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Crimson Concussa - Initial Treatment of The Polytraumatized Patient in Hemorrhagic Shock

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Abstract

This comprehensive article, titled 'Crimson Concussa', delves into the complexities of hemorrhagic shock in the context of polytrauma. It offers an in-depth analysis of the pathophysiological processes, diagnostic challenges, and treatment modalities associated with this critical condition. Emphasizing the importance of the 'golden hour' and the Advanced Trauma Life Support (ATLS) protocol, the paper underscores the necessity of a swift, systematic approach and a multidisciplinary team in managing polytraumatized patients. The review explores the nuances of initial treatment strategies, including airway management, breathing and ventilation assessment, circulatory function, and hemorrhage control. Additionally, it addresses the intricate details of pathophysiology, etiology, clinical stages, and treatment options, including fluid therapy, blood transfusions, and coagulopathy management. The article aims to enhance understanding and provide insights into the latest advancements in trauma care, highlighting the importance of ongoing research for improving therapeutic approaches and patient outcomes in the realm of emergency medicine.

Keywords: Hemorrhagic Shock; Polytrauma; Emergency Medicine; ATLS Protocol; Golden Hour; Pathophysiology; Trauma Management; Fluid Therapy; Blood Transfusion; Coagulopathy.

The Crimson Challenge: Navigating the Complexities of Polytraumatic Hemorrhagic Shock

The phenomenon of hemorrhagic shock in the setting of polytrauma presents both a formidable challenge and a pivotal focus of study. This paper, aptly titled 'Crimson Concussa, delves into the intricate interplay between systemic hemorrhage and multiple traumatic injuries. The term 'Crimson' refers to the color of blood, vividly symbolizing the hemorrhagic element, a critical and often life- threatening aspect of polytrauma, signifying the urgency and severity of blood loss. 'Concussa,' a term derived from Latin, meaning 'shaken' or 'struck,' encapsulates the traumatic nature of these injuries, portraying the physical impact and subsequent physiological turmoil. Together, these synergistically describe the complex scenario where systemic hemorrhage and multiple traumas intertwine, leading to a cascade of clinical challenges. Shock is a syndrome caused by different etiologies, which, despite our better understanding of complex pathophysiological disturbances and necessary technological development/progress, still represent a substantial therapeutic problem associated with high morbidity and mortality. Hemorrhagic shock is a leading cause of mortality in polytrauma patients [1]. Trauma is one of the leading causes of death, which is why it represents an essential problem for the healthcare system and society in general [2]. One of the most severe outcomes, next to the lethal one, is shock, which is why it is essential to quickly and successfully treat the wounded. For optimal treatment, a multidisciplinary and well-educated team is necessary [3].

The Anatomy of Trauma: Understanding Physical Impact and Injury Dynamics

Trauma can be defined as physical damage at the organic level, which occurs due to exposure to energy in a range exceeding the threshold of physiological tolerance. According to the type and number of etiological factors, injuries are divided into isolated, multiple, combined, and associated. In contemporary literature, the terms severe trauma and polytrauma are also mentioned. Severe trauma is traumatic damage to a vital organ or organ system that can lead to destruction or severe and permanent damage to an essential part of the body or an important organ, as well as to the death of the injured person. There are several definitions of polytrauma. According to the latest Berlin definition, polytrauma is an injury to at least two body regions with an Abbreviated Injury Scale (AIS) >3, associated with one or more of the listed physiological parameters: hypotension (SBP < 90mmHg), state of consciousness (GCS < 8), acidosis (BE

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< -6), coagulopathy (INR >1.4 or PTT> 40s), age (> 70 years). [4] The proposed definition, with the application of the mentioned parameters, implies the mortality of the traumatized between 11.4% and 38%. A trimodal distribution of death characterizes polytrauma patients. In the first period, within the first hour, patients die due to the severity of their injuries, most often at the scene of the accident or during transport to the hospital. In the second period, one to two hours after the injury, they die from traumatic shock and organ failure, most often upon arrival at the hospital or during emergency surgery. In the third period, which lasts several days or weeks after the injury, patients die due to organ failure [5]. The consequences of trauma can be different, from damage to complete healing to immediate death.

Golden Hour Tactics: Strategizing the First Line of Defense in Polytrauma Care

Initial treatment of polytrauma patients requires a systematic approach in the shortest interval (a couple of minutes) and is based on the principle of the golden hour. There are several international guidelines and courses for treating trauma patients, and the most widely accepted is the ATLS protocol — Advanced Trauma Life Support, which is used in more than 60 countries worldwide [6]. The protocol implies/suggests the treatment of life- threatening injuries first without a mandatory detailed anamnesis and definitive diagnosis [7]. Assessment is based on the ABCDE criteria (Table 1), and appropriate interventions are implemented/executed without delay [8].

Monitoring vital parameters (ECG, pulse oximetry, body temperature), diuresis (catheterization), and regular control blood workup and arterial blood gas analyses are routine in initial treatment [9]. Time-dependent phases of trauma treatment in the first 24 hours imply 1. The initial approach with basic diagnostic procedures and immediate life- saving procedures according to the ABC algorithm; 2. Damage control surgery in patients who do not respond to initial resuscitation measures (life-saving surgery), 3. Secondary assessment in hemodynamically stable patients with detailed diagnostics, including a “head to toe” examination and further radiological diagnostic procedures (CT, angiography, etc). The golden standard for diagnostics in polytrauma patients is computerized tomography (CT). In hemodynamically unstable patients, eFAST (extended focused assessment ultrasonography in trauma) is used to quickly review the presence of free fluid in the abdomen, which can determine the leading cause of blood loss [10]. 4. “Delayed primary surgery,” which includes decontamination, surgical exploration and treatment of non-life- threatening injuries, and temporary fracture fixation.

Following the ATLS (Advanced Trauma Life Support) protocols, patient treatment starts with a comprehensive evaluation and prompt intervention in vital domains. The first step is airway management, which includes maintaining the cervical spine. This is essential in determining obstructions that foreign materials may cause. These blockages may appear as stridor, hoarseness, or indications of laryngeal injury such as hematoma, emphysema, or dislocation. These symptoms usually occur with dyspnea and tachypnea. Airway obstruction management may require clearing upper airways, suction, or oropharyngeal tube insertion in cases that are more critical, interventions such as endotracheal intubation or surgical cricothyroidotomy might be taken all the time with a conscious awareness of cervical spine integrity.

Second, the protocol involves the evaluation of breathing and ventilation. This also consists of searching for signs of more severe conditions such as tension pneumothorax, massive hemothorax, rib fractures, subcutaneous emphysema, or thoracic contusion. Treating respiratory distress may include needle chest decompression, especially in the second intercostal space at the mid-clavicular line, to quickly relieve tension pneumothorax. Furthermore, a thoracic drain must be inserted to treat hemothorax or pneumothorax. Oxygen therapy is essential, especially in patients with rib fractures who are intubated. Circulatory function and hemorrhage control is the third step. This includes looking for shock signs, including cerebral, peripheral, and renal perfusion problems, tachycardia, pallor, mucous membrane changes, cold extremities, and delayed capillary refill. A blood loss above 30-40% generally indicates hypotension. The strategies used in treatment are focused on the restoration of volumes and surgical management of internal and external hemorrhage. This method uses clinical assessments, eFAST ultrasound, and chest and pelvis X-ray imaging. Disabilities are quickly evaluated through neurological examination, using tools such as GCS and the pupillary light reflex. A GCS score of less than

9 requires immediate medical care. Finally, exposure and environmental control involve completely disrobing the patient for a thorough examination and implementing strategies to prevent hypothermia. These strategies may include using warming blankets and administering warmed intravenous fluids [6].

A secondary assessment is done after the stabilization of vital functions. It encompasses a detailed physical examination, a request for diagnostic procedures, and anamnesis, where information about -existing allergies, drugs the patient takes, chronic illnesses, and pregnancy [7].

Crimson Tide: Deciphering Hemorrhagic Shock in Polytrauma

Shock is a state of organic hypoperfusion that, if not correct, results in cellular dysfunction and death. Hemorrhagic shock is a type of hypovolemic shock that appears as a consequence of sudden, rapid, and massive blood loss [11]. As a result of reduced tissue perfusion due to a disorder in the delivery of oxygen and its utilization, inadequate oxygenation of peripheral tissues occurs, which represents the essential characteristic of hemorrhagic shock.

The Inner Storm: Unraveling the Pathophysiological Mysteries of Hemorrhagic Shock

Intracellular metabolism depends on the delivery of oxygen to tissues (DO2) and its utilization in tissues (VO2). In a state of long- term hypoxia, when metabolic changes become irreversible (cellular death), an increase in oxygen flow does not affect the normalization of intracellular metabolic processes [12]. Pathophysiological events occur in 4 stages: 1. The Initial stage of systemic hypoperfusion leads to hypoxia and reduced ATP production. The cell switches to anaerobic metabolism with a consequential increase in CO2, lactates, and pyruvates concentration and the development of metabolic acidosis. Activation of leukocytes entails the release of inflammatory mediators (cytokines, leukotrienes, TNF) and tissue damage factors. Because of the inflammatory response, vasodilation and hypovolemia occur [13]. 2. Compensatory stage — in a state of massive hemorrhaging, the hematopoietic, cardiovascular, renal, and neuroendocrine systems are activated. At the place of injury, the coagulation cascade also activates the blood vessel

contracts, and peripheral vasoconstriction redirects blood flow to vital organs. Bleeding then leads to a drop in cardiac output and pulse pressure, stimulating baro and chemoreceptors and thus activating the sympathetic nervous system. An increase in sympathetic activity results in an increase in cardiac frequency, further vasoconstriction, and blood redistribution [14]. A drop in blood pressure and sodium concentration triggers an increase in Anti-Diuretic Hormone (ADH) secretion, which results in water and sodium reabsorption in the tubular system of the kidneys. Renin is secreted in the juxtaglomerular apparatus and participates in the compensatory vasoconstriction of the arteriolar musculature and further stimulation of aldosterone secretion. 3. Progressive stage — the cellular reaction to ischemia depends on their anaerobic capacity. Thus, it differs from organ to organ. Cells in the kidneys and colon can lower their metabolic activity, which is not valid for neurons and myocardial cells [15]. As a result of hypoxia and an accumulation of intracellular toxins, an increase in water reuptake occurs, and edema follows, further decreasing the tissue perfusion in surrounding cells. 4 — Refractory stage — characterized by progressive organ dysfunction.

Tracing the Roots: Etiological Factors Behind the Crimson Curtain

Etiological factors that lead to hemorrhagic shock are traumas, gastrointestinal bleeding (e.g., esophageal varices, peptic ulcers), vascular disorders (e.g., aortic dissection, rupture of abdominal aneurysm), urgent conditions in gynecology and obstetrics (e.g., placenta previa, rupture of an ectopic pregnancy) [16].

Shades of Crimson: Staging and Symptomatology in Hemorrhagic Shock

The clinical picture of hemorrhagic shock depends on the amount of blood lost and is thus classified into four distinct stages. Hemorrhages are classified according to the amount of blood loss and its physiological impact on the organism, which can be estimated across several points. First-degree hemorrhage is defined as less than 750 mL of blood loss with a pulse rate greater than 100 beats per minute, normal arterial blood pressure, and diuresis exceeding 30 ml/hr accompanied by an unchanged state of consciousness. Blood loss is between 750 and 1,5 mL in the Second degree, the pulse stays above 100 beats per minute, arterial blood pressure may change to a descending process, diuresis drops to 20-30 mh/hour, and agitation and confusion may develop. In the third-grade hemorrhage, blood loss ranges between 1500 to 2000 mL, and pulse rate above 120 beats/ min; the third arrow indicates a further reduction in arterial pressure, and diuresis drops markedly up to 5-15 ml/hr; the patient becomes apathetic and somnolent.

The worst, the Forth degree hemorrhage, occurs when blood loss is more significant than 2000 mL., the person's pulse rate exceeds 140 times per minute as shown by three down arrows, and signs of shock are present. Arterial blood pressure becomes significantly low; diuresis is minimal or inapparent, and consciousness decreases. These categories are essential in determining the degree of blood loss and guiding emergency medical treatment [17].

The clinical picture depends on the age, previous physiological state, possible comorbidities, and the stage of shock the patient is in. Shock is easy to diagnose if we see a patient with profuse bleeding, injuries, and weakly palpable peripheral pulses. A problem arises when symptoms and signs are present but discrete, and the outcome depends on early recognition and treatment. Early signs and symptoms of shock include pale and cold skin, generalized weakness, thirst, tachycardia, hypotension, and cold sweat. [18] Special care should be given to a state of consciousness that evolves from average to anxious, lethargic, and finally comatose. In young, otherwise healthy trauma patients, tachycardia and hypotension do not necessarily manifest in the initial phase of shock, as these patients have a high capacity for vasoconstriction and maintenance of normal blood pressure (due to sympathetic solid stimulation and good sympathetic tone). Older patients with altered physiological compensatory mechanisms and associated chronic illnesses have a significant reduction in acute loss of circulatory blood volume compensation [3].

Decoding Crisis: Diagnostic Pathways in the Labyrinth of Shock

Diagnosis is made based on data gathered in anamnesis, clinical features, and clinical findings. Initial diagnosis should primarily point to the presence of a life-threatening condition in the patient and is based on quick and orientation examination. After starting initial treatment or parallel to it, additional diagnostic procedures are done, and a multidisciplinary approach is taken. Anamnesis implies obtaining exact information about the mechanism of the sustained injury (fall from height, traffic accident, gunshot wounds, explosions). The injuries associated with blood loss may be apparent, as is the case in external bleeding, or there may be internal bleeding that is not visible. Physical examination of the patient in hemorrhagic shock should be immediate. Inspection methods such as palpation, percussion, and auscultation are applied and should include all organs and organ systems. Before examination, the source of bleeding should be determined, and the amount of blood lost should be estimated. Radiological and laboratory diagnostics allow for detailed evaluation of all injuries when clear signs of bleeding are absent. Laboratory tests are usually the most reliable indicators of hypoperfusion in the early phases of shock. The most basic set of laboratory testing includes a complete blood count with several platelets, blood group, hemostasis tests, arterial blood gas analyses, and lactate and electrolyte levels. Determination of gas analyses, base excess, and pH can approximate the degree of shock the patient is in. Lactate levels can indicate the depth of shock, and on admission, this is a sensitive predictor of outcome in patients in shock. It is done to detect lactic acidosis and prevent it. Hemoglobin and INR can be detected as predictors for massive transfusion needs [20]. The first-line panel includes parameters such as PT, aPTT, TT, and fibrinogen concentration in the hemostasis tests group. The second-line panel includes coagulation factors, euglobulin lysis time, plasmin-antiplasmin complexes, tPA (tissue polypeptide antigen), PAI-1 (plasminogen activator inhibitor), and TAFI (thrombin activatable fibrinolysis inhibitor). The second panel is not routine and is done depending on the first-line tests.

Battling the Crimson Wave: Comprehensive Treatment Strategies and Coagulopathy Prevention

Treatment of patients in states of shock must be quick as all delays lead to further progression (tissue hypoperfusion and hypoxia), and the length of duration of hypoperfusion and its' intensity are directly correlated with mortality. Therapeutic goals in hemorrhagic shock include bleeding control (hemostasis), intravascular volume compensation to correct tissue perfusion and oxygenation, and rational use of blood and blood com-

ponent transfusions in coagulopathy regulation [21]. Before treatment begins, an initial assessment of the patient's state must be done. If severe instability is observed on admission, airway management should be done, and adequate ventilation and circulatory support should be provided according to the ABC model. Guidelines for initial treatment and prevention of further bleeding imply quick patient transport to the nearest health center. In uncontrolled bleeding amid severe injuries to the extremities (penetrating, blast injuries) and traumatic amputations, it is advised to use tourniquets for bleeding control until definitive surgical hemostasis is reached [22].

Damage control strategy is a method that is used in severely injured patients and indicates all actions that need to be taken to increase the chances of survival. The concept consists of 3 phases: 1. Limited surgery — bleeding control (ligation, tamponade), infection prevention, i.e., quick laparotomy, and control of spillage of intestinal contents. 2. Rescuscitation is done in the intensive care unit where volume compensation is done using warm solutions of crystalloids and colloids, warming up of the patient, and applying the massive transfusion protocol. 3. Re-operation — definitive treatment of previously sustained injuries. Before infusion therapy is begun, good intravenous access must be secured to allow for prompt fluid compensation. It is achieved through peripheral vein cannulation and internal jugular or subclavian vein line placement [3]. Many studies have shown that aggressive fluid replacement increases hydrostatic pressure, which leads to even heavier bleeding, and the upkeep of permissive hypotension leads to a better treatment outcome [23]. Permissive hypotension aims to achieve the lowest pressure necessary to perfuse vital organs. An 80-90 mmHg range for systolic pressure is preferable for early coagulation. Important quantities of crystalloid solutions may lead to the destruction of blood clots and the reoccurrence of hemorrhage. Thus, minimal usage is advised in the early stages, 3 liters or less in the first six hours [24].

Fluid Dynamics: Navigating Through Infusion Choices in Shock Management

The most commonly used are crystalloids and colloid solutions. Crystalloid solutions include Ringer's Lactate (RL), physiological saline solution (0.9% NaCl), 0.45% NaCl, and glucose solutions. These solutions quickly redistribute in the extracellular space, so more significant volumes must be used to expand the intravascular space. Ringer lactate is the fluid of choice for fluid replacement therapy in hemorrhagic shock. Physiological saline is the second fluid of choice in treatment. Increased use of sodium chloride leads to hypernatremia and hyperchloremic acidosis [25].

Colloid solutions are extremely potent plasma expanders that increase the intravascular volume by pulling free fluid into the vascular bed. Colloid solutions are divided into natural (albumins, plasma) and synthetic (dextranes, HES). Albumins lower the risk of kidney damage in hemodynamically unstable patients. In hypovolemic conditions, albumins are highly efficient in correcting colloidal-osmotic pressure (COP) and upkeep of an adequate gradient between COP and pulmonary artery occlusion pressure (PAOP) compared to crystalloid solutions [26]. Dextranes are characterized by an effect of intravascular plasma expansion lasting 6 hours. In fluid replacement, it is preferable to use crystalloid solutions [27]. The use of synthetic colloids has been linked to increased incidence of coagulopathy, acute kidney injury, and worse outcomes in critically ill patients [28]. Vasoactive drugs are implemented in the treatment of hemorrhagic shock if hypotension persists despite fluid replacement therapy. Vasopressors such as norepinephrine and epinephrine, as well as inotropic drugs such as dobutamine, are the drugs of choice.

Transfusion Decisions: The Lifeline in a Sea of Crimson

Whole blood transfusions are indicated only in massive bleeding, usually on battlefields and in the army. Recent experiences from Iraq and Afghanistan have shown that patients receiving whole blood and blood derivatives transfusions had better clinical outcomes in comparison to those receiving treatment with blood derivatives only [29]. For this reason, their use in these cases remains justified.

Erythrocytes are the most commonly used blood component in transfusion therapy. Erythrocytes increase the ability to transport oxygen in anemic patients and can thus improve platelet function by stabilizing turbulent blood flow and marginalizing them. Concentrated erythrocytes are used as compensation for hemoglobin, and their use is indicated if Hb values are in the range of 70- 90g/L, after which false elevation may occur due to hemoconcentration. Using fresh frozen plasma in bleeding patients can stabilize fibrinogen levels and prevent further decrease. However, FFP cannot increase fibrinogen levels unless applied in significant quantities [30]. The indication for FFP use is PT and aPPT >1.5 times the average value. The risks of using substantial amounts of FFP include circulatory burden, allergic reaction, and acute lung injury following blood transfusion (Transfusion- Related Acute Lung Injury). Platelet transfusions are indicated if their level drops below 50 x 10^9 /L, except for patients with neurotraumas, whose cut-off value is below 100 x 10^9 L. Recent studies show that low platelet numbers predict increased mortality [30]. Cryoprecipitate contains factor VII, von Willebrand factor, factor XII, fibronectin, and fibrinogen. If fibrinogen is lower than 1.5g/L, a cryoprecipitate transfusion is indicated [31].

When Every Drop Counts: Strategies for Massive Transfusion in Critical Times

Massive transfusion protocols have been developed as a solution to high rates of multi- organ dysfunction and mortality in polytrauma patients with hemorrhagic shock and coagulopathy on admission to the trauma bay.[30] Massive transfusion may be defined as 1. Complete patient blood volume replacement within 24 hours; 2. Replacement of 50% or more of a patient's blood volume within 3 hours, 3. Blood loss replacement at a rate of

>150mL/minute [32]. Although the decision to use massive transfusions in a patient depends mainly on the clinical features, specific scores exist to help estimate if the patient needs an enormous transfusion. The three most famous scores used to estimate bleeding are TASH, ABC, and McLaughlin. However, none of these scores provide perfect estimation and have limits in their usage, so assessment for massive blood transfusions still largely depends on the physician. The ABC score assesses the presence of a penetrating mechanism, systolic arterial blood pressure <90 mmHg, heart rate >120/minute, and positive ultrasound findings for abdominal trauma [33]. Before a transfusion, it is mandatory to obtain the patient's blood group and Rhesus antigen, as well as plasma compatibility of the patient and the donor's erythrocytes. In massive hemorrhaging with little time to spare, O RhD-negative blood is given to prevent adverse reactions. A proactive approach in treating hemorrhagic

shock implies early prevention of coagulopathy. Massive transfusion protocols suggest using transfusion packages (concentrated red blood cells, platelets, and fresh frozen plasma) in the ratio of 2:1:1 or 1:1:1 [34]. Early use of blood and blood derivatives is necessary not only for volume compensation but also to increase oxygen transport and improve hemostatic capacities.

Tranexamic acid is an antifibrinolytic drug used today as part of pre-hospital care. The CRASH- 2 study showed that early use of this agent in the first hour of trauma significantly reduces the risk of death due to hemorrhage [35]. As this drug has a high therapeutic index, it must be administered within the first 3 hours in all polytrauma patients with evident bleeding, a systolic pressure lower than 110mmhg, and an HR >110/minutes or both, regardless of the type of injury.

Acute coagulopathy linked with trauma represents a multifactorial primary state that arises due to a combination of hemorrhagic shock, the creation of thrombin- thrombomodulin complexes as a result of the injuries and activation of the coagulation and fibrinolytic cascades. The thrombin- thrombomodulin complex activates protein C, which inhibits coagulation as it blocks the activation of factors V and VIII, thus stimulating fibrinolysis by lowering plasminogen activating inhibitor, which speeds up plasmin creation and leading to hyperfibrinolysis [36]. Massive bleeding, followed by a drop in circulatory volume, decreases the ability of the body to maintain its temperature as the flow of oxygenated blood to organs is reduced. A drop in temperature is followed by a reduction in thrombin production (factor IIa), which results in longer bleeding time. Once the body's temperature falls below 33 degrees Celsius, tissue factor and factor VIIa's coagulation activity is reduced, directly influencing fibrinogen synthesis. Acidosis and a decrease in ionized Ca also disturb the coagulation process. In liver damage, a deficit in ionized calcium, which contains citrate as an anticoagulant, occurs after blood component transfusions [37]. Coagulopathy is a part of the triad of death, along with acidosis and hypothermia [38]. Early coagulation monitoring is critical for the detection of coagulopathy and for defining the main causative agents, including hyperfibrinolysis. Standard coagulation tests have limited use in a patient with active bleeding. These tests are inadequate predictive tools for massive transfusions and have limited capacity for transfusion therapy guidance. Point-of-care coagulation tests, thromboelastometry, and thromboelastography give quick information about the formation of a clot, its strength, and degradation time in the blood. They are shown to be effective in early identification and targeted monitoring of coagulopathy [39]. To quickly establish a diagnosis, rotation thromboelastometry (ROTEM) uses different reagents that activate or inhibit platelets, heparin, and fibrinolysis. ROTEM is thus used to assess the ability of clot formation, its' stability, and platelet function [40]. Early therapeutic intervention improves coagulation tests and decreases the need for transfusion. Erythrocyte transfusion and intravenous solution for fluid replacement usage do not contain coagulation factors and platelets, which may lead to diluted coagulopathy.

Beyond Crimson: Concluding Reflections on Polytrauma and Hemorrhagic Shock

In conclusion, 'Crimson Concussa' is a profound exploration into the critical and intricate world of hemorrhagic shock amidst polytrauma. This phenomenon, symbolized by the vivid imagery of 'Crimson' – the lifeblood lost in these emergencies – coupled with 'Concussa,' reflecting the traumatic impact, underscores the gravity and complexity of managing such medical crises. Our comprehensive review underlines the necessity for rapid, systematic intervention within the 'golden hour,' the significance of protocols like ATLS, and the importance of a multidisciplinary approach in treating polytraumatized patients.

The journey through the pathophysiology, diagnosis, and management strategies of hemorrhagic shock reveals a landscape fraught with clinical challenges yet ripe with opportunities for advancement. The evolving understanding of therapeutic techniques, especially those aimed at promptly achieving hemostasis, has been instrumental in turning the tide towards more favorable outcomes in these perilous situations. As we continue to delve deeper into the nuances of trauma care, we must focus on areas such as primary prevention, early recognition, and enhanced resuscitation techniques. The quest for knowledge in these domains remains paramount, as they are crucial to unlocking further improvements in patient care and survival rates. 'Crimson Concussa' highlights the current state of trauma management and paves the way for future research and development in this vital field of medicine.

References

1. Chang R, Holcomb JB. Optimal Fluid Therapy for Traumatic Hemorrhagic Shock. Crit Care Clin. 2017; 33: 15-36.
2. Global, regional, and national age-sex specific all- cause and cause-specific mortality for 240 causes of death, 1990-2013: a systematic analysis for the Global Burden of Disease Study 2013. Lancet. 2015; 385: 117-71.
3. Bumbasirević V, Jovanović B, Palibrk I, Karamarković A, Radenković D, Gregorić P, et al. [Hemorrhagic shock]. Acta Chir Iugosl. 2007; 54: 63-70.
4. Rau CS, Wu SC, Kuo PJ, Chen YC, Chien PC, Hsieh HY, et al. Polytrauma Defined by the New Berlin Definition: A Validation Test Based on Propensity- Score Matching Approach. Int J Environ Res Public Health. 2017; 14: 1045.
5. Sobrino J, Shafi S. Timing and causes of death after injuries. Proceedings (Baylor University Medical Center). 2013; 26: 120-3.
6. Radvinsky DS, Yoon RS, Schmitt PJ, Prestigiacomo CJ, Swan KG, Liporace FA. Evolution and development of the Advanced Trauma Life Support (ATLS) protocol: a historical perspective. Orthopedics. 2012; 35: 305- 11.
7. Milenković M, Terzioski Z, Hadzibegovic A, Stanisavljevic J, Petro-vic K, Nikolic J, et al. Evaluation of Independent predictors of in-hospital morality in patients with severe trauma. Srpsk arhiv za celokupno lekarstvo. 2019; 147: 455-460.
8. Stahel PF, Heyde CE, Wyrwich W, Ertel W. [Current concepts of polytrauma management: from ATLS to "damage control"]. Orthopade. 2005; 34: 823-36.
9. Parker M, Magnusson C. Assessment of trauma patients. Int J Orthop Trauma Nurs. 2016; 21: 21-30.
10. Kameda T, Taniguchi N. Overview of point-of-care abdominal ultrasound in emergency and critical care. Journal of intensive care. 2016; 4: 53.
11. Cannon JW. Hemorrhagic Shock. N Engl J Med. 2018; 378: 370-9.
12. Simmons J, Ventetuolo CE. Cardiopulmonary monitoring of shock. Curr Opin Crit Care. 2017; 23: 223-31.
13. Zhang Q, Raoof M, Chen Y, Sumi Y, Sursal T, Junger W, et al. Circulating mitochondrial DAMPs cause inflammatory responses to injury. Nature. 2010; 464: 104- 7.

14. Gutierrez G, Reines HD, Wulf-Gutierrez ME. Clinical review: hemorrhagic shock. Critical care (London, England). 2004; 8: 373-81.

15. Erecińska M, Silver IA. Tissue oxygen tension and brain sensitivity to hypoxia. Respir Physiol. 2001; 128: 263-76.

16. Ruseckaite R, McQuilten ZK, Oldroyd JC, Richter TH, Cameron PA, Isbister JP, et al. Descriptive characteristics and in-hospital mortality of critically bleeding patients requiring massive transfusion: results from the Australian and New Zealand Massive Transfusion Registry. Vox Sang. 2017; 112: 240-8.

17. Advanced trauma life support (ATLS®): the ninth edition. J Trauma Acute Care Surg. 2013; 74: 1363-6.

18. Bonanno FG. Clinical pathology of the shock syndromes. Journal of emergencies, trauma, and shock. 2011; 4: 233-43.

19. Jonishi K, Sakamoto Y, Ueno Y, Matsumoto H, Hara Y, Kutsukata N, et al. Examination of the utility of serum lactate and base deficit in hemorrhagic shock. Critical Care. 2010; 14: P161.

20. Callcut RA, Cotton BA, Muskat P, Fox EE, Wade CE, Holcomb JB, et al. Defining when to initiate massive transfusion: a validation study of individual massive transfusion triggers in PROMMTT patients. J Trauma Acute Care Surg. 2013; 74: 59-65 67.

21. Stainsby D, MacLennan S, Thomas D, Isaac J, Hamilton PJ. Guidelines on the management of massive blood loss. Br J Haematol. 2006; 135: 634- 41.

22. Lakstein D, Blumenfeld A, Sokolov T, Lin G, Bssorai R, Lynn M, et al. Tourniquets for hemorrhage control on the battlefield: a 4-year accumulated experience. J Trauma. 2003; 54: S221-5.

23. Waydhas C. [Preclinical management of multiples injuries: S3 guideline]. Unfallchirurg. 2012; 115: 8- 13.

24. Shafi S, Collinsworth AW, Richter KM, Alam HB, Becker LB, Bullock MR, et al. Bundles of care for resuscitation from hemorrhagic shock and severe brain injury in trauma patients-Translating knowledge into practice. J Trauma Acute Care Surg. 2016; 81: 780-94.

25. Yunos NM, Bellomo R, Hegarty C, Story D, Ho L, Bailey M. Association between a chloride-liberal vs chloride-restrictive intravenous fluid administration strategy and kidney injury in critically ill adults. Jama. 2012; 308: 1566-72.

26. Alderson P, Bunn F, Lefebvre C, Li WP, Li L, Roberts I, et al. Human albumin solution for resuscitation and volume expansion in critically ill patients. Cochrane Database Syst Rev. 2002: Cd001208.

27. Bulger EM, May S, Kerby JD, Emerson S, Stiell IG, Schreiber MA, et al. Out-of- hospital hypertonic resuscitation after traumatic hypovolemic shock: a randomized, placebo-controlled trial. Ann Surg. 2011; 253: 431-41.

28. Lewis SR, Pritchard MW, Evans DJ, Butler AR, Alderson P, Smith AF, et al. Colloids versus crystalloids for fluid resuscitation in critically ill people. Cochrane Database Syst Rev. 2018; 8: Cd000567.

29. Chandler MH, Roberts M, Sawyer M, Myers G. The US military experience with fresh whole blood during the conflicts in Iraq and Afghanistan. Semin Cardiothorac Vasc Anesth. 2012; 16: 153-9.

30. 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.

31. Robinson S, Harris A, Atkinson S, Atterbury C, Bolton-Maggs P, Elliott C, et al. The administration of blood components: a British Society for Haematology Guideline. Transfus Med. 2018; 28: 3-21.

32. Shaz BH, Dente Cj Fau - Harris RS, Harris Rs Fau - MacLeod JB, MacLeod Jb Fau - Hillyer CD, Hillyer CD. Transfusion management of trauma patients. 2022; 28: 725-731.

33. Nunez TC, Voskresensky IV, Dossett LA, Shinall R, Dutton WD, Cotton BA. Early prediction of massive transfusion in trauma: simple as ABC (assessment of blood consumption)? J Trauma. 2009; 66: 346-52.

34. Kleinveld DJB, Wirtz MR, van den Brink DP, Maas MAW, Roelofs J, Goslings JC, et al. Use of a high platelet-to-RBC ratio of 2:1 is more effective in correcting trauma-induced coagulopathy than a ratio of 1:1 in a rat multiple trauma transfusion model. Intensive Care Med Exp. 2019; 7: 42.

35. Shakur H, Roberts I, Bautista R, Caballero J, Coats T, Dewan Y, et al. 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.

36. Moore HB, Moore EE, Liras IN, Gonzalez E, Harvin JA, Holcomb JB, et al. Acute Fibrinolysis Shutdown after Injury Occurs Frequently and Increases Mortality: A Multicenter Evaluation of 2,540 Severely Injured Patients. J Am Coll Surg. 2016; 222: 347-55.

37. MacKay EJ, Stubna MD, Holena DN, Reilly PM, Seamon MJ, Smith BP, et al. Abnormal Calcium Levels During Trauma Resuscitation Are Associated with Increased Mortality, Increased Blood Product Use, and Greater Hospital Resource Consumption: A Pilot Investigation. Anesth Analg. 2017; 125: 895-901.

38. Cosgriff N, Moore EE, Sauaia A, Kenny-Moynihan M, Burch JM, Galloway B. Predicting life-threatening coagulopathy in the massively transfused trauma patient: hypothermia and acidoses revisited. J Trauma. 1997; 42: 857-61.

39. McCullough AL, Haycock JC, Forward DP, Moran CG. Early management of the severely injured major trauma patient. Br J Anaesth. 2014; 113: 234-41.

40. Schöchl H, Nienaber U, Hofer G, Voelckel W, Jambor C, Scharbert G, et al. Goal- directed coagulation management of major trauma patients using thromboelastometry (ROTEM)-guided administration of fibrinogen concentrate and prothrombin complex concentrate. Crit Care. 2010; 14: R55.