1. OVERVIEW

This is the first in a proposed series of review articles on Critical Care in Surgery, with special reference to critical care in major trauma. Further articles will follow an ABCD approach and we plan to discuss airway management, chest trauma, post-operative hypoxia, shock, other topics related to circulation such as fluid therapy, cardiac complications in surgical patients and oliguria, causes of disability such as head injuries, spinal injuries and sepsis, and finally important adjuncts such as patient safety in critical care, communication skills and pain management. The reviews will be presented in sequential order on Surgery in Africa, but it is likely that reviews will not appear consecutively as some elective surgical topics will be interspersed. The current review on critical care and patient assessment applies equally to patients injured outside hospital, as well as to those recovering from major elective surgery.

2. INTRODUCTION TO CRITICAL CARE

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 musculo-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. (See the February 2011 Surgery in Africa review for details). 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**:  
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 haemorrhax, 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) [©RCSEd];

b. 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. Using a systematic approach gives the best chance of success;

d. remembering that most of us are bad at managing unexpected events and it is therefore better to prevent morbidity through
   o prediction,
   o repeated clinical assessment,
   o early detection of deterioration or failure to progress.

e. gathering all available information about a patient yourself through
   o good clinical observations,
   o rapid clinical assessment of deteriorating patients, using ABCDE;
   o emergency support of ABCD to allow time for more thorough assessment and treatment;
   o 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 Situation, Background, Actions or Assessment undertaken, and your Recommendation for help, or the Results of your Assessment).

**3. 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 anti-inflammatory 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 or overwhelming infection with sepsis produce the same physiological response.

3.1 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.

3.2 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)(10) to synthesise and release ACTH and growth hormone. 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.

3.3 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 leucocyte endothelial adherence(13). A cascade of local responses is initiated, characterised by TNF-alpha production, other pro-inflammatoty cytokines released(14), and increased nitric oxide production. All this leads to microcirculatory vasodilation and severe endothelial dysfunction with tissue and cell damage resulting from accumulation of metabolites and tissue hypoxia(15).

3.4 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(6).

Proinflammtory 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, however there comes a point that where if left unchecked, these responses can become counter-productive to patient survival.

3.5 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. Unchecked, the response to the ‘triad of death’ amplifies, 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).

4. Damage control resuscitation:
Damage control resuscitation (DCR) is directed at preventing these 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 includes 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. 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 hypertensive 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 clot3. 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 supraphysiological concentration of chloride. Normal saline is 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(22).
5. 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.

6. Patient Assessment:

6.1 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 Advanced Trauma Life Support (ATLS) course(3). 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 assessment and resuscitation, followed by a more detailed secondary survey and further definitive treatment.

The initial 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.

6.1.1 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 hyperflexion 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 < 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.

6.1.2 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 contusions 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 flank chest to be identified. A flank chest occurs due to multiple rib fractures, paradoxical inward motion of the flank segment will be seen with inspiration. This result 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 intercostal 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 usually 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.
6.1.3 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 is essential to correct it before proceeding with the rest of the initial assessment. This involves restoring any intra-vascular 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[21]. 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 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.

6.1.4 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 Alert, responding to Voice or Pain stimuli, or being Unresponsive).

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.

6.1.5 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, as discussed previously. 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 exist.

7. Adjuncts to Primary Survey

This depends on availability in local hospitals:

1. 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 be completed.

2. Take blood for:
   a. Cross match of urgent blood products;
   b. Electrolytes, Basic haematology, blood clotting, 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 will need emergency damage control surgery as part of resuscitation.

8. Secondary Survey

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 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.

In a critically ill surgical patient it is important to remember that a secondary survey depends on much more than just examining the patient(1). 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:
   o Speak to the patient.  
   o Check the case notes: current notes, previous history, drug history.

c. Check the Charts:
   o Observation chart/HDU/A&E Charts.  
   o Fluid balance chart: Intake-Output; think of unrecorded or hidden losses.  
   o Check the trends, not single values.  
   o 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 (MIMS.com) yourself).

d. Examination:
   o Examine the patient systematically (head to toe) yourself.  
   o Remember spinal column and rectal examination in trauma.  
   o Remember to look at drains and bags.  
   o Think what you are looking for and why.

e. Extra Information:
   o Check on all results yourself (Imaging, biochemistry, haematology, microbiology).  
   o Speak to colleagues: microbiology, laboratoriess, radiology, pharmacy.  
   o 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, RCEEng] on what to do next:

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.
9. 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 systems, 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.

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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