
- children have decidual teeth, which may be loose and may then be inhaled.
- relatively larger tongue, less oro-pharyngeal volume, more prone to obstruction.
- produce more secretions, obscuring airway view and may be aspirated.
- infants have a relatively long, floppy epiglottis; *direct laryngoscopy more difficult*. A straight-bladed laryngoscope is usually used for tracheal intubation in infants.
- infants and small children have a higher, more anterior larynx, *direct laryngoscopy more difficult*.
- narrower laryngeal inlet, risks injury with bleeding or oedema. Diameter of tracheal tube important.
- shorter trachea, length of tracheal tube important to avoid bronchial intubation.

Selection of tracheal tube, length and diameter is important. One formula for tube internal diameter (ID) and length (of insertion to lips) of an oral tube, based on age, is:

- Neonate to 3 months: 3.0mm, length 10 cm
- 3 to 9 months: 3.5mm, length 11 cm
- 9 to 21 months: 4.0mm, length 12 cm
- >21 months: [(age in years)/4 + 4]mm; length = [(age in years)/ +12]cm.

These calculations are not very accurate, with suboptimal size selection reported for nearly one in three cases.

Traditionally it has been advised that paediatric tracheal tubes were cuffless (unlike those for adults). This was due to technical issues regarding tube and cuff wall thickness. This meant that these tubes leaked with positive-pressure ventilation and provided less protection from aspiration. Newer paediatric tubes with thin-walled cuffs are available but are expensive. Since the inflated cuff may produce excessive pressure on the airway mucosa (risking damage), it is advised that cuff pressure is measured and maintained below 20cm water.

Other important anatomical considerations are relevant to vascular access. Paediatric veins (peripheral or central) are relatively small and occasionally obscured by adipose tissue. Vascular access may be technically demanding. For the critically ill or injured paediatric patient peripheral venous access is needed urgently. Central venous access is not advised for initial resuscitation for a number of reasons: the procedure may be technically challenging, time consuming and risks important complication, such as pneumothorax or vascular haematoma. If peripheral access cannot be readily achieved (within minutes), the intraosseus approach is advised, which may usually be continued for up to 24 hours [1].

Suitable sites include the proximal tibia (anterior surface 2-3cm below tibial tuberosity, avoiding the epiphyseal plate) and the distal femur (anterolateral surface 3 cm above lateral condyle). Do not use a fractured limb.

Fluid and drug treatments can then be easily given, but these will not flow under gravity and requires administration with pressure e.g. using a syringe while observing for extravasation. Additionally, marrow may be aspirated for blood testing, including standard biochemical analysis. The laboratory should be told that the sample is from marrow. There are two reasons: firstly, marrow may block the testing machines and must be processed differently from serum, and secondly because the sample (if assumed to be blood) may be interpreted as from a leukaemic patient because of the high number of immature cells present.

PHYSIOLOGICAL CONSIDERATIONS

There are two major general differences in physiology between the paediatric and adult patient: *metabolic activity* and *physiological reserve*. These differences are most apparent for the very young (neonate and infant), less so for the small child and again less marked for the older child.

Because of these differences it is important not to assign "adult" values of normality to the paediatric patient. It is important to remember that all values may vary (as in adults but with a greater relative range of variation) and they should be reviewed in the context of other clinical findings.

Metabolic activity: Compared to the adult, metabolic rate is higher, with higher oxygen consumption (at rest, 200ml/kg/min, about two or three times higher than an adult) and carbon dioxide production per kg. There is proportionately greater heat production (neonatal metabolic rate is twice that of an adult). This is a result of growth and more physical activity. The transport functions of the cardio-respiratory system are generally more active.

So, compared to the adult, the physiology of a child is "hyperdynamic", with tachycardia, tachypnoea and vasodilation.

Physiological reserve: In general, reduction in physiological reserve means that the paediatric patient is less able to compensate for illness or injury. This immaturity is seen in all organ systems, more obvious for the neonate, infant and smaller child:

<u>Respiratory system:</u> The alveolar system is markedly immature, mostly thick-walled with only 10% of the adult number. Airway resistance is much higher (30 cm water/litre/sec compared to 2 cm water/litre/sec for an adult), and lung compliance lower (i.e. the lungs are "stiffer"). The work of breathing is about 15% of total oxygen consumption, compared to 5% in the adult. For the young patient, the closing capacity (the volume of the lungs at which small airways collapse) is about the same as the functional residual capacity (the resting volume of the lungs). This means that the lungs are limited in their capacity to enhance gas exchange. This, together with the high metabolic demand for oxygen results in a lower arterial oxygen tension than in the adult (about 9.5kPa compared to about 13kPa breathing air at sea level).

So, at birth, respiratory rate is about 30-40/ minute, falling to 15-20/ minute in the older child.

A consequence of paediatric dependence for oxygen is the approach to life support. Paediatric resuscitation guidelines advise (up to) five "rescue" breaths (lung inflations) before starting chest compressions. This is not advised for the adult patient.

<u>Cardiovascular system:</u> Compared to the adult, cardiac index (cardiac output referenced to body surface area) and heart rate are higher, systemic resistance and blood pressure are lower. Infant heart rate is between 110 and 160/ minute, falling to 60-80/ minute in the older child. For the young patient, myocardial reserve is limited. Cardiac output is "rate dependant", since increases cannot be achieved by increase in stroke volume. Bradycardia is dangerous in the young. With little ability to compensate by an increase in stroke volume, cardiac output falls precipitately. If heart rate falls to 60/min or below in the young, life support with cardiac compressions should be started. Systolic blood pressure in the infant is 70-90 mmHg, 100-120mmHg in the older child. For this measurement, the size of cuff (for both auscultation and automated devices) should be measured as cuff width >80% of the upper arm length and >40% of upper arm circumference.

One formula to estimate expected systolic blood pressure is:

Systolic blood pressure (mm Hg) = 80 + (patient's age in years X 2)

An important consideration is the response of the paediatric patient to hypovolaemia. In general, blood pressure is maintained even with a high grade of shock, followed by a precipitate fall in the pre-terminal phase. Therefore blood pressure measurement has little diagnostic value in managing the paediatric patient. More useful measures are those related to perfusion such as skin temperature, capillary refill time and urine output.

The response to septic shock is also different than in adults [2]. Most adults present with a hyperdynamic reponse to sepsis ("warm shock"). These patients have a tachycardia with vasodilatation. Paediatric patients, however, more often present with "cold shock". Since there is a relative inability to raise heart rate to restore cardiac output a compensatory response is vasoconstriction to maintain cardiac filling and output. These patients are often severely hypovolaemic. Aggressive fluid therapy, systemic antibiotics

and adrenaline infusion (peripherally if there is no central vascular access) is indicated for management of paediatric "cold shock".

<u>Haematological system</u>: at birth, blood volume is 90 ml/kg, decreasing to 80 ml/kg at age 5 and 70 ml/kg in adulthood.

<u>Hepatobiliary system:</u> liver function is immature at birth. There is relatively less protein synthesis with reduced plasma proteins and a lower capacity for drug binding. This results in higher free fractions and drug action is relatively more potent. Some drugs, such as diazepam and vitamin K displace bilirubin from protein binding sites with resulting jaundice. Intrahepatic metabolism is also less developed, so that drugs such as morphine have lower clearance and therefore prolonged activity. Liver growth develops quite rapidly, so by about two years of age the relative size of the liver is about twice that of the adult.

<u>Nervous system</u>: at birth, most of body fat is within the central nervous system. Lipid soluble drugs (which include most anaesthetic agents) reach equilibration quicker than in older children and adults. The blood-brain barrier is less developed allowing greater drug penetration with increased activity and potential for toxicity. Neuronal cell metabolism requires a constant source of glucose. Only in periods of prolonged starvation can the older brain adapt to using ketone bodies as an alternative source of fuel. This means that hypoglycaemia is poorly tolerated, with seizures. One treatment schedule for hypoglycaemia (<3.3 mmol/l) is 5ml/kg of 10% dextrose by intravenous or intraosseus route.

<u>Renal system</u> is also immature at birth, with relatively lower capacities for glomerular filtration and tubular reabsorption. The ability to handle sodium (most drugs are formulated as the sodium salt) and water loads is reduced, so oedema and overload are a risk. Drugs excreted by the kidney (e.g. penicillins, morphine) may accumulate. Renal capacity improves quickly during the first year of life.

<u>Fluid therapy:</u> For the neonate and infant, there is proportionately more body water than the adult (80%, 75% and 65% of total body weight respectively). This is contained within an expanded extracellular volume (35%, 30% and 20% respectively). Intracellular volume remains constant at 40% and so does plasma volume at 5%.

Fluid therapy (with electrolyte and energy) concerns management of fluid *deficit*, ongoing *losses* and *maintenance* needs. The first two vary greatly with pathology. Regarding maintenance for infants and children, one formula is shown in Table 5:

Body weight (kg)	Fluid need per day (ml/kg)	Fluid need per hour (ml/hr)
First 10 kg	100	4
Next 10 kg	50	2
Subsequent kg	20	1

Table 5: Maintenance fluid requirements for infants and children (not neonates):

For example: a 25kg child is (10kg + 10kg + 5kg):

A. First 10 kg: (10 X 4)	= 40ml/hr	
B. Next 10kg: (10 X 2)	= 20ml/hr	
C. Subsequent 5 kg (5 X	(1) = 5ml/hr	
ach haur cha would nood t	bo cum of (Λ)	P + C = 40 + 20 + 5

For each hour she would need the sum of (A + B + C = 40 + 20 + 5) = 65 ml/hr.

There are a variety of fluids available. One example is half-strength Hartmanns solution (Ringers) with 5% dextrose.

Fluid management of the neonate is different, since less fluid is needed for the first week of life. One scheme is shown in Table 6.

Day of life	Maintenance fluid needed (ml/kg/day)
1	Zero
2 and 3	50
4 and 5	75
6	100
7	120

Table 6. Maintenance fluid requirements for the neonate:

PSYCHOLOGICAL CONSIDERATIONS

The two main psychological considerations relate to *communication* and *compliance* with treatment, which generally is easier as the child gets older. For the young patient, verbal communication is not possible, co-operation may be limited and some aspects of care, such as intravenous access, may be more difficult. For the older child, verbal communication is often possible, but the child may lack "capacity" to understand treatment and its implications.

Consent for treatment of the paediatric patient should be sought from parent or guardian. When a child attains capacity to consent, they should be actively involved in this process.

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16. ANAESTHESIA FOR SURGEONS

16A.SAFE PRACTICE OF ANAESTHESIA

DAVID R BALL

INTRODUCTION

Anaesthesia achieves two therapeutic goals:

- provides safe conditions for effective surgery and
- *protects* the patient from the rigours of surgery, since this is a controlled form of trauma.

The term *"anaesthesia"* stems from the Greek "an" without and "aesthesia", feeling. There are three aims: safety, efficacy and reliability. There are two main forms: general anaesthesia and local anaesthesia, and they may be combined. The main features of general anaesthesia are unconsciousness, muscular relaxation and reflex suppression; this forms the so-called "triad of anaesthesia". Suppression of somatic reflexes results in muscular relaxation; suppression of autonomic and endocrine reflexes result in the reduction (but not abolition) of the "stress

response". This is a variety of cardiovascular effects (often sympathetic, such as tachycardia, hypertension and vasoconstriction), endocrine effects (secretion of stress hormones, such as catecholamines and cortisol) and immune effects.

The variety of local anaesthetic techniques are covered in part 19.B. as mentioned, they may be combined with general anaesthesia and where appropriate, sedation (covered in part 19.C).

For any form of anaesthesia, both technical and non-technical skills must be applied appropriately. Technical skills involved the correct use of equipment, drugs and techniques. Non-technical skills involve appropriate communication, decision-making and teamwork.

Clear communication, verbal and written, is important at all times.

THE PHASES OF ANAESTHESIA

Anaesthetic practice may be considered in three stages: **pre-operative**, **peroperative**, and **post-operative**. A systematic approach at all stages optimises outcome, with

prioritised management of the patient's Airway, Breathing, Circulation, drug-induced Disability and Environment.

THE PRE-OPERATIVE PHASE

This is a time of planning and preparation, which may be divided into two: *generic* and *specific*. Generic preparation involves patient identification, consent, communication, investigations and premedication where appropriate, with pre-operative fasting. Correct guidelines for elective adult surgery advise no solid food six hours pre-operatively, with clear up to two hours before surgery. Specific patient concerns are addressed at this stage, which may involve particular attention to restoring optimal physiology using drugs (often antibiotics, use of analgesics) or intravascular fluid therapy. Other considerations may include seek help or advice, ensuring that the anaesthetic equipment available matches the needs of the patient and that blood for transfusion is ready when (or if) needed.

THE PEROPERATIVE PHASE

During this time, correct selection and use of drugs, equipment and techniques should permit safe surgery with patient protection. The aims are **safety** for the patient and **satisfaction** for all. The patient's immediate welfare is dependent on the anaesthetist.

The peroperative phase is further divided into three; *induction, maintenance*, and *recovery* (this merges with the post-operative phase). Most forms of general anaesthetic produce reflex impairment of the patient's airway, breathing and circulation. Ketamine is unusual in that administration is not usually associated with the loss of airway control and ventilation, and circulatory reflexes are preserved.

At all times, the patient's **A**irway, **B**reathing, **C**irculation, drug-induced **D**isability and **E**nvironment is managed in sequence.

Management of the Airway is considered in detail elsewhere (part xxx).

Management of **B**reathing involves either *spontaneous* or *controlled* ventilation. Spontaneous breathing techniques are usually selected for relatively minor surgery involving body surface procedures or fracture reductions. *Controlled* techniques are often selected for major procedures involving surgery which enters body cavities. Profound muscular relaxation is usually needed and muscle relaxant drugs (neuro-muscular blocking or paralysing drugs) are given rendering the patient apnoeic. For this, airway control is usually *via* a tracheal tube.

Management of **C**irculation is matched to surgery. Vascular access is important for administration of drugs and fluids. This is usually by the peripheral intravenous route, occasionally interosseus. Central venous access is reserved for administration of irritant drugs or when thorough distribution in the systemic circulation is nedded (e.g. for controlled infusion of vasopressor drugs, such as adrenaline). *Preload* is managed by appropriate infusion of fluids, bearing in mind that preoperative, perioperative losses, together with maintenance requirements must be addressed.

Afterload may be managed by drugs. High afterload (often vasoconstriction, manifesting by high blood pressure) may be managed by first ensuring "adequate" anaesthesia (treating "light" patients with increased dose of anaesthetic agent) and then reducing

blood pressure the hypotensive agents if needed. Low afterload (often vasodilation produced by anaesthesia, manifested by low BP) is managed again by ensuring "adequate" anaesthesia (treating "deep" patients by reducing anaesthetic dose). The combination of general anaesthesia with hypovolaemia may produce profound falls in pressure and needs immediate action with appropriate fluid infusion. Treatment of low blood pressure with vasoconstrictive drugs must only be done if all potential causes of hypotension are quickly excluded. In this setting it is *vital* to exclude other cases of low BP, which may actually be the result of airway failure (terminal hypoxia), breathing failure (tension pneumothorax), circulatory failure (hypovolaemia or cardiac dysfunction or vena cava compression in the pregnant mother) or failure of drug management (overdose).

Drugs are used to provide a therapeutic **D**isability. The aim is to produce the triad of anaesthesia (unconsciousness, muscle relaxation and reflex suppression). Often a combination of drugs are used to produce "balanced" anaesthesia matched to the needs of the patient, using hypnotic, relaxant, analgesic and adjunct (extra) drugs.

Hypnotic drugs (or agents) confer reversible unconsciousness (hyponosis). They include induction agents (propofol, ketamine) to start anaesthesia and maintenance agents are used to sustain unconsciousness during the procedure. Common maintenance drugs are inhalational vapours (such as halothane, isoflurane) Halothane may also be used as an induction agent in a patient without vascular access, a "gas induction".

Relaxants are neuromuscular blocking or paralysing drugs. They cause apnoea and must always be used with great respect. Safe, effective airway control is mandatory. Examples include suxamethonium (generally short-acting, unless a specific patient sensitivity), vecuronium, alcuronium (longer-acting). The latter group requires reversal (antagonism) with intravenous neostigmine. This reversal agent causes salivation, bronchorrhoea, and gut spasm, which are reduced by the co-administration of an anticholinergic agent (atropine or glycopyrollate).

"*Analgesics*" are often administered during the case, although a more a more accurate term "antinociceptive", an agent which reduces the patient response to "noxious stimuli" such as surgical incision, tracheal intubation, etc.

Potent opioids are used; therapeutically they reduce the sympathetic and stress responses, pathologically they cause dose-dependent respiratory depression, apnoea, nausea and vomiting.

Adjuncts include administration of agents, to reduce effects of other agents, and as extra analgesics (NSAIDs) and prophylactic use of antiemetics.

The immediate patient Environment within the operating room must be managed during surgery. Important considerations are positioning and pressure care, with temperature maintenance.

THE POST-OPERATIVE PHASE

This begins upon completion of surgery, otherwise called the recovery phase. This is a process by which the patient regains independence from care givers, and varies in duration, depending on the overall health of the patient and the magnitude of the surgery. This is a time of risk, because the patient needs to regain vital reflexes. Trained staff with appropriate training and equipment is vital. Access to further help and advice

with clear communication is crucial. Again, a systematic approach to care in the order Airway, Breathing, Circulation, drug-induced Disability and Environment is effective.

16B. LOCAL ANAESTHETIC TECHNIQUES

MARTIN CLARK

INTRODUCTION

Local anaesthetic (LA) techniques offer many advantages to general anaesthesia both in terms of patient safety and comfort. It avoids the risk of general anaesthesia and dramatically improves post operative pain control. It is highly recommended for operations which are suitable for this technique, and may be used with general anaesthesia or sedation.

MECHANISM OF ACTION

Local anaesthetics work by blocking the sodium channels of neurones, interrupting the conduction of action potentials. Small unmyelinated nerves, such as c-fibres, are easier and quicker to block. Large myelinated nerves such as motor nerves display salutatory conduction where action potentials may jump up to 3 Nodes of Ranvier so both a longer length of nerve and also more channels have to be blocked to achieve anaesthesia. This means pain relief and operative anaesthesia lasts longer and comes on quicker than muscle paralysis, but also weaker concentrations of local anaesthesia can be used to produce a differential block where analgesia is produced but muscle power remains, this can be seen in post operative epidural analgesia.

DRUGS, DOSES, DURATION OF ACTION.

Local anaesthetic duration varies with total dose of local anaesthetic administered, the site of injection which is essentially due to bloodflow removing the agent and whether adrenaline has been administered to reduce this blood flow. Sites with fast uptake of LA and therefore shorter duration of action are in descending order, intercostal, extradural, brachial plexus, femoral/sciatic with removal of LA being lowest with local infiltration. The addition of adrenaline (never use concentrations greater than 1:200,000 which is 5 microgrammes/ml) will with some agents prolong block duration and allow larger total doses to be used. Adrenaline is **ABSOLUTELY CONTRAINDICATED** for blocks close to end arteries such as ankle, penis and fingers, as well as intravenous anaesthesia, as it may produce ischaemia distal to the block. Maximum doses quoted below are conservative but safe, the actual maximum dose for these agents, at the varying sites of injection, are not known. Doses of local anaesthetics are quoted in percentages with 1% concentration equalling 10mg/ml agent. Therefore the maximum dose of plain lignocaine

would be 3mg/kg, which for a 70kg person would be 210mg, which equals 21mls of 1% plain lignocaine or 10.5mls of 2% plain lignocaine.

LIGNOCAINE (LIDOCAINE)

Maximum dose: 3mg/kg no adrenaline, 7mg/kg with adrenaline. Onset 2-20 minutes dependant on injection site. Duration 200-400 minutes dependant on site and adrenaline use.

PRILOCAINE

Maximum dose 6mg/kg no adrenaline, 9mg/kg with adrenaline. Duration of action 1.5 times plain lignocaine. Prilocaine is intrinsically safer than lignocaine making it the ideal agent for Biers blocks. It is rapidly metabolised in the liver and lung. Doses over 600mg to an adult may precipitate methaemoglobinaemia which responds to the administration of 1-2 mg/kg of methylene blue.

BUPIVICAINE

Maximum dose 2mg/kg no adrenaline, 4mg/kg with adrenaline; onset 10-30 minutes, duration 5-16 hours. More cardiotoxic than other agents so contraindicated for Bier's block. Useful for long lasting regional anaesthesia, infiltration and neuraxial blocks.

TOXICITY AND TREATMENT

Local anaesthetics block sodium channels in all conductive tissue so they exert the same conduction blocking effects on brain and cardiac tissue as they do on peripheral nerves. This effect becomes important if they reach these tissues in sufficient concentration. Early features of toxicity are numbness or tingling of the lips or tongue, as well as a metallic taste. Features of a severe toxicity are sudden alteration in consciousness, agitation or loss of consciousness plus or minus seizures. Cardiac features are hypotension, sinus bradycardia, conduction block, ventricular tachycardia or asystole.

Management of a severe overdose consists of an ABC approach and administration of intralipid if required: STOP administering the DRUG, call for help, ensure oxygenation and maintain an airway. Confirm / obtain iv access.Use small boluses of benzodiazepines, thiopentone or propofol to treat seizures. If cardiac arrest then start CPR according to standard protocols. CPR may need to be continued for over 1 hour but good outcomes have been reported following CPR in this situation. If available give intralipid intravenously. If no cardiac arrest, use conventional therapies to support the circulation, i.e. fluid and small boluses of adrenaline 10-20 microgrammes boluses as necessary.

LOCAL INFILTRATION.

Local anaesthesia can be infiltrated into local tissues and wound edges to produce local tissue anaesthesia for minor operations or partial post operative analgesia. Most operations involve damage to deeper structures such as muscle or bone and skin infiltration on its own will not affect pain from these sources, instead local must be

injected into the tissues or nerves proximal to the operative field. Suitable agents are lignocaine / prilocaine for short procedures or bupivacaine for longer procedures / analgesia.

REGIONAL ANAESTHESIA

Regional anaesthesia involves anaesthetising a large area of tissue by blocking the Examples are axillary block for arm operations, nerves supplying that tissue. interscalene block for shoulder surgery, superficial cervical plexus block for carotid surgery or ankle blocks / popliteal/ femoral/ sciatic for lower limb surgery. All these blocks if done well allow the full range of surgical techniques to be performed without the use of general anaesthesia.Nerves can be blocked by using a landmark technique but due to anatomical variation it is better to confidently identify the nerves location by eliciting parasthesiae, using a nerve stimulator or ultrasound guidance. Local anaesthetic is then injected around the nerve with larger volumes being used for landmark techniques (to increase the chances of LA contacting the nerve) and smaller volumes for stimulator or ultrasound guided techniques. As large doses of LA may be used the patient should be attached to a cardiac monitor, BP cuff and oxygen saturation probe (as they would be for a general anaesthetic). To minimise the risk of intravascular injection the needle should be aspirated prior to any injection and if blood is elicited the needle withdrawn and repositioned. All LA injections should beadministered as repeated 5ml boluses with needle aspiration in-between boluses to again minimise the risk of intravascular injection. The patient should also be asked if they are experiencing any LA toxicity symptoms and the ECG monitored for new tachy or bradycardia. Once the block is finished sufficient time must be allowed for its onset prior to surgery commencing. A patchy block can be rescued by further LA injection around any missed nerve, local infiltration or light intravenous analgesia or sedation. Patient should be warned they will be aware of movement, vibration and noise but should experience no pain. Suitable agents are lignocaine / prilocaine for short procedures and bupivacaine for prolonged procedures.

INTRA VENOUS REGIONAL ANAESTHESIA (IVRA OR BIER'S BLOCK)

IVRA is suitable for short procedures involving any distal limb. A suitably sized padded orthopaedic tourniquet is applied to the limb to be blocked. All connections must lock and the pressure gauge must be calibrated. The patient should be fully monitored with ECG, NIBP and SpO₂. An intravenous cannula is inserted into a non-operative limb in case emergency drugs need to be given. A distal vein (preferably dorsum hand / foot) is cannulated on the operative limb distal to the tourniquet. The operative limb is then exsanguinated and the tourniquet inflated to 100mmHg above systolic blood pressure. In an adult 40mls of 0.5% prilocaine is injected into the cannulae in the operative limb. Analgesia is complete within 10 minutes but perception of touch (but not pain) is often preserved and the patient should be warned of this. The tourniquet must NOT be released until at least 20 minutes after injection, even if surgery is completed before this time. This is to allow enough prilocaine to diffuse into tissues to prevent dangerous plasma levels on tourniquet release. Tourniquet pain can be a problem with procedures longer than 40 minutes, options are intravenous analgesia or siting a second tourniquet distal to the first tourniquet, inflating the 2nd tourniquet to 100mmHg above systolic BP then deflating the original tourniquet. The new tourniquet should be over an anaesthetised area and so not painful. If the block has to be repeated within 30 minutes of tourniquet release, this is possible using the technique as described above but with just 50% of the original prilocaine dose.

SPINAL ANAESTHESIA (SUBARACHNOID BLOCK)

Spinal anaesthesia is a tremendously useful technique for any procedures below the umbilicus. It must be remembered that the core of the patients nervous system is at the end of your needle and great care must be taken with its performance. **Contraindications** are coagulopathy (INR or APTT >1.5 times normal or platelets < 70), local or systemic sepsis, moderate / severe aortic stenosis or significant hypovolaemia.

TECHNIQUE

Obtain IV access, sit patient up if possible. Full monitoring is applied. Full sterile technique (mask, cap, gown, gloves, skin prep, operative drapes). All drugs should be preservative free and drawn up with a filter needle to prevent inadvertent glass injection. Position the patient on the operating table with their back arched outwards, clean the skin with ideally 0.5% chlorhexidine solution and allow the liquid to dry before attempting needle insertion. Identify the iliac crests and draw an imaginary line between them, this line should cross the 4th lumbar vertebrae at the midline of the back. Palpate the spinal spaces directly above (L3/4) and below this line (L4/5). Choose whichever space feels larger and infiltrate a small volume (<1ml) of lignocaine into the skin via a 22 or 25 G needle, you do not want to infiltrate too much LA as you will lose the ability to palpate the space. You can however infiltrate a further 1ml of lignocaine into deeper tissues after this but be careful not to insert a green needle to the hilt as in some patients this may actually enter the space and precipitate a spinal headache. A 19G introducer needle is inserted into the middle and midline of either the L3/4 or 4/5 space taking care just to insert it into the interspinous ligament and not insert it too far where again it may inadvertently enter the CSF. A 24 to 26 Gauge Whitacre type needle (pencil point, non cutting) is inserted via the introducer needle and directed in the direction of the umbilicus. If the needle encounters bone it should be withdrawn slightly and the angle of insertion altered either caudal or caudad then readvanced. Entry into the subarachnoid space is identified by a distinct pop sensation (when using a pencil point Withdraw the stilet and observe the needle hub for backflow of clear needle). cerebrospinal fluid (CSF) and there should be no blood visible in the fluid. If clear CSF is visible, attach the LA syringe and perform a gentle aspiration, CSF should flow easily into the syringe, local anaesthesia can then be injected over a few seconds, the needle subsequently withdrawn and the patient then laid supine.

AGENTS:

Lignocaine or prilocaine can be used for short <1hr duration procedures but bupivacaine either plain or hyperbaric is the most commonly used agent and 3mls provides 3-4 hours of operative time. Reduced doses should be used for cesarian sections where 2 to 2.4 mls of bupivicaine (depending on patient height) produce good analgesia up to the T6 dermatome.

COMPLICATIONS OF SPINAL ANAESTHESIA

Acute, hypotension due to vasodilation which can be treated with fluids (for hypovolaemia) and vasopressors (for the vasodilation). A "total spinal" occurs when the block spreads very high due to spinal stenosis / overdose/ pregnancy or other cause, this leads to profound hypotension, respiratory paralysis and loss of airway control. Fluids, vasopressors, atropine and intubation are usually required.

Post operative complications include urinary retention (especially if large volumes of intraoperative fluid have been given); numb and heavy legs which are prone to injury until reflexes have returned

Post Dural Puncture Headache occurs after 1:200 spinal anaesthetics especially in younger patients. It is due persistent leak of CSF from the dural puncture site resulting in reduced pressure of CSF around the brain. It presents 1-7 days post procedure and can last weeks to months. It is much more likely if a sharp (Quinke type) needle has been used and much less common if a small gauge pencil point needle has been used, so please use these. The headache is worse on sitting / standing and relieved by abdominal compression or lying flat. Simple analgesia is the first line of management but occasionally a blood patch is required for symptom control. It is important to diagnose post dural puncture headache and once diagnosed AVOID further diagnostic lumbar puncture (to rule out meningitis) as this will increase the leak and make the headache worse.

Meningitis is very rare, about 1:1,000,000 spinals if proper asepsis observed.

Spinal haematoma is also rare, about 1:500,000 if clotting normal during procedure.

EPIDURAL ANAESTHESIA (EXTRADURAL BLOCK)

Epidurals can be used for operative and post operative anaesthesia /and analgesia. Insertion is a specialist technique and will not be discussed here.

POST OPERATIVE CARE / COMPLICATIONS OF EPIDURAL ANAESTHESIA

Hypotension: Due to vasodilation. Epidurals do not cause NEW hypotension, if a patient with an epidural infusion and a stable block level, develops new onset hypotension, then it is much more likely that hypovolaemia, rather than the epidural is the cause. Assess the block level to see if it has increased by several dermatomes and if it has not suspect bleeding.

Epidural abscess: Occurs in approximately 1:20,000 patients. Inspect epidural site daily and assess for signs of back pain / sepsis. As a rule epidurals should not stay in for more than 3 days.

Epidural haematoma: about 1:20,000 patients, especially if abnormal clotting. This can occur at insertion, whilst in situ and at epidural catheter removal. Catheters should not be removed if platelets <70, or INR or APTT>1.5 times normal. Catheters should also not be removed if on high dose low molecular heparin (LMWH). Catheters should not be removed within 12 hours of low dose LMWH and LMWH should not be given within 2 hours of catheter removal. Haematoma presents with back pain and decreased leg

power (uni or bilateral). Stop the epidural infusion if patients cannot lift their knee of the bed. If no better within 2 hours patient needs an urgent MRI and consideration of neurosurgical laminectomy to decompress. Irreversible neurological damage occurs approximately 6 hours after first symptoms.

Urinary retention: Almost inevitable, epidural patients require catheterisation.

Post Dural Puncture Headache: approximately 1:200 patients, as a result of inadvertent dural puncture.

16C. KETAMINE AND SEDATION

MARTIN CLARK

KETAMINE

INTRODUCTION

All general anaesthesia should, ideally, be administered by a trained anaesthetist but sometimes in remote settings this is not possible. If faced with this situation, Ketamine is the safest choice for a general anaesthetic agent. This is because Ketamine preserves respiration, airway reflexes / tone and maintains / increases blood pressure. Thus airway, breathing and circulation problems (which are by far the biggest risks during anaesthesia) are less likely to occur with Ketamine, than with other agents.

MONITORING

A dedicated staff member should be allocated to monitor the patients airway breathing and circulation throughout surgery, as it is impossible to adequately focus on operating and simultaneously monitor the patients airway, breathing and circulation. If available, all anaesthetics should be monitored by saturation probe, ECG, blood pressure and capnography. Intravenous access and facilities for resuscitation are mandatory.

AIRWAY EFFECTS

With ketamine, airway maintenance and reflexes are generally preserved, however this is not guaranteed and close attention to airway patency and respiration must be maintained. If the patient is an aspiration risk, intubation is still required, as it would be for any other anaesthetic agent. Ketamine stimulates pharyngeal and trachea-bronchial secretions, this can be counteracted by administering an anti-muscarinic drug such as atropine or glycopyrrolate.

RESPIRATORY EFFECTS

When Ketamine is given slowly, respiration is generally maintained, although it may cease briefly after a rapid i.v bolus.

CARDIOVASCULAR EFFECTS

Ketamine increases heart rate, blood pressure and cardiac output. This effect reaches a maximum about 2 minutes afterinjection and settles over 15 - 20 minutes. Hypertension usually responds to doses of i.v. diazepam (1-2mg for an adult). If possible, Ketamine should be avoided in patients with severe angina.

CENTRAL NERVOUS SYSTEM EFFECTS

Ketamine produces a state of dissociative anaesthesia in which the eyes remain open with a slow nystagmus and reflex body movements may occur, although the patient is not aware of this.

Ketamine is a strong painkiller and can be used as the sole analgesic agent *intra-operatively* although local anaesthesia infiltration or block is very useful as an intraoperative adjunct or for post operative analgesia. Administration of opiates or tramadol intra-operatively can also reduce the amount of ketamine required for maintenance of anaesthesia. This increases the risk of hypoventilation / apnoea but does decrease the risk of emergence phenomena which can occur in between 5- 30% of patients. This risk can also be reduced by premedication with benzodiazepines and by recovering the patient in a quiet area.

Intra-ocular pressure is raised and this along with the continued eye movement during ketamine anaesthesia, this makes it unsuitable for ophthalmic surgery.

GASTROINTESTINAL EFFECTS

Post emergence vomiting occurs in 10-15% of patients.

OBSTETRIC EFFECTS

Ketamine crosses the placenta and so will produce anaesthesia in any foetus which should be borne in mind with any operative delivery.

MISCELLANEOUS

Transient erythematous rash lasting about 20 minutes occurs in 5-20% patients. This does not recur with further dosing.

ADMINISTRATION

Ketamine can be administered intravenously intramuscularly, orally or rectally and comes in 3 concentrations 10mg/kg, 50mg/kg and 100mg/kg. Although the recipes below include atropine to reduce secretions some investigators feel it is unnecessary for sedative procedures¹.

Intravenous dose is 1-2mg/kg ketamine, plus atropine 10-20 microgrammes/kg plus diazepam 0.1mg/kg for induction followed by Ketamine 1-2mg/kg/hr infusion or 0.5mg/kg boluses as required for patient emergence, doses should be adjusted dependent on patients sensitivity. Onset 30 seconds to 5 minutes, duration 5-10 minutes.

Stop any infusion 20 minutes before end of surgery to prevent a prolonged recovery period.

Intramuscular Ketamine is especially useful in non co-operative children where i.v access is difficult to obtain awake, naturally iv access must then be obtained once anaesthetised. A suggested mixture for induction is Ketamine 5-10mg/kg, atropine 20microgrammes/kg and diazepam 0.1mg/kg. Onset of action 2-8 minutes, duration of action 10-20 minutes. An alternative is to sedate the child with i.m. ketamine (2mg/kg) +

atropine (20mcg/kg) + diazepam (0.1mg/kg). After 5 minutes you will have a docile child who can cooperate with either cannulations or a gas induction¹.

Oral sedation:for burns changes in children6-15mg/kg plus 0.2mg/kg diazepam, tastes unpleasant.Can be mixed with a small volume (20ml) of orange juice.Can also add in 20microgrammes per kg atropine.Effects are less predictable via oral route, onset 20-30 minutes but may be slower. For adult 500mg ketamine plus 5 mg diazepam. Patients must be monitored and observed by a dedicated member of staff till they become responsive to verbal stimuli.

SEDATION

Conscious sedation has been defined as:

"A technique in which the use of a drug or drugs produces a state of depression of the central nervous system enabling treatment to be carried out, but *during which verbal contact with the patient is maintained throughout* the period of sedation. The drugs and techniques used to provide conscious sedation should carry a margin of safety wide enough to render loss of consciousness unlikely." Royal College of Anaesthetists (2002)

The patient should have:

- Minimally depressed consciousness
- Be easily roused
- Protects own airway
- Maintains own breathing independently
- Responds to verbal commands
- Remains co-operative

Deep Sedation is essentially similar to and carries the risks of general anaesthesia and should only be attempted by those able to manage a general anaesthetic and its attendant complications. The features are:

- Not easily roused .
- Partial loss of protective reflexes.
- Partial or complete inability to protect airway (< GCS 9).
- No purposeful response to stimuli or commands

As a general rule in all sedation, the less sedated the patient the better. This is because patients tend to become less co-operative and move more in response to stimuli the deeper the sedation becomes, until this ceases when they are essentially anaesthetised. This makes the operators job more, not less difficult, better an awake co-operative patient who stays still or a general anaesthetic with a trained practitioner rather than a messy semi-anaesthetised, moving patient with partial airway obstruction, which is often the end product of deep sedation.

Basic clinical monitoring must be provided during all levels of sedation and during the recovery period. and must include the observation and documentation of:

- Level of consciousness
- Airway patency, rate and pattern of breathing; oxygenation and colour
- Heart rate and rhythm
- Pain

Anxiety levels

Advanced monitoring must be provided for all levels of sedation deeper than minimal sedation/anxiolysis. Basic clinical monitoring (as outlined above) must be continued during the procedure and recovery period, until discharge from the facility. Monitoring should be commenced prior to the administration of sedation. Trained personnel, equipment for monitoring, and resuscitation drugs and equipment must be available throughout this period.

Diazepam is commonly used for i.v. procedural sedation. It is administered with an initial bolus of 2.5 to 5.0 mg. Incremental doses of 2.5 mg can be given in 3- to 4minute intervals. Symptoms of overdosage include respiratory depression, hypotension, coma, stupor, confusion, and apnea.

Ketamine 1mg/kg i.v. or 2mg/kg i.m.is an alternative agent.

RECOVERY FROM SEDATION

It is **VITAL** that all sedated patients are recovered in a monitored environment as often the stimulation of a procedure can keep a patient at a higher level of consciousness than they would otherwise be. Once this stimulation ceases (i.e. endoscopy) the patient may become less conscious (even though no more drugs have been given) and then obstruct their airway, become quickly hypoxic and die. There have been several deaths in the UK from just this circumstance.

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16D. PRE-OPERATIVE PATIENT PREPARATION

ERIN MCILVEEN, KHURRAM S KHAN, ALISTAIR BROWN, ABEBE BEKELE

The surgical care of patients has been a key component of health care provision worldwide for over a hundred years. The impact of surgical intervention on public health systems will grow as the incidence of traumatic injuries, cancers and cardiovascular disease continues to rise. The aim of safe surgery is to ensure the well-being of surgical patients by minimising the most common and avoidable risks that endanger live and to identify patients most at risk. Preoperative preparation and assessment is a vital part of care for patients scheduled for both routine and emergency surgery. The preoperative preparation of an individual patient will depend on the results of a thorough clinical assessment and on the particular operation to be undertaken. This will allow specific measures to be taken so that the patient is in the best possible condition for both anaesthesia and surgery.

Preparing a patient for surgery requires co-operation between surgical and anaesthetic teams. It also requires an understanding of the patients' pre-operative status, the nature of the surgery, the anaesthetic techniques required for surgery, as well as the risks that a particular patient may face during the operative period. When a patient presents for surgery they need to be in the best possible condition to help ensure the success of the procedure.

AIMS OF PRE-OPERATIVE ASSESSMENT:

- Establish rapport with patient and family.
- Ensure patient fully informed about their procedure and risks so they can make an informed decision about whether to proceed with surgery.
- Identify co-morbidities and optimally prepare patients, depending on urgency.
- Identify patients with high risk of complications and define appropriate postoperative level of care (day care unit, inpatient, general ward, HDU, ICU) and invasive monitoring required.
- Discuss anaesthetic technique, premedication, pain relief and risks .
- Plan discharge timing and requirements.

PATIENT ASSESSMENT

Clinical assessment is best conducted through the standard format of history, examination and then further investigations. The latter will, in the majority of cases, serve to confirm the impression gained from an accurate history and examination, and should not be used to replace a thorough clinical assessment.

HISTORY AND EXAMINATION

History and examination is the most efficient and accurate method of detecting significant co-morbidities that may affect the peri-operative period. Risk prediction can be used to guide patients' pre-operative care and determine whether the patient needs to see an anaesthetist before surgery. The anaesthetist is essential in assessing, optimising and estimating risk and supporting patients in the decision whether to proceed with surgery and anaesthesia.

THESE VARIABLES PROVIDE INDEPENDENT PROGNOSTIC INFORMATION:

- Age risk of dying and risk of mortality from ischaemic heart disease (IHD) doubles every 7 years from age 10.
- Sex men more likely to die than women.
- Socioeconomic status impoverished twice as likely to die as rich.
- IHD: Myocardial infarction (MI) and Angina each increase risk by 1.5.
- Heart failure increase risk by 1.5.
- Ischaemic brain disease (Stroke and TIA) increase risk by 1.5.
- Renal failure (Creatinine > 150 µmol/l) increase risk by 1.5.
- Peripheral arterial disease.

In short, these relate mostly to the respiratory and cardiovascular systems.

RESPIRATORY SYSTEM

Symptoms of significant respiratory disease are repeated coughing, shortness of breath, haemoptysis, production of purulent sputum and the presence of wheeze. The functional ability of the patient can be assessed by questions such as "*how far can you walk before you get short of breath?*" or "*what activities make you short of breath?*". Valuable information may be revealed about a patient's cardio-respiratory reserve.

The presence of a productive cough is associated with an increase in postoperative chest complications and if it is of recent onset then consideration should be given to postponing surgery and starting appropriate treatment with antibiotics and chest physiotherapy. If the patient has a chronic productive cough then elective surgery should be postponed only if the patient has additional signs suggesting an infection, provided that the patient is kept as dry as possible.

The risk factors for pulmonary complications include:

- Known pulmonary disease
- Abnormal pulmonary function tests (FEV1 and FVC < 60%)
- Smoking
- Age > 60 years
- Obesity
- Upper abdominal and thoracic surgery
- Long operation time
- Preoperative steps
 - Usual history
 - CXR if over age of 40 years.
 - Preoperative Spirometry and arterial blood gases if necessary.
 - FEV1 and maximal breathing capacity
 - Decrease or stop smoking and increase or optimize bronchodilator therapy.

Post operative pulmonary problems

- Adult respiratory distress syndrome
- Pulmonary edema
- Fat embolism
- Atelectasis
- Pneumonia
- Aspiration

Ways to decrease complications

- Incentive spirometry
- Chest physiotherapy
- Postural drainage when needed
- Humidified oxygen
- Bronchodilator therapy
- Antibiotics when necessary

CARDIOVASCULAR SYSTEM

When assessing the cardiovascular system, it must be remembered that patients can have heart disease without symptoms or signs. Symptoms of valvular heart disease include breathlessness on exertion, paroxysmal nocturnal dyspnoea, palpitations, haemoptysis, dizziness, fainting and angina. Obtain historical information concerning previous MI, angina, cardiac medications and arrhythmias. Examine patient to assess the rate and rhythm of the pulse, determine the origin of any murmur and note crackles, peripheral oedema, or any sign of heart failure. The most accurate method of diagnosing the cause of a cardiac murmur is Echocardiography. In general all diastolic murmurs and loud systolic murmurs which are accompanied by a thrill are abnormal and indicate underlying structural heart disease. When the cardiac function is seriously compromised then symptoms and signs of cardiac failure will become apparent.

2. History of ischemic heart disease			
History of myocardial infarction			
History of positive exercise test			
Current complaint of chest pain considered secondary to myocardial ischemia			
Use of nitrate therapy			
ECG with pathological Q waves			
4. History of cerebrovascular disease			
History of transient ischemic attack or stroke			

Lees revised cardiac risk index: Each risk factor is assigned one point.

6. Preoperative serum creatinine > 2.0 mg/dL (175 µmol/l)

Risk of *major cardiac event peri-operatively			
Points	Class	Risk	
0	I	0.40%	
1	П	0.90%	
2	III	6.60%	
>3	IV	11%	

***Major cardiac event** includes myocardial infarction, pulmonary oedema, ventricular fibrillation, cardiac arrest, and complete heart block

PAST HISTORY

Enquiries should be made about previous operations and anaesthetics and any other known diseases should be noted. A history of diabetes, rheumatic fever or sickle cell disease is of particular importance as is epilepsy. A full drug history and details of any allergies should be sought. Particular importance should be paid to drugs that may interfere with anaesthetic agents such as beta blockers (which may cause bradycardia), anti-hypertensive agents, diuretics (hypokalaemia may prolong neuro-muscular blockade as well as induce arrhythmias) as well as those such as warfarin that may need dose alteration prior to surgery.

MEDICATIONS

Know all the medications the patient is taking. As a general rule:

- Continue preoperative antihypertensive medication except for diuretics and possible ACE inhibitors.
- Anti-platelet drugs create controversy: Aspirin alone can be continued for most surgery but some surgeons prefer to stop it for thoracic or pelvic surgery. If a patient is on both aspirin and clopidogrel one should usually be stopped, but if the patient is on both drugs due to coronary artery stenting, you have to discuss with a cardiologist; it might be necessary to postpone non-essential surgery until the patient can safely come off clopidogrel. Anti-platelet drugs should be stopped for at least 6 days to be effective.
- Stop NSAIDs unless absolutely necessary.
- Stop oral anticoagulants (warfarin) and change to heparin infusion.
- For major surgery oral hypoglycaemic drugs are stopped a day before surgery and patients started on an insulin sliding scale. For minor surgery diabetics can continue on oral medication but they need close glucose monitoring.

AIRWAY ASSESSMENT

The anaesthetist must assess the airway in every patient prior to anaesthesia. There are various methods of assessing the likelihood of a difficult intubation at the bedside. The airway should be examined with the patient sitting upright with the mouth open as wide as possible (Mallampati classification). Neck mobility, jaw position, the presence and condition of teeth will all help predict a possible difficult airway or intubation.

PREOPERATIVE CARE AND EVALUATION: PRACTICAL SUMMARY

<u>Aims</u>: Psychological and Physical preparation of the patient for surgery

- **Psychological**: Patient information, obtaining written consent, allaying fears
- Physical: Ensure diagnosis is correct and symptoms have not changed

ANAESTHETIC RISK ASSESSMENT (ASA CLASSIFICATION)

Class*	Physical Status			
P1	A normal, healthy patient			
P2	A patient with mild systemic disease			
P3	A patient with severe systemic disease that limits activity but is not life threatening			
P4	A patient with severe systemic disease that is a constant threat to life			
P5	Moribund; Not expected to survive but is submitted to operation in desperation			
P6	A declared brain-dead patient whose organs are removed for donor purpose			
E:	Indicates emergency surgery "E" used in addition to the above "P" codes.			

Groups 1 to 3 have no or little increased risk with normal anaesthesia. It is important to note that none of these are an absolute contraindication to anaesthesia. Instead, they are ways of comparing the wellbeing of the patient with the importance of the procedure: the production of a risk-benefit concept for surgery and anaesthesia.

CHARACTERISATION OF MILD AND SEVERE CO-MORBIDITY CORRESPONDING TO ASA GRADES 2 AND 3:

	ASA Grade 2	ASA Grade 3			
Current angina	Occasional use of GTN spray	Regular use of GTN spray			
	2-3x/month	2-3x/week			
	Does not include unstable angina Unstable ang				
Myocardial infarction	> 1 month ago	< 1 month ago			
Heart failure	Compensated, comfortable at rest	Decompensated, symptoms at rest			
Valve disease	n/a	Exercise-induced syncope, Angina, dyspnoea, orthopnoea			
Exercise tolerance	Not limiting activity >1 flight of stairs	Limiting activity			
Hypertension	Well-controlled	single anti-hypertensive Multiple anti-hypertensives controlled Not well controlled			
	Uses single anti-hypertensive				
Diabetes	Well controlled No obvious complications				

CARDIOVASCULAR DISEASE

Previous coronary	Not directly	relevant,	depends	on	Not	directly	relevant,	depends
Revascularisation	current signs and symptoms			on c	urrent sig	gns and sy	mptoms	

RENAL DISEASE

ASA Grade 2	ASA Grade 3		
Elevated creatinine (> 100umol/l and			
< 200umol/l)	(>200umol/l)		
Dietary restrictions	Regular dialysis programme		
	(peritoneal or haemodialysis)		

SUMMARY: ROLE OF THE SURGEON

A. THOROUGH HISTORY AND EXAMINATION

- Focus on operative, anaesthetic and patient healing factors.
- Cardiovascular and respiratory fitness.
- Pre-existing medical conditions which influence wound healing: e.g. nutrition, diabetes mellitus.
- Drug therapy and allergies.
- Previous medical and surgical/ anaesthetic history.
- Relevant advice: Lose weight, stop smoking, reduce alcohol.

B. ORDER RELEVANT PRE-OPERATIVE TESTS:

Results should be ready before surgery.

GENERAL TESTS CARRIED OUT IN MOST PATIENTS:

1. Full Blood Count: To establish baseline haemoglobin; exclude evidence of infection (total white cell count/neutrophil count); platelets .

2. Urinalysis: to look for urinary tract infections, diabetes, ketosis or proteinuria.

3. Electrolytes, Urea and Creatinine: Metabolic picture of patient reflecting imbalances, dehydration, ultimately reflecting renal function which is largely responsible for metabolic aberrations.

4. Blood group, Cross match/Save: For anticipated intra-operative blood loss.

5. Chest X Ray: to find occult chest pathology compromising anaesthetic, recovery.

6. ECG: in older patients and young patients with cardiac history.

SPECIFIC BLOOD TESTS WHERE INDICATED

- Liver Function Tests
- Amylase in acute abdominal pain

- Blood Glucose
- Clotting Studies
- Echocardiography
- Spirometry
- Blood gas analysis

C. PREOPERATIVE PREPARATION:

PRE-OPERATIVE PRIMARY CARE

Primary care can help optimise patients' fitness before surgery:

- A. Smoking cessation
- B. Exercise
- C. Weight reduction or weight gain
- D. Optimise treatment of chronic conditions
- E. Stopping certain medications before surgery

PRE-OPERATIVE PREPARATION

- Obtain consent for the procedure
- Discuss with patient and the family the risks, benefits and alternatives.
- Keep NPO after mid-night for morning surgery
- Commence IV hydration with fasting
- Optimize patient medically, treat infection and stabilise diabetes
- Preoperative incentive spirometry.
- Prophylactic antibiotics in theatre
- Skin preparation
- Bowel preparation as necessary
- DVT prophylaxis: all patients should be risk assessed
- Catheterise and/or insert NG tube as indicated (do under anaesthetic if possible for patient comfort)
- Arrange intra-operative X Rays or Frozen section as required

URGENCY OF OPERATION: ELECTIVE VS EMERGENCY

ELECTIVE

When a patient is admitted electively, the patient should have had adequate preoperative assessment so that the anaesthetist and surgeon can proceed safely. Highrisk patients will need to be managed by experienced anaesthetists and may need postponed if such expertise is not available. Operative sessions will have been planned appropriately to allow time for the anaesthetist to review the patient pre-operatively before arrival in the anaesthetic room. If patient is cancelled the reasons for this should be explained and documented in the notes with recommendations for optimisation for surgery in the future.

EMERGENCY

Patients who present as an emergency and are requiring anaesthesia are at a higher risk of peri-operative complications. Despite this the aims are to maintain a high standard of care, prevent excessive periods of starvation and fluid deprivation, especially in vulnerable groups and allow clear communication between surgeons, anaesthetists and nurses. There needs to be a balance between prevention of deterioration caused by delaying surgery and the benefit of optimising medical conditions pre-operatively. The risks and benefits of surgery should be explained clearly to the patient and documented especially when a decision is made to either:

- 1. Proceed with surgery when potentially important investigations have been omitted or when patients condition not optimised; or
- 2. Not proceed with surgery when correctable serious co-morbidity is present, i.e. if the risks of the procedure outweigh the benefit to the patient.

FUNCTIONAL CAPACITY

Exercise tolerance is estimated from the patient's ability to perform normal activities of daily living (ADL) based on the Duke Activity Status Index. When measured correctly, it is an important tool for survival prediction in addition to traditional risk factors such as hypertension, smoking, COPD, diabetes and malnutrition. It can be estimated by the incremental shuttle walk test or more precisely with cardiopulmonary exercise testing, if available, which can also diagnose whether aerobic performance is limited by pulmonary, cardiac or peripheral disease, and may be a good screening test for IHD. In usual practice this is seldomly done.

ON THE DAY OF SURGERY

PRE-OPERATIVE FASTING GUIDELINES IN ADULTS:

<u>'2 – 6 rule'</u>

- '2' intake of water up to 2h before induction of anaesthesia
- '6' fasting for 6h (solids, milk, milk-containing drinks)

Note chewing gum is treated as a solid food and is not to be taken up to 6 hours before. The anaesthetic team should consider further interventions for patients at higher risk of regurgitation and aspiration.

POST-OPERATIVELY RESUMPTION OF ORAL INTAKE

Patients should be encouraged to drink when ready, providing there are no specific contraindications.

PREVENTING PERI-OPERATIVE CARDIOVASCULAR EVENTS

• Identify high risk patients for peri-operative cardiovascular events (as above)

- DVT prophylaxis
- Good diabetic control
- Adequate analgesia and anti-emetics
- Maintain renal function

DVT PROPHYLAXIS

There should be standardised assessment for risk i.e. age, risk factors, type of operation and patients should be classified into low, medium or high risk; mechanical and/or chemical prophylaxis should be put in place based on this risk assessment. Mechanical prophylaxis includes elastic compression stockings, foot impulse devices and intermittent pneumatic compression devices. Chemical prophylaxis includes low molecular weight heparin and un-fractionated heparin.

GOOD DIABETIC CONTROL

Hyperglycaemia is an independent predictor of cardiovascular risk and the severity is directly related to mortality rate during MI. The risk of IHD is 2-4x higher in diabetics than the general population. They also have higher wound infection rates and higher aspiration risks due to gastric neuropathy.

ADEQUATE ANALGESIA AND ANTI-EMETICS

Crucial to remove stress, adverse haemodynamic and hypercoagulable responses as the majority of cardiac events occur post-operatively.

MAINTAIN RENAL FUNCTION

Identify patients at risk of acute kidney injury, especially if elderly or with other comorbidities. Keep patients well hydrated, avoid certain drugs if possible (e.g. NSAIDS, amino-glycosides, cephalosporins) and check urea, creatinine routinely.

PREVENTING SURGICAL SITE INFECTIONS

HAIR REMOVAL

If at all possible, avoid hair removal. If necessary, use clippers rather than razors and remove hair in theatre immediately before skin-preparation for surgery.

PROPHYLACTIC ANTIBIOTICS

Ensure proper prescribing and administration of prophylactic antibiotics within 60 minutes before first surgical incision (see WHO Safe Surgery Checklist).

BLOOD GLUCOSE IN DIABETICS

• Maintain normal range intra-operatively = 5.0 to 10.0 mmol/l, check hourly

- HbA1c at pre-operative assessment for long-term optimisation
- Prioritise on list minimising fasting time
- Start variable rate insulin infusion on morning of surgery if going to miss > 1 meal
- Stop variable rate insulin infusion when eating, glucose levels stable and given background subcutaneous insulin

BODY TEMPERATURE

- Keep temperature 36.5-37.5'C (excluding cardiac surgery)
- Monitor in hour before surgery, before induction, every 30 minutes during surgery, on arrival in the recovery room and every 15 minutes during the recovery period
- Identify those at risk of developing hypothermia i.e. surgery > 30mins and start forced air warming intra-operatively. Keep theatre temperature high, use warming blankets, hot air blankets, IV fluid heaters.
- Reduces peri-operative cardiac events

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17. PAIN MANAGEMENT

DAVID R BALL

INTRODUCTION

Pain is an unpleasant sensory and emotional perception interpreted as actual or potential body damage [1]. This definition emphasises the dual nature of pain: sensation with emotion. The experience of pain therefore is a subjective one where feelings are important. Indeed, there is no objective measure of pain. There are different types of pain experience, which in part reflect the different pain pathologies, resulting from the various traumatic, inflammatory, thermal or chemical injuries that provoke pain. There are recognised distinct anatomical and biochemical nerve pathways within the spinal cord and brain which carry "nociceptive" information (those stimuli that may be interpreted as pain by a patient). This means that there is no single treatment for pain and different therapeutic approaches are available which can realistically improve treatment.

The word "analgesia" (from the Greek "*an*-", meaning without and "*algos*", pain) is a therapeutic state achieved by a variety of interventions, as described below. "Anaesthesia" is also from the Greek, meaning "without feeling".

There is a conventional distinction between acute and chronic pain states. In general, chronic pain is a condition of persistent pain for three to six months, often persisting after an original insult. For pain associated with surgical practice, most acute pain states resolve with time. Poorly managed pain can, however, increase the risk of developing chronic post-surgical pain [1].

We treat pain for two main reasons: *humanitarian* and *therapeutic*. The humanitarian approach is based on the ethical principle: the relief of human suffering. The therapeutic approach is based on improving outcome. A patient with unmanaged pain is at much greater risk of complication, such as delayed healing, impaired mobilisation, poor feeding, etc.

Successful analgesia aims to achieve pain control for both *static* (at rest) and *dynamic* pain (on movement, including the ability to cough and clear respiratory secretions).

The efficacy of pain control (and possible side effects, such as sedation and respiratory depression) should be assessed and recorded [2].

A SIMPLE FOUR-POINT SCALE FOR PAIN CONTROL IS:

- 0 none, patient is comfortable at rest and movement.
- 1 mild, patient comfortable at rest, pain on movement
- 2 moderate, patient has intermittent pain at rest, worse on movement.
- 3 severe, patient has continuous pain at rest, worse on movement.

A SIMPLE SCALE FOR SEDATION IS:

- 0 none, patient is alert
- 1 mild, patient occasionally drowsy but easily rousable to voice.
- 2 moderate, patient frequently drowsy but easily rousable to voice.
- 3 severe, patient difficult to rouse, needing physical stimulus.

The term "S" can be used to describe sleep for both scales.

THERE ARE THREE THERAPEUTIC COMPONENTS TO PAIN MANAGEMENT: PSYCHOLOGICAL, PHYSICAL AND PHARMACOLOGICAL. THESE MAY BE USED IN COMBINATION TO ACHIEVE OPTIMAL EFFECT.

The *psychological* component involves a caring, compassionate approach to patient care in all of its elements. This professional bond between patient and care-giver enhances mutual trust and respect and can provide powerful therapeutic gain, enhancing the efficacy of other interventions [3].

Physical treatments are often helpful, especially for orthopaedic injuries. Fixation, splintage, elevation and traction of limb fractures reduce the burden of pain.

Pharmacological treatments involve the administration of appropriate analgesic drug or drugs. The aim "is to deliver the correct drug at the correct dose at the correct time by the correct route to the correct patient for the correct reason." Drugs are often given in combination for additive or synergistic effect: this is termed "multi-modal" therapy.

THERE ARE THREE CONSIDERATIONS FOR RATIONAL DRUG SELECTION:

First, *pharmacodynamic*: selection on the basis of efficacy (effect, the ability to do good) and toxicity (side effect, the ability to do harm), with the difference between these called the therapeutic index. Generally, drugs with higher efficacy usually are coupled to higher toxicity. For instance, morphine is a more potent analgesic than codeine (higher efficacy) but has more risk of respiratory depression (higher toxicity). Drugs also interact. As mentioned, combinations of analgesics may enhance pain relief, but also other agents, especially sedatives and anxiolytic agents enhance the analgesic effect (and side effects).

Second, *pharmacokinetic:* these are the processes of drug administration, uptake and distribution by the circulation to reach the biophase (the site of drug action).For optimal effect the drug must be delivered at sufficient dose at appropriate time. Removal of drug from the biophase involves redistribution followed by combination of metabolism (usually by the liver) and excretion (usually by the kidney). Drugs have kinetic interactions. For example, drugs that have emetic toxicity will impair the efficacy of orally administered analgesics. Analgesics generally have two major sites of action' peripheral (at an injury site, for example, non-steroidal anti-inflammatory agents) and central (the brain and spinal cord for opioid drugs)

Patients vary considerably in their response to any administered drug, in both dynamic and kinetic terms. This is due to a variety of physiological and pathological process unique to an individual. In general, ill and injured patients and those at the extremes of age require lower doses for an equivalent effect, compared to healthy matched controls.

Third, *pharmacoeconomic*: an assessment of utility of the drug based on economic considerations, such as cost and availability.

There are two main groups of drugs available for pain control, the *analgesics* and the *anaesthetics*.

ANALGESIC DRUGS

There are two main types of drug within this group: the NSAIDS (non-steroidal antiinflammatory drugs) and *Opioids*.

NSAIDS.

These are inhibitors of cyclo-oxygenase, an enzyme involved in the production of prostaglandins from arachidonic acid. These are inflammatory mediators produced a sites of tissue injury. The prototype drug is aspirin. Others include ibuprofen, and diclofenac. Paracetamol is a related agent which has central activity. NSAIDS can be used alone and are particularly useful for bone, joint and dental pain. They can also be used with opioids and local anaesthetic techniques. They are usually administered by the oral or rectal route.

NSAIDS may provoke bronchospasm, gastic ulceration and renal impairment. They should be withheld from the very ill patient at risk of renal failure or coagulopathy.

OPIOIDS.

These are natural or synthetic derivatives of morphine acting at specific receptors (μ 1 type) within the brain. Morphine is the prototype drug; others include pethidine, codeine and diamorphine. They are useful for most pain states, especially visceral pain and are available as enteral or parenteral formulations. NSAIDS and/or anaesthetic techniques may be used with opioids as part of a "multi-modal technique. Some opioids are co-formulated with paracetamol, such as co-dydramol (codeine with paracetamol). Tramadol is a μ receptor agonist that also has central α adrenoceptor activity.

The World Health Organisation (WHO) has developed the "**WHO analgesia ladder** " (initially for cancer pain but now extended for wider use) [4] advising on sensible analgesia regimes for various levels of pain, broadly similar to the pain rating scale described above.

- Mild pain: Simple analgesics. e.g. NSAIDS
- Moderate pain: Intermediate analgesics. e.g. codydramol or tramadol
- Severe pain: Strong analgesics. e.g. morphine, diamorphine, pethidine.

The main side effects of opioids are dose dependent sedation, respiratory depression, nausea, vomiting and constipation. The decision to give strong opioid analgesia is based on a risk - benefit assessment. Use of strong opioids for management of severe pain does **NOT** lead to addiction. Patients can have variable and unpredictable response to treatment, so it is important that accurate, timely recording of vital signs, including pain and sedation scores are kept, together with a robust alert and response process to detect and treat complications. When opioid therapy is prescribed sensibly, the risk of complication is, however, low, and should not deter their use for patients who need them.

A common concern is the administration of strong opioids to ill or injured patient. The considerations are:

- If hypovolaemic, the patient will have greater sensitivity to an intravenous analgesic dose because a greater proportion of drug will be delivered to the brain.
- Increased sensitivity of opioids means there is increased efficacy (analgesia) and toxicity (respiratory depression, nausea, vomiting)
- That a patient will absorb oral, subcutaneous and intramuscular morphine poorly because of reduced drug uptake as a result of circulatory redistribution away from skin, muscle and gut (as part of the acute adaptive response to protect vital organs, such as the brain, kidney and liver).

THE ADVICE IS:

- Patients can benefit from incremental, intravenous morphine.
- This should be done together with a sensible, systematic approach to management, based on support of the "airway, breathing, circulation, etc", coupled with regular recording and review.
- A reasonable analgesic approach is to give increments of intravenous morphine up to 0.1 mg/kg over ten minutes. This may be repeated for more robust individuals. Oxygen therapy is indicated and response, with vital signs recorded.

ANAESTHETIC DRUGS

Local anaesthetic agents can be used to provide conduction blockade of sensory nerves, including those subserving pain. They are sodium ion channel blockers, including the short-acting lidocaine (1 to 2 hours) and bupivicaine (4 to 6 hours) Since these agents block conduction in all nerve fibres, there is usually motor, sensory and autonomic blockade within the territory of the block, with small-diameter fibres more sensitive: these include those fibres carrying information about pain and temperature. Local anaesthetics can be administered at a variety of anatomic sites. The techniques vary in simplicity, efficacy and risk. For all techniques, the important considerations are:

DOSE SELECTION:

for bupivicaine, the maximum advised dose is 2mg/kg for every four hour interval. For lidocaine, 4mg/kg. Adrenaline-containing solutions allow these doses to be doubled. Adrenaline-containing solutions must **NOT** be infiltrated in fingers, toes or the penis, since these structures are supplied by end-arteries and gangrene is a recognised complication.

DOSE ADMINISTRATION:

this should be injected at a relevant anatomical site to achieve blockade in the required area (the "territory of the block") The important concern is to avoid intravascular or intraneural injection. The calculated dose should be divided (fractionated) and administered in volumes of 3 to 5 ml, aspirating at each step. Aspiration of blood must prompt stopping the injection and the approach reviewed. Any resistance or pain on injection must also prompt stopping the injection.

DOSE LOCATION:

Sites include:

- Simple infiltration (including field block)
- Nerve block, single or multiple (e.g. femoral, sciatic, ankle in the lower limb and digital, ulnar, median in the upper limb)
- Plexus block (e.g. brachial plexus block for the upper limb)
- Paravertebral block is useful for unilateral analgesia for operations on the trunk, e.g thoracotomy, open cholecystectomy, open nephrectomy. There is usually little autonomic blockade.
- Neuraxial Block. This includes spinal (subarachnoid) block, most useful for operations below the umbilicus and epidural (extradural) block, useful for procedures in chest, abdomen and lower limbs. These often produces sympathetic blockade in the block territory, with resulting hypotension and bradycardia. Close cardiovascular monitoring is needed. A caudal block is a form of sacral epidural bock, used for perineal analgesia.

This list, from distal to proximal, increases in technical complexity and risk the more proximal the block is sited. As well as providing post-operative analgesia, these techniques may, when well conducted, be used per-operatively, alone (e.g. spinal) or combined with general anaesthesia.

These techniques may be conducted once ("single shot"), repeated (which involves a repeat injection) or, with the positioning of a suitable catheter, allow continuous infusion or interval administration of drug.

Intravenous regional anaesthesia (IVRA or Bier's block) is usually done for the upper limb. It is a single-dose operative technique which does not offer further analgesia.

DOSE TOXICITY:

Local anaesthetics are dangerous in overdose, either absolute (exceeding the recommended limits) or relative, when agent is injected into a blood vessel. The cardiovascular and nervous systems are affected with dose-dependent toxicity. Signs range from patient agitation, confusion and tingling (often peri-oral) to unconsciousness and seizures. Hypotension, arrhythmias with cardiac arrest can alsooccur. Standard Life support protocols should be used and seizures treated with benzodiazepines or sodium pentothal if experienced in its use. More recently administration of lipid solutions ("Lipid Rescue") to sequester the anaesthetic may help, but only in conjunction with robust life support. It is important to conduct any block where there are resources to manage these complications, with the right drugs, equipment and people available.

Nagaratnam *et al* provide a brief guide to analgesic prescription for the surgical patient [5].

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18. NUTRITION IN SURGICAL CRITICAL CARE

MARCIA MCDOUGALL, ANNEKA MAXWELL

INTRODUCTION

Management of the critically ill patient requires a multifactorial approach. One important factor in the care of the critically ill patient is ensuring adequate and timely nutrition. This chapter aims to discuss important aspects to be taken into consideration in the nutritional management of the adult critical care patient. The content of this chapter is based on evidence-based guidelines and UK practices. Clinical judgement and multi-disciplinary team work however should take precedence along with development of local guidelines and protocols. After reading this chapter you should be able to:

- 1. Identify malnourished patients;
- 2. Understand the complex metabolic situation in critically ill patients;
- 3. Describe routes of feeding and their complications.

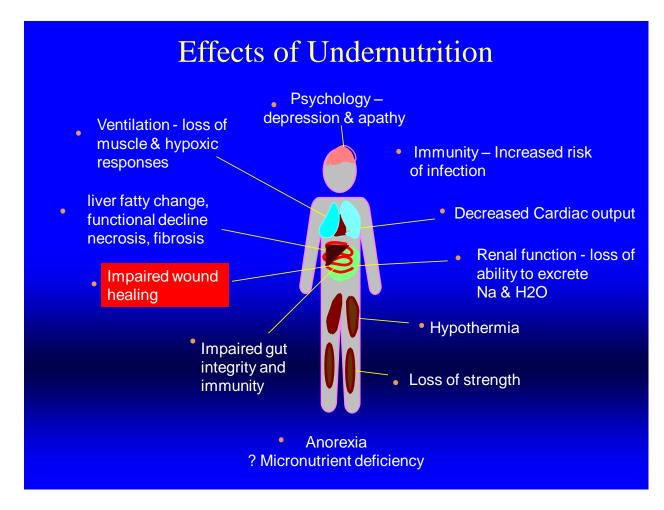
CHALLENGES IN FEEDING SICK PATIENTS

PRE-EXISTING MALNUTRITION

Providing nutrition to the critically ill patient is a challenge for a number of reasons. Many patients are already malnourished on admission to hospital or to critical care as a result of their acute illness which can quickly render even healthy people malnourished. Other causes of pre-existing malnutrition are chronic cardiac, respiratory, endocrine, gastrointestinal or parasitic disease, poverty, HIV or AIDS, alcoholism, illicit drug use, self-neglect or psychiatric illness. We must identify which patients are already malnourished as this will affect how they are fed.

Malnutrition is a broad term used to cover both over and under-nutrition. Currently there is no universally accepted definition. The British Association of Parenteral and Enteral Nutrition (BAPEN) describes malnutrition as; "a state of nutrition in which a deficiency or excess (or imbalance) of energy, protein and other nutrients causes measurable adverse effects on tissue / body form (body shape, size and composition) and function and clinical outcome". Although over-nutrition comes with its own challenges, the term malnutrition is used within this context to refer to under-nutrition. It is well documented that malnutrition include impaired wound healing and immune function, muscle wasting which may contribute to increased time on mechanical ventilation, increased length of stay, higher rates of readmission and increased morbidity and mortality (Figure 1) [1,2,3]. To minimise these complications it is important to identify patients at risk or those who are already malnourished early.

Figure 1: The Effects of Malnutrition:



ACUTE ILLNESS

Even if a patient is well nourished until becoming acutely ill, the illness itself usually results in anorexia or inability to eat, so that patients who are not fed rapidly become malnourished. After ten days without food a person is defined as malnourished. Being severely ill results in rapid muscle wasting of up to 30% in seven days in patients with multiple organ failure, despite adequate early nutrition. The mechanisms for this wasting are complex and poorly understood and at present there is little that can be done to prevent the wasting, but the provision of nutrition is essential to prevent even more catastrophic tissue loss. Wasting appears to be most severe in patients with prolonged hypoxaemic respiratory failure and multiple organ failure.

CATABOLISM AND INFLAMMATION

METABOLIC RESPONSE TO INJURY

It is well established the acute phase response induced by shock, trauma or sepsis has a significant impact on body metabolism. In 1932 Cuthbertson *et al* discussed the increase in metabolic rate and nitrogen losses associated with injury. With an increase in energy demands, liver glycogen stores are quickly exhausted. Catabolic hormones

are released in response to stress to break down body fat and skeletal muscle for energy substrates. In general the magnitude of the stress response is dependent on the degree of the insult/sepsis. Whilst a patient is in a catabolic state the breakdown of muscle and associated weight loss cannot be reversed simply by increasing nutrition. The cause of the stress therefore has to be identified and treated first before the hormonal balance can be restored to allow weight gain and muscle stores to be replenished.

Ongoing inflammation from any source, such as acute pancreatitis with pseudocyst, multiple trauma, burns or prolonged acute respiratory distress syndrome, therefore perpetuates the catabolic state and hence survivors of intensive care frequently emerge weak and wasted even though nutrition has been provided. With no nutrition at all a severely catabolic patient is likely to die within a month, as opposed to about ten weeks for a previously well person suffering complete starvation but no catabolic process. The time at which nutrition should be instituted is discussed later.

MAINTAINING ADEQUATE NUTRITION

Maintenance of adequate feeding in changing clinical situations is another challenge. Patients rapidly stop absorbing enteral feed if they become more unwell; iatrogenic complications of drug treatment such as antibiotic-related diarrhoea and opioid-related ileus are frequent barriers to adequate feeding. Displacement or blockage of tubes and lines and delay in their replacement, transfer out of the unit for procedures or investigations, inadequate supply of feeds and equipment and lack of expertise may be reasons that feeds are discontinued, missed or given in inadequate amounts.

GIVING THE CORRECT FEED FOR EACH PATIENT

Sick patients have differing nutritional needs depending on their nutritional status, their degree of catabolism, their weight and the effects of their illness. In units where a dietitian is employed, these issues are generally addressed by these professionals. However, in their absence, various techniques may be employed to arrive at an approximate figure for nutritional requirements.

Critically ill patients are often already immunosuppressed due to their illness. Malnutrition will exacerbate this state. Failure to feed patients adequately will worsen the muscle wasting of critical illness and can prevent weaning from ventilation. It contributes to poor wound healing and prolongs organ failure.

When critical illness, with or without organ failure, is superimposed on malnutrition, a complex and deleterious situation arises and it is thus essential to feed critically ill patients. Gastroparesis and ileus are common in the critically ill and lead to poor absorption of feed. Sepsis, sympathetic nervous system stimulation and the systemic inflammatory response lead to increased consumption of oxygen and an increase in resting energy expenditure; a third of energy requirements are met by breakdown of muscle protein and hence sick patients suffer significant loss of muscle bulk, exacerbated by inactivity. This occurs despite the provision of adequate nutrition and in previously healthy as well as malnourished patients but is much worse in the latter. Attempts to prevent muscle wasting in critical illness by hypercaloric feeding have failed and overfeeding can lead to lactic acidosis, persistent pyrexia, raised triglycerides, and fatty liver.

ASSESSMENT OF NUTRITIONAL STATUS

Establishing a patients' nutritional status is important, as this can influence the quantity and delivery of nutritional support. Essential to nutritional assessment are a full history and examination, a history of weight loss, recent food intake and gastro-intestinal symptoms, and weight. It is recommended that nutritional screening be carried out on admission and on a weekly basis thereafter whilst an inpatient [4]. The use of a nutritional screening tool allows the early identification of those under-nourished or those at risk and allows the implementation of an appropriate nutritional care plan. The 'Malnutrition Universal Screening Tool' (MUST) [©BAPEN], developed by the Malnutrition Advisory Group (MAG), is now widely used in hospitals in the United Kingdom [5]. MUST is a validated nutritional screening tool consisting of five steps (Appendix 1) [6]. MUST takes into consideration body mass index (BMI), percentage weight loss and acute disease effect. A score from zero to six is generated, with the greater the score indicating the greater the risk of malnutrition.

As an alternative, especially in the ICU or elsewhere when patients are confined to bed, the Mid Upper Arm Circumference may be used to assess malnutrition (Appendix 1; p6).

It is important to use a measure of some kind as overfeeding of patients carries its own risks (see "Complications" below).

WEIGHT, HEIGHT AND BODY MASS INDEX (BMI)

Measuring weight and height in the critically ill patient can be particularly challenging especially if the patient is sedated and/or ventilator dependent. In those who can be weighed it is important not to overlook amputations, ascites, oedema and plaster casts. These factors can have a significant influence on weight, which in turn may lead to overfeeding if used to calculate nutritional requirements. Often it is not practical to obtain a weight therefore a reported weight from the patient or a family member may provide a useful starting point. Weight as a single measure provides little information on a patient's nutritional status. Instead calculation of body mass index, which takes into consideration a patients weight in relation to their height, can be a useful guide to measure thinness or fatness. Visual estimation of weight may be up to 20% inaccurate but is often used. Height is also required for calculated by dividing weight in kilograms by height in metres squared. It should be noted the BMI cut offs included in MUST are based on the UK population and mainly Caucasians, therefore these measurements should be interpreted with this in mind.

PERCENTAGE WEIGHT LOSS

To help gain further insight into an individual's risk of malnutrition a simple weight history can be beneficial. In comparison to body mass index, unplanned weight loss over 3-6 months is considered a more acute risk factor for malnutrition [7]. Calculation of percentage weight loss helps to establish the degree of unintentional weight loss and its associated clinical significance. Unintentional weight loss greater than ten percent is considered to be clinically significant. Weight loss of 5-10% is more than normal variation and indicates increased risk.

Calculation of Percentage Weight Loss: % Weight loss = (usual weight - current weight) x100 ÷ usual weight.

DURATION OF REDUCED NUTRIENT INTAKE

Consideration into the cause of critical illness can also help to provide insight into a patient's nutritional status. For example, a patient involved in a road traffic accident may present normally nourished due to eating and drinking well prior to the incident. A post-operative patient, however, may present already malnourished due to their acute illness and the symptoms associated with the illness causing altered nutrient intake. Due to the hyper-metabolism induced by the catabolic effects of stress hormones after severe trauma, patients who are normally nourished on presentation can quickly become malnourished. In the critical care setting most patients are at high risk of malnutrition on MUST-score due to the 'acute disease effect'.

REFEEDING SYNDROME

Severely malnourished patients are at risk of the refeeding syndrome. The following patient groups are especially at risk: anorexia nervosa, marasmus, kwashiorkor, chronic malnutrition, chronic alcoholism, morbid obesity with massive weight loss, patients unfed in 7-10 days with evidence of stress and depletion, prolonged fasting and prolonged intravenous hydration [8]. If refeeding syndrome is not recognised it can be fatal. When the body's metabolism shifts from the free fatty acid metabolism of starvation back to utilisation of carbohydrate, acute deficiencies of thiamine and phosphate are precipitated, along with disruption of electrolyte and insulin metabolism. These changes cause intracellular shifts of potassium, magnesium and phosphate, sodium and fluid retention and several adverse clinical effects [9]. These include diarrhoea, arrhythmias, pulmonary oedema, respiratory muscle weakness, hypotension, immune dysfunction, lactic acidosis, and in extreme cases coma and seizures due to cerebral oedema. Prevention of refeeding syndrome includes identification of patients at risk, treatment vitamins B and C prior to feeding and for ten days and careful monitoring of fluid and electrolyte balance with urgent replacement of phosphate, magnesium and potassium as required.

Table Two: NICE Guidelines on Refeeding Syndrome

At Risk

Those who have had little or no nutrition for >5 days

High Risk

Those with any of the following:

- BMI <16kg/m²
- Unintentional weight loss >15% within past 3-6 months
- Very little or no nutrition >10days
- Low levels of potassium, phosphate or magnesium prior to feeding

Those with two or more of the following:

- BMI <18.5kg/m²
- Unintentional weight loss >10% within the past 3-6 months
- Very little or no nutrition >5days
- A history of alcohol abuse or some drugs including insulin, chemotherapy, antacids, diuretics

Extremely high risk

- BMI <14kg/m²
- Very little or no nutrition >15days

Once the likelihood of re-feeding syndrome has been determined the NICE guidelines (2006) suggest that, for those patients 'at risk', one should introduce nutrition at a maximum of 50% of total energy requirements for the first two days before increasing to full requirements if there are no biochemical abnormalities [4]. It is to be noted that although pre-feeding electrolytes may be normal, a patient can still be at risk of Refeeding Syndrome and electrolyte levels can quickly fall once feeding is commenced. For those at 'High Risk' it is recommended to consider starting nutrition at a maximum of 10kcal/kg and increase slowly to meet requirements by 4-7 days. Any increase in feed should be dependent on trends in biochemistry. In a patient identified to be at 'extremely high risk' guidelines suggest considering starting nutrition at 5kcal/kg and monitoring cardiac rhythm.

Guidelines suggest patients identified to be at high risk of refeeding syndrome should be given oral thiamine, vitamin B co-strong and a multi-vitamin if the oral route is available. These should be given 30 minutes before and during the first ten days of nutrition. Where the oral route is not available intravenous vitamin B and C preparation is recommended, e.g. Pabrinex 300mg/day for 10 days [4]. Electrolytes should be monitored daily and corrected with oral, enteral or intravenous supplementation of potassium, phosphate and magnesium where appropriate. If electrolytes remain low the feeding rate should be kept low until these stabilise.

CALCULATION OF NUTRITIONAL REQUIREMENTS

The method in which energy requirements are estimated in the critically ill patient continues to be an area of debate. Estimation of an individual's nutritional requirements is often undertaken by a specialist dietitian following detailed nutritional assessment. Energy requirements can be calculated through indirect calorimetry, the use of published predictive equations or through more simplistic equations based on calories per kilogram body weight.

Indirect calorimetry is the most reliable way of estimating calorie needs therefore it is classed as the 'gold standard'. Indirect calorimetry measures a patients carbon dioxide production and oxygen consumption but it is time consuming and often unavailable.

Predictive equations are an alternative method in calculating energy needs to allow estimation of basal metabolic rate (BMR). There are a large number of published predictive equations available. Examples include Henry, Schofield, Ireton Jones and Harris Benedict equations which take into consideration a patients weight, age and gender. Not all predictive equations are designed specifically for critical care use. Since they are based on healthy individuals' the addition of stress factors is necessary which can introduce error. The level of stress or activity factor should be determined by clinical judgement and thorough nutritional assessment. Assessment should include (but is not exhaustive) disease state, infection, pyrexia, nutritional losses, biochemical and inflammatory markers and consciousness level.

The use of 'per kilogram body weight' to estimate calorie requirements for the critical care patient has been suggested. The ESPEN guidelines propose feeding the critically ill patient no more than 20-25kcals per kilogram body weight per day during the acute phase. Once the patient is in the anabolic recovery phase this should be increased to 25-30kcals per kilogram body weight per day [10]. Burned patients require an addition of 60% to the basic energy expenditure figure (see Chapter 13).

Protein – Protein requirements are elevated in the critically ill patient; it is therefore important to supply adequate amounts to minimise nitrogen losses. As mentioned previously, nitrogen balance cannot be achieved simply by increasing nutrition while a patient is catabolic. The ideal amount of protein required is unknown however it will be influenced by the degree of hyper-metabolism. In patients with a BMI below 30kg/m² a range of 1.2-2.0g per kilogram actual body weight per day has been suggested [11].

Fluid – It is important to consider fluid requirements on an individual patient basis particularly in the critically ill patient and in those with renal impairment. An individual's fluid requirement should be reviewed on a daily basis and set depending on fluid balance targets. Maintenance fluid requirements are traditionally set at 25-30ml/kg/24hours, including drugs, feeds and infusions but this is overly simplistic in critically ill patients and can lead to seriously wrong calculations. Consideration of the volume that enteral and/or parenteral nutrition provides is important to avoid excessive amounts. Additional losses such as pyrexia, diarrhoea, excessive stoma output, wound exudate and surgical drains can all significantly impact on an individual's fluid requirement, therefore each critical care patient's fluid requirements should be calculated individually daily [12].

Extra fluid losses should be replaced with appropriate solutions depending on the type of loss. Upper gastrointestinal losses may be replaced with 0.9% sodium chloride solution; for losses above the pylorus potassium needs to be added and the patient has to monitored for metabolic alkalosis. Diarrhoea volume is replaced with a balanced solution or saline if balanced solutions are unavailable. Serum potassium may drop with diarrhoea. This can be replaced intravenously as a 20mmol KCI/500ml saline solution over four hours or with oral supplements. White or kidney beans, spinach, dried fruit, vegetable squashes, some fish, and bananas are a reasonable source of potassium if other preparations are unavailable.

ROUTES OF FEEDING

ORAL FEEDING

If a patient is able to eat normally, this should be encouraged. Many sick patients may be either too weak to feed themselves, however, or lose their appetites. They will require help and encouragement to eat. Frequently oral intake alone is not enough to maintain adequate calorific intake. Nutritional supplement drinks can be commercially available; some contain 300 kcal in 200ml.

ARTIFICIAL FEEDING

Artificial feeding refers to tube feeding into the gut (enteral feeding) or intravenous feeding into peripheral or central veins (parenteral feeding) and is required for patients who are unable to take oral nutrition due to their being obtunded, ventilated, having had surgery to the upper gastrointestinal tract, having an unusable gut or inadequate

absorption of nutrients. Artificial feeding of any type carries with it the risk of complications and any feeding programme in critical care must be set up with full regard to safety precautions to prevent harm and minimise risk.

It is more physiological to feed enterally than parenterally. Lack of enteral feeding leads to loss of enterocyte integrity, mucosal atrophy, reduced gut immunity and increased permeability [13]. In severe illness gut flora become pathogenic and contribute to systemic sepsis and ventilator-associated pneumonia. Enteral nutrients promote normal gastro-intestinal function.

Critically ill patients with trauma and burns should be fed within 24 hours of admission, preferably by the enteral route [10, 14]. Most enteral feed is administered naso-gastrically, but in patients with gastro-paresis but a working small bowel the post-pyloric route can be used via a naso-jejunal or jejunostomy tube. For gastric feeding confirmation of the tube position is paramount before use, to ensure the tube is in the stomach and not the lungs. The use of chest x-rays and checking the pH of gastric aspirates using pH graded paper are common practice to help establish tube position. It is recommended that the position of the nasogastric tube is confirmed before each use [4]. Naso-jejunal tubes may be placed at the bedside, by endoscopy or by interventional radiology with screening during placement. Jejunostomy tubes are placed by surgeons intra-operatively directly into the small bowel. (Routes of feeding Fig. 2)

ENTERAL FEEDING

The type and composition of enteral feed should be determined on a case by case basis following detailed nutritional assessment, often undertaken by a dietitian. Assessment should consider fluid balance, biochemistry (including the risk of refeeding syndrome), renal impairment, bowel movements, nutritional requirements and tolerance. Recent studies have suggested permissive underfeeding or trophic feeding to reduce the prevalence of gastrointestinal intolerance. It is not appropriate to advocate underfeeding as a routine strategy, especially in higher risk patients as it may lead to impaired recovery and worse clinical outcome; therefore complete nutrition to calorie and protein targets will benefit critically ill patients [13]. Commercially available feeds contain varying numbers of kilocalories per millilitre (1, 1.5, or 2 kcal/ml) and differing proportions of proteins, carbohydrates and fats. Types of feed include low sodium or fibre feeds, elemental or peptide-containing feeds for malabsorption, and oral supplements. (Fig. 1b) The more concentrated feeds are useful for patients who require fluid restriction, for instance those who have renal impairment and a reduced urine output, or who are receiving large volumes of fluid for other reasons. If commercial feeds are unavailable it is possible to administer processed liquid food via a nasogastric tube. If a meal is balanced in protein, carbohydrates and fat it will provide adequate nutrition for most patients. However the risks of this approach are under- or overfeeding as the calorific value of the food will be unknown, and there is also a possibility of introducing bacterial infection into the patient if the food is contaminated prior to administration. Strict food hygiene standards should be implemented where feeds are prepared for use in this way.

The website <u>http://www.healthaliciousness.com</u> contains extensive lists of foods with a high content of certain vitamins and nutrients and may be useful in planning hospital menus or specific feeds for sick patients.

A review of a patients' medication can allow the identification of any possible drugnutrient interactions which may alter the type and delivery of enteral feed. The absorption of some medications are known to be reduced by enteral feed or sometimes more specifically components of enteral feed eg fibre, therefore incorporating appropriate breaks in feed prior to the administration of medication is important. For those requiring insulin administrations feeding continuously over 24hours is deemed safer and more practical [4].

TOTAL PARENTERAL NUTRITION

Intravenous or total parenteral nutrition (TPN) is required for patients with a nonfunctioning or gastro-intestinal tract or who require bowel rest to allow healing. TPN may also be used to supplement enteral feed to achieve calorific goals. TPN is usually administered via a dedicated central venous catheter; meticulous attention to asepsis in line insertion and the care of such lines must be taken to avoid catheter-related blood stream infection. If infection is suspected the line must be removed. A PICC line is a peripherally inserted central catheter, inserted into the basilic or cephalic vein in the arm and extending to the central veins. Peripheral TPN may be given via the shorter Peripherally Inserted Catheter (PIC) but the formula must be of a lower osmolality that that of central TPN as otherwise thrombosis of the veins will quickly occur. Long-term TPN is given via tunnelled lines.

TPN bags are ready made by nutrition companies in a three-chambered form with a lipid, carbohydrate and protein section to maintain stability of the ingredients. These are mixed together immediately before use. Vitamins, trace elements and additional electrolytes are added by pharmacists in an aseptic chamber; if such a facility is not available these essential nutrients should be given separately.

Parenteral nutrition should be delivered using a volumetric pump with in-line air alarm. It should be introduced gradually, taking into consideration the risk of refeeding syndrome. In severely ill patients continuous administration should be considered [4]. Careful monitoring is essential, e.g. biochemistry, fluid balance, clinical picture, blood glucose levels, PN access route and nutritional requirements to minimise complications.

WHEN TO FEED, BY WHAT ROUTE AND SUPPLEMENTAL OR TOTAL?

As mentioned previously, enteral nutrition is the preferred route of nutrition in critical care and complies with: 'if the gut works, use it' approach. Total parenteral nutrition (TPN) should be considered when enteral nutrition is not an option. In some circumstances, for example poor gastrointestinal tolerance, enteral feed may not be adequate to meet the patients' nutritional needs therefore supplementary parenteral nutrition may then have a role to play [4]. The optimal time to initiate parenteral nutrition in a patient receiving inadequate enteral nutrition is unclear. There are contrasting opinions in the guidelines around when supplemental and total parenteral nutrition should be considered. The Society of Critical Care Medicine and American Society for Parenteral and Enteral Nutrition suggest initiation of supplemental PN after 7-10 days. In cases where enteral feeding is not feasible over the first 7-10 days following admission, it is suggested no nutrition support therapy should be provided, with parenteral nutrition only being considered after the first seven days in those previously healthy with no evidence of protein-calorie malnutrition. In those with evidence of protein-calorie malnutrition on admission and where enteral nutrition is not feasible, initiation of parenteral nutrition is recommended as soon as possible following adequate resuscitation [14]. The European Society of Parenteral and Enteral Nutrition on the other hand recommend consideration of supplementary PN in those receiving less than their targeted enteral feeding after two days. For all patients not expected to be on normal nutrition within three days and enteral nutrition is contraindicated, parenteral nutrition should be considered within 24-48 hours [15].

COMPLICATIONS OF NUTRITIONAL SUPPORT

DIFFICULTIES ASSOCIATED WITH THE MANAGEMENT OF CRITICAL ILLNESS

Frequent interruptions to feeding occur due to procedures; antibiotics and other drugs cause diarrhoea, and inotropes and opioids cause reduced gastric emptying. Feeding protocols are used to ensure optimum delivery of feed in the critically ill (e.g. Appendix 2). One study found patients only received 51.6% of their nutritional needs via enteral tube feeding due to different factors which impeded adequate delivery. These factors included increased residual volumes, tube displacement, routine nursing practices and gastrointestinal intolerance [11]. Looking for signs of poor tolerance to feed, for example abdominal distension, diarrhoea, vomiting, regurgitation of feed and complaints of nausea (in the conscious patient), can help to assess tolerance to enteral nutrition. One measure commonly used as an indicator of feed tolerance is monitoring of gastric residual volumes (GRV's). There is no agreed cut-off volume for GRV's however previously a 200ml cut-off was proposed but one study suggested rhat it can be increased to 500ml without risk of aspiration, although these patients all received metoclopramide initially [16].

ASPIRATION RISK

In all patients commenced on enteral nutrition, strategies should be employed to reduce the risk of aspiration. Strategies to minimise aspiration risk include providing a continuous feed instead of a bolus feed, elevating the head of the bed to 30-45°, using pro-motility agents (metoclopramide 10mg tds or erythromycin 250mg bd) and considering the placement of a post pyloric feeding tube [14].

OVERFEEDING

Overfeeding can lead to refeeding syndrome, lactic acidosis, raised triglycerides, fatty liver, hypercapnia, azotemia and hyperglycaemia. Insulin resistance is already a feature of critical illness and causes hyperglycaemia, which needs glucose control with intravenous insulin.

NG TUBE POSITIONING

Patients have died due to feed being administered into the lung, mediastinum or pleural cavity. If an aspirate of pH <5.5 is not obtained from the tube after insertion, a chest x-ray must be obtained to check position of the tube. The tip of the tube must be seen below the diaphragm on the patient's left side. Blockage of the tube, diarrhoea, vomiting, skin and mucosal damage are other complications of enteral feeding.

PARENTERAL NUTRITION

Complications of line insertion (pneumothorax, bleeding, vessel damage, air embolism), line infections, endocarditis, thrombosis, deranged liver function, cholestasis and gut atrophy may all occur.

SUMMARY

In summary, nutrition in the critically ill is a complex matter and must be carefully delivered and monitored by a multi-disciplinary team which ideally should include a physician/surgeon/intensivist, nurse, pharmacist and dietitian. The critical care patient population is not a homogeneous group, therefore 'a one size fits all' approach is not appropriate. Protocols should be used to ensure that all critical care patients are nutritionally assessed and their needs tailored on a case by case basis, feeding them by the most effective route. Close monitoring and review of their nutritional needs should be undertaken daily by the multidisciplinary team [17,18].

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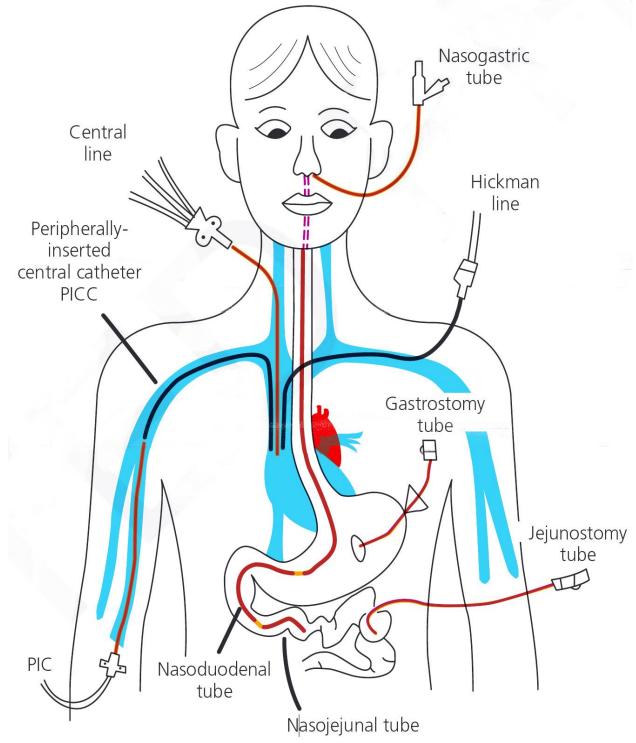
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Figure 2: Routes for Enteral or Parenteral Nutrition:





'Malnutrition Universal Screening Tool' 🛛 💆



BAPEN is registered charity number \$023927 www.bapen.org.u

'MUST'

'MUST' is a five-step screening tool to identify **adults**, who are malnourished, at risk of malnutrition (undernutrition), or obese. It also includes management guidelines which can be used to develop a care plan.

It is for use in hospitals, community and other care settings and can be used by all care workers.

This guide contains:

- A flow chart showing the 5 steps to use for screening and management
- BMI chart
- Weight loss tables
- Alternative measurements when BMI cannot be obtained by measuring weight and height.

The 5 'MUST' Steps

Step 1

Measure height and weight to get a BMI score using chart provided. If unable to obtain height and weight, use the alternative procedures shown in this guide.

Step 2

Note percentage unplanned weight loss and score using tables provided.

Step 3

Establish acute disease effect and score.

Step 4

Add scores from steps 1, 2 and 3 together to obtain overall risk of malnutrition.

Step 5

Use management guidelines and/or local policy to develop care plan.

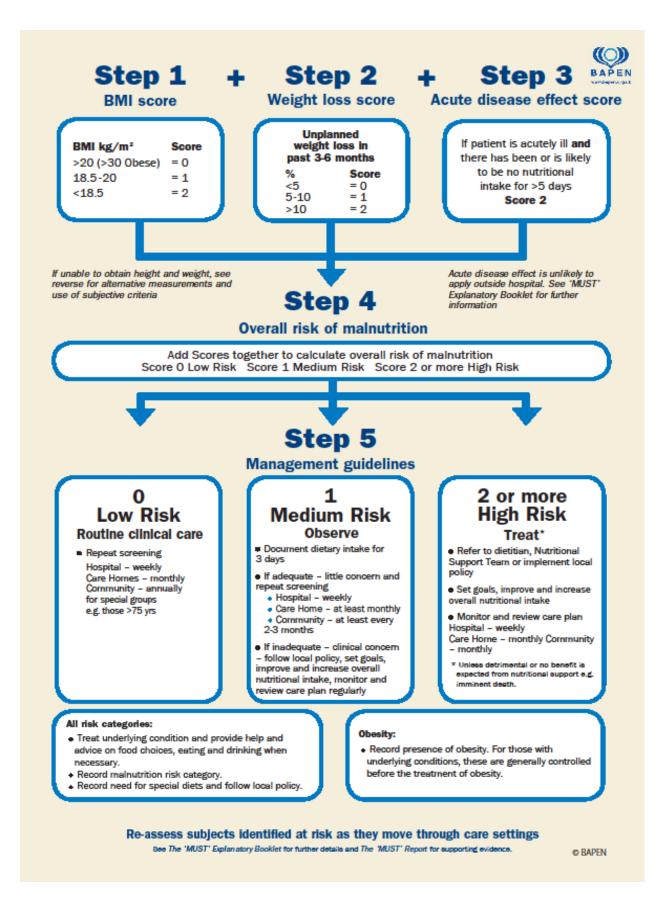
Please refer to *The 'MUST' Explanatory Booklet* for more information when weight and height cannot be measured, and when screening patient groups in which extra care in interpretation is needed (e.g. those with fluid disturbances, plaster casts, amputations, critical illness and pregnant or lactating women). The booklet can also be used for training. See *The 'MUST' Report* for supporting evidence. Please note that 'MUST' has not been designed to detect deficiencies or excessive intakes of vitamins and minerals and is of **use only in adults**.

Step 1 - BMI score (& BMI)



Height (feet and inches)

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Step 2 – Weight loss score

		Score 0	Score 1	Score 2						
		Wt loss < 5%	Wt loss 5 - 10%	Wt loss > 10%						
			ght loss in							
		3 to 6 months								
	ka	Less than	Between	More than						
	kg	(kg)	(kg)	(kg)						
	30	1.6	1.6 - 3.3	3.3						
	31	1.6	1.6 - 3.4	3.4						
-	32	1.7	1.7 - 3.6	3.6						
-	33	1.7	1.7 - 3.7	3.7						
	34	1.8	1.8 - 3.8	3.8						
	35	1.8	1.8 - 3.9	3.9						
	36	1.9	1.9 - 4.0	4.0						
	37	1.9	1.9 - 4.1	4.1						
	38	2.0	2.0 - 4.2	4.2						
-	39	2.1	2.1 - 4.3	4.3						
	40	2.1	2.1 - 4.4	4.4						
	41	2.2	2.2 - 4.6	4.6						
	42	2.2	2.2 - 4.7	4.7						
Current weight	43	2.3	2.3 - 4.8	4.8						
÷.	44	2.3	2.3 - 4.9	4.9						
Š.	45	2.4	2.4 - 5.0	5.0						
ŧ.	46	2.4	2.4 - 5.1	5.1						
2.	47	2.5	2.5 - 5.2	5.2						
Ъ.	48	2.5	2.5 - 5.3	5.3						
Ο.	49	2.6	2.6 - 5.4	5.4						
	50	2.6	2.6 - 5.6	5.6						
	51	2.7	2.7 - 5.7	5.7						
	52	2.7	2.7 - 5.8	5.8						
	53	2.8	2.8 - 5.9	5.9						
	54	2.8	2.8 - 6.0	6.0						
	55	2.9	2.9-6.1	6.1						
	56	2.9	2.9 - 6.2	6.2						
	57	3.0	3.0 - 6.3	6.3						
	58	3.1	3.1 - 6.4	6.4						
	59	3.1	3.1 - 6.6	6.6						
	60	3.2	3.2 - 6.7	6.7						
-	61	3.2	3.2 - 6.8	6.8						
	62	3.3	3.3 - 6.9	6.9						
	63	3.3	3.3 - 7.0	7.0						
	64	3.4	3.4 - 7.1	7.1						

	Score 0 Wt loss < 5%	Score 1 Wt loss 5 - 10%	Score 2 Wt loss > 10%
		ght loss in	
		to 6 month	
	Less than	Between	More than
kg	(kg)	(kg)	(kg)
65	3.4	3.4 - 7.2	7.2
66	3.5	3.5 - 7.3	7.3
67	3.5	3.5 - 7.4	7.4
68	3.6	3.6 - 7.6	7.6
69	3.6	3.6 - 7.7	7.7
70	3.7	3.7 - 7.8	7.8
71	3.7	3.7 - 7.9	7.9
72	3.8	3.8 - 8.0	8.0
73	3.8	3.8 - 8.1	8.1
74	3.9	3.9 - 8.2	8.2
75	3.9	3.9 - 8.3	8.3
76	4.0	4.0 - 8.4	8.4
77	4.1	4.1 - 8.6	8.6
78	4.1	4.1 - 8.6	8.7
79	4.2	4.2 - 8.7	8.8
80	4.2	4.2 - 8.9	8.9
81	4.3	4.3 - 9.0	9.0
82	4.3	4.3 - 9.1	9.1
83	4.4	4.4 - 9.2	9.2
84	4.4	4.4 - 9.3	9.3
85	4.5	4.5 - 9.4	9.4
86	4.5	4.5 - 9.6	9.6
87	4.6	4.6 - 9.7	9.7
88	4.6	4.6 - 9.8	9.8
89	4.7	4.7 - 9.9	9.9
90	4.7	4.7 - 10.0	10.0
91	4.8	4.8 - 10.1	10.1
92	4.8	4.8 - 10.2	10.2
93	4.9	4.9 - 10.3	10.3
94	4.9	4.9 - 10.4	10.4
95	5.0	5.0 - 10.6	10.6
96	5.1	5.1 - 10.7	10.7
97	5.1	5.1 - 10.8	10.8
98	5.2	5.2 - 10.9	10.9
99	5.2	5.2 - 11.0	11.0

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Alternative measurements and considerations



Step 1: BMI (body mass index)

If height cannot be measured

- Use recently documented or self-reported height (if reliable and realistic).
- If the subject does not know or is unable to report their height, use one of the alternative measurements to estimate height (ulna, knee height or demispan).

Step 2: Recent unplanned weight loss

If recent weight loss cannot be calculated, use self-reported weight loss (if reliable and realistic).

Subjective criteria

If height, weight or BMI cannot be obtained, the following criteria which relate to them can assist your professional judgement of the subject's nutritional risk category. Please note, these criteria should be used collectively not separately as alternatives to steps 1 and 2 of 'MUST' and are not designed to assign a score. Mid upper arm circumference (MUAC) may be used to estimate BMI category in order to support your overall impression of the subject's nutritional risk.

1. BMI

 Clinical impression – thin, acceptable weight, overweight. Obvious wasting (very thin) and obesity (very overweight) can also be noted.

2. Unplanned weight loss

- Clothes and/or jewellery have become loose fitting (weight loss).
- History of decreased food intake, reduced appetite or swallowing problems over 3-6 months and underlying disease or psycho-social/physical disabilities likely to cause weight loss.

3. Acute disease effect

Acutely ill and no nutritional intake or likelihood of no intake for more than 5 days.

Further details on taking alternative measurements, special circumstances and subjective criteria can be found in *The 'MUST' Explanatory Booklet*. A copy can be downloaded at www.bapen.org.uk or purchased from the BAPEN office. The full evidence-base for 'MUST' is contained in *The 'MUST' Report* and is also available for purchase from the BAPEN office.

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Alternative measurements: instructions and tables

If height cannot be obtained, use length of forearm (ulna) to calculate height using tables below. (See The 'MUST' Explanatory Booklet for details of other alternative measurements (knee height and demispan) that can also be used to estimate height).

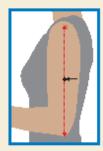
Estimating height from ulna length



Measure between the point of the elbow (olecranon process) and the midpoint of the prominent bone of the wrist (styloid process) (left side if possible).

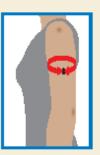
5e	men (<65 years)	1.94	1.93	1.91	1.89	1.87	1.85	1.84	1.82	1.80	1.78	1.76	1.75	1.73	1.71
Heigh (m)	men (≥65 years)	1.87	1.86	1.84	1.82	1.81	1.79	1.78	1.76	1.75	1.73	1.71	1.70	1.68	1.67
	Ulna length (cm)	32.0	31.5	31.0	30.5	30.0	29.5	29.0	28.5	28.0	27.5	27.0	26.5	26.0	25.5
통 순	Women (<65 years)	1.84	1.83	1.81	1.80	1.79	1.77	1.76	1.75	1.73	1.72	1.70	1.69	1.68	1.66
Height (m)	Women (≥65 years)	1.84	1.83	1.81	1.79	1.78	1.76	1.75	1.73	1.71	1.70	1.68	1.66	1.65	1.63
-	mon (kRE upper)	4.00	4.07	4.00		4.00	4.00	4.50	4.57	4.55	4.50	4.54	1.10	4.40	4.40
ξ.	men (<65 years)	1.69	1.67	1.66	1.64	1.62	1.60	1.58	1.57	1.55	1.53	1.51	1.49	1.48	1.46
Height (m)	men (≥85 years)	1.65	1.63	1.62	1.60	1.59	1.57	1.56	1.54	1.52	1.51	1.49	1.48	1.46	1.45
	Uina length (cm)	25.0	24.5	24.0	23.5	23.0	22.5	22.0	21.5	21.0	20.5	20.0	19.5	19.0	18.5
(m)	Women (<65 years)	1.65	1.63	1.62	1.61	1.59	1.58	1.56	1.55	1.54	1.52	1.51	1.50	1.48	1.47
μĘ	Women (≥85 years)	1.61	1.60	1.58	1.56	1.55	1.53	1.52	1.50	1.48	1.47	1.45	1.44	1.42	1.40

Estimating BMI category from mid upper arm circumference (MUAC)



The subject's left arm should be bent at the elbow at a 90 degree angle, with the upper arm held parallel to the side of the body. Measure the distance between the bony protrusion on the shoulder (acromion) and the point of the elbow (olecranon process). Mark the mid-point.

Ask the subject to let arm hang loose and measure around the upper arm at the mid-point, making sure that the tape measure is snug but not tight.

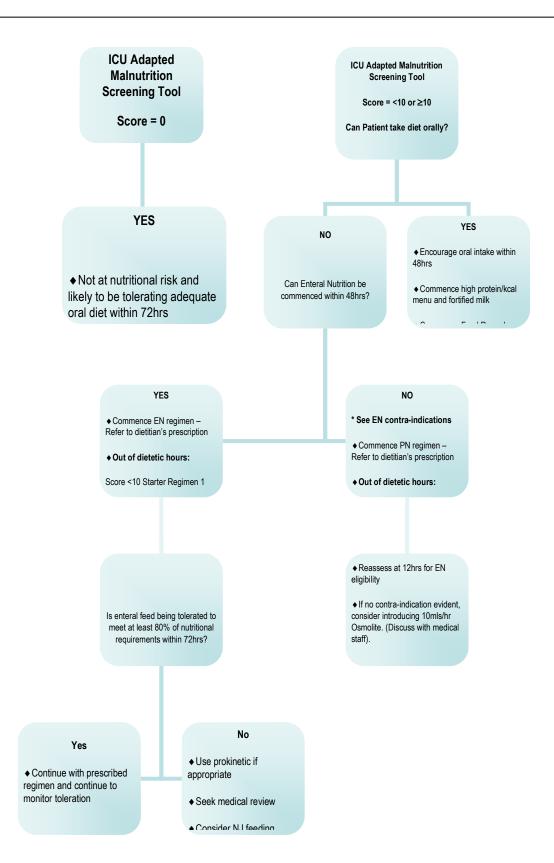


If MUAC is <23.5 cm, BMI is likely to be <20 kg/m². If MUAC is >32.0 cm, BMI is likely to be >30 kg/m².

The use of MUAC provides a general indication of BMI and is not designed to generate an actual score for use with 'MUST'. For further information on use of MUAC please refer to The 'MUST' Explanatory Booklet.

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ICU Nutrition Care Pathway



*Relative Contra-indications for EN: Surgical instructions, Severe Pancreatitis, Enteric anastomosis, Possible Ischaemic bowel, Enteric fistula, Consistently high aspirates, Severe IBD, Severe malnutrition – non functioning GI tract, Very high dose inotropes: caution with enteral feed due to risk of ischaemic bowel.

19. ADVANCED ICU CARE IN SURGERY AND AFTER MAJOR TRAUMA

D. WAYNE WRATHALL

Intensive care units (ICU) evolved to provide support of failing organ systems. In order to provide such support the ICU requires

- Medical expertise
- Nursing expertise and high nurse to patient ratio
- Equipment for supporting failing organs
- Intensive monitoring, usually continuous and often invasive

In this chapter we will consider the support of failing organ systems.

ASSESSMENT OF THE CRITICALLY ILL PATIENT

A structured assessment of a critically ill patient should include:

- **Primary assessment**: A rapid scan using the ABCDE approach to detect and manage immediately life threatening conditions
- Secondary assessment. A structured assessment of each organ system. This should include any available history, recent progress and observations, a clinical examination and consideration of any relevant test results

There are two main areas to consider:

- **The underlying disease** and its progression. If progress is not as expected then the diagnosis may need to be reconsidered; further investigations may be necessary or a change in therapy may be required.
- Optimising **support of failing organ systems.** In general as the patient improves the degree of organ support required reduces if organ support is requiring to be increased then the treatment of the underlying condition should be reconsidered

THE RESPIRATORY SYSTEM:

1. AIRWAY

Securing the airway in the critically ill patient is considered in chapter 5, 'The Airway in Trauma and Critical Care.'

Airway patency is compromised in many patients after major trauma due to the trauma itself, oedema or decreased conscious level from trauma or sedatives. Many such

patients will require an artificial airway. The commonest reason for requiring an artificial airway however is the need for mechanical ventilation due to respiratory failure.

Most patients will initially need a cuffed oral tracheal (endotracheal) tube.

Desirable features:

- straight, kink resistant (usually PVC)
- the largest diameter that comfortably passes through the glottis
- Position of the tip of the tube ideally confirmed on CXR
- Length of the tube at the lips should be noted and documented
- Cut to length 2-4cm beyond the lips
- Secure fixation by a method that is secure but easy to undo and minimizes trauma, especially to the corner of the mouth. Tape tied in a 'larks foot' knot is widely used [1]

Nasal endotracheal tubes are infrequently used in adults in intensive care due to the following disadvantages:

- longer length
- smaller diameter
- increased incidence of sinusitis which may increase the risk of ventilator associated pneumonia (VAP) [2]
- Risk of intracranial passage in basal skull fracture
- Risk of bleeding in patients with a significant coagulopathy

However they have some advantages:

- Use in oral surgery/trauma
- More secure fixation
- Better tolerated in awake patients: helpful when weaning an agitated patient
- Commonly used in paediatric patients: ease of fixation; lower risk of sinusitis

All endotracheal tubes have several disadvantages:

- They are long and relatively narrow
- Usually require general anaesthesia to insert.
- Usually require some sedation for the patient to tolerate their presence
- Promote micro-aspiration of pharyngeal secretions which is the major cause of ventilator associated pneumonia

Tracheostomy tubes may overcome some of these disadvantages. They may be required as a primary airway in maxillofacial trauma but are commonly used if prolonged intubation and ventilation are expected. There is no clear evidence that a policy of early (within the first week of ventilation) or late tracheostomy improves outcome [3] but there are several benefits of tracheostomy in patients requiring prolonged respiratory support:

- Usually tolerated without sedation: the patient can then co-operate with their care including communication, physiotherapy and mobilization.
- Lower resistance to breathing
- Easier to pass suction catheters and keep clean and patent.
- May be re-inserted without anaesthesia once track is formed (approximately 1 week after percutaneous insertion but immediately with some surgical techniques

Several types of tracheostomy tubes are available:

- **Cuffed or uncuffed** A cuffed tube allows a full range of ventilatory modes to be used and may protect to some degree from gross aspiration of material within the pharynx. An uncuffed tube is only suitable for limited ventilatory support but does allow speech and swallowing under certain conditions
- **Fixed or adjustable length.** Standard tracheostomy tubes have a fixed flange and therefore fixed length and will suit the majority of patients. However in obese patients or those with other forms of neck swellings a longer tube may be necessary and an adjustable flange tube can be used
- Inner cannula. These are desirable as they can be removed for cleaning or if they become blocked
- **Speaking valves** may be attached to the airway connector of tracheostomy tubes or may come as part of the inner cannula. They are one way valves that allow exhalation over the vocal cords *but only in uncuffed tubes or if cuff is deflated.* They improve speech and strength of cough

Insertion of a tracheostomy tube in ICU is usually performed under general anaesthesia and may be either open surgical or percutaneous Seldinger. The percutaneous 'Seldinger' (needle/guidewire/dilator) technique is usually preferred if available as it can be performed at the bedside and complication rates are lower. To perform safely, however it requires that oral intubation is straightforward and that the anatomy of the neck is not grossly distorted.

2. VENTILATORY SUPPORT

Ventilatory support may be necessary in respiratory failure but is supportive rather than therapeutic: the underlying condition needs to be effectively treated to return the patient to health.

Patients in respiratory failure require support of either oxygenation, ventilation or both.

In **Type 1 or hypoxic respiratory failure** oxygenation is improved by supplementing oxygen, strategies to reduce V/Q (ventilation / perfusion) mismatch and supporting ventilation when advanced ventilator modes are required or if patient becomes fatigued

In **Type 2 or hypercapnic respiratory failure** the patient is usually hypoxic *and* hypercarbic. The mechanism is lack of *alveolar ventilation*:

arterial PCO_2 is inversely proportional to the alveolar ventilation.

As carbon dioxide is a sedative in its own right the severely hypercarbic patient becomes less conscious and ventilation drops further, requiring progression to support of ventilation.

TYPES OF VENTILATORY SUPPORT:

• Face mask oxygen will reduce hypoxia in both Type1 and Type 2 respiratory failure

Non invasive ventilation (NIV). This is delivered to awake patients via specialized face masks, nasal cannula/prong or hood. It avoids many of the complications of invasive ventilation but requires a co-operative patient and has a limit of maximum pressure delivered of around 20cm H₂O. If the pressure is kept constant it is known as Continuous Positive Airway Pressure (CPAP), and is useful to reduce V/Q mismatch in Type 1 (hypoxic) respiratory failure

If inspiration triggers a higher pressure than in expiration it is called **BPAP** (Bilevel Positive Airway Pressure). This increases tidal volume and hence minute ventilation and is used in Type 2 (hypercapnic) respiratory failure

• **Invasive ventilation** is delivered via endotracheal tube or tracheostomy. It may be necessary in severely hypoxic patients to allow advanced ventilator modes, or in the tiring patient. Higher pressures and volumes can be delivered than in NIV for severely hypercapnic patients

MONITORING DURING RESPIRATORY SUPPORT:

- **Clinical observation** is the most important monitoring modality.
- **Pulse oximetry** is the most useful monitor of oxygenation subject to the limitations outlined in chapter 'Hypoxia in the Surgical Patient'
- **Capnometry** is particularly of benefit in assessing correct placement of an endotracheal tube (see chapter Airway Management in Trauma and Critical Care). The end tidal CO₂ tension correlates with the arterial PaCO₂ in health, however in severe lung disease the end tidal CO₂ increasingly underestimates the arterial PaCO₂
- Arterial blood gases may be helpful in assessing oxygenation if pulse oximetry is unavailable / unreliable. Assessment of the PaCO2 does give a good indication of adequacy of alveolar ventilation, however in most circumstances keeping the PaCO₂ within close limits is unnecessary. The principal exception to this is in neurotrauma where normal PaCO₂ should be targeted as it is a major determinant of intracranial blood volume and intracranial pressure
- **Chest X ray** is useful to check the position of endotracheal tube and other invasive devices such as nasogastric tubes, chest drains and central lines. It is also useful in monitoring the progression of any underlying lung condition but is rarely used to guide the level of respiratory support

INITIATING INVASIVE VENTILATORY SUPPORT

In most circumstances invasive ventilatory support is initiated by inducing general anaesthesia and muscle relaxation and inserting an endotracheal tube as described in detail in the Chapter 'Airway Management in Trauma and Critical Care. Under some circumstances awake intubation or tracheostomy under local anaestetic are options.

Capnometry is very useful to confirm airway postioning and effective ventilation. Initial hand ventilation with a self inflating bag or manual circuit gives the experienced operator a 'feel' of resistance and compliance of the lungs and a fail safe if the mechanical ventilator malfunctions

TYPICAL INITIAL VENTILATOR SETTINGS

The different modes of invasive ventilation are beyond the scope of this chapter. Ventilator settings should be titrated to effect. Clinical observation of the patient is important, particularly in children where delivered tidal volume is often much less than set tidal volume due to compliance in the tubing and leaks around non-cuffed ET tubes

Tidal volume	Respiratory rate RR	Inspiratory time	Peak inspiratory pressure	PEEP	F ₁ O ₂
5-10ml/kg Adults and children	10-20 in adults 20-40 in children, faster in younger age groups	1 – 2s or 35- 50% of total breath time Inspiratory time should not exceed expiratory time.	30-35 cm H ₂ O in volume control. In pressure control adjust pressure to achieve desired tidal volume/chest movement	5 cm H_2O Adjust upwards if requires high F_1O_2	1.0 initially Titrate down rapidly according to SpO ₂

WEANING AND LIBERATION FROM MECHANICAL VENTILATION

The term 'weaning' is a misnomer. The patient will be liberated from ventilation provided the lung disease is treated, the respiratory muscles are strong and the patient is conscious with good ventilatory drive

Progression to liberation typically follows the following steps:

- 1. Ensure lung disease is adequately treated
- 2. Encourage spontaneous breathing:
 - a. Reduce sedative drugs
 - b. Reduce mandatory mechanical breaths
 - c. Establish on spontaneous breathing mode
- 3. Ensure hypoxia is absent on low F_1O_2 (< 0.5)
- 4. Reduce PEEP to less than $10 \text{cm} H_2O$
- 5. Reduce any pressure support or ASB (assisted spontaneous breathing) to less than 10-15cmH₂O
- 6. Assess strength of cough and sputum production
- 7. Reduce/stop sedation to allow consciousness to return [4]
- 8. Consider use of a 'T Piece trial': a period of 30 minutes spontaneous breathing through ETT with supplemental oxygen but without mechanical ventilation [5]
- 9. Clinical assessment prior to extubation

COMPLICATIONS OF MECHANICAL VENTILATION

1. **Pneumothorax** is now less common as it is recognized that high tidal volumes (volutrauma) and high inflation pressures (barotrauma) should be avoided. A simple pneumothorax often progresses rapidly to a tension pneumothorax during mechanical ventilation and should be considered in any case of ventilatory and cardiovascular collapse

- 2. **Dynamic hyperinflation**, where insufficient time for expiration leads to incomplete emptying of the lungs typically occurs in patients with bronchospasm and if unrecognised may lead to cardiovascular collapse and barotrauma
- 3. Ventilator associated pneumonia (VAP) is usually due to colonization of the pharynx with pathological micro organisms which gain access to the lung through micro-aspiration past the ET tube. It is a cause of prolonged ICU stay and increased morbidity and mortality It can be reduced by sitting the patients 30° head up, ensuring good mouth hygiene including oral antiseptics and reducing duration of intubation [6]. Daily sedation breaks when appropriate reduce the accumulation of sedatives and promote early extubation [4]
- 4. Critical Illness Polymyoneuropathy (CIP) may lead to a profound flaccid weakness of all skeletal muscle with prolonged and incomplete recovery of respiratory and neuromuscular function. It is particularly prevalent in patients treated with steroids and neuromuscular paralyzing drugs for prolonged periods. It is important to limit the use of these drugs if possible and to recognize CIP as it is a cause of failure to liberate from mechanical ventilation [7]

THE CARDIOVASCULAR SYSTEM

All patients require that sufficient oxygenated blood is delivered to their tissues to meet metabolic need. Circulatory failure that does not meet this demand is **shock**. See chapter 'Shock and Haemorrhage'

Circulatory shock is caused from failure of either or both of the following:

1. Adequate cardiac output

2. Appropriate distribution of cardiac output

1. CARDIAC OUTPUT

Cardiac output = stroke volume x heart rate

The determinants of stroke volume are:

- Preload: the stretch on the cardiac muscle fibres *prior to onset* of contraction and affects stroke volume according to the Starling curve. Preload is closely related to venous return and lack of preload is the mechanism of shock in hypovolaemia
- **Contractility**: the force of contraction of the cardiac muscle fibres
- Afterload: the tension in the cardiac fibres *during* contraction. It is mainly determined by the aortic pressure and degree of arterial tone but is also increased by dilatation of the ventricles and stenosis of the aortic/pulmonary valves. Reduced afterload (ie vasodilation) favours a higher cardiac output but tends to lower blood pressure

2. THE DISTRIBUTION OF CARDIAC OUTPUT

The main distributing force of cardiac output is the **blood pressure**. Each organ has a range of blood pressure over which it can *autoregulate* its own blood flow. If the pressure drops below the lower limit of autoregulation, decreased organ blood flow and shock will ensue. The **mean** blood pressure is the relevant parameter, except for coronary circulation which is perfused during diastole.

In certain conditions there is inappropriate vasodilation or vasoconstriction in certain organs or vascular beds leading to an inappropriate distribution of cardiac output. An example of this is in high spinal cord injury where loss of thoracic sympathetic outflow causes inappropriate vasodilation below the level of injury.

MONITORING THE CIRCULATION

- Clinical observation can detect abnormal circulatory parameters such as tachycardia and hypotension. Signs of abnormal organ function such as poor skin perfusion, oliguria and altered conscious level may be additional evidence of shock
- ECG will detect arrhythmias, the treatment of which can improve cardiac output
- **Invasive Arterial Blood Pressure** via an arterial cannula gives beat to beat measurement of blood pressure and a means of sampling arterial blood. The measurement is prone to many errors:
 - Inappropriate setting of zero measurement
 - Resonance which exaggerates pulse pressure, artefactually elevating systolic BP
 - o Damping which reduces pulse pressure, artefactually lowering systolic BP

The latter two errors affect systolic and diastolic pressure but mean pressure is relatively unaffected and therefore is usually recorded: it also correlates better with non-cardiac organ blood flow.

Additional hazards of arterial cannulae include local infection, thrombosis and disconnection leading to major blood loss. For the latter reason arterial cannulae should always be transduced: loss of the waveform giving a warning of disconnection

- **Central venous pressure**, usually using an internal jugular cannula is often used as an indirect measure of preload, however it correlates very poorly with subsequent response to fluid challenge [8] and is often of questionable clinical value. The principal use of central venous cannulae is for administration of inotropes and vasopressors (see below)
- Arterial blood gases are useful in the assessment of tissue perfusion: a metabolic acidosis without other clear cause, especially in the presence of a raised blood lactate level is an indication that shock may exist. Blood lactate is

an important prognostic marker and indicator of the effectiveness of treatment of shock

• **Cardiac output measurement**. A variety of methods are available. The pulmonary artery catheter is rarely used now due to evidence of harm and lack of clear evidence of benefit [9]. Devices using Oesophageal Doppler, arterial waveform analysis, impedance plethysmography are available, all with advantages and disadvantages. The principal use of such monitors is assessing the response to a fluid bolus

ASSESSMENT OF THE CIRCULATION

The circulation cannot be assessed in isolation but as part of a structured clinical assessment of all organ systems. The heart and circulation itself should be clinically examined, the level of circulatory support assessed and the results of investigations and advanced monitoring reviewed. Other organ systems should be assessed for evidence of impaired function due to shock but also for a cause for the circulatory abnormality such as sepsis.

SUPPORT OF THE FAILING CIRCULATION

1. OPTIMISING PRELOAD

The optimal preload is that at which cardiac output is maximized but the undesirable effects of fluid overload are avoided. Determining the optimal preload or 'volume' status is not always straightforward in a complex critically ill patient.

METHODS INCLUDE:

• The response to a fluid challenge

250-500ml of crystalloid (e.g. normal saline or Ringers lactate) is administered as a rapid bolus over a few minutes with clinical observation before and after. An increase in blood pressure by more than 10% suggests that the cardiac output has increased and further fluid boluses are indicated until no further increase in blood pressure is seen. An increase in respiratory rate, decrease in SpO₂ or decrease in lung compliance may indicate that pulmonary oedema is developing.

• Passive leg raise

This has the effect of increasing venous return and preload and can simulate a fluid bolus without administering fluid [10]

Recent fluid balance history and urine output

This is typically inaccurate and reflects total body fluid rather than intravascular volume.

• The Central Venous Pressure (CVP) has very poor correlation with optimal left ventricular preload. (see above)

• Assessment of the arterial pressure waveform may be helpful in fully ventilated patients: a variation of more than 10% in systolic or pulse pressure during the respiratory cycle indicates that stroke volume is responsive to changes in preload. This can be visually seen as a fluctuation in the pressure waveform with respiration [11].

2. TREATMENT OF ARRHYTHMIAS:

Inappropriately **slow heart rates** will cause shock because

cardiac output = stroke volume x heart rate. Depending on the cause, atropine; adrenaline infusion or cardiac pacing may be effective

Inappropriately **fast heart rates** will also reduce cardiac output by reducing diastolic filling. Supra ventricular tachycardia, ventricular tachycardia and atrial fibrillation may be treated with anti-arrhythmics or DC cardioversion. Cardioversion has added benefits in atrial fibrillation, particularly in valvular heart disease where atrial contraction significantly augments cardiac output.

3. INOTROPES AND VASOPRESSORS:

An **inotrope** is a drug that improves force of cardiac contraction. Most inotropes are also *chronotropes* : they increase the heart rate. Inotropes increase myocardial oxygen consumption and can therefore worsen myocardial ischaemia. They also tend to exacerbate arrhythmias. Some inotropes (e.g dobutamine, milrinone) also cause some vasodilatation and are termed **inodilators.** This may have a further beneficial effect on cardiac output by reducing afterload, however it may exacerbate hypotension.

A **vasopressor** is a drug that increases blood pressure by causing peripheral arterial vasoconstriction. 'Pure' vasopressors increase afterload and therefore reduce cardiac output but many vasopressors also have some inotropic action which offsets this effect.

Most inotropes and vasopressors are potent short lasting drugs that best given by continuous pump controlled infusion, preferably through a central venous catheter but if not possible then by peripheral infusion of a dilute solution. As they are short acting drugs blood pressure measurement should be frequent and preferably continuous using an arterial cannula. At higher doses significant hypotension will occur when changing infusion syringes/bags, this can be avoided by using two pumps

Commonly used drugs include:

- **Noradrenaline.** A vasopressor with some inotropic action. If available considered first choice in septic shock and most other vasodilatory shock [12]
- Adrenaline. An inotrope, chronotrope and vasopressor. Highly potent and can be used first line in septic or cardiogenic shock. Intense vasoconstriction and increase in after load limits use in cardiogenic shock, tachyarrhythmias may limit use also. Increases lactate production, reducing value of lactate monitoring. The first line drug in anaphylaxis
- **Dopamine**. An inotrope, chronotrope and vasopressor. Can be given peripherally. Tends to cause tachyarrhythmias. Increases urine output but does *not* improve outcome in acute renal failure

- **Dobutamine.** An inodilator. Good choice in cardiogenic shock. Increases cardiac output but may decrease blood pressure. Often used in combination with a vasopressor e.g. noradrenaline
- **Phenylephrine.** A 'pure' vasopressor. Increases afterload hence decreases cardiac output. Tends to cause reflex bradycardia

Typical 'recipes' for inotropes:

• Adrenaline/noradrenaline

3mg in 50ml saline or dextrose at 2 – 50ml/hr via central line 1ml/hr=1 microgram per minute (Dilute 10 fold for peripheral administration)

Dobutamine/dopamine
 200mg in 50ml saline or dextrose, 0-20ml/hr
 1ml/hr = 1microgram per kg per minute for a 67kg adult

RESUSCITATION FROM SHOCK IN CLINICAL PRACTICE

- 1. Administer intravenous fluids by repeated bolus until preload is optimized. Use blood and blood components if indicated
- 2. Establish central venous access if possible.
- 3. If blood pressure still low after fluid resuscitation clinically assess cardiac output
 - If cardiac output thought to be high (most cases of fluid resuscitated shock) then choose a **vasopressor**
 - If cardiac output is thought to be low then choose an **inotrope**
- 4. Titrate rate of infusion to effect, increasing or decreasing every 2-5 minutes aiming for:
 - Mean arterial pressure of 55-80 mmHg
 - Evidence of improved organ function
- 5. Reconsider repeat fluid boluses

FLUIDS, ELECTROLYTES AND ACUTE KIDNEY INJURY

1. FLUID BALANCE

Close attention to fluid balance is one of the cornerstones of ICU practice. Untreated hypovolaemia and fluid overload are both harmful.

All decisions on fluid therapy should take account of the formula:

Fluid requirement = deficit + maintenance + ongoing losses

DEFICIT

This may be estimated from clinical observations, for example trauma guidelines for acute blood loss [13] or from knowledge of recent fluid losses, changes in body weight etc. All such estimates are prone to large error and more usually fluid boluses are administered whilst looking for clinical effect. It may be hazardous to replace deficit

rapidly and typically deficit is replaced over 24-48hr. This is particularly the case when deficit is associated with gross abnormalities in serum sodium levels where correction over several days may be indicated.

MAINTENANCE

The 4-2-1 rule may be used for children and adults in many clinical circumstances:

4m/kg/hr for first 10kg body weight

2ml/kg/hr for next 10kg body weight

1ml/kg/hr for all weight above 20kg

e.g. a 75kg adult would receive $4ml/hr \times 10 + 2ml/hr \times 10 + 1ml/hr \times 55 = 115ml/hr$.

Sodium requirements are typically 1mmol/kg/day so our 75kg adult would require 75mmol i.e. 500ml of 0.9% saline with the rest of the fluid being given as free water (dextrose 5% when given iv).

However in the critically ill individual neuroendocrine response to stress, particularly cortisol and ADH; retains sodium and water. Maintenance requirements are usually *less* than in health and as much sodium is often administered with drugs it is often rational to reduce maintenance fluid by 30-50% and administer as free water only. The exception to this is in febrile and tachypnoeic patients who have additional insensible losses.

ONGOING LOSSES

These may be **external** or **internal**:

- External losses are usually easier to measure and estimate. In general they are replaced with sodium containing crystalloid fluids. GI losses may require other electrolyte replacements
- Internal losses are more difficult to estimate. Most critically ill patients develop a capillary leak and expand their interstitial fluid space (the erroneously named 3rd space losses) Such loss occurs early in their illness and fluid to replace this loss should be reduced early with the best approach being to examine for deficit frequently and replace with bolus. The same applies to loss into the GI tract in ileus

2. ELECTROLYTE BALANCE

Sodium is predominantly distributed to extracellular space and the plasma concentration is similar to that throughout the extracellular space. The blood brain barrier is relatively impermeable to sodium thus rapid changes in serum sodium cause osmotic expansion or contraction of the brain. Severe hyponatraemia may cause seizures. Sodium maintenance and replacement requirements are discussed above

Potassium is predominantly an intracellular ion and plasma levels reflect movements in and out of cells. Acidosis is associated with movement of potassium out of cells, insulin therapy and beta adrenergic agents favour movement into cells. Renal failure and rhabdomyolysis are common causes of severe hyperkalaemia in ICU, diuretics are common causes of hypokalaemia. Hypokalaemia may precipitate arrhythmias, particularly atrail fibrillation. Hyperkalaemia causes bradycardia, conduction block and asystole.

Severe hyperkalaemia is a medical emergency with the following treatment options

- 1. Protect the heart: Intravenous calcium e.g. calcium chloride 10ml 10%
- 2. *Move potassium into cells*: intravenous bicarbonate; insulin/dextrose; salbutamol nebulisers
- 3. *Move potassium out of body:* diuresis; ion exchange resins enterally; dialysis

Daily potassium requirements are 0.5-1mmol/kg but vary enormously in critical illness

Calcium is an extracellular ion. Severe hypocalcaemia may cause arrhythmias, decreased cardiac contractility and tetany. Intravenous calcium should be given slowly, particularly in the presence of tissue ischaemia

Magnesium is an intracellular ion. Low plasma and intracellular levels are frequently found in critical illness, particularly when feeding a patient after prolonged nutritional deficit. Low levels are associated with arrhythmias, hypokalaemia and hypocalcaemia. If arrhythmias are present magnesium 4-8mmol may be given rapidly over 2-5min but for uncomplicated replacement (e.g. 8-16mmol) should be infused slowly e.g. over 12 hrs otherwise much is lost in the urine.

Phosphate is an intracellular ion that is excreted by the urine. High levels are found in renal failure and rhabdomyolysis, are associated with hypocalcaemia but rarely cause severe short term problems in. Low levels are associated with muscular weakness and decreased cardiac contractility. Phosphate can be replaced orally or intravenously at up to 50mmol/day.

3. ACUTE KIDNEY INJURY

Acute kidney injury (AKI) is defined by a reduction in glomerular filtration rate (and associated rise in serum creatinine) and/or a decrease in urine output. The **RIFLE** criteria were created in 2002 by the Acute Dialysis Quality Initiative (ADQI) [14] with the primary goal of developing consensus- and evidence-based guidelines for the treatment and prevention of Acute Kidney Injury.

Stage	GFR ^{**} Criteria	Urine Output Criteria	Probability
R isk	SCr [†] increased × 1.5	UO [‡] < 0.5 ml/kg/h × 6	High sensitivity
	or GFR decreased	h	(Risk >Injury
	>25%		>Failure)
Injury	SCr increased × 2	UO < 0.5 mL/kg/h x	High sensitivity
	or	12 h	(Risk >Injury
	GFR decreased		>Failure)
	>50%		
Failure	SCr increased × 3	UO < 0.3 ml/kg/h × 24	High sensitivity
	or	h	(Risk >Injury
	GFR decreased 75%	(oliguria)	>Failure)
	or	or	
	SCr ≥4 mg/dl; acute	anuria × 12 h	
	rise ≥0.5 mg/dL		

Loss	Persistent acute renal failure: complete loss of kidney function >4weeks		High specificity
End stage renal disease		Complete loss of kidney function >3 months	

- **GFR—glomerular filtration rate; †SCr—serum creatinine; ‡UO—urine output
- Note: Patients can be classified either by GFR criteria or by UO criteria. The criteria that support the most severe classification should be used. The superimposition of acute on chronic failure is indicated with the designation RIFLE-F_C; failure is present in such cases even if the increase in SCr is less than 3-fold, provided that the new SCr is greater than 4.0 mg/dl (350 µmol/l) and results from an acute increase of at least 0.5 mg/dl (44 µmol/l).

When the failure classification is achieved by UO criteria, the designation of RIFLE- F_0 is used to denote oliguria. The initial stage, "risk," has high sensitivity; more patients will be classified in this mild category, including some who do not actually have renal failure. Progression through the increasingly severe stages of RIFLE is marked by decreasing sensitivity and increasing specificity. Acute Kidney Injury is common in the ICU. Managing early stage AKI may prevent progression to more advanced stages.

THE CAUSES OF AKI ARE CLASSIFIED AS:

- **Prerenal:** any condition leading to a decrease in kidney perfusion predominantly hypotension and decreased cardiac output
- Renal: Intrinsic kidney disease or drugs/toxins adversely affecting renal function
- **Post renal:** Any obstruction to urine flow either within or outside of the kidney

MANAGEMENT OF ACUTE KIDNEY INJURY

- 1. Treat shock. Correct hypovolaemia with fluid therapy, treat persistent hypotension with inotropes/vasopressors.
- 2. Exclude obstruction of urine flow: examine for enlarged bladder, place or check patency of urinary catheter. Ultrasound examination of renal tract to exclude ureteric obstruction is ideal.
- 3. Consider other causes of intrinsic renal disease such as streptococcal infection, any septicaemia, blood transfusion reaction, rhabdomyolysis.
- Review drugs for presence of renal toxins. Non steroidal anti inflammatory drugs (NSAIDS), angiotensin converting enzyme inhibitors (ACEI), Xray contrast media are commonly used renal toxins.
- 5. Review drugs to adjust doses. Many drugs are excreted by the kidney and doses need to be reduced accordingly.

- 6. Look for and treat electrolyte abnormalities, particularly hyperkalaemia (see above).
- 7. Consider commencing renal replacement therapy if available.

TYPES OF RENAL REPLACEMENT THERAPY

Peritoneal dialysis is a simple renal replacement method where several litres of balanced dialysis fluid are infused into the peritoneal cavity for a period of hours then drained. Exchange of small molecules occurs across the peritoneal membrane. It does not have a high efficacy, particularly in a critically ill patient with high metabolic rate and is not suitable in abdominal trauma and sepsis.

Haemodialysis/haemofiltration. A variety of different modes are available. They are usually performed through a large bore double lumen central venous line with a pump driving blood through the dialyser/filter. Treatment can be intermittent or continuous, the latter is helpful for fluid balance but there is no clear outcome benefit for either [15].

Haemodialysis is an efficient method for removal of small electrolytes and toxins and combined with ultrafiltration can remove fluid rapidly. **Haemofiltration** is less efficient and therefore requires longer treatment but removes small molecules and some larger molecules. Equipment may be simpler but costs may be higher as large quantities of haemofiltration fluid are often used. Combined modes of filtration and dialysis are commonly used

In patients with chronic renal failure 3-4 hrs of haemodialysis 3-4x per week may be sufficient. Critically ill patients often require longer 'gentle' treatments as they are less tolerant of high ultrafiltration rates and electrolyte movements. Daily treatments of 12hrs or longer may be optimal.

Anticoagulation may be necessary to prevent the dialysis circuit clotting however the anticoagulation can cause problems in the surgical/trauma patient.

INDICATIONS FOR INITIATING HAEMODIALYSIS/FILTRATION:

None of these indications are absolute and the timing of initiation of renal support are much debated [16].

- Severe hyperkalaemia with acute kidney injury, failure or loss
- Severe fluid overload with oliguric kidney injury not responding to diuretics
- Dialysable life threatening toxin such as lithium, ethylene glycol
- Severe metabolic acidosis with acute kidney injury
- RIFLE: F especially if anuric and situation not expected to improve

NUTRITIONAL SUPPORT

Patients who are not supported nutritionally will ultimately not survive through malnutrition. However overzealous attempts to feed may cause more harm than good.

There is much research still to be done to determine the correct strategy for feeding critically ill patients.

Nutritional support in critical illness should follow the following widely accepted principles:

- 1. Oral feeding in the conscious patient is preferable to artificial enteral or parenteral feeding for reasons of oral hygiene, patient morale and ease of administration
- 2. Enteral tube feeding is preferable to intravenous feeding if the enteral route is available
- 3. Initiation of feeding should be cautious in malnourished patients with close electrolyte monitoring and supplementation to avoid 'refeeding syndrome', where electrolytes move into cells when feeding is commenced, leading to critically low extra cellular levels of Potassium, Magnesium and Phosphate
- 4. Overfeeding is potentially more hazardous than underfeeding. Overfeeding increases metabolic rate, oxygen demand and carbon dioxide production and increases fat deposition without increasing muscle mass
- 5. High gastric volumes predispose to regurgitation and aspiration of gastric contents, especially in intubated patients. Regular aspiration of the stomach should be performed and if residual volume is high (e.g >250ml) then consideration should be given to using prokinetic drugs or reducing rate of feeding
- 6. High blood glucose associated with artificial feeding is undesirable although the optimal level of glucose in uncertain [17]
- 7. Daily nutritional targets of 25-30kcal/kg and 1-1.5g/kg of protein are reasonable

NEUROLOGICAL SUPPORT AND SEDATION

Support of the injured brain is considered in the chapter 'Trauma Neurosciences'

SEDATION AND ANALGESIA IS OFTEN REQUIRED IN ICU FOR THE FOLLOWING REASONS:

- To manage delirium and agitation associated with critical illness
- To treat pain associated with injury or illness
- To allow the patient to tolerate certain therapies, particularly oral intubation and mechanical ventilation.

HOWEVER SEDATION AND ANALGESIA HAVE UNDESIRABLE EFFECTS:

- Decreased level of consciousness and airway protection in non-intubated patients
- Decreased respiratory drive
- Decreased ability to co-operate with therapies such as mobilization
- Decreased ability to communicate with patient

- Immobility and increased risk to pressure areas and risk of venous thrombosis
- Decreased gastrointestinal function
- Delay in liberation from mechanical ventilation
- Withdrawal effects

ANALGESIC DRUGS

Paracetamol may be used alone or in combination. Dose should be adjusted to body weight and used with caution in liver disease

NSAIDs can be very effective in traumatic and surgical pain however they increase risk of gastrointestinal bleeding and acute kidney injury and should not be used in those at high risk of these conditions

Opioids are effective and often necessary. Most accumulate in critical illness, particularly in renal failure and when used by continuous infusion.

SEDATIVE DRUGS

All common sedative drugs accumulate in critical illness and their use is associated with prolonged tracheal intubation and increased risk of ventilator associated pneumonia. This can be minimized by the use of **daily sedation breaks** to allow conscious level to improve.

Benzodiazepines are effective sedatives and have a strong amnesic effect. Large doses may be required in children. They are effective by enteral or parenteral route.

Propofol is given by infusion and accumulates less than benzodiazepines. It is considerably more expensive. It should be used cautiously in low body weight patients and children as the lipid carrier is toxic in excess.

Major tranquillizers such as haloperidol are of use in agitated delirium. Most have a dose limit and may cause cardiac arrhythmias

HAEMATOLOGY

1. HAEMOGLOBIN LEVEL

Anaemia in ICU is common and associated with poorer outcomes in most diseases. Anaemia is multifactorial and can be reduced by

- Minimising blood loss during surgery
- Preventing/treating on going bleeding from trauma and GI tract
- Reducing frequency of blood tests and minimizing volume of blood drawn
- Good nutrition and use of haematinics such as iron and folate

Transfusion of red blood cells may be necessary but may cause fluid overload, transfusion reactions, inflammatory response and worsening of acute lung injury. In the majority of patients a trigger for transfusion of a Hb level of 70g/l (7g/dl) is as safe as more aggressive transfusion [18]. Exceptions to this may be patients with severe

ischaemic heart disease and patients with ongoing bleeding where a target Hb around 100g/l is more appropriate.

2. PLATELETS

Low platelets in critical care may be caused by

- Sepsis
- Blood loss
- Disseminated Intravascular Coagulation (DIC) and other consumptive coagulopathies
- Heparin Induced Thrombocytopenia and Thrombosis (HITT)
- Hypersplenism

In addition platelet function is impaired in renal failure and with the use of NSAIDs and other drugs.

Blood clotting is impaired with platelet levels below $50x10^{9}$ /l and spontaneous bleeding more likely below $20x10^{9}$ /l.

High platelet levels are often seen as part of the stress response to inflammatory conditions. Risk of venous and arterial thrombosis is increased and antiplatelet drugs such as aspirin may be indicated.

3. CLOTTING FACTORS

Impaired coagulation from low levels of clotting factors occurs in:

- Liver disease
- Blood loss
- DIC and other consumptive coagulopathies
- Drugs, particularly warfarin

The tests of coagulation, APTT and INR, are reasonable measures of heparin and warfarin activity respectively but are less good measures of coagulation in critical illness.

Impaired coagulation can be offset by managing the above conditions and ensuring good nutrition and vitamin replacement, particularly vitamin K.

PROPHYLAXIS

Several conditions can be acquired during ICU stay and the risk of these can be reduced with certain prophylactic measures:

- **GI bleeding**, usually due to stress ulceration. Prompt treatment of shock; avoidance of coagulopathy; limitation of NSAID use and restoring normal GI function will reduce bleeding. H₂ antagonists such as ranitidine or proton pump inhibitors (PPIs) such as omeprazole can be used as prophylaxis in higher risk cases. Sucralfate is of little benefit and is now rarely used [19]
- Venous thrombosis/embolus is increased due to immobility and the procoagulant inflammatory response. Encouraging mobility, protecting pressure

areas, adequate hydration are important. Compression stockings may help. Heparin subcutaneous at 'prophylactic' dose reduces the risk with a small increased risk of bleeding. It is not usually used in significantly head injured patients for this reason

- Ventilation induced lung injury (VILI). This is a combination of high pressure injury (barotrauma) and high volume injury (volutrauma) and is reduced by adopting 'protective lung ventilation' strategies [20]
- **Critical illness polymyoneuropathy** is a heterogenous condition leading to hypotonic muscular weakness which delays weaning from mechanical ventilation. It is exacerbated by the use of steroids and muscle relaxants so the use of these drugs should be limited in dose and duration [7]
- **Hospital acquired Infections.** All of these can be reduced by good hygiene practices, in particular hand washing:
 - Ventilator associated pneumonia (VAP) more accurately termed 'intubation associated pneumonia' is associated with regurgitation of gastric contents, bacterial overgrowth in the pharynx and microaspiration past the ET tube. The following measures reduce the risk:
 - Head up posture of 20-30°
 - Restoration of normal GI function
 - Mouth hygiene including topical antiseptics such as chlorhexidine
 - Reducing duration of intubation.
 - Catheter related blood stream infections can occur with central lines or peripheral venous lines. It increases with the duration the line has been in situ: peripheral venous access sites should be rotated and central lines changed if there are unexpected signs of sepsis or local skin infection. Anti bacterial coated central lines have a lower incidence
 - Clostridium difficile infection. This causes diarrhoea and pseudomembranous colitis. Its incidence is increased by prolonged antibiotic use, particularly clindamycin, cephalosporins and quinolones.

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20. THE SURGICAL HUMANITIES IN CRITICAL CARE

JACOB S DREYER

The surgical ICU/ITU is not only a unit for intensive care or therapy, but also a place of intense stress for patients, relatives and staff, difficult decision making, emotional extremes and death. Severe surgical sepsis has a 50% mortality, multi-system trauma carries a high mortality, especially in countries with limited pre-hospital trauma systems and for elective major intra-thoracic, intra-abdominal and pelvic surgery mortality can be 5-10%. Traditionally surgeons have not been trained in managing dying patients, managing a stressful environment, non-technical skills or reflection on outcomes and safety. Professionalism in decision making and behaviour is put under the same strain as other aspects of patient care in managing a continuum of critically ill patients.

We therefore think it is important to dedicate some time to discussing the humanities' place in critical care. Successful working in a stressful environment needs high levels of integrity and professionalism, good non-technical skills such as situation awareness, leadership, decision making, teamwork and communication skills. Correct decision making depends not only on clinical knowledge, but also on knowledge of systems, care processes and best practice to keep patients safe from hospital-acquired infections and injuries. Such knowledge can only be acquired through rigorous quality control and audit of outcomes, openness and transparency in discussing outcomes, mortality and morbidities, and through maintaining a very high standard of medical ethics and professionalism, based on the universal principles of excellence, accountability, humanism, altruism and human relations within the workplace where all team members are regarded as equal.

It is essential to have a structured approach to managing the dying patient in ICU. Patients and their loved ones want a dignified "good" death for the patient who reaches the end of their life, but hospital deaths are often the exact opposite. The cultural environment greatly influences how dying patients are managed but the principles of openness, respect, ethical behaviour, symptom relief and non-abandonment should be universal. Managing dying or very ill patients does have a psychological impact on health workers, and they need to know how and where to ask for support. Going through major trauma or illness, especially with a stay in ICU, often alters brain function and surgeons who visit the ICU must understand these derangements.

This section on Surgical Humanities in critical care therefore discuss the principles of surgical professionalism, how good non-technical skills can help to lessen the risk of surgical error and manage the situation if error occurs, communication skills, with special reference to a model of structured communication in stressful environments (SBAR), how to use audit, quality control and simple patient safety bundles to deliver safer patient care, the psychological impact that critical illness or major trauma has on patients, families and staff, and compassionate care of the dying patient. The authors and editors consider the humanities as an important component of modern surgical critical care. When teaching critical care courses in Africa we have found that frank, open and unbiased discussion around these topics are always much appreciated and empowers

participants to be more assertive in managing these difficult aspects of patient care in the critically ill.

20A. PROFESSIONALISM IN SURGERY

JACOB S DREYER

The contents of this chapter is based on qualitative research the author completed for his dissertation as part fulfilment of the requirements for a Master's Degree in Surgical Education at Imperial College London (University of London), 2008 [1].

INTRODUCTION

Professionalism is a much maligned word. Most doctors, trainees, students, educators, patients and other health professionals have an idea what the term means, but it is often difficult to describe and, for most, impossible to define precisely. In the literature there is a plethora of definitions, ranging from short moral statements to elaborate descriptions of the elements or characteristics that could make up professionalism. Often authors revert to using Justice Potter Stewart's description of pornography to help them out: "I cannot define it but I know it when I see it" [2]. The impression is easily created that professionalism is something vague, something that medical professionals, including surgeons, should have and use in daily practice, but cannot be specific about. Patients and staff definitely recognise when it is absent in doctors. Professionalism seems to be something intangible and tacit that makes some doctors better at their work and in human interactions, which happens at the interface between surgeons and patients and between surgeons and co-workers.

DEFINING PROFESSIONALISM

Etymologically "profession" means *"to declare aloud, to proclaim"*; it is thus much more than a job, but is an identity, which implies:

giving much of yourself;

a lifelong role of dedication to the welfare of others; a commitment to excellence [3].

The Oxford English Dictionary defines a profession as:

"An occupation whose core element is work based upon the mastery of a complex body of knowledge and skills. It is a vocation in which knowledge of some department of science or learning or the practice of an art founded upon it is used in the service of others. Its members profess a commitment to competence, integrity and morality, altruism, and the promotion of public good within their domain. These commitments form the basis of a social contract between a profession and society, which in return grants the profession the right to autonomy in practice and the privilege of self-regulation. Professions and their members are accountable to those serviced and society."

Freidson sees two main elements in professional work [4-7].

1. COMMITMENT TO A SPECIFIC BODY OF KNOWLEDGE AND SKILL:

Professionals' work is esoteric, complex and discretionary in character and requires judgement. Their work is especially important for the well-being of individuals and society, and its value cannot be measured by money alone; i.e. it is "good work". Training develops such commitment to a particular body of knowledge and skill that work becomes a central life interest with its own intrinsic rewards. Intellectual interest and belief in its value encourage professionals to further develop and refine this body of knowledge. They not only exercise a complex skill but identify with it; work is done not only to generate income but has inherent pleasure and can become play, e.g. as demonstrated through surgeons' love of new tools and technology.

2. MAINTAINING A SPECIAL FIDUCIARY RELATIONSHIP WITH CLIENTS:

Due to the complexity of the skills and judgement required patients have to trust doctors. Recently scientific and technological advances have been so rapid that doctors had to become super-specialised to maintain individual competence. Despite better access to medical information patients still need a high level of trust in individual doctors. In return patients' needs should take precedence over the doctor's need to make a living.

FUNDAMENTAL PRINCIPLES OF PROFESSIONALISM [8,9]:

Doctors will have a commitment to:

1. Professional competence: Individual lifelong learning.

2. Honesty with patients: Give information honestly before consent. Patients do not have to be involved in every minute decision, but should be empowered to decide on the course of therapy. Be honest when patients are injured; report and analyse medical mistakes.

3. Patient confidentiality which earns the trust and confidence of patients. Confidentiality is becoming more difficult to maintain due to pressure from the state and healthcare corporations.

4. Maintaining appropriate relations with patients: do not exploit their vulnerability and dependency.

5. Improving quality of care: Not only through clinical competence, but also by reducing error, improving patient safety, better outcomes of care and measurement of quality of care.

6. Improving access of care: must individually and collectively reduce barriers to equitable health care, promote public health and disease prevention, provide public advocacy without concern for self-interest.

7. Just distribution of finite resources: advocate cost-effective care.

8. Scientific knowledge: uphold scientific standards, promote research, create new knowledge and ensure its appropriate use.

9. Managing conflicts of interest to do best for the patient.

10. Professional responsibilities to colleagues and other health professionals: work collaboratively, be respectful of each other and accept self-regulation, including mediation and discipline of members.

A PRACTICAL MODEL OF MEDICAL PROFESSIONALISM

Louise Arnold and David Stern have produced an encompassing model [10]. In this model professionalism is based on the three-part foundation of Clinical Competence (knowledge of medicine), Communication Skills and Ethical & Legal Understanding. On these stand four pillars, the principles of Excellence, Humanism, Accountability and Altruism which collectively carry Medical Professionalism.

Excellence implies a conscientious effort to exceed ordinary expectations and includes the concepts of lifelong learning, reduction of medical error & patient safety, and integrity in the promotion of scientific knowledge and technology. Our own research showed that excellence was rated highest by consultants and by senior surgeon-educators in surgical Royal Colleges.

Humanism includes respect, compassion and empathy, honour and integrity. *Humanism* has been described as "the passion that animates professionalism" [11]. Nurses who worked with surgeons rated humanism highest in our study; they especially valued surgeons' behaviour towards other health care team members.

Accountability includes personal responsibility in being available for patients and accepting personal inconvenience, regulation, and public duty. Surgical trainees rated accountability highest in our study.

Altruism means that patients' best interest guide doctors' behaviour. Patient groups rated this the most important in our research project. The research also showed that, whereas doctors tend to describe professionalism in abstract concepts (respect, empathy, honesty, altruism, etc) patients describe specific behaviours e.g. *"look me in the eye when you talk to me"* or *"be prepared to walk the extra mile with me"*.

PROBLEMS WITH PROFESSIONALISM

The ten most common examples of unprofessional behaviour that had been encountered amongst students, residents and staff physicians were [12]:

- Dishonesty: fabrication of research results and laboratory values; plagiarism; cheating in examinations.
- Arrogance and disrespectfulness.
- Prejudice.

- Abrasive interactions with patients and co-workers; usually due to exaggerated sense of self-importance.
- Lack of accountability regarding errors or administrative oversights. ("We do not want physicians to ascribe every adverse outcome to 'stuff happens'").
- Fiscal irresponsibility.
- Lack of sustained commitment to self-learning.
- Lack of due diligence: carelessness, laziness, inattention to detail, failure to follow through on clinical commitments.
- Personal excesses.
- Sexual misconduct.

A survey in six USA medical schools reported that 98% of medical students had heard physicians speak of patients in a derogatory manner, and 61% had seen examples of unethical behaviour in the medical team [13].

CAN WE ASSESS PROFESSIONALISM?

There are a number of problems in the evaluation of professionalism [14]:

- 1. Evaluation of behaviours, including professionalism, is often implicit, unsystematic and therefore inadequate.
- 2. Students who display unprofessional behaviour may not be identified in the current system.
- 3. A disservice is done to students for not giving them feedback on their behaviours, missing opportunities for greater awareness and improvement.
- 4. Evaluation methods that use abstract concepts of professionalism (such as altruism, duty, respect etc) lead us to evaluate people instead of behaviours.
- 5. Specific personality traits do not predict behaviour.
- 6. Behaviours are often due to attempts to resolve conflict between two opposing professional or personal values.
- 7. Professional behaviours are highly context dependent.
- 8. An individual's behaviour in a difficult situation does not tell us how he/she had arrived at that decision. It is thus necessary to understand the process of thinking.
- 9. Evaluators are very reluctant to report unprofessional behaviour in students or trainees, especially if lapses are regarded as minor.
- 10. In medical schools there is too much "top down" focus on abstract concepts of professionalism rather than focus on concrete sets of actions.

From various studies in this field one could summarise that an effective programme to assess professionalism in surgical trainees could have four components [15]:

1. A simulated patient OSCE;

2. A portfolio of reflective writing on experiences or observations of critical incidents in professional behaviour, either positive or negative;

3. A quantitative assessment of specific elements of professionalism through multisource feedback in the work place;

4. A longitudinal record of episodes of unprofessional behaviour, with very strict criteria to avoid unfair reporting, held by the dean or programme director responsible for training.

LEARNING PROFESSIONAL BEHAVIOUR

Positive role models are probably the most influential in teaching students and trainees professional behaviour [11, 12, 16]. Mentor was not only responsible for the education of Telemachus while Odysseus was away, but also taught him about life [17].

Wear and Kuczewski expressed serious concerns about the professionalism discourse and how the specialised language of academic medicine defines, organises and fixes certain attitudes, values and behaviours under the label "professionalism" [18]. In their opinion there are four issues addressed inadequately through the current professionalism literature:

1. THE DISCOURSE OF PROFESSIONAL DEVELOPMENT:

- The discourse contains too many abstractions (students and residents have more insight into their relations and needs than in abstract concepts such as altruism or duty).
- It pays too little attention to social factors, especially gender (the rules and expectations for doctors have been developed by men for male physicians).

2. A CURRICULAR THEORY OF PROFESSIONAL DEVELOPMENT:

- Students and residents should help develop an institution's theory of professionalism through reflection on activities that the learners view as important.
- Educators should not take a direct route between defining professionalism and assessment but firstly create a culture of professionalism throughout the curriculum, with support from the top down.

3. THE LEARNING ENVIRONMENT:

- The professionalism of students should be fostered by encouraging and rewarding positive behaviour the relationships of patient care. The current push towards professionalism is inauthentic given the widespread mistreatment of medical students. This includes belittlement, humiliation, sexual harassment and racial or ethnic harassment. At least 40-50% of medical students report incidents of abuse during medical school [19].
- Too often students and residents learn unprofessional behaviour from their teachers. "We as leaders cannot expect our students to succeed while we model failure" [20].
- Professionalism is not only a personal, but also an institutional responsibility [21].

4. THE DUTY OF THE PROFESSION TO ADVOCATE FOR THE WELL-BEING OF SOCIETY:

Professionalism should be tied directly to social justice. This must include a
macrocosmic picture of the economics of healthcare and a microcosmic picture of
how injustice is reflected in social and cultural barriers that patients encounter
daily. The "patient" is not just an individual who needs care anymore, becomes

the public at large, with special attention to the poor, the repressed and the indigent [16].

- The perspectives of the least advantaged should be incorporated in the curriculum. This section of community usually has no input and no power to influence the care they receive. Professionalism projects should learn from the experiences of the disadvantaged.
- Many teaching hospitals serve poor populations and all that students see are the negative aspects of these communities that bring patients to hospital e.g. violence and drugs. Students should learn to know these communities as communities, not simply as problems that present at the emergency department.
- Students should be encouraged to take risks, ask difficult questions about society and culture, and commit themselves as public intellectuals [22].

SUMMARY

"We need to encourage students to look inward and reflect on how we not only struggle with our diagnostic and therapeutic challenges, but also with ourselves and our inevitably flawed humanity." (Coles 1986 JAMA 256: 2124-25). In the final analysis it is this author's opinion that professionalism simply describes how we deal with daily conflicts of interest when we look after vulnerable patients who need care.

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20B. UNDERSTANDING ERROR AND NON-TECHNICAL SKILLS IN SURGERY

JOHN S RUTHERFORD

INTRODUCTION

Adverse outcomes are common in medicine, and been recorded at least since the time of Hippocrates. Surveys of adverse events in medicine include Harvard practice study [1], an Australian study based on the Harvard study [2], and UK health committee report in 2009 [3]. The incidence of adverse outcomes varies depending on the method used to measure, but is reported to be between 3.7% and 16.6% in the Harvard and Australian papers for conditions that prolonged discharge or produced disability. It is assumed that at least 50% of these events are avoidable [4]. More patients die from complications of medical treatment than die from road traffic accidents [5]. Transmission of infection between patients by medical staff has been noted to be a problem by Florence Nightingale in 1863, whilst in 1857 Ignaz Semmelweiss in Vienna found the rate of death from puerperal sepsis to be 3% on the midwife lead ward (where he could ensure hand-washing) and 29% on the ward with medical students (where he was unable to convince his colleagues to wash their hands) [6].

Medical education has traditionally concentrated on passing on the knowledge required to practice medicine, and assumed that the skills and attitudes would be picked up in the fashion of an apprentice. This assumed that the trainee surgeon was fortunate enough to have good role models and could implicitly learn how to do the job, in addition to what to know. Amongst the technical skills learnt by a surgeon are the ability to tie knots, make incisions etc. Successful surgery also involves the ability to work in a team (good interpersonal skills) and to make good decisions (cognitive skills).

MEDICAL ERROR

James Reason described error as "...all those occasions in which a planned sequence of mental or physical activities fails to achieve its intended outcome, and when these failures cannot be attributed to the intervention of some chance agency." [7]

Patients may suffer an adverse event, which may be an unintentional consequence of their treatment, such as a patient developing a rash secondary to medication, or a patient who develops a wound infection despite sterile precautions. Some adverse events are avoidable, and due to human error. **Human error is inevitable**, everybody makes mistakes. The number of ways something can be done incorrectly is huge, whilst there may only be one or a few ways to perform a procedure correctly. There are many ways errors are made, but some are done much more commonly than others, and form recognisable patterns. These are the errors that we need to focus our attention on reducing.

We cannot eliminate error, but we can aim to minimise harm.

BURDEN OF UNSAFE CARE.

The outcome from surgery can be unpredictable. Some patients will have abnormal anatomy, or will bleed more than others, or fail to heal as expected. You need to have some confidence and self-belief to operate. This attitude that we can control the outcomes of our endeavours by our efforts is natural, but the risk is that it can result in a culture of blame when a patient has an adverse outcome. Staff don't set out to have an adverse event when they start an operation or come to work.

CLINICAL EXAMPLE:

A nurse gave a child a premedication intended for the adult who was to follow later in the same list, and was criticised by the senior nursing management in her hospital. She gave the medicine to the wrong patient – and was blamed for it. She did not set out to make a mistake nor harm this child.

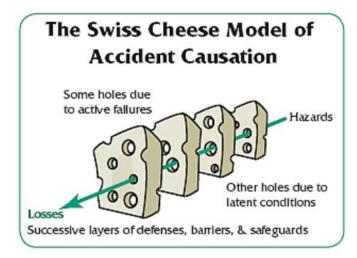
So what happened that morning? The child was on a mixed adult and childrens' theatre list despite requests to the surgeon to run dedicated paediatric surgical lists which were separate from the adult lists. The child was admitted to the same ward as the adults despite there being a children's ward in the hospital. The patients for the theatre list had arrived at 8 a.m. for a 9 a.m. theatre list, and needed admitting. The drugs for the patients already in the ward were prescribed to be administered at 8 a.m. The breakfasts had arrived at the same time, and needed to be given to patients not going to theatre. There were only two trained nurses on the ward, and to get any chance of getting everything done, and not holding up the theatre list the nurses gave medicines without double-checking with each other. The hospital procedure was that nurses should double-check the administration of drugs.

Could this error be prevented in future by criticising the nurse who made the error? As in a theatre, if you only change the actors, the play will replayed the same next time. Unless the way the ward is organised is altered, it is inevitable that some other nurse will make the same mistake in future. Looking at the other events that predisposed the nurse to make the error is called a **systems approach to error**.

The example above was of a **blame culture**. In a blame culture the person/people nearest to the adverse event are criticised, and are therefore less likely to report adverse events. One American hospital had a policy that if a nurse administered three drug errors the nurse would be sacked. The hospital administrators thought this was a successful policy because they had never had to sack a nurse. The hospital managers were not aware that the hospital still had a problem, nor able to devise better means of administering their drugs because the nurses would not report their errors for fear of losing their job.

In a **just culture**, staff are able to report adverse events without hazard providing they were not malicious. The nurse in the clinical example above did not deliberately give the child an adult premed. The Australian Incident Monitoring System is an anonymous reporting system used by anaesthetists in Australia and New Zealand to report critical incidents and near misses so that the events can be analysed to learn how it might be possible to avoid them in future.

A useful model to illustrate critical incidents is James Reason's **"Swiss cheese model"**. A critical incident will normally have a number of latent factors or systemic factors (lack of staffing to cope with the activity, failure of senior management to support and rearrange scheduling of activities, mixed ward with children and adults when there was a paediatric ward in the hospital and so on), as well as the slip or mistake that was the last event in a chain. The latent factors have often been present for some time, providing the environment in which an adverse event is more likely to happen. It is easy to see who was nearest to the accident, and takes more effort to look behind at the precursors to the incident. Improving the system is more likely to prevent further critical incidents in the future however.



From <u>http://thereliabilityroadmap.com/html/articles_pg_2.html</u> Accessed 29th October 2012.

Each layer of cheese represents a barrier to an adverse outcome. No defence is perfect, which is why the barriers are shown as Swiss cheese with holes. When all the holes line up, an incident can pass through the barriers to become an adverse event. One example of defences being imperfect is maintenance. If equipment is not adequately maintained it may not be safe to use. However, equipment which has been sent for servicing may be taken to pieces and reassembled incorrectly, and introducing a new hazard.

Different researchers have described error in various ways. An unsafe act may be an intended action or unintended. If unintended it may be a **slip** (something done at wrong time or sequence, i.e. visible) or a **lapse** (something forgotten and not visible to an observer). If the action was intended it may have been a **mistake** or a **violation**. A mistake may occur with choosing the wrong rule or misapplying a correct rule or with a failure of knowledge. Violation is a deviation from procedure, and may be routine (as was the case for the nurse in the example above to allow the work to proceed at all), exceptional (and may be appropriate in the circumstances) or sabotage (thankfully rare in healthcare, but remember Dr Harold Shipman, GP in England).

Serious adverse events are normally rare, and not immediately visible from their cause. If you don't give antibiotic prophylaxis for a total hip replacement, there is an increased risk of joint infection. This may not present for weeks or months after the antibiotic prophylaxis was omitted. The patient may present to another surgeon and you are not even aware that the antibiotic prophylaxis was omitted or given too late. But if you delay starting the operation to give the antibiotics you and those in theatre can immediately see that you are being delayed. Hence there is a tension between improving safety (which takes an investment of time and resources) and the pressures to carry on with work.

NON-TECHNICAL SKILLS

Non-technical skills are the cognitive and social skills used in the safe and efficient completion of a task [8]. They are what we all do on a good day, what the best of us do consistently, and what we fail to do on a bad day.

They were first described and researched in aviation. Towards the end of the Second World War it was noticed that pilots on certain makes of planes (Boeing B-17, North American B-25, and the Republic P-47 but rarely on the Douglas C-47) pulled up the undercarriage when they were supposed to be altering the wing flaps. The controls for both the undercarriage and the wing flaps were close to each other and looked similar in the former planes. When the controls were moved further apart, and the wing flap control altered to look like the wing flap, and the under carriage like a wheel, the error diminished markedly. In the 1970s and 1980s there were a number of high profile plane crashes which were not solely due to mechanical failures, such as the flight of an Eastern Airlines TriStar into the Everglades, Florida in 1972. When approaching Miami a light which indicated if the landing gear was down failed to light up. The pilot, co-pilot and engineer all focused their attention on the malfunctioning light, with the result that no one was watching their altitude, and their autopilot was accidently disengaged so the plane flew down into the swamp. The plane had an alarm which sounded to let them know they were approaching the ground, but it is not clear if it was heard whilst they were focused on fixing the light. The Air Traffic Control noticed that they were losing height and asked them if everything was alright but did not specifically mention the loss of altitude. The crew replied "Everything is alright" seconds before they crashed; 101 people died in the crash [9].

Aviation disasters such as this clearly illustrated the contribution of role of human factors , and lead to the development of Crew Resource Management (CRM) in aviation. The CRM training was integrated into flight training in general.

NOTECHS was a **behavioural marking system** developed in 1996 which described non-technical skills used by pilots which could be directly observed, and a marking system. It was divided into categories such as 'Teamwork' which comprised various elements for which good and poor behavioural examples were described.

The aviation industry and CRM provided an inspiration for medicine. David Gaba developed the Anaesthetic Crisis Resource Management and used simulation to train staff. In Aberdeen, the Anaesthetic Non-Technical Skills (ANTS) taxonomy was developed around a task analysis of anaesthetists in the operating theatre, rather than taking NOTECHS and putting it into medical terms. This was followed by the development of Non-Technical Skills for Surgeons (NOTSS). See below.

A SURGICAL CLINICAL EXAMPLE:

A trainee surgeon working with a consultant surgeon removed the wrong kidney from a patient, who subsequently died of sepsis [10]. The wrong side was written in the paper which was used for arranging admission and writing the theatre operating list. The correct side was noted on the surgical clerking on admission to hospital, but the patient was asleep when seen by the trainee surgeon prior to theatre so he did not confirm the side with the patient (and presumably also failed to mark the side with an indelible pen). The consultant surgeon put the X-rays up the wrong way round in theatre, and neither trainee nor consultant surgeon checked the consent. The trainee thought he was being

supervised, and the consultant had positioned the patient; whilst the consultant thought it was the responsibility of the surgeon operating, that is the trainee. A medical student was in theatre and pointed out that it was the wrong side, but her comment was ignored and she did not speak up a second time. What are the latent factors that led to this catastrophe? What were the non-technical skills present?

NON-TECHNICAL SKILLS – THE BACKGROUND:

SITUATION AWARENESS

Situation awareness is being aware of what is going on around you, and what this implies. It has three stages:

- 1. gathering information;
- 2. recognising the significance of the information or making a diagnosis;
- 3. anticipating what is likely to happen next.

1. GATHERING INFORMATION:

This is the first level at which we may have problems. We may fail to gather the information we need, or be distracted by focusing on other things that are happening. We may be tired and so less likely to take in information in. If we don't have all the necessary information, our interpretation of events may be incorrect. We have a limited amount of mental capacity and if this is being used for something else, we will be less aware of what else is going on in the periphery e.g. a surgeon doing a difficult part of an operation not noticing that the patient is bleeding from another part of the abdomen. Drawing up antibiotics or other drugs, do we always check the name of the drug, its dose and route; or do we rely on the fact that the packaging looks similar to what we were expecting?

2. RECOGNISING SITUATIONS:

We have a number of cognitive biases impairing our ability to recognise what is going on. At a very simple level we can look at a line with arrows pointing in or out, and although we know they are the same length, they appear different. Look at the following video clips on YouTube® for other examples of our failure to recognise what is going on: http://www.youtube.com/watch?v=Ahg6qcgoay4

http://www.youtube.com/watch?v=voAntzB7EwE

When our routine changes and we come across something unexpected it is very easy to ignore the changes, and try to explain theT away. Does the patient have abnormal anatomy or am I operating in the wrong plane? The recognition can range from seeing a patient and knowing that they have a club foot to the constellation of signs that allows you to realise a patient may have a tension pneumothorax. A patient may have a high heart rate and low blood pressure with a wide variety of different diagnoses such as haemorrhage, myocardial infarction, anaphylaxis or tension pneumothorax. Whilst some of these diagnoses are very common, others are rare and more likely to be missed if they do not come to your attention.

3. ANTICIPATION:

When flying a plane at a few hundred miles per hour, it is not enough to know where you are, you need to be aware of what is in front of you that you might need to avoid. Similarly a surgeon needs to know what structures he might damage, what is just beyond your scalpel, and how not to get into the situation where that might happen, or what to do if there are complications.

DECISION MAKING

Most of the time we make decisions rapidly and intuitively with minimal effort, and this is called using System 1. If we come across something we haven't seen before, or if we decide to give the question some consideration, we can think about the problem in a logical fashion. This takes more mental effort, is slower but less prone to some of the mental biases that occur when we make a decision intuitively. This is called using System 2. We naturally avoid excess effort, and so will default to using System 1 where possible [11].

When we come across something new we may work it out using system 2. If, however, we are exposed to the situation repeatedly, it will become familiar and become automatic, such as overcoming the difficulty learning to ride a bike or drive a car. Once it has become automatic, we can then add other activities such as holding a conversation at the same time. One can mentally over-ride the automatic decision of system 1 by mental effort, or conversely our emotional response may prevent us acting on a decision from system 2. It is clear how distorted situation awareness can impair decision making.

RECOGNITION PRIMED DECISION MAKING:

As described above, decision making is most commonly done by system 1. It has been given a number of names depending on the researcher but includes "recognition primed decision making" or "naturalistic" decision making [12]. When we start our career we have experienced fewer situations and so do not recognise as many events. Some decisions may be straightforward such as which suture you plan to close the skin with, to more complex ones like whether to operate for possible appendicitis in someone with right iliac fossa pain. There are a number of logical errors we make, but more commonly we have problems with **psychological biases** such as availability, anchoring and attribution. In availability bias, we remember things which we see frequently and underestimate the likelihood or rare causes. In anchoring, we are affected by the initial presentation or previous setting such as being unduly influenced in our behaviour by our last bad case. Attribution bias can happen when we follow the stereotype of the patient, and ignore the base rate of the condition.

RULE BASED DECISION MAKING:

Our decision making deteriorates when we are under severe stress. One option is to use rule based decision making.

The advantages of rule based decision making include that the decision has been thought about in advance, and if the right rule is applied correctly for the right situation there is a high likelihood of making a good decision. It can be very rapid, which is useful in time-critical situations, such as in a patient with cardiac arrest. It is easy to use a rule based system to train a lot of people with minimal input. It is easy to defend why you did something if you have followed the protocol.

The potential disadvantages of rule based decision making include that staff may not think for themselves, that the wrong rule may be applied, or it cannot be remembered or retrieved.

Rule based decision making is used extensively in aviation by pilots. It also forms the basis for medical systems such as the Advanced Trauma Life Support course.

OPTION APPRAISAL:

When we don't recognise immediately what we need to do, we may try to work out the answer from first principles. This can be done in a structured fashion to reduce the risk of a poor decision. The pilots in British Airways are taught T-DODAR as a reminder of the stages of option appraisal:

- Time, how much time do you have in which to make the decision?
- Diagnosis, what is the problem, is there a problem?
- Options, what are the available options, and what are their risks and benefits?
- Decide, what are you going to do?
- Allocate tasks and resources
- Review, has it achieved what you wanted? If not, did you have the right diagnosis?

It is common that we fail to generate as many options as possible, but stop with the first one or two that we can recall. The review is also frequently done poorly, and is a good opportunity to pick up faulty situation awareness.

GENERATING A NOVEL SOLUTION:

This is the least commonly used means of making a decision. It tends to be used when there is no previous experience to guide us. It takes considerable mental effort and is unlikely to reach an optimal solution to the problem. A medical example of novel decision making was when an intensive care team had to look after 6 research volunteers who had developed multi-organ failure after being given a monoclonal antibody (TGN1412) which had only been tested on animals before [13].

TEAMWORK AND COMMUNICATION

Teamwork occurs when the outcome of a group is greater than the sum of their individual efforts. Communication is described here, although it is used in all non-technical skills. The elements of teamwork include exchanging information, co-ordinating activities, supporting others and resolving conflicts.

EXCHANGING INFORMATION:

Exchanging information is a theme which runs through all the non-technical skills. If information is not shared, staff may have a different perception of what is going on, what is likely to happen, which decisions have been taken, with consequent delays, frustrations and irritation.

CO-ORDINATING ACTIVITIES:

Failure to co-ordinate with other members of the theatre team may result in delays taking the patient to theatre, or the wrong equipment being available.

SUPPORTING OTHERS:

We all have a limited mental capacity, and the demands of the situation may exceed what we can do. Support can be given in a variety of forms, such as emotional support ("you're doing well, there is enough time!"), cognitive support ("have you thought whether the patient may be septic?") and practical support ("would you like me to take some blood cultures whilst you are getting venous access?"). Different people show their stress differently, and it may take experience of working with someone to learn how they react. One of my colleagues described working with a surgeon who whistled whilst operating if things were going badly.

LEADERSHIP

ROLE MODEL:

The team looks to its leader as its role model, and in the operating theatre this will often be the surgeon. Positive words are completely undermined by behaviour that is not congruent. If you are rude and dismissive to staff who point out a problem, you cannot be surprised if they remain silent when you are in difficulty next time.

RESOLVE CONFLICTS:

Staff have different priorities and perspectives and it is natural that there will be disagreements at times. The focus should be on what is right for the patient, rather than who is right.

USE PROBLEM SOLVING TECHNIQUES:

There will be differences of opinion and values between different members of a team, and the leader will need to resolve these to enable the team to function.

SUPPORT AND ENCOURAGE THE TEAM:

Support for a team can range from recognising when staff are task saturated, and if possible redistributing tasks to reduce the pressure. A team which knows what is expected and told when they have achieved it, is much more likely to work well together in future.

STRESS MANAGEMENT

Performance is often poor when there is little arousal, and improves with increasing demands until a point is reached where the demands of the situation lead to a deterioration in performance (Yerkes Dodson curve) [14]. The ability of staff to cope with stress will vary depending on the time of day, their health and whether they feel they are

in control of the situation. Staff can improve their ability to cope with situations by training in situations with graded exposure to stress, and by reframing their view of the situation. It is much easier to remain calm if those around you are behaving calmly as well.

FATIGUE MANAGEMENT

We have a circadian rhythm and with peak arousal occurring in the morning and later in the afternoon. Staff are more likely to make errors at night, and in the UK it is recommended that only surgery which is for life or limb-threatening conditions are operated upon late at night. The organisation of surgical services may need addressing at a departmental or hospital level to reduce the risks associated with unnecessary late night operating.

The effect of lack of sleep has been compared with the decrease in manual skills by alcohol in the following table [15].

Hours sleep lost	Number of US beers
8	10-11
6	7-8
4	5-6
2	2-3

PRACTICAL APPLICATIONS AND SUGGESTIONS

Non-technical skills need to be practised rather than just read about to have an impact!

SITUATION AWARENESS

GATHERING INFORMATION.

- At critical times in an operation, focus any conversation on the task in hand. Can you delegate other staff to answer any calls whilst you are at a stage of an operation where mistakes are possible?
- Avoid interrupting others when they need to be focused don't ask the anaesthetist about antibiotics just as he/she is putting a patient off to sleep or intubating them.
- Pause occasionally from your task to scan the environment to check all is well.
- Checklists are valuable for reducing errors from omissions. The WHO checklist was shown to reduce surgical complications in a wide variety of hospitals around the world [16], and more recent work by van Klei shows that the benefits occurred for the teams that used the checklists consistently [17].

RECOGNISING SITUATIONS.

- What information is available to you (from the patient, family, other staff, notes etc)? What information is not available to you? What can you reasonably expect?
- If information does not fit or make sense, find out why. Has the patient that you thought had pancreatitis got an abdominal aortic aneurysm instead?

ANTICIPATING.

• What might go wrong? Have you prepared for this, and do your staff know what you should do? If you know that an operation has a risk of blood loss, have you prepared fluids and cross-matched blood in preparation?

DECISION MAKING

- Communicate your thinking so others know what you are deciding and why. If you make an error it gives others an opportunity to correct you.
- Pause before you make a decision. Have you thought of alternatives? What are the risks and benefits?
- What are you trying to achieve? If you are giving fluids for a shocked patient, what outcome do you want? e.g: Heart rate down and if so how much? Blood pressure up, if so how much? What is success?
- When are you going to review to see if your treatment has worked?
- If your treatment does not have the desired effect, was it too little of the right thing, or not working because the diagnosis is wrong?
- Actively seek information which might prove your diagnosis is wrong!
- Avoid irreversible decisions when possible.

TEAMWORK

- Learn to recognise when you are task-saturated, and where you can get help.
- Learn to recognise when others are task-saturated and offer or delegate help.
- Use closed-loop communication:
 - This is where someone makes a request (preferably by their name),
 "Nurse Smith, can you get a unit of blood for this patient!"
 - The person receiving the request repeats the message back, "I am getting a unit of blood for this patient." (The person requesting now knows the right message has been heard.)
 - The person who made the request now says, "That is correct." (The person receiving the message now knows that they heard the correct message too.)
- Speaking up can be done with graded assertiveness. You need to learn to recognise when to apply which level, and to listen for other staff using it to communicate with you. Generally you should be working with observations and suggestions. Use of a challenge or emergency intervention implies the need to review events afterwards to find out why things turned out like this.
 - Observation, "I notice the bleeding is a bit heavier." (Expecting that the person listening will do something about it, tie off a vessel or use direct pressure etc.)
 - Suggestion, "I notice the bleeding is a bit heavier, would you like me to cross-match some blood?" (This makes it more obvious what outcome you would like to see).

- Challenge, "You must control the bleeding now!" (The haemorrhage is potentially life-threatening).
- Emergency, "Stop! I will not let you give penicillin to this patient, he is allergic to it!" (The condition is so immediately life threatening that you must be prepared to directly intervene).

LEADERSHIP

- Model the behaviours you are looking for in your team.
- When you give instructions, describe what you are trying to achieve. If the condition of the patient changes, staff can then consider altering their care to achieve what you planned to do.
- Prioritise the life-threatening.
- Encourage staff to speak up to let you know if you make an error, and thank them when they do.
- Speak aloud what you are thinking, so your team know what is coming next.
- Use problem solving techniques. Focus on what is right for the patient, not who is right!
- Are any of your team task-saturated? Can you redistribute the workload?

NON-TECHNICAL SKILLS FOR SURGEONS (NOTSS)

This is a behavioural rating system developed for surgeons. It can be used to provide a language to help discuss non-technical issues in the operating theatre, both for training junior surgeons and reflecting on your own practice. Suggestions for its use are included in the NOTSS document. This behavioural rating scale is available at www.abdn.ac.uk/iprc/NOTSS

SUMMARY

Surgical teams have made use of non-technical skills for many years, but now have a vocabulary with which to discuss them. These are practical skills, and just as one cannot become a great musician by reading a book, so one cannot become a good surgeon without practice, feedback and reflection.

Failures of non-technical skills are associated with an increased rate of complications. Good non-technical skills will not eliminate error, but will minimise the harm which ensues.

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20C. COMMUNICATION SKILLS FOR SURGEONS

ANDREW HOWARD

Surgeons are not regarded as excellent communicators – in fact we may be caricatured as the opposite – and yet communication is key to our clinical lives.

The most common clinical procedure most of us will perform is a diagnostic interview. We then need to convey diagnosis, prognosis, and treatment options in a way patients understand. Our patients are often under stress and time pressure. We must break bad news while building a therapeutic relationship and maintaining hope. Many of us will be involved in end of life care and we must learn to be sensitive and direct with our patients.

Communication with our colleagues occurs in a fast paced and distracting environment where errors can cause significant harm. Many medical and surgical errors have their roots in team communication and team functioning. Structured communication tools designed for such environments, for example SBAR communication, are being promoted to improve clinical care and reduce error.

Communication skills have been directly linked to better patient outcomes in many practice settings [1], and communication can be taught, learned, and evaluated [2]. COSECSA has recently introduced OSCE stations evaluating communications skills into the MCS examinations. The purpose of this review is to discuss the literature related to the teaching, learning, and evaluation of communication skills in African surgical practice.

COMMUNICATION WITH PATIENTS

HISTORY TAKING, COUNSELING, CONSENT FOR SURGERY

The current paradigm of 'patient centred care' places the patient at the centre of the clinical encounter and emphasizes shared decision making between patient and surgeon. A review of communications skills for patient centred care published in 2005 [3] gives some very practical tips on conducting the medical interview. They describe the 4 E's of communications – Engage, empathize, educate, and enlist – to supplement the 2 F's of clinical surgical practice – Find the problem, Fix the problem.

Engaging means making a personal connection with the patient by smiling, relaxing, sitting at eye level, and sharing some preliminary chat. Six simple, powerful words "How can I help you today?" are recommended to provide engagement in the clinical task. The authors then emphasize sitting back and listening. Patients will tell you 80% of what you need to know in two minutes of history, yet the average physician interrupts after 18 to 23 seconds and begins asking simple, pointed questions. Such questions may not reveal what is of greatest concern to the patient, or how the patient views or responds to their condition. Instead, phrases like "tell me more about that", or "what concerns do you have about that" or "how do you think that affects you" allow the patient to tell their story and provide their own meaning.

Empathy refers to detecting and acknowledging or sharing the emotional content of the communication, without necessarily sharing the emotion itself. Patients have an emotional response to their illnesses and surgeons must show empathy. Sharing the emotion fully would be sympathy, and while we may be somewhat sympathetic with strong emotions at appropriate times, we generally do not share the emotions of most patients we are seeing. Often we are, in fact, too detached and fail to empathize. When patients express emotion, we should reflect it in our conversation and it can help to normalize it. For example "It was painful for you passing that kidney stone. Many people feel that way"

Educating the patient means providing the diagnosis, the prognosis, and the options for therapy in a language that the patient understands. This is best done as a dialogue with the patient setting the pace. Some patients want more information and some want less – allow them to tell you. Ask if they understand. Ask 'Does that match what you have been thinking about this problem" to allow the patient to bring up other outstanding issues. Surgeons tend to be better 'givers' of information than listeners – and they tend to have a lot of detail to convey. Remember to make sure that the important details have been received. Consider providing supplementary written material – especially about important preoperative or postoperative instructions. Remember that patients forget half of what they have been told within minutes.

Enlisting means making the patient a willing and empowered participant in the care process. Different surgeons and different patients will vary in their preferences for who is really 'calling the shots' but it is important that the patient recognize that she and her surgeon are working together. Orienting people to the process of care, discussing procedures and complications, discussing risks frankly but positively all help to empower patients facing surgery. It can be useful to foreshadow the next visit "I will see you in the hospital in a few weeks time" or "next time I see you I will ask how much better you knee is feeling -10%, 50%, 90%, or whatever". End on a positive note - with an expectation of a good outcome, appropriately phrased. Closing in a manner that gives patients hope is a very powerful technique.

BREAKING BAD NEWS

Bad news is a routine part of many surgical practices – from the oncology service to the trauma room and everywhere in between. Extra care needs to be taken with patients and relatives who may feel at their most vulnerable. A good surgeon will offer both the words and the actions appropriate in the settings of a dangerous operation or a devastating diagnosis. Patients need to be able to absorb bad news, and they need to be able to prepare for the worst while hoping for a cure. This requires careful and balanced communication. Bradley, in a review on core competencies in surgical palliative care [4], suggested the SPIKES approach described by the oncologist and humorist Robert Buckman:

- Setup
- Perceptions
- Invitation
- Knowledge
- Emotions
- Summarize and Strategy

Setup refers to finding an appropriate time and place for a discussion. Quiet, private, seated, at eye level, and free of distractions such as cellular telephones all help. Perceptions refers to the useful technique of asking the patient/family what they know or believe about the situation. This is always the starting point for the discussion and they may already know the bad news or have guessed it, but will still usually need some additional information and some support. Invitation means asking the patient/family what else they would like to know or whether they would like to know more. It lets them decide whether they are ready – whether the right people are present, and so forth. It allows a patient to control when they are told what. Knowledge means conveying the information. Many patients want to know all that the doctor knows or understands - and also what the doctor might not know or understand. Knowledge is power and it should be shared. Emotions are commonly expressed when facing loss of health or loss of life. These emotions should be acknowledged and empathized with. Different people will have very different means of expressing the same emotions depending on their personal, family, and cultural background. Silence on behalf of the doctor may constitute effective communication here. 'I wish' statements can be used to convey empathy and also to bring people to realistic and practical steps. Finally, Summarize and Strategy means what it says. Remember that the strategy is always positive and reflects hope. Aggressive, timely, and appropriate treatment of symptoms can be offered even when a curative option does not exist.

DISCUSSING MEDICAL ERROR

Medical and surgical error seems pervasive today, mostly because it is increasingly sought and increasingly recognized. This is important because identifying error or potential for error is a necessary step in making a dangerous environment – the hospital – as safe as possible for patients. It was once controversial whether medical error should be disclosed to patients, for fear of losing trust or increasing lawsuits. Current evidence suggests that disclosure of medical error, done properly, actually maintains trust and decreases the risk of lawsuits. Framing a discussion according to the SPIKES mnemonic above is often appropriate when discussing a major error that has resulted in patient harm. Offering an apology without taking blame is appropriate "I am sorry this has happened to you" or "We are sorry this has happened to you". Many patients also want to know that steps are being taken to continue their care or rectify the error, and that steps are being taken to minimize similar errors in the future.

DISCUSSING DEATH

Discussing death will fall often to some surgeons. A caring attitude must be clearly conveyed to the patient and the family. Again, the SPIKES mnemonic can be used to give appropriate structure to the conversation. Time without interruptions must be found. An offer to be available and a means of making contact with the doctor is very important to most patients and families, and many families will want to contact the doctor once more after a patients death as part of a closure process. In discussions about death it is important to repersonalize the patient and resist the tendency to talk only about the disease. Leaving time and space for rituals is obviously important, as is involving a chaplain from the patients own faith. Most surgeons are far better off leaving chaplaincy to the chaplains.

EVIDENCE SUPPORTING COMMUNICATIONS SKILLS

Stewart [1] performed a systematic review of papers which examined physicians communications skills and related them directly to patient outcomes. She limited the design to randomized controlled trials and analytical studies and found high quality evidence that better communication improved health outcomes including anxiety, role functioning, physical functioning, blood pressure, and blood glucose - mainly through better understanding of and adherence to treatment plans. A randomized trial specific to surgical communication showed that patients undergoing abdominal surgery had better pain scores and lower narcotic use postoperatively when communication was better preoperatively.

Wendy Levinson published a foundational paper on communications skills in the Journal of The American Medical Association in 1997 [5]. She taped and analyzed 1265 patient office visits with 59 primary care practitioners and 65 surgeons, to determine if the doctors communication style was related to malpractice claims. For surgeons, it was not – but for primary care physicians it was – with physicians who spent longer (18 minutes vs 15), oriented their patients more, and laughed more being less likely to be sued. A description of the surgeons communication style from these tapes showed that the surgeons talked more than the patients, empathized infrequently, and spent brief or no time on social interactions [6]. A later analysis found malpractice claims among surgeons were associated with the surgeons' tone of voice – as judged from a ten second clip of each tape [7] although the implications of this are unclear.

Wright performed a qualitative study among 39 breast cancer patients in England who were dealing with either surgeons or oncologists for care [8]. The premise of this work was to find out what patients need in communication rather than what professionals think they need. Patients did not specifically discuss or value 'communication skills' and in fact were willing to discount poor communication in order to form attachments with their doctors. What patients wanted was technical expertise, plus and individual relationship, plus respect. They valued eye contact, smiling, and when appropriate touch. They needed to know that the doctor knew and valued them as a person. Patients differed on how much information about their disease they wanted – some complained about being subject to too much information. If they wanted information it was usually for a purpose – such as to establish trust or to inform hope.

COMMUNICATION WITH COLLEAGUES

COMMUNICATION AND MEDICAL ERROR

(Also see Chapter 23)

Communication lapses are responsible for up to 70% of sentinel events and medical errors [9]. Standardization of communication has been suggested in order to promote concise and complete messages, and to 'level the hierarchy' by encouraging nurses and junior staff on the front lines to convey their analyses and recommendations to the doctors and consultants they are contacting. The Joint Commission on Health Care Accreditation in the United States has recommended SBAR communication – situation, background, analysis, and recommendation – to meet these needs [10-12].

SBAR COMMUNICATION

SBAR is mnemonic for situation, background, analysis and recommendation to prompt a structured communication and a shared conceptual framework between health care providers. An example given in [11] is Situation: I am the ward nurse calling a doctor about Mr X who has been having breathing difficulties all day but has deteriorated substantially in the past 20 minutes. Background: The patient is a 59 year old male admitted with an exacerbation of obstructive lung disease yesterday. Pulse rate is 100, blood pressure is 130/90, respiratory rate is 35 and shallow. Last auscultation of the chest revealed absent breath sounds on the right side. Analysis: He may have developed a pneumothorax. Recommendation: I would like the doctor to attend and consider whether a chest tube is indicated. Notice in the example that the nurse is prompted to give necessary current and background information but also to present his view of the analysis and recommendation.

PATIENT HANDOVERS

Patient handovers during shift change are responsible for about 60% of communication lapses that lead to errors or near misses [9]. Reduced resident work hours are making handovers more common. Despite this, there is little evidence base to draw on describing the best practice for patient handovers. Cheung published a review article in 2009 focusing on the emergency department setting and describing what is known about handovers. He cited information about high performing flight crews, who routinely spend a third of their handover time on questions and answers, compared with 5% of time for low performing crews. Question and answer sessions ensure that people who are sharing information are really sharing it and both understand it the same way. Any message mixes signal with noise. Noise can be distractions from the environment, or ambiguity within the message itself. Issues to be decided during handover include the balance between concise communication and complete information, the lack of reliable triggers or 'red flags' for high risk handovers, and the appropriateness of a multidisciplinary handover (doctors, nurses, other professionals) compared with a single discipline handover. There are many aids and mnemonics to structuring handovers -SBAR is only one of them, at least 20 others have been described in the literature none seems to be followed routinely and none has a high level of research or evidence supporting it . Computer aids to handoff will need to be studied in the future, as factual information may be facilitated electronically but interpretation and analysis will still likely need to be discussed among professionals.

TEACHING AND LEARNING COMMUNICATION

COURSES

Communication is a skill that can be taught, learned, and tested. We can all improve our communication skills and this will improve our performance and enjoyment in clinical practice.

McManus described in the BMJ [13] how to teach communications skills to clinical learners in a two day course. He emphasized using concise lectures and lots of practice with feedback because communication is far better taught with experiential than instructive methods. Lectures can cover the importance of communication, can increase

students knowledge about specific steps, tasks, or useful mnemonics, and can provide conceptual tools that the students can use to discuss and analyze their own communication and others. Showing many brief videos of clinical communication in various settings helps people to become comfortable with analyzing and giving feedback on communication skills. Scenarios can then be used in which the patients alternate in the roles of doctor, patient, and observer who gives feedback. Students quickly warm to the task provided they are not humiliated with the feedback. It is useful to provide sandwiched feedback – a positive observation, a constructive criticism, and another positive observation. In addition to having role play, it is possible to get actors (standard patients) who have scripts and who are often better than students at getting into a role – because they are trained (and sometimes paid!) to do it.

Aspegren performed a systematic review of teaching and learning communications skills focusing on high quality and medium quality evidence [2]. She found 31 randomized controlled trials, 38 open comparative studies, and 19 descriptive studies which met inclusion criteria. Together these studies established that 1) Communications skills should be included in clerkship curricula 2) Doctors are effective at teaching communications skills after brief training 3) Experiential teaching methods (practice and feedback) are necessary, instructive methods do not work, 4) The content of courses should be problem defining rather than prescriptive 5) Men learn communications skills more slowly than do women 6) Anyone can be taught – those with the lowest scores on a pre test showed the greatest gain in a course 7) a ten minute OSCE is the best format to evaluate communications skills.

STANDARDIZED PATIENTS – ACTORS

Moulton performed a randomized trial on having standardized patients (actors) give immediate feedback (versus none) to students who were performing clinical tasks involving communication [14]. Students given feedback showed a significant increase in their communication scores in later tasks compared with those who did not.

Yudkowsky in 'Current Surgery' evaluated OSCE type exams using standardized patients and found them to be a reliable, effective, and fair way of evaluating communication skills [15].

COMMUNICATIONS SKILLS AND ORAL EXAMINATION SUCCESS

An interesting study regarding communication may be motivating to those students facing examinations. Rowland-Morin designed an experiment in which actors re-read transcripts of actual oral examination answers given on surgical exams. Actors were instructed to deliver the same information in one of two styles – Style A with direct eye contact and a moderate pace of delivery, and Style B with indirect eye contact and slow, hesitant responses. The responses were taped and sent to examiners for evaluation and scoring using five point scales for individual items and overall. Given exactly the same answers, word for word, Style A scored an average of 4.5 on a 5 point scale while Style B scored an average of 3.5. Although examiners are meant to examine only on content, they cannot – and communication style again shows its importance [16].

SUMMARY

- 1. Communications skills are a key component of the practice of surgery.
- 2. Communications skills can be taught, learned, and improved.
- 3. Learning should be through practice with feedback.
- 4. Patients value being regarded as individuals and being treated with respect in all clinical encounters.
- 5. Special methods are appropriate when discussing death or catastrophic loss.
- 6. Interprofessional miscommunication is the root cause of many medical errors.
- 7. Structured communication protocols, such as SBAR, are designed for efficient and complete communication.
- 8. Efficient and complete handover requires dialogue including questions, answers, and clarifications.

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20D. PATIENT SAFETY, QUALITY CONTROL AND AUDIT IN CRITICAL CARE

DEWI WILLIAMS, JACOB S DREYER

INTRODUCTION

To be safe means to be "free or protected from risk or danger"; in an active sense it means "not causing harm or allowing anything that results in harm". In healthcare this implies reducing risk and avoiding needless, potentially avoidable harm due to healthcare provision.

Common causes of preventable morbidity and mortality in healthcare are

- Hospital acquired infections;
- Errors with medicine;
- Venous thrombo-embolism;
- Poor communication and handover.

For example, risk of medication error is common; sometimes this causes unintentional harm such as over-anticoagulation. In a complex system like healthcare it is not possible to eliminate all risk but can be significantly reduced by changing working culture and systems. Recently there has been extensive work on improving health care systems and working culture; the process has been named "healthcare improvement". The **Institute of Healthcare Improvement** in the USA have outlined six improvement aims that patient care should be focussed on [1]:

- 1. Safety
- 2. Effectiveness
- 3. Patient centred care
- 4. Timeliness
- 5. Efficiency (reducing waste)
- 6. Equity (of access)

Another way of describing these aims is the **no needless list**: No needless deaths, no needless pain or suffering, no helplessness, no unwanted waiting, no waste, no one left out.

A STRUCTURED APPROACH TO REDUCING RISK

Severity scoring for risk of death of certain conditions has been studied extensively and has led to scores such as APACHE, POSSUM, and Injury Severity score. It implies that more complex pathology increases risk of death or complications. Risk cannot be

eliminated due to the complexity pathology and treatments that surgeons deal with but risk can be significantly reduced in by having a structured approach to patient safety, even in the critically ill patient. One way of doing this is to get things **RIGHT** as many times as possible, which means having a reliable approach to all patients with no exceptions or omissions. Asking the following questions during every patient journey will lessen avoidable harm significantly:

- 1. Right Patient?
- 2. Right Assessment?
- 3. Right Diagnosis?
- 4. Right Treatment?
- 5. Right Time?
- 6. Right Side?
- 7. Right place for best care?
- 8. Right Outcome?

RIGHT ASSESSMENT

See **Chapters 1 and 2** for structures that support such decision making.

RIGHT PLACE OF CARE, RIGHT TIME

These can be very difficult to judge and junior doctors may often feel intimidated about discussing their concerns with seniors or specialists in other disciplines (e.g. ICU). This handbook describes a series of structured approaches to common conditions that cause critical illness, e.g. hypoxia, sepsis, as well as a structured approach to communication (SBAR) which should guide both decision making and judgement in these patients, and allow assertive communication without being aggressive.

RIGHT PATIENT, RIGHT TREATMENT (OPERATION), RIGHT SIDE

These are essential components of the WHO Safe Surgery Checklist [2,3]. Safe Surgery is a specific WHO programme to improve peri-operative patient safety, dependent on using an evidence based safety checklist that is completed for each patient.

About 234 million operations are done globally each year. At mortality and morbidity rates of 0.4-0.8% and 3-16% respectively there at least 1 million deaths and 7 million annual disabling complications worldwide after surgery. This makes surgery and its complications a significant public health concern. More people die each year worldwide from surgical emergencies and complications than from malaria. We know that safety in surgery means much more than a single checklist, but include a wide range of topics such as: Patient selection; Patient pre-assessment; Patient preparation; Surgeon's preparation; Equipment check and maintenance; Anaesthetic planning; Patient positioning on the table; Safe use of diathermy; Safe sharps management; Awareness of

allergies (anaesthetic drugs, antibiotics, plaster); Post-operative fluids, drugs, pain relief and on-going patient care.

The checklist is based on the WHO's 10 objectives for Safe Surgery:

- 1. The team will operate on the correct patient at the correct site.
- 2. The team will use methods known to prevent harm from administration of anaesthetics, while protecting the patient from pain.
- 3. The team will recognize and effectively prepare for life-threatening loss of airway or respiratory function.
- 4. The team will recognize and effectively prepare for risk of high blood loss.
- 5. The team will avoid inducing an allergic or adverse drug reaction for which the patient is known to be at significant risk.
- 6. The team will consistently use methods known to minimize the risk for surgical site infection.
- 7. The team will prevent inadvertent retention of instruments or sponges in surgical wounds.
- 8. The team will secure and accurately identify all surgical specimens.
- 9. The team will effectively communicate and exchange critical information for the safe conduct of the operation.
- 10. Hospitals and public health systems will establish routine surveillance of surgical capacity, volume and results.

There are, however, two more important points to make Safe Surgery work:

- 1. Adapt the checklist for local circumstances and get wide "buy-in". Short 1-2 week PDSA cycles (Plan, Do, Study, Act) allow rapid progress of this process.
- 2. Success depends on mature non-technical skills, emotional intelligence and good theatre teamwork.

It is important to realise that "perhaps the most distinguishing feature of high reliability organisations is their collective preoccupation with the possibility of failure. They expect to make errors and train their workforce to recognise and recover from them" [4]. As surgeons we tend to think that we can control everything and think that, as long as we are well prepared and concentrate hard enough, we will avoid errors. Subconsciously we know that is not the case. When we do make a mistake we tend to say "If you do not get complications it means that you do not operate enough", or we try to blame someone or something else for such errors. We are usually hardest on ourselves and live with our skeletons in the cupboard. Recognition and use of non-technical skills is a way to accept that we are fallible, but work in a team that can help us recognise and correct for errors before they become catastrophic.

In NOTSS surgeons are assessed in theatre in teamwork, situation awareness, decision making and leadership [5]. Of these I think leadership style is the most important. We have all experienced teaching by humiliation and sometimes we perpetuate this process.

Surgery has always depended on a very strict hierarchical structure, and maybe the time has come to challenge that.

Because patients are all individuals with varying anatomy and pathology we need to be adaptable in theatre. This includes listening to the concerns of others, including the most disempowered members of the theatre team. For this reason the first point on the "Time Out" is "Have we all introduced ourselves by name?" In 2002 in Wales a surgeon accidently removed a patient's healthy kidney. A medical student noticed that the surgeon was operating on the wrong side and after an initial attempt to tell the surgeon was told that she had it wrong; she did not feel empowered enough to push the point [6]. We must therefore ask ourselves "Am I such a strong character that I induce fear with demanding respect, or am I open enough that members of my team will make me aware of my potential errors?" Our patients would prefer us to be safe.

The main learning points from the Safe Surgery process therefore are:

- 1. The Safe Surgery Checklist (SSC) should not stand alone, but be part of a series of safety checks along the patient journey [7,8].
- 2. The SSC will not work without awareness of non-technical skills and a change in theatre culture, especially in hierarchical structure.
- 3. Using the SSC strengthens team work and actually induces culture change in theatre so that team members become more open with each other.

RIGHT DIAGNOSIS

Getting the right diagnosis is challenging and will depend on local factors of disease prevalence and resources.

There has been work on types of thinking (or cognition) and decision making (See also **chapter 20B**). Two main subtypes are recognised.

- Type 1 (sub-conscious thinking) which is impulsive, rapid, instinctual, relies on first impression (for example chest pain is a heart attack rather than a dissecting aortic aneurysm). This type is prone to error due to biases in our own experiences. Although essential to make such decisions in an immediately life-threatening situation, it is important to realise its weakness.
- Type 2 (conscious thinking) which is more considered, analytical, produces a differential diagnosis and weighs up each possibility. A critical part of conscious thinking is to slow down in data processing and decision making. I also means one can adapt as new information becomes available over time from repeated clinical assessment or investigations. Type 2 thinking is very important in managing patients with critical illness after primary assessment and resuscitation (See **chapter 2**).

Cognitive errors are mistakes that a clinician makes despite "knowing better". Mental shortcuts allow physicians to make decisions quickly but may be responsible for errors. Simple decision making checklists can reduce error and monitor thought processes [9].

RIGHT OUTCOME

To improve and become safer many lessons are learnt from the manufacturing industry. Outcome is the final product (such as a new car). Patient outcome in its simplest form is life or death. Life can be further defined by "quality life years", and its absence by "DALYs (Disability Adjusted Life Years)"; quality of death can be seen as "a good death" or "dignified death" which minimises suffering (see **chapter 20F**).

To get the right outcome you need a structure and a process, very similar to understanding anatomy and physiology.

STRUCTURE + PROCESS = OUTCOME

Structure in the healthcare setting involves buildings, equipment, drugs, staff, and finance. Process is how we do things and involves teamwork, communication, protocols, checklists, data gathering and audit. The crucial point is whether we comply with these processes and not simply "know about" them.

Structure is difficult to change quickly but may have a huge influence on outcome, e.g. putting all your theatres together, having a "complex airway trolley " for all theatres, having a regular pharmacist on the ICU ward round, improving transfer links, providing pulse oximeters. But it is not enough on its own.

Process can be improved more rapidly but in smaller steps. To understand your process you need data, because *"data is power"*. Data on misdiagnosis, wrong treatments, drug errors, hospital acquired infection, thromboembolic disease, wrong site surgery and anaesthetic mishaps are key examples.

One of the simplest and widespread processes is hygiene. Hands of healthcare workers transport microbes. Good hand hygiene is logical and works. You need to know how good your institution is. Data will inform you and will track improvement. If you don't realize there is a problem you cannot improve it. Tell your staff about the data, post it on the walls so everyone can play their part.

QUALITY CONTROL

Quality control depends on good data collection and processing, making outcomes available to all users in a way that they can understand, and in working together in a culture where team members do not blame each other for errors but work together in a supportive way to improve outcomes.

THREE TYPES OF DATA MEASURES

Use a balanced set of data for all improvement efforts in outcomes, processes and balancing measures.

1. OUTCOME DATA:

Examples are:

- Intensive Care Unit (ICU) unadjusted mortality percentage
- Adverse drug events per 1,000 doses
- Number of Staphylococcal bacteraemias

2. PROCESS MEASURES

Are the parts/steps in the system performing as planned? Are we on track in our efforts to improve the system?

- Compliance with bundle implementation
- Effective use of SBAR on handovers from the Emergency Department
- Successful hand hygiene opportunities

3. BALANCING MEASURES (LOOKING AT A SYSTEM FROM DIFFERENT DIRECTIONS):

Are changes designed to improve one part of the system causing new problems in other parts of the system, e.g?

- For reducing time patients spend on a ventilator after surgery: Make sure reintubation rates are not increasing
- For reducing patients' length of stay in the hospital: Make sure readmission rates are not increasing

Managing safety and risk requires data to be gathered by clinicians within a "no blame" culture. Organizations should be encouraging their staff to raise safety concerns. Such data collection may be done prospectively in the form of critical incident/ accident monitoring where a person reports an event shortly after it happens and it is analyzed. Or it can be done retrospectively by case note review, for example mortality and morbidity reviews, or using systems to screen notes for clues on adverse events.

BUNDLES OF CARE

It is well known that research findings are poorly implemented by clinicians. Patients therefore potentially miss out on new developments that have been shown to improve outcome. Treatments are also not reliably carried out for very patient. Recently "bundles" of evidence based interventions have been implemented and have been shown to improve outcome in a selected group of patients. An example of this is the **"Sepsis six"**, a simplification of the original Surviving Sepsis Campaign care bundles [10].

Sepsis Six is simple set of six tasks which can be completed by any team of healthcare professionals. All nurses, doctors and allied health professionals have a part to play in the delivery of the Sepsis Six. When delivered within one hour following recognition of sepsis, Sepsis Six save lives. It has been shown that the chances of an individual dying from sepsis can be halved (Also see **Chapter 11**).

THE SEPSIS SIX:

- 1. Give high flow oxygen
- 2. Take Blood cultures
- 3. Give IV antibiotics
- 4. Start IV fluid resuscitation
- 5. Check haemoglobin and lactate
- 6. Monitor hourly urine output.

It seems that there is an additive effect to each intervention. Any healthcare system that delivers this close to 100% of the time is working well. There are immeasurable effects of improved teamwork and communication that come into play. Perhaps better recognition, diagnosis and correct empirical antibiotic administration are an additional result.

REDUCING HOSPITAL ACQUIRED INFECTION

Significant results can be achieved by focussing on:

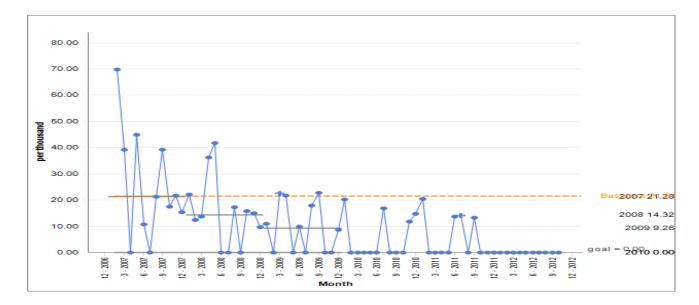
- Hand hygiene
- Reducing intravenous catheter related blood stream infections both peripheral (PVC) and central (CVC)
- Reducing ventilator associated pneumonia (VAP)
- Reducing surgical site infection

(A) THE VENTILATOR ASSOCIATED PNEUMONIA (VAP) BUNDLE.

The Scottish Intensive Care Society recommends the following Bundle Elements [11]:

- 1. Sedation to be reviewed and, if appropriate, stopped each day.
- 2. All patients will be assessed for weaning and extubation each day.
- 3. Avoid supine position, aiming to have the patient at least 30° head up
- 4. Use Chlorhexidine as part of daily mouth care.
- 5. Use subglottic secretion drainage in patients likely to be ventilated for more than 48 hours.

Applying these interventions has reduced VAP from 8 per year in 2006 to none for 14 months in 2012 in the author's ICU. See graph below of VAP rates per thousand ventilated days.



(B) CENTRAL VENOUS CATHETER INSERTION BUNDLE

- 1. Ensure that a surgical scrub is performed immediately before donning maximal sterile barrier precautions (i.e. gloves and gown)
- 2. Ensure that maximal sterile barrier precautions are used, including headwear, mask, sterile gown and sterile gloves and a sterile body drape for the patient.
- 3. Ensure that the subclavian site or internal jugular vein is used if possible (the femoral site should be avoided whenever possible).
- 4. Ensure that 2% chlorhexidine in 70% isopropyl alcohol is used for skin preparation of the insertion site and allowed to dry, before CVC insertion/skin puncture (do not wipe or blot dry).
- 5. Ensure a sterile, transparent, semi-permeable dressing is used to cover the catheter site.

Additional related good practice considerations:

- Coagulation status
- Site of other lines
- Potential for pneumothorax
- Operator skill and the use of ultrasound guidance
- A fresh site rather than a guide wire change should be applied whenever possible.
- There is strong association between the number of needle passes and complications (though not infection related). Aim to keep the number of needle passes to less than three.
- After skin preparation the patient should be covered as much as possible with sterile drapes allowing only a small opening at the site of insertion.

Figure 1 is an example of sticker enclosed in a CVP pack to act as a reminder.

Central Line Insertion BundleName:GradChecklist to be completed at time of centralnotes.	
Date:	
Time:Full asepsis (including Hat, mask, gloves, gown)2% Chlorhexidine in 70% Isopropyl alcohol	Line:1 Lumen:PICC:2 Lumen:Vascath:3/4/5 Lumen:
	Right femoral vein Left femoral vein Peripheral Vein
Left internal jugular vein Methods used to avoid ARTERIAL placement in addition to pulsatile flow and colour (use at least one in order of preference) <i>Pre dilation</i> 1.Wire visualized in vein 2. Needle/ small catheter transduced 3. Arterial gas <i>Post dilation</i> 4. CVP line transduced post insertion Radiological confirmation Fluroscopy	(avoid subclavian vascaths is possible) Use of ultrasound recommended (in order of preference) 1. Real time 2. Pre-insertion scan 3. Not used CXR N/A
Final Line Position Check Time:	Signature:

C. CARE AND MAINTENANCE OF CVC LINES

While not part of a Central Line Insertion bundle, maintenance and care of the line should be seen as equally important:

Key recommendations:

- Ensure that the need for the CVC in situ is reviewed and recorded on a daily basis. Assess patient daily to determine continued necessity for the central line. Document this on the central line bundle sticker. Lines should be removed as soon as possible
- 2. Maintain sterile technique when using the line
- 3. Do not routinely replace lines
- 4. Blood should not be drawn from the line for sampling if possible
- 5. Ensure daily that the CVC dressing is intact.
- 6. Ensure that the CVC dressing has been changed in the last seven days.

- 7. Check the insertion site during dressing changes.
- 8. Ensure that hand hygiene is performed immediately before accessing the line/site.
- 9. Ensure that an antiseptic containing 2% chlorhexidine gluconate in 70% isopropyl alcohol is used to clean the access hub. Prior to accessing rub the access hub for at least 15 seconds ('scrub the hub').

Other examples of care bundles in trauma exist for managing acute head injury and rapid sequence induction.

COMMUNICATION IN HANDOVER OF CRITICALLY ILL PATIENTS

As discussed previously, SBAR is an invaluable tool to have a structured approach to communication. It allows team members to be assertive without being aggressive in high-pressure situations where things are happening quickly and patients are at high risk of rapid deterioration and death. Some tips to improve SBAR structure in such situations are:

<u>S</u>ITUATION:

- Who I am. Where I am.
- Who is the patient.
- What is wrong

BACKGROUND:

- Admission diagnosis
- Relevant previous medical history
- Summary of treatment so far

ASSESSMENT/ACTION

- Vital signs/MEWS/GCS
- I think the problem is...
- I have done...

RECOMMENDATION

- What I would like you to do.
- What do you want me to do in the meantime?

In the ICU daily goal setting improves care through careful decision making and communication. The tasks of data gathering, process audit, bundle compliance and communication have come together in a simple checklist that is done on every patient everyday (Figure 2). This is similar to pre-flight checklists in the aviation industry.

THE TOP 10 TIPS FOR KEEPING PATIENTS SAFE:

- 1. If you see something unsafe (or potentially unsafe), do something NOW.
- 2. If it does not feel right, get HELP.
- 3. If you don't know how or what to do, ASK.
- 4. Prior to any procedure or administration of drugs, CHECK: the right patient, right dose, right route, no allergies.
- 5. Believe the PATIENT/relative.
- 6. Take special care with HIGH RISK patients (intoxicated, elderly, children, language barriers, learning difficulties).
- 7. Review ALL relevant and available information (see Chapter 2).
- 8. Review the RESULTS of all investigations and document these.
- 9. LISTEN to the patient and encourage them to participate in their safety.
- 10. Use structured communication (SBAR).

CLINICAL AUDIT IN QUALITY CONTROL

Clinical audit is an essential part of effective data collection that leads to improved care quality and patient safety. In Audit we ask "What happens?" and in Research we ask "How" or "Why does it happen?".

There are many definitions of clinical audit. The British audit support group defines clinical audit as "a process that improves the quality of patient care through systematic review of care against explicit criteria and supports changes in practice to meet those criteria" [12]. As a research tool audit means that you are "assessing individual practices honestly enough to notice differences in outcome and report such data." It implies that we will be honest when we look at our practice data and recognise what can be improved, that we will be big enough to admit deficiencies and make the necessary improvements, and then audit our patients' outcomes again. It means that audit should not be threatening.

The benefits of doing audit projects in Safe Surgery are:

- It is cheap.
- It does not need ethics approval although you still need to write a proposal.
- It is based on clinical work.
- It improves practice and patient care.
- It makes you used to analysing clinical outcomes and reporting these honestly.
- It takes you away from a "blame culture" for mistakes and teaches you to analyse the cause of errors.
- It will improve patient safety.

Trainees might wonder what research or audit can be done in Safe Surgery. In my opinion you can look at one of the following:

- 1. Audit the process of using the Safe Surgery Checklist (SSC): e.g. How the SSC is implemented and/or accepted; Practical problems in using different parts of the SSC; Developing a protocol for swab and sharps counts.
- 2. The effects of the SSC on changing patient outcomes: e.g. on wound infection or DVT incidence; drug allergic reaction incidence; in speeding up the time it takes to get blood to theatre.
- 3. Qualitative research on how the SSC affects practice: e.g. How did introducing the SSC affect theatre teamwork or communication, or: How difficult was it to change theatre practice?

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20E. THE PSYCHOLOGY OF CRITICAL ILLNESS, MAJOR INJURIES AND ICU CARE: IMPACTS ON PATIENTS, RELATIVES AND STAFF.

MARTIN CLARK

INTRODUCTION

Intensive care units treat patients with the most severe medical and surgical conditions in the hospital and it is not surprising therefore that they often present the most challenging psychological conditions for the treating staff, patients and their families. This chapter will discuss the latest evidence which demonstrates how improved psychological care can not only alleviate distress but actually reduce both mortality and long term morbidity.

THE PATIENT

It is important to remember that patients in the ITU are in a non escapable life threatening situation, which is highly stressful, and will react in a number of different ways based on their pre-existing coping mechanisms. They may also have to cope with feelings of loss, be that a limb or having a colostomy, or more generally loss of independence or maybe family members following an accident etc. In addition, and very importantly, their brain and cognitive processes may be affected by their acute illness. Just as a pneumonia may produce multi-organ failure in the shape of renal failure, it can produce a brain failure which will present as altered level of consciousness and delirium.

DELIRIUM

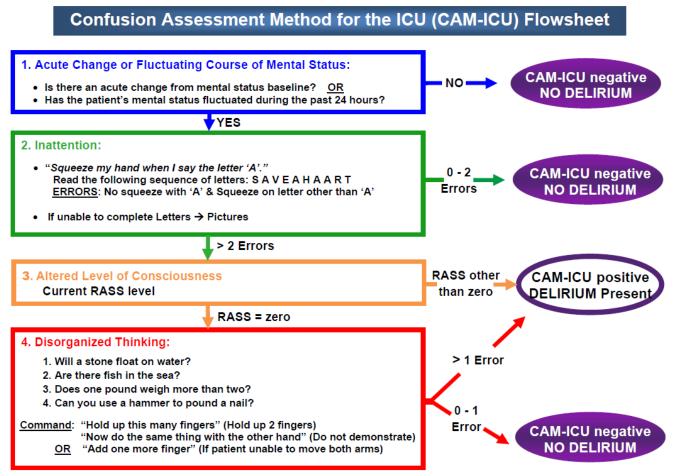
Delirium is a common life threatening condition which should be given the same consideration as any other organ failure. After correcting for confounding variables Ely demonstrated delirious patients have a threefold mortality risk (34 % vs 15%) at six months compared to non delirious patients, they also spend on average 10 days longer in hospital and have increased long term cognitive deficits compared to non delirious patients [1]. Finally delirium is associated with significantly higher ICU and hospital costs (30% more] [2]. Therefore in any ICU, but especially in a resource poor environment delirium should be avoided if possible and should it occur, be recognised and treated as quickly as possible.

Delerium is a primary organic brain dysfunction characterised by an acute onset over hours to days, there is a fluctuating level of consciousness, patients display inattentiveness and may have disorganised thinking with perceptual disturbances of real objects, or frank visual hallucinations. They may be disorientated to time and place. Patients may have delusions and misinterpret stimuli. For example they may not realise they are in hospital, may see frightening animals which could attack them at any time, may feel they or other patients are being tortured or think the staff are trying to kill them. It is important to realise that the patient may be absolutely convinced of this. Often a patient who is delirious will still recognise friends and family although they will not generally believe their reassurances. Patients with delirium can find it very difficult to understand or retain information – so even if they appear to understand what is happening, or may be joining in a conversation, they may not remember what has just been said to them.

From the above description it would seem easy to recognise the delirious patients but it isn't. Delirium occurs in 25%-82% of all ICU patients and 65% of ventilated patients but this number seems very high to most ICU physicians [1,3,4]; this is because only 5-23% of delirious patients present with the classical agitated hyperactive delirium characterised by agitation, restlessness, combativeness, attempts to remove lines etc that we all recognise. By far the commonest presentation of delirium is actually hypoactive delirium (45-64% of cases) or a mix of the hypoactive and hyperactive states. Hypoactive delirium presents as a generally apathetic, lethargic withdrawn state in which patients may appear to the unsuspecting clinician to be depressed tired or lack motivation. The important features to look for in making the diagnosis are disordered thinking and inattention. It is important to realise that whilst the patient with hypoactive delerium appears calm and is less of a physical challenge to manage, they have just the same poor long term prognosis as those with the apparently more challenging hyperactive form.

The pathophysiology of delerium is poorly understood and various studies disagree on the predisposing factors. However it is clear that benzodiazepines and especially lorazepam are strong risk factors for developing delirium, with a cumulative dose of 20mg Lorazepam producing delirium in 100% of ICU patients [5]. A recent large study has reported that increasing age, increasing apache scores, increasing blood urea, infection, metabolic acidosis, morphine, sedatives, urgent admission and if the patient is a medical, trauma or neurosurgical admission are all risk factors for developing delirium [4].

Diagnosis of delirium is clinical and best performed using the CAM-ICU test as detailed in the flow diagram below. Essentially there must be inattention and either altered consciousness or disordered thinking in order to diagnose delirium.



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RASS=Richmond agitation sedation score, a score of zero = alert and calm

The test takes 1-2 minutes and a video of the procedure can be viewed at <u>http://www.mc.vanderbilt.edu/icudelirium/assessment.html</u>.

PREVENTION AND TREATMENT OF DELIRIUM

As can be seen from the above predisposing factors most ICU patients have multiple risk factors for developing delirium but there are some approaches which may reduce the risk.

Non pharmacological approaches involve regular orientation of the patients, ensuring the patients wears their glasses or hearing aids (these should be taken out at night to aid sleep), consistency of nursing staff, keeping the bed-space dark at night with minimal noise to encourage normal sleep patterns, ambulate / keep patient active / stimulated during day, maintain a normal body physiology by avoiding hypoxia, hypoglycaemia, uraemia, electrolyte abnormalities, dehydration etc.

Pharmacological approaches involve, if possible, stopping unnecessary anticholinergic drugs and sedatives, especially benzodiazepines, the delirious patient may enter a vicious cycle of sedation for agititation which in turns lead to increased delerium and further agitation once the sedation wears off. Studies have been equivocal on the risk of propofol causing delirium but it is probably safer than benzodiazepines. Dexmedetomidine an α_2 adrenoreceptor agonist has recently been shown in two studies to significantly reduce delirium and duration of ventilation and is now being used increasingly for sedation in the ICU as well as for sedation of those patients already delirious [6,7].

Haloperidol and other antipsychotics have been advocated to treat delirious patients although this area remains contentious and is still largely an area of expert opinion. However the use of haloperidol in ITU patients has been shown to reduce mortality in a large cohort study, whilst the use of Quetiapine (an atypical antipsychotic) in addition to haloperidol has been shown to speed resolution of delirium and increase the chances return to home or rehabilitation [8,9]. Care must be taken if giving haloperidol to monitor the QTc on the ECG and cease giving Haloperidol if it increases to over 450msec. Haloperidol also increases extrapyramidal symptoms and must be avoided if theses become manifest or the patient has Parkinsons diseas

LONG TERM COGNITIVE DEFICIT

Approximately 1 in 3 survivors of intensive care will suffer long term cognitive impairment. Common problems are poor memory, concentration, fine motor skills, executive function (planning, organisation, decision making) and verbal fluency. Amongst patients with ARDS the prevalence of cognitive impairment was 70% at hospital discharge and 46% at one year, with all affected patients falling below the 6th percentile for the normal distribution of cognitive functioning [10]. Another study of ARDS patients found 25% had cognitive impairment at 6 years with only 46% of all patients having returned to full time employment [10]. In middle aged trauma patients 22% of survivors had trouble with employment, managing financial affairs or making travel arrangements [10]. Thus LTCI is a common and serious problem in ICU survivors.

The pathophysiology of LTCI is poorly understood but there is increasing evidence of a link between delirium and subsequent development of LTCI. Girard reported in a prospective cohort study that "increasing duration of delirium was an independent predictor of worse cognitive performance" at both 3 and 12 months follow up [11].

POST-TRAUMATIC STRESS DISORDER (PTSD)

PTSD is an anxiety disorder caused by witnessing or experiencing a traumatic event where the patient experiences extreme fear or helplessness. This could be the event that precipitates the admission to the ICU, such as a trauma, but quite easily it can also be caused just by having a life threatening illness and the treatment delivered on the ICU such as ventilation. PTSD may present as nightmares and flashbacks as well as feelings of guilt, anger and irritability. Typically reminding the patient of the traumatic event can lead to extreme reactions and should be avoided. Other signs and symptoms include avoidance of any situation that may trigger a flashback, hyper-arousal, depression, anxiety, insomnia, poor concentration and autonomic features such as sweating, shaking, chest pains and GI disturbances. The patient may also develop problems with drug or alcohol misuse and may suffer relationship problems.

The evidence for causation is poor, with most studies being relatively small, but there is developing evidence that recall of delirious memories is a risk factor for PTSD with patients who had either no recall of ICU or the most (non delirious) recall of ICU, being the most protected from developing PTSD [12]. Whilst Treggigari has demonstrated more disturbing memories and a trend towards increased PTSD in a trial comparing deeply sedated patients (rousable to voice or pain) versus those titrated to a level of awake and calm [13]. Thus it seems that avoidance of delirium and careful titration of sedation to achieve a comfortable, alert and orientated may reduce the risk of developing PTSD

Potential therapies are out-with the scope of this chapter but include cognitive behavioural therapy, psychotherapy, eye movement desensitisation and reprocessing and certain antidepressants.

ANXIETY

Intensive care is a stressful environment for patients and they will have anxieties regarding whether they will survive, their own and their families futures and concerns regarding treatment and any suffering it may cause. One of the commonest fears is "what if the ventilator fails or the endotracheal falls out?" and as a result of this many patients report being left alone as the most frightening event that can happen to them in the ICU. Patients may also have abnormal expectations based on previous experiences or even television, for instance a diagnosis of cancer may equate to a death sentence for some patients, when actually the operation has been curative. A useful approach to elucidate these fears is to ask "most people in this situation have some worries or concerns, what are yours?"

Signs and symptoms of anxiety are overt fearfulness, hypervigilence, excessive speech, increased startle response, tachycardia, hypertension and tremor. History from the patient, their medical notes or their family may suggest previous problems with anxiety and this will almost certainly be exacerbated by admission to the ICU. Anxiety can occasionally be confused with delirium but the important differentiation is that cognition and attention should not be impaired in anxiety, whereas they are in delirium.

If the admission is elective, and often even if the admission is an emergency, these anxieties can be reduced by telling the patient what to expect during their ICU stay and that the ICU staff will be monitoring them, even if not constantly by the bedside. Interventions on the ICU include appropriate reassurance from medical and nursing staff, a nursing presence by the bed, relaxing music and distraction by books or television. Rarely this will not be enough and in these instances there is a place for short term, low dose benzodiazepines but care must be taken not to precipitate delirium.

DEPRESSION

Depression is common in intensive care patients but this is often appropriate to the situation they find themselves in. Patients have an often life changing illness and have lost control over their daily life. Such a depression may reduce motivation and delay recovery. More importantly depression is common post discharge from ICU as the patient struggles to come to terms with the aftermath of critical illness experiencing problems such as, weakness, poor sleep, PTSD, anxiety, chronic illness etc. It is important to realise that pharmacotherapy plays only a very small role in treatment in the ICU although it is useful in the longer term post discharge depression.

In the ITU firstly ascertain the cognitive state, patients may state they feel down but can see they will eventually get better and still value themselves as a person. Non pharmacological treatments are the mainstay of therapy in these cases, with no real benefit from antidepressants. Patients should be given as much autonomy as possible, allow them to plan their day with their nurse so they chose, within reason, when to bathe, sit out in a chair etc, Provide books, television, radio to alleviate boredom. Encourage visits from family or close friends. If a patient has been in the unit for a prolonged time, a trip outside the front doors of the hospital, to see the sun and the sky can work wonders, this is possible even if the patient is on a ventilator. One patient I dealt with stayed 2 years in our unit and the biggest difference we made in his mental state was sneaking his dog into the unit for visits.

However when the patient has altered cognition and sees themselves as a bad person, or a burden to their families i.e. "I'd be better off dead" this is of more concern and should be investigated more thoroughly by a psychiatrist.

Patients may also develop depression after discharge from the ICU as they struggle to come to terms with the long term morbidity common after critical illness such as weakness, fatigue, PTSD, chronic illness etc.

RELATIVES

Intensive care is psychologically demanding on patients relatives. They have no real control over events, deteriorations in the patient can happen rapidly and at any time. The treating staff may not have a firm diagnosis and they may harbour doubts about the competency of the hospital staff as their relative may have been admitted to the ITU as a result of a complication of an earlier treatment. Communication is the vital factor here to produce a good relationship between the staff and relatives.

The treating doctor should meet with relatives shortly after the patient has been admitted to the ICU to update the family on the situation and ascertain their beliefs. It is important not to offer false reassurance or downplay the state of the patient. Relatives should be given a realistic prognosis followed by the assurance that appropriate treatments and investigations have been commenced. It is important to state that the patient might die during this admission but that everyone will do their best to try and prevent this. Warn them the patients' condition will fluctuate with good and bad days and finally describe the

appearance of the patient (endotracheal tube, lines etc) so they are not shocked when they first see them. If a prolonged admission is expected state that relatives will need to ensure they have plenty of sleep and periods away from the hospital themselves, statements such as "you will be no good to your relative if you haven't slept" are useful here.

Relatives can feel more empowered if allowed to assist in caring for the patient, i.e.feeding, shaving etc. They should have regular updates from the staff so they maintain realistic expectations at all times. This will make any discussions around withdrawl of therapy much easier should it become clear the patient will not survive.

STAFF FACTORS

Working in an intensive care unit can be very stressful for the staff involved. You are dealing with multiple, critically ill patients simultaneously. You also have to make decisions regarding who to admit and who to refuse. The conditions requiring admittance to intensive care are varied and you will not be an expert on them all. Furthermore the diagnosis in many patients with critical illness may be unclear leading to concerns and uncertainty regarding the appropriateness of investigations, patient management and prognosis. Finally many staff working in ICU's, like many other medical staff, have high often unrealistic expectations of their personal clinical performance.

Critically ill patients by definition have less physiological reserve to cope with any incorrect decisions, for instance choosing the wrong antibiotic for a well patient with an infected leg is not a disaster as you can simply change the agent when the infection fails to clear, but the same situation in a critically ill patient may well result in a rapid deterioration and death before you have time to realise that the chosen antibiotic is ineffective. As long as you have investigated and treated the patient sensibly, to the best of your efforts and in accordance with local best practice, it is important to realise that such a **death is not your fault**, sometimes patients die in the ICU despite our best efforts. In view of this it is important to have a colleague that you can approach for advice regarding any patient you have concerns about, or for their opinion on how you performed in a particular situation, this will allow you to seek help and evaluate yourself as others see you in a low key manner. Asking another clinician is not a sign of weakness but strength and they will most likely be flattered that you value their opinion. Remember to dwell on your successes as much as any failures to keep a sense of perspective.

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20F. MANAGING THE DYING PATIENT IN CRITICAL CARE

VINCENT PERKINS

Critical Care Units can support severely ill patients through a period of significant organ dysfunction. Despite the increasing complexity of technology available the fact remains that a large proportion of patients admitted to a unit will die. In the United States, with its relatively large amount of critical care beds, it is estimated that 20% of all deaths occur in the ICU [1].

Most of these severely ill patients are incapacitated and thus unable to voice their opinions concerning appropriateness of continuing support, which highlights the importance of fully considering the ethics of end of life care. The medical team and the family (seen as *surrogate* decision makers) face many challenging questions concerning probability of survival and the likely quality of life associated with that survival. The very concept of defining "good" quality of life for that individual patient may present difficulties in shared decision making.

In order to attempt to address some of these issues it is important to discuss the basic tenets of medical ethics. These include autonomy, beneficence, non-maleficence, justice, dignity and honesty [2].

Autonomy refers to a patient's right to accept or refuse treatment. In many ICUs surrogate decision making is the norm because of patients' inability to participate in decision making processes. Occasionally advanced care directives ("living wills") will have been made at some time in the past by the patient. These may afford some help for both doctor and surrogate in protecting autonomy. If no such directives exist there is evidence to show significant differences in decision making, depending on geography and culture. Limitations of life sustaining treatments in European ICUs are common and variable, and associated with the patient's age, diagnosis, ICU length of stay, geographic and religious factors [3].

Beneficence implies providing care which is in the best interests of the patient whereas **non-maleficence** means doing no further harm to the patient. It is of utmost importance that full and frank communication between the clinical team, the patient (if possible) and surrogates take place, because what the doctor views as in the best interests of the patient might be entirely contrary to the surrogates' ideas of the "right" thing to do. The approach by which a doctor discusses concepts of limiting life support is very important. Professional Critical Care Societies advise that physicians should offer recommendations on limitations to life support yet there is little consensus amongst surrogate decision makers as to whether that should be the physician's role. This implies that the physician should ask the surrogate whether they wish to receive recommendations regarding life support decisions [4]. Some surrogates prefer physicians to adopt a paternalistic approach whereas others feel that the role of the

doctor is to simply provide the facts and not offer their own recommendations, leaving decisions entirely to the family.

The American College of Physicians advocate a 3 step approach to decision making:

Justice implies the equal distribution of limited health care resources. Decisions should be made on clinical need and on the patients capacity to benefit and should not be based on criteria such as age, race or status The ethical care of the patient should also include concepts of dignity and honesty with the physician having no real or perceived conflicts of interests

Many national bodies will have formulated codes of ethics to assist physicians. In the UK the General Medical Council has recently issued guidance, summarised as:

- Show respect for human life;
- Protect the health of the patient;
- Treat with respect and dignity;
- The Care of the patient is your first concern [6].

Having an understanding of ethical considerations leads the ICU clinician on to making more considered judgements for the patient. In dealing with the critically ill one has to assess overall benefit in continuing care. Many ICU scoring systems have been developed in order to aid prognostication. These include **APACHE II** (Acute Physiology and Chronic Health Evaluation), looking at the 12 worst physiology variables in the first 24 hours of ICU admission; **SAPS** (Simplified Acute Physiology Score), using 14 variables and **SOFA** (sequential organ failure assessment), based on 6 systems determining the rate and extent of organ failure. These scoring systems are useful in determining relationships between disease severity and outcome and case mix for research but should not be used for individual patient prognostication [7].

A clinician simply using subjective assessments can incur "prognostic pessimism". In the UK the National Institute for Health and Clinical Excellence (NICE) suggests that consultants (i.e. the most senior doctors available) should always be involved in the decision to admit to an ICU [8]. Such decisions have significant effects on end of life care. Yet even senior doctors can be inappropriately pessimistic on the likelihood of survival when using subjective assessments; the implication is that some patients who

might otherwise survive are possibly being denied admission because of unwarranted prognostic pessimism. A study with 832 admissions for severe exacerbation of chronic obstructive lung disease compared outcome predicted by physicians against outcome at 180 days. The fifth of patients with poorest prognosis had a predicted survival rate of 10% but their actual survival rate was 40% [9].

Attempts to improve predictions in certain patient groups have resulted in new prediction models to help support decision making [10].

Once a patient has been admitted to the ICU, objective assessment of organ dysfunction will assist in the process of further decision making. The actual goal of therapy should be clarified, **medical futility** being defined as a clinical action serving no useful purpose in attaining a specified goal for the patient [11]. An Intensive Care Unit provides a clinician with an array of tools such as intubation to improve oxygenation and dialysis to remove fluid and toxins but if the patient is dying from, for example, an inoperable cancer these actions serve no useful function and should be considered medically futile. Just because a clinician <u>can</u> do something it does not follow that they <u>should</u> do something. Physicians are not obligated to provide treatment they believe are ineffective or harmful to patients even if surrogates wish these treatments to occur. There may be situations, however, when even medically futile treatment may be of short term benefit to the family through, for example, allowing time for a relative to arrive and attend the patient.

If a clinician feels that a treatment is medically futile it is in the interests of honesty and transparency to explain his reasoning to the surrogate. In discussing limitation of treatment the family must be made aware that basic medical care will continue. These include appropriate palliation ("comfort care"), pain control and dignity. If a family insist that "everything should continue to be done" then it is important for the clinician to explain the concept of medical futility and that one should minimise treatments that would simply prolong suffering. Continued communication and empathy with the family to eliminate misunderstandings and misconceptions are cornerstones of care at this critical phase in the patient's illness. Despite this there will be occasions when the family continue to disagree with a proposed limitation of treatment. It is imperative that the clinician then involves other senior colleagues and consideration should be made, if available, to involve the hospital ethics committee.

Although many ethicists and critical care societies agree that there is **no** distinction between *withholding* and *withdrawing* life sustaining treatments this view is not universal. Orthodox Jewish Law for example allows withholding of treatment but views withdrawal of continuous life sustaining treatment as an act to shorten life. The religious and cultural views of the patient, doctor and family will all influence end of life decision making.

Current US consensus on withdrawal of life-sustaining treatment is based on 3 principles [12]:

- 1. Withholding and withdrawing life support are equivalent;
- 2. There is an important distinction between killing and allowing to die;
- 3. The doctrine of double effect allows for appropriate symptom relief even when this may have the foreseen but not intended consequence of hastening death.

WITHDRAWING LIFE SUSTAINING INTERVENTIONS

Communication with the family must remain paramount. Once a decision has been reached that the patient is not benefitting from ongoing support a process should be initiated which may include withdrawal of continuing interventions but not withdrawal of care itself. Affirmation of **non-abandonment** by stressing the continuation of care for the patient and family is an essential aspect of this process. Use of accepted pathways to guide appropriate palliative care in this setting enables all members of the team to understand what the expected aims of treatment are. One such example is the *Liverpool Care Pathway* developed by the Marie Curie Palliative Care Institute, Liverpool, UK .This provides a framework for good practice and should be tailored to a person's individual needs, including consideration of their physical, social, spiritual and psychological requirements.

Relatives of patients dying in ICU are at increased risk of developing symptoms of post traumatic stress [13]. Consequently an important aspect of any approach to care of an ICU patient must be consideration of the effects of that care on the relatives.

Appropriate levels of sedation and analgesia are important components of withdrawal of support and medicines chosen should be used to achieve patient comfort. Common intravenous agents include sedatives such as propofol or benzodiazepines and analgesic agents such as morphine; dosages should be adjusted to achieve this aim.

Removal of inappropriate attachments such as ECG wires, invasive lines and saturation probes should be done sensitively and will allow the family to focus on the patient rather than fixating on a monitor. Clear explanations must be given as to the reason for doing this as the family, particularly following a prolonged ICU stay may have become very accustomed to observing every trend.

Putting a stop to unnecessary further investigations such as blood testing and radiological interventions must be made clear to all staff.

Touching and holding the patient by relatives should be encouraged, particularly as many will fear dislodging medical equipment and thus avoid attempting contact. If not done already, the family can bring in treasured mementos such as photographs to further attempt to humanise the situation. If appropriate, dressing the patient in their own clothes can assist in aiming to demedicalise the final hours as can the use of favourite pieces of music

Privacy is often difficult to achieve in a busy unit but, where possible, curtains should be drawn and family members allowed by the bedside. Unit rules that state, for example, only two members by the bedside should be relaxed as much as is practical within the confines of the unit. Acknowledging the patient's and family's spiritual needs may allow the visit of an appropriate religious representative.

Decisions must be taken on how to withdraw support. If a patient is intubated there is wide variance in what may be seen as appropriate for that patient. This may include continued intubation but with reduction/removal of ventilatory support. This could involve

reducing inspired oxygen down to 21% and removal of any additional positive end expiratory pressure, PEEP.

Extubation may be considered, particularly if the endotracheal tube is contributing to the patients discomfort. Another option may be to extubate the patient but then insert an oro- or nasopharyngeal airway to minimise obstructive symptoms. Whatever mode is chosen, it should be explained to the family first and done to minimise the patient's discomfort and avoid distressing dyspnoea. There is no place for the initiation of the use of neuromuscular blocking agents (muscle relaxants) in this process as they have no analgesic or sedative effect and mask any ability for the medical and nursing team to assess the patients level of comfort. Ideally, if a patient has been receiving these drugs they should be withdrawn. The possibility of prolonged neuromuscular blockade secondary to reduced renal and hepatic metabolism should be considered.

Other ongoing support may include cardiovascular infusions such as vasopressors and inotropes. Once a decision to withdraw has been made then there is no benefit to the patient in a gradual tailing off of these groups of drugs and thus they should be discontinued. Arterial and venous pressure traces at the bedside should be switched off. Other therapies including antibiotics, intravenous fluids, enteral and parenteral nutrition are now also considered medically futile and should in general be stopped. Some clinicians may argue that some of these treatments are minimising patient discomfort e.g. antibiotics for a urinary tract infection. This highlights the importance of individualised decision making.

If the dying process is expected to take more than a few hours it may be appropriate to consider transfer of the patient out of the Intensive Care Unit. This could be to a side room on a ward or, if available, a bed in a palliative care unit. This transfer must be agreed with the family who may fear the loss of the "protective" atmosphere of the unit and incur feelings of abandonment. Transfers out of the unit must be fully discussed with other members of hospital staff to minimise potential errors as a result of lack of communication. These errors may include inappropriate calling of the cardiac arrest team. An appropriate means of communicating important issues might include the SBAR system which includes a brief summary of the current situation, the background leading to this situation, an assessment of current needs and recommendation for ongoing care [14]. Moving a patient out of the unit can be a challenging decision because of the potential for the patient dying during the transfer process itself. This could lead to significant additional psychological trauma to the family. If the decision to transfer out of the unit is made then sedative and analgesic regimes will need to be modified to regimes that are more applicable the ward area. This may include administration of appropriate drugs via subcutaneous administration.

Occasionally, relatives may request the patient's transfer home to allow death to occur in familiar surroundings. If this is considered feasible then appropriate support should be made available to the family. If available this should include discussion and agreement with the family doctor and community nursing staff so that palliative care can be continued.

Depending on religious and cultural background the relatives may wish to prepare/cleanse the body following death. These wishes should be explored sensitively.

ORGAN DONATION

Worldwide organ donor rates vary widely but there remains a shortage of suitable donors resulting in premature deaths in potential recipients.

Consideration of deceased organ and tissue donation should, if facilities and infrastructure allow, be viewed as an integral aspect of end of life care in the Intensive Care Unit. Both patients who satisfy criteria for *brain stem* death or *circulatory* death can be viewed as potential donors. Both must demonstrate the irreversible loss of the capacity for consciousness combined with the irreversible loss of the capacity to breathe.

(A) DONATION AFTER BRAIN DEATH (DBD):

Diagnosis of death of a patient in coma whose cardio-respiratory activity is being supported by mechanical ventilation includes several key components. (There are differences worldwide on some of the criteria used such as levels of carbon dioxide rise in blood gases and duration of maintained cardio-pulmonary arrest). These components include:

- An established aetiology e.g. anoxic brain injury;
- Exclusion of reversible conditions include metabolic, endocrine and drug induced causes of coma;
- Clinical examination including presence of coma (Glasgow Coma Scale 3), absence of brain stem reflexes and continued apnoea following disconnection from the ventilator despite a significant stimulus to spontaneous respiration as measured by a specified rise in arterial PaCO2.

(B) DONATION AFTER CIRCULATORY DEATH

Includes absence of brain stem reflexes and specified duration of maintained cardio respiratory arrest.

Approaching the topic of organ donation with relatives can be emotionally challenging particularly if the patient had not made their views on the subject known beforehand. It is important that appropriately trained staff including specialist donor transplant coordinators are involved in these discussions at an early stage. They should not be viewed as having conflicts of interest (they should not be part of a transplant team). Access to organ donation registers can assist in assessing whether the patient had made previous choices on the matter. Specific ethnic and cultural considerations should be taken into account when in discussion with the family.

COMMON REASONS FOR FAMILY REFUSAL TO CONSENT [15]:

- Relatives not wishing surgery to the body/concerns regarding disfigurement;
- Feelings that the patient has suffered enough already;
- Uncertainty regarding patient's wishes;
- Disagreements amongst family members;
- Religious/cultural reasons;
- Concerns over delay to funeral/burial process;
- Unable to accept death, lack of understanding of brain death;
- Long standing negative views on organ donation;
- Relatives had decided on their own that organs were not suitable;
- Emotional exhaustion .

Full explanations should be given as to what the process of donation following death will involve, including transfer to the operating theatre and, in the case of circulatory death, a minimum of delay to retrieval of organs to preserve their function. Families should be continued to be supported through this very stressful time

SUMMARY

Intensive Care Units have much to offer in terms of organ support but the availability of medical technology should not obscure our primary aims as physicians: to care for the sick patient in a humanitarian and holistic fashion and to know when and how to take a palliative approach to care.

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ADDENDUM: SYLLABUS FOR TWO DAY CRITICAL CARE COURSE

Registration		08h10-08h40
1.1 Welcome & Introduction		08h40
1.2 Introduction to Critical Care:		09h10
1.3 Assessment of Critically ill surgical patient A. Practical demonstrations by faculty (20 min) B. Lecture (20 min)		09h30
1.4 CPR	(A) BLS/ALS <u>tutorial</u> and (B) BLS <u>demonstration</u>	10h10-10h45
<u>Tea</u>		10h45-11h05
1.5 ALS Practical		11h05-11h50
Practice CPR in groups of 3 under guidance		
1.6 ALS in Children (tutorial)		11h50-12h15
Lunch		12h15-13h00
Meet with Mentors		13h00-13h15
AIRWAY, BR	EATHING: Rotate through 3 tutorials (30 min each)	13h15-14h45
1.7 Ad	lvanced Airway management	
1.8 Trauma causes of breathlessness: life threatening chest inj		injuries
1.9 Pc	st-operative hypoxia in surgical patients	
Tea		14h40-15h05
CIRCULATION: Rotate through 3 tutorials (35 min each)		15h05-16h50
1.10 S	hock and Haemorrhage	
1.11 N	ew approaches to fluid therapy and Oliguria	
1.12 C	ardiac complications in surgical patients	
Feedback with Mentors		16h50-17h10
END OF DAY 1		
<u>DAY 2:</u> 2.1 In	08h00	
DISABILITY:	08h10-09h40	
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2.2 Confusion in surgical patients

2.3 Head injuries

2.4 Spinal injuries and patient transfer

2.5 Practical: Handling the patient with spinal trauma	09h40-10h10		
<u>Tea</u>	10h10-10h30		
Rotate through 3 tutorials (35 min each)	10h30-12h15		
2.6 Surgical Sepsis			
2.7 Obstetric critical care for surgeons			
2.8 Emergency care of Burns			
Lunch	12h15-13h00		
Rotate through 3 tutorials (30 min each):	13h00-14h30		
2.9 Anaesthesia for surgeons: Ketamine; Local and Regional anaesthesia			
2.10 Pain management			
2.11 Monitoring in critical care			
<u>Tea</u>	14h30-14h50		
EXTRAS: Rotate through 3 stations (30 min each): 14h50-16h20			
2.12 SBAR Communication intro + scenarios (in small groups):	14h50-15h30		
2.13 Quality control in critical care (interactive lecture with all)	15h30-15h50		
2.14 End-of-life care in critical illness (open discussion with all)	15h50-16h20		
10 minute break			
TEST: MCQs and EMQs	16h30-17h00		
2.15 Course Summary and Feedback	17h00-17h20		
CLOSE AND END OF COURSE			