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DAMAGE CONTROL BEYOND THE ABDOMEN (E DEGIANNIS, SECTION EDITOR)

Damage Control Resuscitation: Restarting the Machinery of Life

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Abstract

Purpose of Review Resuscitation of the patients suffering major haemorrhage has seen a marked change in mind-set in the current millennium. This article contains a perspective on the history of shock, the detrimental effects of the traditional sequential resuscitation reactive approach of using clear fluids for volume expansion followed by blood and component therapy, and the current philosophy of the early proactive transfusion of red blood cells, plasma, and platelets in a recommended ratio. The debate concerning pharmacological intervention for fibrinolysis and percutaneous mechanical control of haemorrhage will be discussed.

Recent Findings Although the initial goal should be to achieve a ratio of 1:1:1 for packed red blood cells, plasma, and platelets, thromboelastometry is an essential point of care tool to determine the need for and effect of component therapy. This point of care tool is superior to the traditional laboratory assessment of coagulation. The use of tranexamic acid has come into question with the discovery of fibrinolytic shutdown and the detrimental effects on physiological fibrinolysis. The benefit of mechanical control of haemorrhage using percutaneous aortic occlusion is debatable.

Summary Major haemorrhage remains the commonest potentially correctable cause of early in-hospital death following major trauma. Minimising the use of clear resuscitative fluids and the early use of blood and component therapy is essential to maximise oxygen delivery, reverse the oxygen deficit and debt, and correct the coagulopathy of trauma and shock. The optimal ratio remains elusive but should be guided by thromboelastometry which will also dictate the need for antifibrinolytic therapy. The benefit and extent of permissive hypotension in those with ongoing haemorrhage until surgical control of haemorrhage can be obtained is unclear. The use of endovascular balloon occlusion of the aorta is controversial. Throughout resuscitation and damage control surgery, hypothermia must be corrected by core rewarming. Commensurate with damage control resuscitation is damage control surgery, the two being complementary for maximal benefit.

Keywords Damage control resuscitation · Damage control surgery · Major haemorrhage

Introduction

"A momentary pause in the act of death" was how John Collins Warren, the surgeon who operated during the first public demonstration of ether anaesthesia by William Morton in 1846, described severe haemorrhage. His contemporary, Samuel Gross, considered the consequences as, "A rude unhinging of the machinery of life". Their observations are not surprising for in that era of the late nineteenth century, intravenous access, resuscitation fluids, blood transfusion, and the ability to provide advanced life support were interventions of the future. Furthermore, even if available, for many years shock was not considered to be the result of hypovolaemia but rather an affliction of the nervous system. Remedies included alcohol, ammonia, and turpentine intended to act as stimulants and ironically, venesection [1]. The above notwithstanding, Gross inadvertently proposed the concept of damage control surgery (DCS) when he stated, "The indications presented in all wounds of whatever nature are first to relieve shock, secondly to arrest haemorrhage, thirdly to remove foreign matter, fourthly to approximate and retain the parts, and fifthly to limit the resulting inflammation". Controlling haemorrhage and contamination followed later by definitive surgery and attempts to minimise

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the systemic inflammatory response syndrome are precisely the essential manoeuvres employed today in the critically injured.

Although the nervous system theory persisted during the First World War, doubts were being cast. Walter Cannon recognised that shock was due to a marked reduction in blood volume but thought this resulted from pooling of blood within the body [2]. Nevertheless, he cautioned against overzealous fluid resuscitation stating, "If the pressure is raised before the surgeon is ready to check any bleeding that may take place, blood that is sorely needed may be lost". This seminal statement is now the tenet of damage control resuscitation (DCR) and permissive hypotension.

DCR and DCS, although independent concepts, are inseparable when managing the critically injured; without one, the other will almost invariably fail [3••, 4]. In brief, DCR entails minimising the use of clear fluids, a massive transfusion policy with blood and component therapy in prescribed ratios, the acceptance of permissive hypotension until surgical control can be achieved, and correcting hypothermia. The primary aims of DCS are to control haemorrhage, limit contamination if present, and delay definitive surgery until physiology allows. Initially designed for major abdominal trauma, the concept has been expanded to other anatomical regions [5, 6].

Damage Control Resuscitation

Should a critically injured person survive to reach hospital, haemorrhage remains the commonest cause of potentially preventable death. Following the realisation that after major trauma hypotension equates with hypovolaemia, fluid resuscitation became standard practice. Shires and colleagues advocated crystalloid in three times the estimated volume of blood loss [7] whereas Hamilton Bailey condemned such an approach and favoured colloids [8]. Whichever fluid was chosen, the goal was to achieve a normal blood pressure before surgery was contemplated. There are a number of major flaws in this approach. In the face of ongoing haemorrhage, as suggested by Cannon, driving up the blood pressure will result in persistent bleeding. Fluids alone will aggravate the acute coagulopathy of trauma and shock (ACoTS) following excessive blood loss, and neither crystalloids nor colloids transport oxygen, the most crucial intervention in resuscitation.

Unhinging of the Machinery of Life

Severe haemorrhage results in hypoperfusion and a marked reduction in oxygen delivery and therefore consumption. Lack of oxygen prevents pyruvate from entering Kreb's cycle with a resultant increase in lactate and an anaerobic metabolic acidosis. Failure of pyruvate to enter the cycle results in a massive reduction in the manufacture of adenosine triphosphate (ATP) the production of which, in the most severe scenario, may be reduced by 95%. This molecule is responsible for almost every energy-requiring process within the cell. The average daily turnover of ATP is in the region of 12×10^{25} molecules, the number of cupsful of water in 200 Pacific Oceans, and a mass equivalent to 100 kg [9]. The consequences on cellular function are catastrophic with apoptosis and necrosis, intracellular messenger dysfunction, the formation of oxygen free radicals, endothelial disintegration, and mitochondrial implosion [10••].

Although a vicious lethal triad of acidosis, coagulopathy, and hypothermia has been cited frequently, this is a late stage from which recovery is rare [11]. Earlier identification of at risk patients is essential, and in the absence of traumatic brain injury, there are three independent predictors of death in the critically injured namely hypoxia, hypoperfusion, and hypothermia; the "Triple H Syndrome" [12]. Hypoxia may be absolute with a low PaO_2/FiO_2 ratio or be manifest by a severe metabolic acidosis indicating anaerobic metabolism and cellular hypoxia. Hypoperfusion is reflected by a low blood pressure or a normal mean arterial pressure while using vasopressors. A core temperature < 35 °C defines hypothermia. The goal is to restore aerobic metabolism and correct hypothermia.

Airway and Ventilation

If not already performed in the prehospital setting, rapid sequence intubation and ventilation is mandatory. Drugs which do not further compromise cardiac output are essential and etomidate and ketamine are the preferred induction agents [13]. Following successful airway control, there is a limited role for protective lung ventilation in the acute setting. ARDS does not occur in the resuscitation room and data from trials in ICU have been incorrectly extrapolated to conditions outside the ICU without any proof of benefit. An adequate tidal volume is necessary to compensate for the metabolic acidosis, an elevated $PaCO_2$ must be avoided if traumatic brain injury is present, and in the presence of a pulmonary contusion, tidal volume is a recruitment manoeuvre whereas PEEP maintains volume after recruitment. A tidal volume of 8–10 mls/kg may be required in the acute setting [14•].

Correction of Hypovolaemia

Although a multi-lumen central venous catheter will be required for fluid and drug delivery, the lumen size precludes their use for rapid fluid infusion. Flow is proportional to the radius of the lumen and inversely proportional to length. Our preference is to insert an 8.5F gauge intravenous sheath into a

central vein. Possible sites are the internal jugular, subclavian or femoral veins. All have associated drawbacks. Although the easiest to access, the femoral route should be avoided in patients with suspected major intra-abdominal haemorrhage or pelvic fractures. On occasion, depending on the patient's body habitus, the sheath is too short for safe subclavian insertion. Accessing the internal jugular vein will require removal of the anterior portion of a cervical collar if in situ.

Once venous access has been secured, all administered fluids must be given through a dedicated warmer which allows high flow rates. Crystalloid solutions must be kept to a minimum and blood, plasma, and platelets are the preferred fluids in a ratio approaching that in whole blood. Both military and civilian experience suggests that this should be 1:1:1 [15–17] although the exact ratio has yet to be determined. Depending on the need for fibrinogen, cryoprecipitate may also be required. To this end, a massive transfusion protocol should have been established in all units treating major trauma. In many centres, blood and component therapy is not immediately available and a vasopressor infusion using either adrenaline or noradrenaline should be commenced to maintain an acceptable mean arterial pressure until the necessary fluids are infused [3••]. Furthermore, in those suffering massive injury, mitochondrial antigen is released into the circulation and may be many thousand times the normal concentration. Mitochondria are intracellular organelles that have evolved from bacteria and are normally hidden from the immune system. Exposure to the innate immune system results in a response similar to septic shock but without an infective component, with vasodilatation and hypotension and management therefore entails the use of vasopressors [18].

Overzealous use of blood and component therapy is not without hazard [19] and the precise requirements should be quantified objectively. Haemostasis involves an interaction amongst the endothelium, platelets, fibrin, clotting factors, and red blood cells. The standard laboratory coagulation tests are performed on platelet and cell free plasma at 37 °C, do not reflect the in vivo haemostatic process, and if abnormal cannot determine the underlying defect. Furthermore, the international normalised ratio (INR), prothrombin time, and partial thromboplastin time assess only the initial phase of coagulation and not progression of clot formation or fibrinolysis. Thromboelastometry assesses coagulation in whole blood and has gained acceptance as the optimal point of care tool by graphically illustrating each phase of coagulation [20, 21, 22•]. A directed choice of the necessary interventions thus minimises overuse of component therapy and has been reported to increase survival [22•]. This is especially relevant with the recent description of the Acute Coagulopathy of Trauma Shock (ACoTS) [23]. Although the original intent should be to achieve the recommended ratio, in addition to thromboelastometry, the clinical scenario must also be taken into consideration. Those in whom total source control of haemorrhage can be achieved surgically, such as splenectomy for splenic trauma, may not require the full protocol whereas liver or pelvic injury which necessitates packing with the risk of ongoing haemorrhage will undoubtedly need all components in the optimal ratio. Furthermore, the presence of a severe acidosis and hypothermia compromises coagulation and these physiological derangements will also dictate the necessity for specific therapeutic interventions. With regard to coagulation, laboratory values of a platelet count of > 50,000, fibrinogen > 1 g/l, and an ionised calcium of > 1 mmol/l are recommended although thromboelastometry is invaluable in determining whether these are sufficient. Based on rheology, the optimal haemoglobin for oxygen delivery is 10 g/dl. The European guidelines suggest between 7 and 9 g/dl [24] based on the TRICC study [25]. Inclusion criteria in this trial however were patients with stable organ dysfunction and normovolaemia, and the exclusion criteria were those with ongoing blood loss or transfusion of more than three units of packed red blood cells within the previous 12 h. These criteria do not apply to the trauma patient with active haemorrhage and as with studies on ventilation and intravenous fluid management in the critically ill already housed within the ICU; data from such trials cannot be extrapolated to the acute resuscitation phase.

Fibrinolysis and Tranexamic Acid

Following the CRASH-2 trial [26], tranexamic acid (TXA) has been incorporated widely into protocols for the management of major haemorrhage. The premise is that this drug will address fibrinolysis in ACoTS. This pathology occurs in a small percentage of patients with major injury and concern has been expressed that thromboembolic events have been under-reported and the drug should be used with caution and only in selected patients [27]. Furthermore, fibrinolysis shutdown has been reported in some patients and in those with physiological fibrinolysis, mortality is increased with TXA [28, 29]. To this end, thromboelastometry is the investigation of choice to determine which patients will benefit [3••, 21, 22••].

Permissive Hypotension

Although the goal of resuscitation is reversal of anaerobic metabolism by restoring mean arterial pressure and oxygen delivery, in patients who are transient responders, Cannon's premise holds true. Attempting to maintain a normal blood pressure in the face of ongoing haemorrhage is futile. In this situation, a degree of permissive hypotension is acceptable until surgical control can be achieved. This is a balance

between haemostasis and perfusion and the optimal blood pressure has yet to be elucidated although most suggest a maximum systolic blood pressure of 90 mmHg [30•]. Whatever pressure is chosen, the shortest time to operative intervention is an absolute necessity. This strategy does not apply, however, in patients with traumatic brain injury where a reduced cerebral perfusion pressure is associated with a marked increase in morbidity and mortality [31••]. Such a scenario requires a precarious course to be navigated between Scylla and Charybdis. The initial goal of DCR, however, remains the same. It is pointless undertaking neurosurgical intervention in the presence of uncontrolled haemorrhage elsewhere. If feasible, a combined surgical approach should be considered and the patient prepared for dual surgery. Once bleeding has been controlled and coagulation acceptable neurosurgical intervention should proceed. Resuscitative endovascular balloon occlusion of the aorta (REBOA) is currently being evaluated as a means of controlling non-compressible haemorrhage prior to surgical intervention [32]. This allows normal cerebral perfusion while controlling distal haemorrhage, best described as regional permissive hypoperfusion. Significant complications have been reported with debatable improved outcome and as with many new techniques, the initial enthusiasm has begun to wane and the precise indications remain unclear [33].

Hypothermia

Primary hypothermia arises when heat production is maintained but core temperature falls as a result of exposure to a cold environment and survivors with temperatures < 32 °C are recorded. Secondary hypothermia arises when the environmental temperature is normal but heat production is diminished as a result of a reduction in oxygen consumption. This is the pathology encountered following major haemorrhage with no survivors documented below 32 °C [34]. Rapid correction of core temperature must commence during DCR. External warming devices such as blankets or convective air rewarmers prevent further heat loss but are ineffective at rewarming the core. Based on the second law of thermodynamics, heat will only flow from the periphery to the core once the peripheral temperature exceeds the core. In hypothermic patients, skin blood flow decreases from 200 to 4 ml/min/m^2. At this flow rate, skin has the conductivity of cork making peripheral rewarming completely ineffective. Although essential, warming inspired air transfers little heat and the most effective and practical method of rewarming is by using heated intravenous fluids. At a fluid temperature of 40 °C, the common level achieved by dedicated warming devices, 5–8 kcal of heat per litre will be transferred to the patient. Should hypothermia persist up to ICU admission the mortality rate quadruples [35].

Restarting the Machinery of Life

If the aim of resuscitation is to reverse anaerobic metabolism, then measuring cardiac output and oxygen delivery is logical. The past few years have seen a trend from invasive to non-invasive cardiac output monitoring by a variety of techniques. Each has advantages and drawbacks and in the haemodynamically unstable patient, all are prone to error. Furthermore, the numbers generated do not necessarily reflect success or failure. Distinction must be made between oxygen deficit and debt [36••]. A normal oxygen delivery and consumption with no oxygen deficit do not mean that the debt has been repaid and other downstream markers are required. Both lactate and base deficit mirror changes in metabolism, the former anaerobic and the latter both anaerobic and aerobic. The ultimate goal should be a lactate within normal limits. If achieved within 24 h, the prognosis is optimistic; if delayed beyond 48 h, survival is uncommon [37]. In addition to documenting the success of resuscitation, serum lactate is a useful indicator of the optimal time for definitive fracture fixation following initial damage control orthopaedics [38]. Despite normal haemodynamics, persistent hyperlactaemia indicates ongoing cellular hypoperfusion and definitive surgery under these adverse circumstances renders the patient at risk of new onset organ dysfunction and prolonged mechanical ventilation.

Conclusion

More than two thousand years ago, the Hindu doctrines of Ayurveda stated, “The best treatment of any lost substance is replacement by an identical expander”. Nowhere is this more relevant than the patient suffering massive haemorrhage who has lost all components of the haematological system. Volume expansion using clear fluids is ineffectual in maximising oxygen delivery or correcting a coagulopathy and has been abandoned in favour of the rapid infusion of red blood cells, plasma, platelets, and cryoprecipitate. The exact ratio is debatable but the current philosophy is 1:1:1 with cryoprecipitate need determined by fibrinogen concentration or thromboelastometry. Although relevant only to a minority of trauma patients, DCR combined with DCS has had a marked benefit on survival. The emphasis on correction of physiology over anatomic reconstruction, minimal use of clear fluids, maximal early oxygen delivery, correction of coagulation defects, abbreviated surgery, further resuscitation in the ICU, and a return to the operating theatre only when physiology allows have been a major paradigm shift in the management of the critically injured.

Compliance with Ethical Standards

Conflict of Interest The authors declare no conflicts of interest relevant to this manuscript.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

References

Papers of particular interest, published recently, have been highlighted as:
- • Of importance
- •• Of major importance

1. Gross SD. Wounds and other injuries. Chapter 5 p45–73. In: Gross SD. A manual of military surgery. Philadelphia: JB Lippincott and Co; 1861.
2. Millham FH. A brief history of shock. Surgery. 2010;148:1026–37.
3.•• Mizobata Y. Damage control resuscitation: a practical approach for severely haemorrhagic patients and its effect on damage control surgery. J Int Care. 2017;5:4. https://doi.org/10.1186/s40560-016-0197-5. **This article contains 100 references and analyses the evidence for damage control resuscitation. The author discusses the acute coagulopathy of trauma, the benefits of a massive transfusion protocol, whether 1:1:1 is the optimal ratio, and pharmacological intervention.**
4. Lamb CM, MacGoey P, Navarro AP, Brooks AJ. Damage control surgery in the era of damage control resuscitation. BJA. 2014;113:242–9.
5. Vargo DJ, Battistella FD. Abbreviated thoracotomy and temporary chest closure: an application of damage control after thoracic trauma. Arch Surg. 2001;136:21–4.
6. Pape HC, Hildebrand F, Pertschy S, Zelle B, Garapati R, Grimme K, et al. Changes in the management of femoral shaft fractures in polytrauma patients: from early total care to damage control orthopedic surgery. J Trauma. 2002;53:452–61.
7. Shires T, Coln D, Carrico J, Lightfoot S. Fluid therapy in hemorrhagic shock. Arch Surg. 1964;88:688–93.
8. Bailey H. The treatment of shock and other methods of resuscitation. Chapter V p43–7. In: Bailey H. Emergency surgery. Bristol: John Wright and Sons Ltd.; 1953.
9. Muckart DJJ, Malbrain MLNG. A Whiter Shade of Pale: the ongoing challenge of haemorrhagic shock. Anesthesiol Intensive Ther. 2017 November 18; https://doi.org/10.5603/AIT.a2017.0060.
10.•• Paxian M, Bauer I, Rensing H, et al. Recovery of hepatocellular ATP and "pericentral apoptosis" after haemorrhage and resuscitation. FASEB J. 2003;17:993–1002. **In an exceptionally elegant animal model, the authors describe the effect of shock on hepatic ATP concentrations using firefly tail organs. The coloured illustrative figures demonstrate succinctly the massive reduction in ATP availability during various shock severity states.**
11. Mitra B, Tullio F, Cameron PA, Fitzgerald M. Trauma patients with the "triad of death". Emerg Med J. 2012;29:622–5.
12. Muckart DJJ, Bhagwanjee S, Gous E. Validation of an outcome prediction model for critically ill trauma patients without head injury. J Trauma. 1997;43:934–8.
13. Lyon RM, Perkins ZB, Chatterjee G, et al. Significant modification of traditional rapid sequence induction improves safety and effectiveness of pre-hospital trauma anaesthesia. Crit Care. 2015;19:134. https://doi.org/10.1186/s1305-015-0872-2.
14.• Hardcastle TC, Muckart DJJ, Maier RV. Ventilation in trauma patients: the first 24 hours is different. World J Surg. 2017;41:1153–8. **Protective lung ventilation has become standard of care within the intensive care unit. The authors challenge this concept during the acute resuscitative phase and offer practical reasons and guidelines for a more aggressive approach.**
15. The Prospective, Observational, Multicenter, Major Trauma Transfusion (PROMMTT) study: comparative effectiveness of a time-varying treatment with competing risks. JAMA Surg 2013;148:127–36.
16. Holcomb JB, Tilley BC, Baraniuk S, Fox EE, Wade CE, Podbielski JM, del Junco D, Brasel KJ, Bulger EM, Callcut RA, Cohen MJ, Cotton BA, Fabian TC, Inaba K, Kerby JD, Muskat P, O'Keeffe T, Rizoli S, Robinson BR, Scalea TM, Schreiber MA, Stein DM, Weinberg JA, Callum JL, Hess JR, Matijevic N, Miller CN, Pittet JF, Hoyt DB, Pearson GD, Leroux B, van Belle G, PROPPR Study Group. Transfusion of plasma, platelets, and red blood cells in a 1:1:1 vs a 1:1:2 ratio and mortality in patients with severe trauma: the PROPPR randomized clinical trial. JAMA 2015; 313:471–482.
17. Cannon JW, Khan MA, Raja AS, Cohen MJ, Como JJ, Cotton BA, et al. Damage control resuscitation in patients with severe traumatic hemorrhage: a practice management guideline from the Eastern Association for the Surgery of Trauma. J Trauma Acute Care Surg. 2017;82:605–17.
18. Sharma SK, Naidu G. The role of danger-associated molecular patterns (DAMPs) in trauma and infections. J Thorac Dis. 2016;8:1406–9.
19. Inaba K, Branco BC, Rhee P, Blackbourne LH, Holcomb JB, Teixeira PGR, et al. Impact of plasma transfusion in trauma patients who do not require massive transfusion. J Am Coll Surg. 2010;210:957–65.
20. Wise R, Faurie M, Malbrain ML, Hodgson RE. Strategies for intravenous fluid resuscitation in trauma patients. World J Surg. 2017;41:1170–83.
21. Abdelfattah K, Thromboelastography CMW. Rotational thromboelastometry use in trauma. Int J Surg. 2016;33:196–201.
22.• Gonzalez E, Moore EE, Moore HB, et al. Goal-directed hemostatic resuscitation of trauma-induced coagulopathy: a pragmatic randomized clinical trial comparing a viscoelastic assay to conventional coagulation assays. Ann Surg. 2016;263:1051–9. **Comparing conventional coagulation assays with TEG, the authors demonstrated the benefits of directed interventions on the use of component therapy with a reduction in both plasma and platelet transfusions. A reduction in mortality was also evident in the goal-directed group.**
23. Frith D, Brohi K. The pathophysiology of trauma-induced coagulopathy. Curr Opin Crit Care. 2012;18:631–6.
24. Rossaint R, Bouillon B, Cerny V, Coats TJ, Duranteau J, Fernández-Mondéjar E, et al. The European guideline on management of major bleeding and coagulopathy following trauma: fourth edition. Crit Care. 2016;20:100. https://doi.org/10.1186/s13054-016-1265-x.
25. Hebert PC, Wells G, Blajchman MA, et al. A multicenter, randomized, controlled trial of transfusion requirements in critical care. N Engl J Med. 1999;340:409–17.
26. CRASH-2 Trial Collaborators. Effects of tranexamic acid on death, vascular occlusive events, and blood transfusion in trauma patients with significant haemorrhage (CRASH-2): a randomised, placebo-controlled trial. Lancet. 2010;376:23–32.
27. Nishida T, Kinoshita T, Yamakawa K. Tranexamic acid and trauma-induced coagulopathy. J Int Care. 2017 January 20;5:5. https://doi.org/10.1186/s40560-016-0201-0.
28. Moore HB, Moore EE, Gonzalez E, Chapman MP, Chin TL, Silliman CC, et al. Hyperfibrinolysis, physiological fibrinolysis,

and fibrinolysis shutdown: The spectrum of postinjury fibrinolysis, and relevance to fibrinolytic therapy. J Trauma Acute Care Surg. 2014;77:811–7.

29. Moore HB. Tranexamic acid is associated with increased mortality in patients with physiological fibrinolysis. J Surg Res. 2017;220: 438–43.

30.• Kudo D, Yoshida Y, Kushimoto S. Permissive hypotension/ hypotensive resuscitation and restricted/controlled resuscitation in patients with severe trauma. Crit Care. 2017;5:11. https://doi.org/10.1186/s40560-016-0202-z. **This study reviews both the human and animal evidence for permissive hypotension. The authors highlight the uncertainty about the benefits to survival and recommended maximal acceptable blood pressure.**

31.•• Nevin DG, Brohi K. Permissive hypotension for active haemorrhage in trauma. Anaesthesia. 2017;72:1443–8. **This editorial summarises the acute coagulopathy of trauma and shock (ACoTS), the evidence for permissive hypotension, suggestions for the optimal blood pressure, and the dilemma of a sustained reduction in blood pressure in the patient with traumatic brain injury and extracranial uncontrolled haemorrhage**

32. Stannard A, Eliason JL, Rasmussen TE. Resuscitative endovascular balloon occlusion of the aorta (REBOA) an adjunct for haemorrhagic shock. J Trauma. 2011;71:1869–72.

33. Morrison JJ, Galgon RE, Jansen JO, Cannon JW, Rasmussen TE, Eliason JL. A systematic review of the use of resuscitative endovascular balloon occlusion of the aorta in the management of hemorrhagic shock. J Trauma Acute Care Surg. 2016;80:324–34.

34. Gentilello LM, Pierson DJ. Trauma critical care. Am J Resp Crit Care Med. 2001;163:604–7.

35. Balvers K, Van der Horst M, Graumans M, et al. Hypothermia as a predictor for mortality in trauma patients at admittance to the intensive care unit. J Emerg Trauma Shock. 2016;9:97–102.

36.•• Barbee RW, Reynolds PS, Ward KR. Assessing shock resuscitation strategies by oxygen debt repayment. Shock. 2010;33:113–22. **This article neatly summarises the pathophysiology of haemorrhagic shock with regard to oxygen delivery and consumption. The concept and difference between oxygen deficit and oxygen debt are illustrated and the benefits of serial lactate analysis I determining successful resuscitation.**

37. Odom SR, Howell MD, Silva GS, Nielsen VM, Gupta A, Shapiro NI, et al. Lactate clearance as a predictor of mortality in trauma patients. J Trauma Acute Care Surg. 2013;74:999–1004.

38. Grey B, Rodseth R, Muckart DJJ. Fracture fixation in the presence of subclinical hypoperfusion. Injury. 2013;44:217–20.