CUTTING-EDGE TREATMENTS FOR HEMORRHAGIC SHOCK IN VETERINARY MEDICINE

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INTRODUCTION

Hemorrhagic shock (**HS**) is a frequent, life-threatening emergency in veterinary medicine, defined by profound systemic hypoperfusion due to significant blood loss. HS can result from various etiologies, including trauma, neoplasia, coagulopathies, or iatrogenic events such as surgical procedures. Immediate, evidence-based recognition and targeted therapy are critical to patient survival and recovery.

CLINICAL ASSESSMENT AND DIAGNOSIS

Accurate and rapid clinical recognition of HS involves integrating a structured physical examination, bedside diagnostic tools, and laboratory assessments. Traditionally, the initial patient assessment follows the ABC (Airway, Breathing, Circulation) sequence, focusing primarily on airway patency, adequate breathing, and stable circulation. While widely used, the ABC approach may delay recognition and control of life-threatening hemorrhage.

In contrast, the MARCH-PAWS protocol, adapted from military and human trauma care, provides a systematic assessment prioritizing bleeding control and rapid stabilization. This structured trauma-specific approach includes:

- **M (Massive hemorrhage):** Immediate identification and direct control of active bleeding through pressure, hemostatic dressings, or tourniquets.
- **A (Airway):** Rapidly evaluate and secure airway patency.
- **R (Respiration):** Ensure effective breathing; intervene as needed (oxygen supplementation, thoracocentesis).
- **C (Circulation):** Clinically, patients in hemorrhagic shock typically exhibit pale mucous membranes, prolonged CRT (>2 seconds), weak peripheral pulses, cold extremities, altered mentation, and elevated heart rate.
- **H (Head injury/Hypothermia):** Assess mentation; actively prevent hypothermia.
- **P (Pain management):** Provide appropriate analgesia.
- **A (Antibiotics):** Consider prophylactic antibiotics for open or contaminated wounds.
- **W (Wounds):** Thorough assessment and temporary management of injuries.
- **S (Splinting):** Stabilize suspected fractures or spinal injuries.

Adopting the MARCH-PAWS protocol, specifically prioritizing rapid bleeding control, substantially improves outcomes by preventing ongoing hemorrhage, accelerating stabilization, and guiding timely and targeted interventions.

Severe cases commonly present with hypotension (systolic blood pressure <90 mmHg), signaling progression from compensated shock (in which vital organ perfusion is initially maintained through compensatory mechanisms) to decompensated shock, marked by overt circulatory failure. However, it is crucial to recognize that direct blood pressure measurement is not always necessary to diagnose shock; clinical signs alone (such as altered mentation, prolonged CRT, weak pulses, and cold extremities) can be sufficient for immediate recognition and initiation of therapy. Additionally, calculating the shock index (heart rate divided by systolic blood pressure) provides a valuable tool to quantify severity, identify patients at risk of rapid deterioration, and dynamically monitor therapeutic responses.

Laboratory diagnostics significantly enhance clinical assessments by providing objective measures of shock severity and response to treatment. Serum lactate (>2.5 mmol/L) and negative base excess values correlate closely with shock severity and patient prognosis. Serial lactate measurements, in particular, provide valuable feedback on the efficacy of therapeutic interventions and the status of resuscitation. However, while these laboratory parameters are beneficial for assessing the extent of shock and evaluating treatment response, they are not mandatory for the initial clinical diagnosis and immediate management decisions, which should be driven by clinical signs.

During the initial stages of hemorrhagic shock, compensatory vasoconstriction dominates to preserve central organ perfusion. However, during resuscitation, especially with restoration of blood flow, patients may experience a paradoxical shift toward vasodilation because of ischemia-reperfusion injury and the release of inflammatory mediators. Clinically, this transition can be recognized by persistent hypotension despite adequate fluid administration, a widening pulse pressure, hyperemic mucous membranes, rapid capillary refill time, and warm extremities. Recognizing this transition promptly is critical, as it often necessitates adjusting resuscitation strategies, including the cautious use of vasopressor support to restore vascular tone and stabilize perfusion.

Point-of-Care Ultrasound (POCUS)

POCUS is indispensable for rapidly diagnosing and monitoring HS, especially when physical examination findings are ambiguous. Ultrasound can promptly identify abdominal or thoracic free fluid (hemoperitoneum, hemothorax), assess cardiac contractility, detect pericardial effusion, and estimate intravascular volume status. Assessment of intravascular volume using POCUS typically involves evaluating the caudal vena cava diameter and collapsibility. A small and highly collapsible caudal vena cava suggests significant hypovolemia, while a distended, minimally collapsible vessel indicates adequate or excessive fluid volume. Additionally, ultrasonographic evaluation of cardiac chambers can help detect underfilling or overdistension, further refining fluid therapy decisions. Serial POCUS facilitates real-time monitoring of fluid therapy effectiveness, rapid identification of ongoing hemorrhage, and guides adjustments in treatment strategy.

THERAPEUTIC STRATEGIES

Initial Hemorrhage Control

Immediate control of external bleeding via direct compression and the use of hemostatic dressings (e.g., combat gauze) is crucial. Non-compressible internal hemorrhages require early surgical intervention (e.g., resuscitative thoracotomy). Clinicians should promptly initiate surgical consultation or interventional procedures for refractory cases.

Fluid Resuscitation and Blood Product Therapy

Fluid therapy remains a cornerstone in managing HS; however, crystalloids should be administered judiciously to avoid potential damage to the endothelial glycocalyx and exacerbation of coagulopathy. While recent veterinary evidence supports controlled, balanced resuscitation with an emphasis on early administration of blood products in severe hemorrhage, clinicians must acknowledge practical limitations such as availability, logistics, and cost. In situations where blood products are initially unavailable or delayed, careful use of crystalloids to achieve initial stabilization can be appropriate, followed by prompt incorporation of blood products as soon as feasible to optimize outcomes.

Fluid Type	Examples	Pros	Cons
Isotonic Crystalloids	Lactated Ringer's Solution (LRS), 0.9% NaCl, Normosol-R	Readily available, inexpensive, easy to administer, suitable for initial volume expansion	May cause glycocalyx damage, dilutional coagulopathy, edema, and transient volume expansion
Hypertonic Saline (7.2%)	Hypertonic saline	Small volume needed; rapid volume expansion; beneficial in head trauma	Short-lived and limited effect; risk of hypernatremia; must follow with additional fluids
Synthetic Colloids	Hydroxyethyl starch (Hetastarch, VetStarch)	Longer intravascular retention; effective rapid volume expansion	Dose-dependent coagulopathy risk; potential for renal impairment; **not ideal in severe hemorrhage**
Fresh Whole Blood	Freshly collected blood	Ideal for massive hemorrhage; provides red cells, plasma, platelets, and coagulation factors	Short shelf life
Stored Whole Blood	Refrigerated whole blood	Provides red cells and plasma proteins	Reduced platelets
Packed Red Blood Cells	Red cells concentrate	Efficiently treats anemia	No coagulation factors or platelets
Fresh Frozen Plasma	Frozen plasma	Provides coagulation factors; effective for coagulopathy management	Does not restore oxygen-carrying capacity; requires thawing
Lyophilized Plasma	Freeze-dried plasma	Long shelf-life, rapid reconstitution, functional in emergencies	Limited availability; no RBCs
Autotransfusion	Collected the patient's blood (e.g., abdominal bleeding)	Immediate availability; reduced transfusion reaction risks	Risk of contamination/infection; requires careful collection

When managing HS, resuscitation with blood products fundamentally differs from routine transfusions, primarily in the urgency and rate of administration. Blood products during resuscitation are typically delivered rapidly, sometimes under pressure, to restore circulating volume and perfusion. This accelerated infusion can significantly increase citrate load (used as an anticoagulant in stored blood products), raising the risk of citrate toxicity and subsequent hypocalcemia. Citrate binds ionized calcium, potentially leading to impaired cardiac contractility, hypotension, and coagulopathy. <u>Therefore, ionized calcium levels should be closely monitored, and supplemental calcium gluconate (0.5–1 mL/kg IV slowly) or calcium chloride (0.2–0.3 mL/kg IV slowly) should be readily administered during rapid, high-volume blood resuscitation.</u>

Correction of Coagulopathy and Use of Antifibrinolytics
Trauma-induced coagulopathy (**TIC**) significantly complicates HS management due to its complex pathophysiology involving the interplay of acute blood loss, tissue injury, hypoperfusion, inflammation, and resuscitation efforts. Immediately following trauma, endothelial injury and tissue damage trigger systemic activation of the coagulation cascade, resulting in rapid consumption of platelets and coagulation factors (consumptive coagulopathy). Concurrently, hypoperfusion and shock induce endothelial glycocalyx shedding, promoting further inflammation and coagulopathy. Trauma and shock also alter fibrinolytic balance, initially causing hyperfibrinolysis that increases bleeding risk, which may subsequently transition to a state of fibrinolysis shutdown, associated with microvascular thrombosis and multiple organ failure. This dynamic coagulopathy complicates fluid and blood product resuscitation, necessitating careful and targeted management to restore hemostatic balance.
Tranexamic acid (TXA), an antifibrinolytic agent, is increasingly utilized in veterinary medicine based on favorable human clinical data.
Tranexamic Acid Dosing: Initial loading dose: 10–15 mg/kg IV slowly (over 10 min). Maintenance dose: 1–2 mg/kg/hr IV continuous infusion for 5–8 hours. TXA should ideally be administered within 3 hours of injury for maximal benefit.
Vasopressor Support
Persistent hypotension despite adequate volume resuscitation may necessitate vasopressor therapy.

Drug	Dose	Comments
Norepinephrine	0.05–0.5 μg/kg/min IV CRI	First-line agent; potent vasoconstrictor
Dopamine	5–15 μg/kg/min IV CRI	Alternative if norepinephrine is unavailable

ORGAN SUPPORT IN INTENSIVE CARE
Post-resuscitation organ support focuses on monitoring and preventing renal dysfunction, evolving coagulopathies, and metabolic derangements. Serial assessment of renal and liver functions, coagulation status, electrolytes, lactate, and acid-base status is fundamental in ongoing intensive care.
NCHECKLIST: KEY STEPS IN MANAGING HEMORRHAGIC SHOCK
- Rapid clinical recognition of shock (physical exam, bedside labs, POCUS)
- Control external bleeding immediately
- Judicious crystalloid resuscitation; prioritize early blood products
- Consider early tranexamic acid (TXA)
- Monitor and supplement calcium levels
- Consider vasopressor therapy if refractory hypotension
- Discuss surgical or interventional management early for uncontrolled hemorrhage
- Intensive monitoring (renal, coagulation, metabolic) post-resuscitation
- Educate owners on prognosis and intensive care expectations

SUMMARY AND CLINICAL PEARLS
Hemorrhagic shock management in veterinary medicine has evolved significantly, now emphasizing targeted resuscitation, rapid diagnosis, and tailored supportive care. Incorporating advanced bedside diagnostics (POCUS) and balanced transfusion strategies significantly enhances patient outcomes. Familiarity with cutting-edge interventions and evidence-based strategies ensures optimal management and improved survival in veterinary hemorrhagic shock patients.

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