Hemorrhagic Shock

Katherine W. Gonzalez¹ Amita A. Desai¹ Brian G. Dalton¹ David Juang¹

¹Department of Pediatric Surgery, Children's Mercy Hospital, Kansas City, Missouri, United States

Address for correspondence David Juang, MD, Department of Surgery, Children's Mercy Hospital and Clinics, 2401 Gillham Road, Kansas City, MO 64108, United States (e-mail: djuang@cmh.edu).

J Pediatr Intensive Care 2015;4:4–9.

Abstract

Hemorrhagic shock has been studied extensively in the adult population, but evidence is lacking in the pediatric population. Unlike adults, pediatric patients tolerate hypovolemia with less hypotension until they have reached significant blood volume loss. It is imperative they receive prompt intravenous access, crystalloid resuscitation, followed by blood product transfusion. A hemoglobin goal of 7 g/dL has been translated to the pediatric population without evidence of poor outcomes. Massive transfusion protocols involving a 1:1:1 ratio of red blood cells:fresh frozen plasma:platelets has been recommended although further evidence is needed. With the transfusion of multiple blood products, consideration must be taken into account for the side effects, including electrolyte imbalance and lung injury.

Keywords

- shock
- massive transfusion
- pediatrics
- hemorrhage

Introduction

Historically, shock and hemorrhage were viewed as two separate entities. In fact, in the early 20th century, shock was defined as hypotension and elevated hematocrit. This concept, endorsed by the National Research Council, led to the relatively delayed acceptance of blood transfusion for the treatment of hemorrhagic shock. Instead, crystalloid solution was first used as resuscitative fluid in the Spanish American war. It was primarily provided via a rectal or subcutaneous route. Even with World War I, only the most severe cases of hemorrhage were provided intravenous resuscitation.¹ With the development of blood typing, transfusion became a safer option. Although utilized in World War I, it was not until late in World War II that the efficient storage and delivery of blood products was fully established.²

Not surprisingly, military conflict and trauma have shaped the understanding and treatment of hemorrhagic shock. However, there is substantially less data available regarding hemorrhagic shock in the pediatric population. This review article summarizes the current strategies regarding the diagnosis and management of life-threatening hemorrhage in children.

Physiology

Massive hemorrhage is quantified as bleeding requiring the transfusion of 10 units or greater (or > 40 mL/kg) of cellular blood products in a 24-hour period. An estimated 5 to 15% of pediatric trauma patients will fall into this category.³ This scenario requires the prompt identification of the etiology, intervention to prevent further loss, and treatment to maintain adequate perfusing circulatory volume. The etiology of blood loss of this magnitude can be divided into two categories: disruption of the vessel structure or deviation in the coagulation cascade.⁴

At the root of the pathophysiology associated with hemorrhage is the resultant mismatch between oxygen delivery and oxygen consumption. As blood volume falls, cardiac output and oxygen delivery decrease as well. Initially there is increased oxygen extraction and hence consumption in response to tissue acidosis and hypoxia. However, with prolonged decreases in oxygen delivery, consumption will also begin to decrease. End organs, such as the brain and intestine, are much less tolerant of this resultant hypoxemia.⁵

received
October 5, 2014
accepted after revision
November 2, 2014

Issue Theme Management of Children
with Trauma in the PICU; Guest Editor:
Pablo Aguayo, MD

Copyright © 2015 by Georg Thieme
Verlag KG, Stuttgart · New York

DOI <http://dx.doi.org/10.1055/s-0035-1554982>.
ISSN 2146-4618.

Presentation

Patients with active hemorrhage may present with symptoms ranging from mild tachycardia to lethargy based on the percentage of blood volume they have lost. The estimated blood volume is dependent on age: 90 to 100 mL/kg for premature infants, 80 to 90 mL/kg for the term infant to 3 months, 70 mL/kg in children older than 3 months, and 65 mL/kg in obese children.⁶ With a loss of less than 15% blood volume (class I hemorrhagic shock), the patient may demonstrate tachycardia or anxiety. With an increase of 15 to 25% loss in blood volume (class II), patients begin to develop oliguria and extremities become mottled in appearance and may become cool to the touch, early signs of diminished end organ perfusion. In class III hemorrhage or 26 to 39% blood volume loss, patients are more notably tachypnic and develop metabolic acidosis. By the time a patient has lost greater than 40% total blood volume (class IV), they become lethargic and may lose consciousness (► **Table 1**).^{5,7} Hypotension is a late finding in children with hemorrhagic shock and is nonspecific in the trauma population as it is often associated with traumatic head injury as well.⁸

Management

The primary goal in pediatric patients with known hemorrhage is the cessation of bleeding. History and physical examination are crucial to elucidate the cause. In some cases, the patient has visible hemorrhage, such as a traumatic extremity wound or gastrointestinal bleeding. When bleeding is visualized, direct pressure, tourniquet, and operative ligation must be performed. Gastrointestinal bleeding may be controlled via endoscopic means or with angiography and embolization. However, other sources may be less obvious. A trauma patient with hemodynamic instability, a distended and tender abdomen warrants imaging via bedside ultrasound and prompt surgical consultation. A patient with altered mental status, asymmetric or unreactive pupils

should undergo emergent computed tomography scan and neurosurgical consultation for possible intracranial hemorrhage evacuation. Postoperative hemorrhage must be entertained as a possibility following any procedure and surgeons should have a low threshold for re-exploration if bleeding is suspected. While these tasks are achieved resuscitation should be initiated.

Peripheral intravenous access is the first line of intervention. In trauma patients, peripheral lines should not be placed in injured extremities. Patients with an obvious injury below the level of the diaphragm should have at least one access in upper extremities, neck, or chest that may drain to the superior vena cava. If peripheral access is unobtainable, central venous or intraosseous routes are appropriate. However, central access is associated with higher complication rates and therefore should only be used when indicated.⁹

The initial management of children with suspected hemorrhagic shock is the prompt delivery of 20 mm/kg of crystalloid fluid. This may be repeated, but if the patient remains hemodynamically unstable or has clinical evidence of continued hemorrhage, resuscitation efforts should be transitioned to cellular blood products.¹⁰

If the patient does not require the immediate delivery of uncrossmatched packed red blood cells, cellular blood products should be leukocyte depleted, irradiated, and cytomegalovirus seronegative. Blood products should be administered through a 170- to 200- μ m filter.¹¹

Often, however, hemorrhagic shock requires the prompt transfusion of uncrossmatched blood products via a massive transfusion protocol. The use of a massive transfusion protocol has been implemented in increasingly varied settings. One institution found nearly 40% of patients receiving transfusion > 10 units (U) were in nontraumatic settings.¹² Numerous studies have addressed massive transfusion protocols in the adult population. These results have slowly been implemented in the pediatric population. A recent proposed protocol recommends a ratio of 1:1:1 units of packed red blood cells: fresh frozen plasma: platelets in patients greater than

Table 1 Hemorrhage classification

Organ system	Class			
	I	II	III	IV
Blood volume loss (%)	< 15	15–25	30–39	> 40
Cardiovascular				
Heart rate	Normal	Tachycardia	Significant tachycardia	Severe tachycardia
Blood pressure	Normal	Normal	Hypotension	Significant hypotension
Respiratory	Normal	Tachypnea	Moderate tachypnea	Severe tachypnea
Renal				
Urine output	Normal	Oliguria	Oliguria	Anuria
pH	Normal pH	Normal pH	Metabolic acidosis	Significant acidosis
Central nervous system	Anxious	Irritable	Irritable or lethargy	Lethargy
Skin	Warm, pink	Cool, mottled	Cool, pallor	Cold, cyanotic

Source: Adapted from Soud et al and Partrick et al.^{7,8}

30 kg, which is the current adult practice. Patients less than 30 kg however, should receive packed red blood cells, fresh frozen plasma, and platelets at a ratio of 30:20:20 mL/kg. Cryoprecipitate is indicated if patients demonstrate continued hemorrhage after receiving one round of each component, or if fibrinogen levels are less than 1 to 1.5 g/L. Although this protocol has not been validated, it is being adopted in many trauma centers.⁶

A prospective protocol was instituted to validate the delivery of packed red blood cells, fresh frozen plasma, and platelets in a 1:1:1 ratio after the patient has received the equivalent to one blood volume in packed red blood cells. There was no difference in mortality, when compared with patients who did not follow the protocol, and the patients outside the protocol had higher incidence of thromboembolic events. In review, the patients within the protocol had received fresh frozen plasma to red blood cell ratio closer to 1:3.¹³

A retrospective review of the delivery of blood products also did not find an increased survival in children with higher plasma to packed red blood cell or platelet to packed red blood cell ratios. They hypothesized this may be related to the pediatric population's superior tolerance of hypovolemia. Pediatric patients are also more vulnerable to complications, such as metabolic derangements, related to blood transfusion.¹⁴

As with the massive transfusion protocol, the goals of transfusion have largely been taken from adult literature. The landmark transfusion requirements in critical care trial, randomized patients to receive a blood transfusion via restrictive, or liberal policies with a trigger to transfuse of 7 versus 10 g/dL. Patients in the restrictive arm had significantly less cardiac events. In addition, mortality was decreased in patients less than 55 years of age and those who were less ill as defined by the Acute Physiology and Chronic Health Evaluation II scores.¹⁵ A multicenter prospective observational study, found the quantity of transfused red blood cells was an independent predictor of worse outcomes. The most frequent adverse effects were fever, hypotension, and fluid overload. Mortality was notably increased in critically ill patients who received greater than 6 U of packed red blood cells.¹⁶

Given the risks associated with transfusion, a restrictive policy has also been accepted in the pediatric intensive care setting. Hemoglobin of 7 g/dL is considered a reasonable goal based on review of current literature.¹⁷ Lacroix et al¹⁸ found no difference in multiorgan system failure or mortality using a restrictive approach in the pediatric intensive care unit. Restrictive transfusion guidelines have also been applied in the pediatric burn population with no increase in mortality, length of stay, or ventilator days. There were, however, double the pulmonary complications in the liberal group.¹⁹

Hemoglobin alone is an inadequate marker of resuscitation. Lactate and central venous oxygen saturation (SvO₂) are routinely used to evaluate end organ perfusion, which is, by definition, decreased in uncontrolled hemorrhagic shock. Higher levels of lactate in the adult population are associated with increased mortality and morbidity. However, measuring

lactate alone is not sufficient. Steps must be taken to improve perfusion. Decreasing lactate can be used as a marker of improved tissue oxygenation. It has been shown extensively that patients with decreasing or normalized lactate levels have better outcomes than those whose lactate remains elevated.²⁰ Central venous oxygen saturation may also be used as a marker of resuscitation. An elevated SvO₂ (> 70%) may be indicative of impaired oxygen extraction while a value less than 70% suggests inadequate perfusion. Lactate and SvO₂ have been shown to best work in conjunction rather than independently in the assessment of the critically ill patient.²¹ Further data are needed in the pediatric population regarding endpoints of resuscitation.

Although fluid therapy has often been a mainstay in decreasing markers such as lactate, limited resuscitation, both crystalloid and blood products, have emerged in the adult literature with increasing acceptance. Permissive hypotension has been championed as a method to decrease hemodilution, limit the magnitude of the ensuing inflammatory cascade, and prevent thrombus dislodgement from increasing intravascular pressures. However, it is crucial that permissive hypotension be avoided in patients with traumatic head injury, as adequate perfusing pressure is required to prevent irreversible damage.²² The theory of permissive hypotension in children may be less applicable given their improved cardiovascular reserve.²³

The impetus behind the development of the massive transfusion protocol is the onset of severe coagulopathy in patients with hemorrhagic shock. Hendrickson et al²⁴ reviewed pediatric trauma patients specifically and found 77% were coagulopathic on arrival. Those patients had longer ventilator days and an increased length of stay in the intensive care unit. Although prior authors hypothesized that hemodilution from resuscitation led to this coagulopathy, this population received on average 13 mL/kg of crystalloid fluids, a fairly minimal amount. Also, only 4% had been transfused before arrival.²⁴ Instead, multiple factors likely lead to the coagulopathic state, including metabolic acidosis from hypoperfusion and endothelial stimulation leading to protein C activation.²⁵ As with transfusion policies, the evidence behind the management of coagulopathy in children lags behind the adult data and requires further work.

Complications

Although cellular blood transfusion is necessary in the treatment of hemorrhagic shock, the delivery of blood products is not without side effects. The rate of complications has been estimated as high as 18 in 100,000 packed red blood cells in patients less than 18 years. This rate increases to 37 in 100,000 packed red blood cells in infants. Both incidences are much higher than the adult population, which is estimated to be 13 in 100,000 packed red cells. The incidence and mortality associated with noninfectious complications are substantially higher than infectious complications.²⁶

One noninfectious complication is related to citrate metabolism, a required component for anticoagulation during product storage. Citrate metabolism has been demonstrated

to lead to metabolic alkalosis in patients receiving blood products via a massive transfusion protocol. This relationship has also been confirmed in patients receiving smaller volume transfusions. In this sample, patients had decompensated metabolic alkalosis and respiratory acidosis. This was believed to be due to carbon dioxide production, leading to intracellular acidosis.²⁷

Blood product transfusion can also lead to electrolyte imbalances such as hyperkalemia. Although rare, cardiac arrest due to hyperkalemia during transfusion has been reported. A review of the Mayo experience, found 11 adults and 5 children who suffered transfusion associated hyperkalemic cardiac arrest in the operating room. Interestingly, a subsequent review of the potassium levels in blood bank samples (independent to these patients) revealed increasing potassium levels as the shelf life increased.²⁸

Transfusion related lung injury has been reported more extensively in adult literature. However, a total of 17 cases of pediatric transfusion-related lung injury were reported to the

Canadian Blood Services between 2001 and 2011. Using an estimate of total blood product transfusion in that region, the calculated incidence was 5.58 in 100,000 children. Patients were either less than 1 year or greater than 14 years, except one.²⁹

Future Endeavors

Strong data regarding the optimal use of blood products as well as endpoints of resuscitation in pediatric hemorrhage, shock requiring massive transfusion is still lacking. As it is unlikely that a prospective randomized trial will be repeated for restrictive transfusion policies, attention may be directed to the coagulopathy associated with hemorrhagic shock. Thromboelastography (TEG) has been used increasingly to direct transfusion in adults. TEG is a validated measure for fibrinolysis and assesses platelet function rather than total quantity. The TEG results can be obtained much quickly than traditional coagulation studies, allowing more rapid

Table 2 Thromboelastography interpretation

Variable	Definition	Hemostatic phase	Cause for abnormalities	Intervention
Reaction time	Time to beginning of clot formation	Initiation of coagulation	Prolonged reaction time	Plasma
			Factor deficiencies	
			Anticoagulants	
			Shortened reaction time	
			Plasma hypercoagulability	
Kinetics	Time from start of clot formation until curve reaches amplitude of 20 mm	Amplification of coagulation	Prolonged kinetics	Cryoprecipitate
			Factor deficiencies	
			Hypofibrinogenemia	
			Thrombocytopenia	
			Platelet dysfunction	
Slope between reaction time and kinetics	Angle between baseline and the tangent to the curve through the starting point of coagulation	Propagation of coagulation	Low slope between reaction time and kinetics	Cryoprecipitate
			Factor deficiencies	
			Hypofibrinogenemia	
			Thrombocytopenia	
			Platelet dysfunction	
Maximum amplitude	Amplitude measured at maximum curve width		Low maximum amplitude	Platelets
			Hypofibrinogenemia	
			Thrombocytopenia	
			Platelet dysfunction	
			Platelet deficiency	
Lysis	Reduction in area under the curve from the time maximum amplitude is achieved until 30 or 60 min after maximum amplitude	Fibrinolysis	Increased lysis	Antifibrinolytics
			Hyperfibrinolysis	

Source: Adapted from Christiaans et al.³²

decision-making in the coagulopathic patient.³⁰ TEG calculates and reports the rate of clot formation, propagation and fibrinolysis in graphical form (► **Table 2**).^{31,32} Whole blood is used in the testing, which facilitates the assessment of platelet function in conjunction with coagulation factors, red blood cells, and white blood cells. TEG results have been used as a tool to predict mortality, need for massive transfusion, and rate of thrombotic events in the adult population.³² Prospective data regarding the use of TEG in children are still lacking.

There is also potential for the use of tranexamic acid in pediatric patients with hemorrhagic shock. Tranexamic acid, an antifibrinolytic that prevents the degradation of fibrin by inhibiting plasmin and plasminogen, has been shown to decrease blood loss in pediatric cardiac surgery. Interestingly, the total transfusion rate was not decreased in this study.³³ Tranexamic acid has also been used in the orthopedic pediatric population, however, it has not been widely implemented in pediatric trauma or for resuscitation for perioperative hemorrhage. Both the Israel Defense Forces Medical Corps and British Columbia Ambulance Service have successfully implemented the use of tranexamic acid in military and civilian adult patients before hospital arrival. Although feasibility has been confirmed, outcome data are less clear.^{34,35} A multicenter, blinded, randomized clinical trial comparing prehospital use of tranexamic acid to placebo will be under way in the near future. Although this trial will only include patients older than 18 years of age, the results will certainly be interesting for prehospital care of the pediatric trauma patient.³⁶

In conclusion, hemorrhagic shock in the pediatric population remains a prevalent issue due to trauma, operative procedures, and spontaneous bleeding. Although largely gleaned from adult literature, a transfusion trigger of hemoglobin 7 g/dL is currently accepted. Patients who receive greater than one equivalent blood volume in packed red cells, should be given fresh frozen plasma and platelets in a near 1:1:1 ratio, although further research is needed in this arena. Given the risks associated with transfusion, the decision to provide cellular blood products should always be determined by individual need and clinical indication.

References

- Hardaway RM. Wound shock: a history of its study and treatment by military surgeons. *Mil Med* 2004;169(4):265–269
- Manring MM, Hawk A, Calhoun JH, Andersen RC. Treatment of war wounds: a historical review. *Clin Orthop Relat Res* 2009;467(8):2168–2191
- Pediatric Trauma Society. Massive transfusion protocol (MTP). Available at: <http://pediatrictraumasociety.org/multimedia/files/clinical-resources/MTP-3.pdf>. Accessed September 29, 2014
- Utter GH, Gosselin RC, Ownings JT. Bleeding and transfusion. In: Ashley SW, ed. *Scientific American Surgery*. Decker Intellectual Properties. Available at: <http://www.sciamsurgery.com/sciam-surgery/>. Accessed September 29, 2014
- Gutierrez G, Reines HD, Wulf-Gutierrez ME. Clinical review: hemorrhagic shock. *Crit Care* 2004;8(5):373–381
- Dehmer JJ, Adamson WT. Massive transfusion and blood product use in the pediatric trauma patient. *Semin Pediatr Surg* 2010;19(4):286–291
- Soud T, Pieper P, Hazinski MF. Pediatric trauma. In: Hazinski MF, ed. *Nursing Care of the Critically Ill Child*. 2nd ed. St. Louis, MO: Mosby Year Book; 1992:829–875
- Partrick DA, Bensard DD, Janik JS, Karrer FM. Is hypotension a reliable indicator of blood loss from traumatic injury in children? *Am J Surg* 2002;184(6):555–559, discussion 559–560
- Alarcon LH, Pyuana JC, Peitzman AB. Management of shock. In: Mattox KL, Moore EE, Feliciano DV, eds. *Trauma*. 7th ed. New York, NY: McGraw Hill; 2013:189–215
- Cooper A. Early assessment and management of trauma. In: Holcomb GW III, Murphy JP, Ostlie DJ, eds. *Ashcraft's Pediatric Surgery*. 6th ed. London: Elsevier; 2014:177–189
- Gibson BE, Todd A, Roberts I, et al; British Committee for Standards in Haematology Transfusion Task Force: Writing group. Transfusion guidelines for neonates and older children. *Br J Haematol* 2004;124(4):433–453
- McDaniel LM, Neal MD, Sperry JL, et al. Use of a massive transfusion protocol in nontrauma patients: activate away. *J Am Coll Surg* 2013;216(6):1103–1109
- Chidester SJ, Williams N, Wang W, Groner JL. A pediatric massive transfusion protocol. *J Trauma Acute Care Surg* 2012;73(5):1273–1277
- Nosanov L, Inaba K, Okoye O, et al. The impact of blood product ratios in massively transfused pediatric trauma patients. *Am J Surg* 2013;206(5):655–660
- Hébert PC, Wells G, Blajchman MA, et al. A multicenter, randomized, controlled clinical trial of transfusion requirements in critical care. Transfusion Requirements in Critical Care Investigators, Canadian Critical Care Trials Group. *N Engl J Med* 1999;340(6):409–417
- Corwin HL, Gettinger A, Pearl RG, et al. The CRIT Study: Anemia and blood transfusion in the critically ill—current clinical practice in the United States. *Crit Care Med* 2004;32(1):39–52
- Secher EL, Stensballe J, Afshari A. Transfusion in critically ill children: an ongoing dilemma. *Acta Anaesthesiol Scand* 2013;57(6):684–691
- Lacroix J, Hébert PC, Hutchison JS, et al; TRIPIU Investigators; Canadian Critical Care Trials Group; Pediatric Acute Lung Injury and Sepsis Investigators Network. Transfusion strategies for patients in pediatric intensive care units. *N Engl J Med* 2007;356(16):1609–1619
- Palmieri TL, Lee T, O'Mara MS, Greenhalgh DG. Effects of a restrictive blood transfusion policy on outcomes in children with burn injury. *J Burn Care Res* 2007;28(1):65–70
- Fuller BM, Dellinger RP. Lactate as a hemodynamic marker in the critically ill. *Curr Opin Crit Care* 2012;18(3):267–272
- Joshi R, de Witt B, Mosier JM. Optimizing oxygen delivery in the critically ill: the utility of lactate and central venous oxygen saturation (ScvO₂) as a roadmap of resuscitation in shock. *J Emerg Med* 2014;47(4):493–500
- Kaafarani HM, Velmahos GC. Damage control resuscitation in trauma. *Scand J Surg* 2014;103(2):81–88
- Hughes NT, Burd RS, Teach SJ. Damage control resuscitation: permissive hypotension and massive transfusion protocols. *Pediatr Emerg Care* 2014;30(9):651–656, quiz 657–658
- Hendrickson JE, Shaz BH, Pereira G, et al. Coagulopathy is prevalent and associated with adverse outcomes in transfused pediatric trauma patients. *J Pediatr* 2012;160(2):204–209.e3
- Choi PM, Vogel AM. Acute coagulopathy in pediatric trauma. *Curr Opin Pediatr* 2014;26(3):343–349
- Lavoie J. Blood transfusion risks and alternative strategies in pediatric patients. *Paediatr Anaesth* 2011;21(1):14–24
- Bıçakçı Z, Olcay L. Citrate metabolism and its complications in non-massive blood transfusions: association with decompensated metabolic alkalosis+respiratory acidosis and serum electrolyte levels. *Transfus Apheresis Sci* 2014;50(3):418–426

- 28 Smith HM, Farrow SJ, Ackerman JD, Stubbs JR, Sprung J. Cardiac arrests associated with hyperkalemia during red blood cell transfusion: a case series. *Anesth Analg* 2008;106(4):1062–1069
- 29 Lieberman L, Petraszko T, Yi QL, Hannach B, Skeate R. Transfusion-related lung injury in children: a case series and review of the literature. *Transfusion* 2014;54(1):57–64
- 30 Spinella PC, Holcomb JB. Resuscitation and transfusion principles for traumatic hemorrhagic shock. *Blood Rev* 2009;23(6):231–240
- 31 Galvez K, Cortes C. Tromboelastografía, nuevos conceptos en la fisiología de la hemostasia y su correlación con la coagulopatía asociada al trauma [in Spanish]. *Rev Colomb Anestesiol* 2012; 40(3):224–230
- 32 Christiaans SC, Duhachek-Stapelman AL, Russell RT, Lisco SJ, Kerby JD, Pittet JF. Coagulopathy after severe pediatric trauma. *Shock* 2014;41(6):476–490
- 33 Shimizu K, Toda Y, Iwasaki T, et al. Effect of tranexamic acid on blood loss in pediatric cardiac surgery: a randomized trial. *J Anesth* 2011;25(6):823–830
- 34 Nadler R, Gendler S, Benov A, Strugo R, Abramovich A, Glassberg E. Tranexamic acid at the point of injury: the Israeli combined civilian and military experience. *J Trauma Acute Care Surg* 2014; 77(3, Suppl 2):S146–S150
- 35 Vu EN, Schlamp RS, Wand RT, Kleine-Deters GA, Vu MP, Tallon JM. Prehospital use of tranexamic acid for hemorrhagic shock in primary and secondary air medical evacuation. *Air Med J* 2013; 32(5):289–292
- 36 Brown JB, Neal MD, Guyette FX, et al. Design of the study of tranexamic acid during air medical prehospital transport (STAAMP) trial: addressing the knowledge gaps. *Prehosp Emerg Care* 2015;19(1):79–86