Managing the trauma patient presenting with the lethal trial

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It is essential that orthopaedic and trauma practitioners develop and maintain their own skills and knowledge in order to help improve and deliver quality care for patients. The utilisation of research in practice development is an important part of the process for nurses endeavouring to deliver evidence based practice to improve care and service user experience.

This new series entitled 'Practice development in orthopaedics and trauma' aims to showcase initiatives and innovations in practice development that have transformed delivery of quality care around the world and to provide readers with brief summaries of current thinking in relation to clinical issues that are common features of orthopaedic and trauma nursing practice. The feature will provide readers with a summary of an evidence base with the aim of empowering them to initiate discussion with colleagues and question their own practice. There will be associated CPD activities incorporating self-directed learning that will enhance the series and provide nurses with an opportunity to extend their learning.

The International Journal of Orthopaedic and Trauma Nursing invites contributions from clinical staff, educators and students normally of between 1000 and 2500 words. Items may focus on, but are not restricted to, best practice and practice development initiatives relating to clinical care issues, implementation of research findings and education and development of the workforce in the clinical environment.

INTRODUCTION

Musculoskeletal injury brings with it a pattern of physical trauma that can result in death in the hours, days and months that follow. It is important that orthopaedic trauma
practitioners have the knowledge and skills to provide care for the injured patient that takes into account the physiological impact of trauma so that they can deliver best evidence based care for their patients. An understanding of the actual and potential physiological response to trauma can help practitioners care for those whose condition deteriorates or who have recently been transferred from a critical care setting to an orthopaedic ward.

Studies have demonstrated a significant reduction in mortality among patients with polytrauma within the last 10 years (MacKenzie et al 2007; 2010, Cameron et al 2008). Advances in medical and surgical care, the emergence of major trauma centres and the development of trauma systems have been associated with improved mortality (Durham et al 2006, Brown et al 2010, Sampalis et al 1997, Harris et al 2010). Research suggests that poly-trauma patients managed at a major trauma centre have a significant reduction in mortality than those patient managed at general hospitals (Cameron et al 2008, McDermott et al 2007). Advances in trauma care generally and care in major trauma centres have focused on early management and “damage control” to reduced immediate life threatening complications.

The “lethal triad” of trauma is a term that describes the relationship between hypothermia, acidosis, and coagulopathy (Mitre et al 2012, Rotondo et al 1997) that can lead to patient death following major trauma. The incidence of patients presenting with the trauma triad is low, but in trauma patients with significant bleeding this combination forms a vicious circle that can be impossible to recover from (Mitre et al 2012). This has not improved in line with the improvement in general trauma care and the management and resuscitation of patients with severe trauma remains controversial.
Orthopaedic and trauma practitioners provide care to individuals following trauma in a variety of clinical settings and at various stages in the recovery trajectory. The aim of this paper is to provide practitioners working outside of the intensive care / critical care setting with an overview of the physiological cascade that can lead to the trauma triad along with an outline of management. Through an enhanced understanding of these, the practitioner will be better equipped to provide care to the deteriorating trauma patient receiving care in the general orthopaedic setting. A list of useful terms is provided in box 1.

**Box 1 - Useful terminology relating to the trauma triad**

- **Acidosis** – an increased acidity in the blood and other body tissue that causes an acid base disturbance
- **Anaerobic respiration** – cellular respiration in the absence of oxygen
- **Coagulation cascade** – Two separate pathways, the intrinsic and extrinsic, that join together to form the common pathway which leads to the clotting of blood through the production of fibrin in order to achieve haemostasis
- **Coagulopathy** – impairment of the blood’s ability to clot
- **DIC (Disseminated Intravascular Coagulation)** – the widespread activation of the clotting cascade that causes blood clots to form in the small blood vessels, reducing blood flow and leading to organ damage
- **Endogenous** – produced within an organism, tissue or cell
- **Fibrinolysis** – the normal breakdown of blood clots that prevents blood clots becoming problematic
Lactic acid – produced in muscles when they are working anaerobically (without oxygen)

Lactic acidosis – increased amount of lactic acid in the blood caused a drop in the blood pH

Metabolic acidosis – a drop in the blood pH caused by build-up of acid or impaired kidney function

Permissive hypotension – the use of restricted fluid resuscitation that increases systolic blood pressure without reaching normal blood pressure

THE LETHAL TRIAD OF TRAUMA

The term “lethal triad” describes the combination of hypothermia, acidosis and coagulopathy (figure 1). Haemorrhage from injured organs, viscera, bones and soft tissue reduces the circulating volume leading to a drop in core temperature and a reduction of oxygen rich blood supply to the tissues. This causes anaerobic metabolism which creates lactic acid production and metabolic acidosis. Anaerobic metabolism limits endogenous heat production which exacerbates any existing hypothermia caused by exposure at the scene and during pre-hospital care and subsequent emergency care and resuscitation (Jansen et al 2009). The combination of acidosis and hypothermia slow the coagulation cascade causing a loss in clotting ability known as coagulopathy (Moffatt 2012). Coagulopathy prevents haemostasis and haemorrhaging continues causing further heat loss and hypoxia. The hypothermia and acidosis continue to deteriorate which further inhibits coagulation and encourages further haemorrhage.
Studies have reported that a core temperature of less than 35°C on admission is an independent predictor of mortality in trauma patients. Several studies also suggest a fourfold increase in mortality when an established early coagulopathy is present (Brochi et al 2003; 2007, McLeod et al 2003, Maegele et al 2007). When acidosis is present alongside hypothermia, the impairment of coagulation is exacerbated (Dirkmann et al 2008, Martini et al 2005, Maani et al 2009).

**HYPOTHERMIA**

Trauma patients may have lost heat through exposure during entrapment, haemorrhage, administration of cold resuscitation fluids, removal of clothing, exhaustion and a cold environment (Lundgren et al 2011). Hypothermia is defined as a core body temperature of less than 35°C (Tortora and Derrickson 2014). Body temperature is regulated by the hypothalamus via a negative feedback loop. The hypothalamus receives feedback from the peripheral nervous system and from its own receptors that body temperature has dropped. This activates heat generating mechanisms to bring the body temperature back within the normal range. The hypothalamus stimulates skeletal muscles to shiver which ultimately leads to increased heat production but which uses up the body’s fuel; adenosine triphosphate (ATP). Blood absorbs this heat and distributes it to other tissues during circulation. As the body temperature continues to fall and the blood vessels that supply the skin constrict blood flow is restricted to the vital organs (Tortora and Derrickson 2014). Heat
can also be lost because of decreased blood flow through organs (hypoperfusion) due to hypovolaemic shock (loss of circulating volume) (Spahn et al 2005). Decreased heat production alongside impaired circulation means that blood loss can have a rapid and significant effect on core body temperature. Hypothermia can cause haemoglobin to release oxygen less readily, reduces cardiac output and can instigate cardiac arrhythmias (Moffatt 2013) which can lead to cardiac arrest.

The reactions of the clotting cascade are all temperature dependant. Both prothrombin time (PT) and partial thromboplastin time (PTT) increase in hypothermic patients (Dirkmann et al 2008). The optimum temperature for the activity of clotting factors is 37°C. Hypothermia prolongs coagulation, leading to increased clotting times and as the temperature drops, bleeding increases dramatically (Moffatt 2013). Hypothermia therefore has significant consequences for the trauma patient with one study finding a core body temperature of less than 32°C associated with a zero survival rate (Jurokovich et al 1987). Strategies for limiting hypothermia include using warmed blood products and resuscitation fluids, limiting patients’ exposure time, air warming blankets and warmed oxygen.

**ACIDOSIS**

The cause of acidosis following trauma is usually inadequate tissue perfusion (the amount of blood that a tissue is getting from the circulation). In homeostasis, aerobic respiration provides the energy required for cellular function in the form of ATP. This is reliant on a supply of oxygen to the tissues that meets their demand. A reduction in oxygen carrying capacity causes hypoxaemia. Anaerobic respiration then occurs which leads to a metabolic
Acidosis usually described as a ‘lactic acidosis’ due to the accumulation of lactic acid (Marieb 2009).

Arguably, the most dangerous effect of acidosis in the trauma patient is coagulopathy. Normally blood is slightly alkaline with a pH of between 7.35 and 7.45 (Tortora and Derrickson 2014). The coagulation system does not function in an acidic environment. The natural qualities of clotting enzymes (activated clotting factors) are altered and when pH levels drop below 7.3, thrombin generation is impaired and clotting factors break down (Sorenson et al 2012). Studies suggest that PT and PTT are increased in acidosis suggesting that acidosis has a negative effect on both the intrinsic and extrinsic clotting pathways (Dunn et al 1979). Potant procoagulant drugs cannot work when the pH is low (Mong et al 2003).

Acidosis also decreases cardiac contractility, causes arrhythmias, reduces cardiac output, causes vasodilation and hypotension and decreases renal and hepatic blood flow (Withdenthal et al 1968). As the acidosis worsens, cardiac output decreases and catecholamines (neurotransmitters such as adrenaline and dopamine) become less effective. Fluid resuscitation with unbalanced crystalloids such as 0.9% normal saline can cause a metabolic acidosis due to its high concentration of chloride. Administration of sodium bicarbonate has historically been used to treat acidosis but there is no evidence to support its effectiveness (Boyd et al 2008). Sodium bicarbonate produces carbon dioxide which can require large increases in respiratory minute volume (volume of gas inhaled or exhaled per minute) to clear. It also decreases ionised calcium concentrations which has a negative effect on cardiac and vascular contractility (Boyd et al 2008).

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COAGULOPATHY

Coagulopathy refers to an INR (International Normalised Ratio – measure of clotting efficiency) of greater than 1.5 times normal (Hodgetts et al 2007). Severe tissue damage such as traumatic amputation and head injuries can result in disseminated intravascular coagulopathy (DIC). Traumatised tissues activate the clotting cascade in an abnormal way that causes fibrinolysis (clot breakdown) out of proportion to the injury and in areas distant to the site of bleeding. DIC is a result of the severe inflammatory response that can occur following trauma which causes clotting factors to become abnormally active resulting in small clots being formed in the blood vessels (Johansson et al 2011). The clots obstruct the blood supply to vital organs causing organ damage and, ultimately, organ failure. DIC uses up the clotting factors leading to a deficiency of these at a time when they are in greatest need. This leads to further haemorrhage and compounds the coagulopathy. This lack of clotting factors can cause failure of haemostasis, haemorrhage from relatively minor injuries, stroke, limb ischaemia and organ failure (Johansson et al 2011).

Blood loss is further exacerbated by the dilution of clotting factors (dilutional coagulopathy) when fluid resuscitation is undertaken with fluids that do not contain clotting factors.

DAMAGE CONTROL RESUSCITATION

Damage control resuscitation combines permissive hypotension and haemostatic resuscitation with damage control surgery (Holcomb et al 2007) (Figure 2). Permissive hypotension involves fluid resuscitation that is limited to a volume sufficient to maintain a
radial pulse (Martin et al 2005). Blood and blood products are used early as primary resuscitation fluids to treat acute traumatic coagulopathy and to prevent the development of dilutional coagulopathy (Duchesne et al 2010). Resuscitation and surgery are undertaken simultaneously allowing an integrated approach to the management of the severe trauma patient.

*Permissive hypotension*

Permissive hypotension is a strategy for restricting fluid resuscitation until haemorrhage is controlled in order to avoid a rise in blood pressure that is likely to lead to worsening haemorrhage while accepting a period of suboptimal organ perfusion (Jansen et al 2009). No published evidence currently exists to support permissive hypotension but the concept has not been disregarded. Whilst replacing lost volume in patients with self-limiting or controlled haemorrhage is appropriate, for patients with uncontrolled haemorrhage, permissive hypotensive combined with expert resuscitation and rapid control of haemorrhage may be more appropriate. In recognition of the challenges seen in trauma following combat situations, permissive hypotension has been incorporated into military trauma management protocols (Hodgetts et al 2007). The National Institute for Health and Clinical Excellence (NICE) and the Advanced Trauma Life Support Guidelines (2012) support the need for a balance between end organ perfusion, the risk of exacerbating bleeding and maintaining an adequate blood pressure. Guidance from NICE regarding the assessment and management of airway, breathing and ventilation, circulation, haemorrhage and temperature control in major trauma is due for publication in February 2016.

*Haemostatic resuscitation*
Proactive management of coagulopathy is crucial to improving the outcome for trauma patients (Hodgetts et al 2007, Kirkman et al 2008). Common tests such as PT and PTT lack sensitivity when guiding treatment in such situations and the decision to replace clotting factors is often a clinical one (Miller 2013, Kirkman et al 2008). In trauma patients expected to require massive transfusion, administration of fresh frozen plasma (FFP), packed red blood cells (PRC) and Platelets in a 1:1:1 ratio is associated with improved survival (Miller 2013, Holcomb 2010, Jansen et al 2009).

**FFP**

Current military practice is to give FFP and PRC in a 1:1 ratio (Miller 2013, Kirkman et al 2008, Holcomb et al 2007, 2010). A retrospective study of military casualties showed a statistically significant reduction in mortality for patients (46%) who been resuscitated with 1:1 ratio compared to a more conventional 1:8 ratio (Borgman et al 2007).

**Platelets**

The administration of platelets to RBC in a 1:1 ratio is recommended by military guidelines (Miller 2013, Borgman et al 2007, Kirkman et al 2008). There is limited evidence to support this with few studies investigating the high platelet to packed cell ratio (Gunter et al 2008, Holcomb et al 2008). These studies show improved survival with a 1:1 ratio compared to the lower rates but further research is needed.

**Cryoprecipitate**

Fibrinogen deficiency develops before any other clotting derangement. Fibrinolysis carries a mortality rate of over 50% in severely injured trauma patients (Kashuk et al 2010, Schochl et al 2009, Theusinger et al 2011). The CRASH -2 trial (Shakur et al 2010, 2011) is the only class
Evidence showing a 30-day benefit from using cryoprecipitate or fibrinogen concentrate as part of resuscitation management. Cryoprecipitate contains fibrinogen, factor VIII, factor XIII and von Willebrand factor. Current British and European guidelines recommend giving cryoprecipitate or fibrinogen concentrate if plasma fibrinogen levels drop below 1.0g/l (Jansen et al 2009).

**Damage control surgery**

Following major trauma patients lack the physiological reserve to survive complex, prolonged or reconstructive surgery. The aim of damage control surgery is to restore normal physiology, not anatomical integrity. Haemorrhage is stopped by temporary clamping, packing, shunting or ligation and hollow injuries are closed or resected without anastomosis (Jansen et al 2009). Unstable musculoskeletal injuries are stabilised using minimally invasive techniques such as external fixation (Roberts et al 2005). Planned re-operation to definitively restore anatomy and achieve definitive repair is undertaken when normal physiology returns. Damage control surgery is associated with potential morbidity and should not be carried out in isolation. There is currently no evidence to support its use despite being an accepted part of the trauma patient’s management (Ball 2014).

**COMPLICATIONS ASSOCIATED WITH THE RESUSCITATION OF THE TRAUMA PATIENT**

Following the initial resuscitation there are numerous complications which can affect the major trauma patient. The management of early post resuscitation complications can affect mortality and morbidity.
**Cardiopulmonary complications**

Transfusion related acute lung injury (TRALI) is the leading cause of blood transfusion related mortality (Vamvakas 2010). Typically TRALI occurs within six hours of a blood transfusion with no evidence of circulatory overload or alternative risk factors. Patients present with acute onset hypoxaemia and bilateral infiltrates on chest xray. ICU admission is required for invasive ventilation and monitoring. Transfusions should be limited to avoid worsening outcomes. In patients where it is not appropriate to restrict transfusion avoidance of plasma with pathogenic antibodies, administration of washed blood products and use of products with the shortest length of storage are recommended (Vamvakas 2010).

Transfusion-associated circulatory overload (TACO) is the second most common cause of transfusion related mortality (Narick 2012, Popovsky 2009). TACO does not rely on large volume transfusion and is seen particularly in children and the elderly. Treatment involves supportive care, diuretic therapy for hypervolaemia and reduced infusion rate for future transfusions.

**Complications of fluid resuscitation**

Reperfusion injury occurs when perfusion is re-established to an area that has been ischaemic. In tissues which have been without oxygen, cellular mechanisms are disrupted. When the circulation is restored these conditions result in oxidative damage which initiates the inflammatory response and involves the mechanisms of apoptotic cell death (Tortora and Derrickson 2014). These factors can lead to acute respiratory distress syndrome (ARDS) and multi organ failure.

**RENAZ AL ELECTROLYTE COMPLICATIONS**
Rhabdomyolysis is the elevation of serum creatinine kinase (CK) due to the destruction or disintegration of striated muscle (Huerta-Alardin et al 2005). Muscular trauma is the most common cause with 10-50% of patients developing acute renal failure (Ward 1998, Better et al 1997). Muscular trauma may be as a result of compartment syndrome. CK and myoglobin levels are used to diagnose and monitor patients with rhabdomyolysis. Treatment involves early and aggressive fluid resuscitation to achieve a target of 100-200mls urine per hour (Shere-Wolfe et al 2012). Renal replacement therapy may be required for those patients concurrently suffering acidosis and hyperkalaemia. With appropriate treatment CK levels rise within the first 12 hours of injury, peak by 3 days and fall 3-5 days following that (Huerta-Alardin et al 2005).

Both hyperkalaemia and hypocalcaemia can become deranged due to resuscitation from haemorrhage and hypovolaemic shock. Hyperkalaemia should be promptly treated with insulin, glucose and calcium to protect the myocardium and increase the shift of potassium into the cells. Renal replacement therapy is indicated for life threatening hyperkalaemia with or without renal failure.

Hypocalcaemia occurs due to the binding of calcium to citrate preserves in blood products. It can contribute to hypotension and haemostasis and should be treated promptly.

**HYPERGLYCAEMIA**

Elevated glucose levels are very common in critically injured trauma patients and are a response to injury and stress. This is caused by acute metabolic and hormonal changes such as increased cortisol, glucagon, catecholamines, growth hormone, gluconeogenesis (the
formation of glucose from non carbohydrate substances) and glycogenolysis (the breakdown of glycogen to glucose). Strict glucose control has been associated with improved mortality and morbidity (DuBose et al 2011). Maintaining blood glucose between 4.4-5.6mmol/L has been shown to decrease length of stay and mortality (Eriksson et al 2011).

**INTRA-ABDOMINAL HYPERTENSION (IAH) AND ABDOMINAL COMPARTMENT SYNDROME (ACS)**

Damage control resuscitation and damage control surgery have improved our understanding of IAH and ACS. IAH is an elevation of intra-abdominal pressure >12mmHg and ACS the elevation of intra-abdominal pressure >20mmHg that are associated with end organ dysfunction (Mohmand et al 2011). Hypothermia, acidosis, anaemia and blood transfusion all increase the risk of ACS. Both IAH and ACS can cause cardiac failure due to vena cava compression and reduced venous return. Oliguria can occur due to compression of the intra-renal blood vessels (Mohmand et al 2011, Balogh et al 2011, Barnes et al 1985). Management includes prompt diagnosis using transduced urinary bladder pressure (Mohmand et al 2011). There are few non-surgical options available and in most cases prompt opening of the abdomen is the only method for restoring end organ function (Leppaniemi 2009).

**SUMMARY**

This article has explored the initial and continued management of the trauma patient presenting with the triad of trauma and the major clinical challenges have been identified. The last 10 years has seen major changes in the way this group of patients are resuscitated,
but there is still work to be done. Despite improvements in trauma care, mortality in patients with the triad of trauma remains high. Nurses caring for trauma patients need to understand the relationship between acidosis, hypothermia and coagulopathy in order to identify and treat patient presenting with the lethal triad of trauma.

### Table 1
**Resources for further learning: the trauma triad**

<table>
<thead>
<tr>
<th>Resource</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>General Resources</strong></td>
<td></td>
</tr>
<tr>
<td>Ball C (2014) Damage Control Resuscitation: history, theory and technique <em>Canadian Journal of Surgery</em> 57(1) 55-60</td>
<td>Ball reviews the current evidence base surrounding the use of damage control resuscitation in the trauma population</td>
</tr>
<tr>
<td>Moffatt S (2012) Hypothermia in trauma <em>Emergency Medicine Journal</em> 30:989-996</td>
<td>Moffatt explores the pathophysiology of hypothermia as a contributor to the triad of trauma and the role that hypothermia has to play in the management of the trauma patient</td>
</tr>
</tbody>
</table>

### Continuing professional development
It is essential that orthopaedic and trauma practitioners develop and maintain their own skills, knowledge and competence in order to improve and deliver safe, high quality care for patients and service users. This section is intended to provide a CPD learning opportunity, helping to support the requirements of your professional bodies in terms of lifelong learning. Continuing professional development serves as a catalyst in helping you to reflect upon and evaluate your practice. This learning activity may support you to make changes to the way you work so that you can improve the quality of care and services you provide. Alternatively, it may endorse existing practice meaning that you work as you did before, but you are more confident that your practice is safe, effective and responsive to the needs of your patients and service users (Health Care Professional Council, 2012).
**Activity: Reflection**

Reflection on your own standard of practice is an integral part of your development. You should reflect on what you have learnt from this article in terms of the trauma triad and its assessment and consider the impact on your patients and the areas in which you work. (Nursing & Midwifery Council, 2011). A useful approach to this may be to write a reflective account of a patient’s care who has been at risk of or experienced the trauma triad. See Box 2 for further guidance.

Health care professionals have a key role in musculoskeletal assessment and management. This article together with the reflective exercise and further reading is designed to maximize the existing knowledge and skills of health care professionals and help them to undertake evidence-based accurate assessment of musculoskeletal injury.

**Box 2 Reflection guide (adapted from Gibbs et al. 1998)**

<table>
<thead>
<tr>
<th>Description</th>
<th>Explain what you are reflecting on; describe your learning expectations and prior knowledge of musculoskeletal assessment in the trauma patient.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Feelings</td>
<td>Discuss your personal thoughts and feelings about your experiences of caring for the severely injured/polytrauma patient in your area of practice.</td>
</tr>
<tr>
<td></td>
<td>Have your opinions about how the severely injured/poly trauma patient is managed changed after reading this article?</td>
</tr>
<tr>
<td>Evaluation</td>
<td>What did you learn? What was helpful, what was missing, how did it make you feel?</td>
</tr>
<tr>
<td>Analysis</td>
<td>What did you gain from reading this article; did it meet your expectations?</td>
</tr>
<tr>
<td></td>
<td>How does the information in this article relate to your current practice?</td>
</tr>
<tr>
<td></td>
<td>Is there further evidence you need to gather relating to caring for the trauma patient?</td>
</tr>
<tr>
<td></td>
<td>What are the potential implications for practice?</td>
</tr>
<tr>
<td>Conclusion</td>
<td>Summarise your thoughts, what are your general and personal conclusions about management of the patient following severe trauma/polytrauma and why?</td>
</tr>
<tr>
<td>Action Plan</td>
<td>Use your reflection to initiate a discussion/debate on management of the trauma patient with your colleagues.</td>
</tr>
<tr>
<td></td>
<td>Recommendations for good practice? What will you do differently?</td>
</tr>
<tr>
<td></td>
<td>How will you disseminate any recommendations to the multi-disciplinary team?</td>
</tr>
</tbody>
</table>
REFERENCES

Ball C (2014) Damage Control Resuscitation: history, theory and technique Canadian Journal of Surgery 57(1) 55-60


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Popovský MA (2009) Transfusion associated circulatory overload: the plot thickens *Transfusion* 49: 2-4


Figure 1 – The lethal triad of hypothermia, acidosis and coagulopathy

Figure 2 – The Damage Control Resuscitation Paradigm

- Permissive Hypotension
- Haemostatic Resuscitation
- Damage Control Surgery