Prehospital Anemia Care

A Review of Symptoms, Evaluation, and Management

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ABSTRACT

The ongoing evolution of prehospital medical care continues to advance beyond immediate triage care. Prehospital care is even more important to consider in theaters with extended evacuation times and limited local medical assets. Although blood loss is often associated with settings of acute traumatic hemorrhage in military medicine, the possibility for other hematology compromise necessitating urgent action requires medics operating in these environments to have a fundamental knowledge of the pathophysiology, manifestations, and stabilization measures of anemia to aid their patients prior to, or in lieu of, evacuation. Continued development of and access to point-of-care testing in increasingly forward-deployed settings further enable medics to perform these tasks. Here, we provide a brief review of hemoglobin function and composition, and presentation and management considerations of anemia, to assist medics in their treatment efforts. We also address specific concerns for battlefield and atraumatic presentations.

KEYWORDS: hemoglobin; anemia; prehospital care; blood loss, hemorrhage; military; laboratory; malaria; hemolysis; bleeding; transfusion

Introduction

Scenarios to consider: A 30-year-old male is carried into your aid station in remote Africa by his teammates concerned by his complaints of dizziness throughout the day. They state that he has been sick recently and was telling people he has been sweating more than usual at night. There has been a significant increase in mosquitos because of increased rain 1 week ago. You have an i-STAT handheld blood analyzer (Abbott) at your disposal among some other basic laboratory testing equipment. What are your concerns? What tests would help you evaluate this patient?

A 26-year-old female comes into your aid station complaining of ongoing vaginal bleeding, with a longer and heavier than usual menstrual cycle that is now on its ninth day. She denies any history of similar events or known bleeding disorders. She is pale, and you notice that her heart rate of 120 beats per minute is not appropriate for an otherwise healthy female who just walked 100 meters to your aid station. As your junior medic prepares to draw a small sample of blood, you try to remember which important laboratory cut-offs are relevant for this patient and how you might provide treatment.

Anemia broadly describes a variety of conditions with decreased proportions of functional erythrocytes, more often referred to as red blood cells (RBCs). This is diagnostically measured by hemoglobin concentration in blood samples. Although the World Health Organization criteria provide a diagnostic threshold for anemia with a hemoglobin level of <12g/dL in adult females and of <13g/dL in adult males, this definition is not used consistently throughout the literature and is inconsistent between demographic groups. These variations demonstrate the need for anemia to be viewed as part of a broader patient assessment for optimal care.

Anemia affects up to one-third of the global population, although prevalence may vary significantly because of geography, age, and gender. Civilian frequencies range widely from 9% in pediatric populations to as high as 47% in geriatric patients, whereas military studies find rates between 12% and 20% in active-duty personnel.

Stimulated by increased demand for oxygenation, renal tubular cells secrete erythropoietin to drive RBC development. This signal triggers bone marrow differentiation, with normoblasts expelling their nuclei during maturation to become reticulocytes, then losing their ribosomal networks to ultimately become functional RBCs approximately 3–4 days later. A healthy RBC averages a circulation of approximately 120 days, after which a natural degradation cycle occurs, eventually leading to destruction by the immune system via the liver and spleen. Approximately 0.8% of all circulating RBCs are destroyed in this process every day, releasing biliverdin (later converted into bilirubin) and free iron. Inside each healthy RBC, four hemoglobin chains bind, exchange, and transport oxygen and carbon dioxide via the iron-containing heme ring complex.

Both hereditary and environmental factors can affect the normal function of hemoglobin and subsequently impair the...
overall oxygen-carrying capacity of RBCs. Hereditary factors include methemoglobinemia and the hemoglobin S variant, the latter of which is present in sickle cell disease. Hemoglobin structural alterations underlie these pathologies, referred to as hemoglobinopathies, and affect not only the oxygen-carrying capacity but also RBC lifespan and tendency toward destruction (i.e., hemolysis). Environmental factors may likewise worsen hereditary disease or alone cause hemoglobinopathy. Examples include sulfhemoglobinemia (i.e., exposure to sulfa), cyanohemoglobinemia (i.e., exposure to cyanide), and carbon monoxide toxicity.1–4,21 Hemoglobinopathy can compromise tissue oxygenation and trigger compensatory responses, such as increased cardiac output and vasoconstriction.1,19 A rightward shift in the oxyhemoglobin dissociation curve increases oxygen release from circulating hemoglobin to supplement tissue oxygenation.9,22 If anemia continues over several weeks or longer, hematologic adaptations occur, such as increasing plasma volume to offset lost intravascular mass and erythropoietin surges to promote RBC production.1,9,22 Increased RBC turnover and a hastened production cycle increase the proportion of circulating immature reticulocytes.1,18,22,24 These physiologic adaptations are important to remember during the initial evaluation because they can prevent compensatory physiologic responses, concealing outward signs and symptoms classically associated with anemia.2,3,6,22

**Classification**

Evaluation of anemia in the forward-deployed setting necessitates a foundational understanding of acute and chronic causes in relation to overall patient stability.1–3,24 Ranges for acute and chronic, often used to describe medical conditions, are not equally well-defined in anemia but rather are broadly used in patient evaluations.

Acute anemia may be most obviously caused by hemorrhage in settings of severe battlefield injury, but occult hemorrhage can occur through numerous pathways. Concerns for these underlying causes are most important in the unstable patient without obvious hemorrhage. Conversely, those with obvious signs of traumatic hemorrhage may not have anemia on initial evaluation because this is a measurement of hemoglobin concentration and not of overall loss. A lack of anemia on initial laboratory evaluation should not necessarily reassure medics.

Every effort should be made to search for hemorrhage in the acutely anemic patient, especially with traumatic presentations, but the atraumatic onset of anemia should prompt consideration for less obvious sources of bleeding. Examples include occult gastrointestinal bleeding from the use of nonsteroidal anti-inflammatory drugs (NSAIDs) or poorly controlled acid reflux or bleeding from the genitourinary system.1,3 Other causes without obvious blood loss can be less apparent on initial evaluation and include sickle cell disease with aplastic crisis, disseminated intravascular coagulopathy (DIC), thrombotic thrombocytopenic purpura (TTP), and hemolytic uremic syndrome (HUS).1,3,20,21 Although most current prehospital guidelines do not advocate for the use of crystalloids in trauma patients, in the setting of triage interventions for unstable vital signs, dilutional anemia should be considered if the patient received crystalloid infusions prior to laboratory evaluation.1,21

Chronic anemia can encompass decreased RBC production, increased RBC destruction, or a combination of both, and in some settings the diagnosis may be found incidentally. An effort should be made to correlate low hemoglobin values with patient history, physical examination, and other laboratory findings, when available, to identify an underlying cause. Although laboratory values such as mean corpuscular volume (MCV) and RBC distribution width (RDW) can narrow possible causes of anemia, these may be difficult to obtain in the austere setting.1–3,21

**Patient Presentation and Assessment**

Initial assessment in suspected or confirmed cases of anemia immediately focuses on hemodynamic stability and the search for possible sources of gross hemorrhage. Priority should be made for stabilization in all patients, with or without significant hemorrhage.1–4,26,27 It is important to note, however, that RBCs can carry up to four times the oxygen required for the body at rest and therefore can compensate for hypoxemia, precluding tachypnea or tachycardia.1,22,26 Stable vital signs should not necessarily reassure medics.1,22,26 When vital signs are unstable in suspected anemia, medics should refrain from reflexive fluid resuscitation with crystalloids or other non-blood products because military and civilian trauma literature continues to illustrate the harmful effect of dilution.28–30

In the anemic patient, medics should consider all possible sources of recent or ongoing hemorrhage, including the nose (epistaxis), lungs (hemoptysis), gastrointestinal tract (hematemesis, hematochezia, melena), and genitourinary (hematuria) systems, whereas others may be based on patient demographics, such as ectopic pregnancy in women of childbearing age.1,3,31 A thorough surgical and medical history may suggest other conditions indicating proneness to bleeding, including peptic ulcer disease, Crohn’s disease, and ulcerative colitis.32 Similarly, acute hemolytic anemia may be triggered by recent illness, often with strongly suggestive symptoms (Table 1).

<table>
<thead>
<tr>
<th>Antibiotics</th>
<th>Analgesics/Antipyretics</th>
<th>Other Medications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amoxicillin</td>
<td>Acetaminophen</td>
<td>Chlorpromazine</td>
</tr>
<tr>
<td>Cephalexin</td>
<td>Aspirin</td>
<td>Furosemide</td>
</tr>
<tr>
<td>Ciprofloxacin</td>
<td>Ibuprofen</td>
<td>Hydrochlorothiazide</td>
</tr>
<tr>
<td>Levofloxacin</td>
<td>Naproxen</td>
<td>Methotrexate</td>
</tr>
<tr>
<td>Penicillin</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trimethoprim/</td>
<td></td>
<td></td>
</tr>
<tr>
<td>sulframethoxazole</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

If able, patients may recall relevant family history suggestive of such hereditary hemoglobinopathies as sickle cell disease or glucose-6-phosphate deficiency (G6PD). Suspicion for G6PD can increase with a known medication history of drugs that can trigger this condition, including common antibiotics such as nitrofurantoin (used in urinary tract infections) as well as such sulfa-based medications as trimethoprim-sulamethoxazole, which are the most common causes of acute hemolytic anemia in patients with G6PD.33–35 These drugs, as well as other medications, including NSAIDs, angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, the antimalarial prophylaxis drug primaquine, and cephalosporins (Table 1), can independently cause drug-induced immune hemolytic anemia.35–38 Military members are now screened for sickle cell disease, but the condition can still present in contracting personnel and local civilians who present for treatment.
Acute or chronically anemic patients can present with a spectrum of symptoms, including dyspnea, weakness, fatigue, irritability, and headache, and most will not demonstrate any visible evidence of active bleeding.1–3,20 Many symptoms may not manifest until hemoglobin levels fall below 7g/dL, given potential compensatory mechanisms, although there is sparse literature to directly correlate laboratory values and symptom onset.1–3,20 Patients with chronic anemia may have significantly lower hemoglobin levels with only mild symptoms, given gradual rates of decline and ongoing adaptations.

Initial findings of the acutely anemic patient may reveal hemodynamic instability, including hypotension, tachycardia, tachypnea, and even hypoxemia.1,20,26,27 The presence of fever and considerations of anemia should prompt concerns for malaria in endemic regions, other infections, or such acute hemolytic anemias as DIC, HUS, or TTP.19 Additional signs and symptoms include decreased urine output, increased thirst, and altered mental status. Vital sign changes in anemia are inconsistent and can change with patient demographics (primarily age), as well as possible comorbidities and medication usage. Pediatric patients may have delayed vital sign changes.1,20 Older patients do not consistently demonstrate compensatory responses, and the use of such medications as beta-blockers may prevent tachycardia.1,6,20 Outward findings may include pallor, scleral icterus, jaundice, and petechiae (Figure 1), the latter of which is concerning for DIC and TTP.1,20,21 Abnormal enlargement of the spleen (splenomegaly), liver (hepatomegaly), thyroid (thryomegaly), and lymph nodes (lymphadenopathy) may be noted, with or without associated tenderness, suggesting idiopathic hemolysis or a malignant process.1,2,20 Auscultation of the chest may reveal a cardiac murmur or inspiratory crackles concerning for pulmonary hemorrhage.1,3,20 Any unexplained joint swelling and/or tenderness should be scrutinized for possible hemarthrosis. Evaluation of all suspected and confirmed anemic patients should include rectal examination for gross blood or melena indicative of a gastrointestinal bleed.1,20

**FIGURE 1** Possible outward signs of hemopathies involving anemia. (A) Scleral icterus and jaundice. (B) Petechial rash.

Images obtained through open access at:
https://commons.wikimedia.org/wiki/File:Scleral_Icterus.jpg
and https://commons.wikimedia.org/wiki/File:Petechial_rash.JPG.

**Laboratory Evaluation**

While medical history and physical examination may suggest anemia, diagnosis relies on laboratory testing, which in most settings comes from hemoglobin levels as part of a complete blood count panel. However, complete testing may not be available in forward-deployed settings, depending on ancillary medical support, and hemoglobin levels may be available only through an iSTAT or similar point-of-care testing device, limiting laboratory evaluation.40 Furthermore, in the setting of acute hemorrhage, initial testing may not demonstrate anemia; therefore, repeat testing should be performed as indicated with vital sign changes or following resuscitation measures.1 If finger prick samples are used for point-of-care testing, medics must remember that inaccuracies may occur because of decreased capillary flow in cold or shock patients, or simple dilution from interstitial fluid shifts.41

Other indices found on traditional laboratory evaluation, such as hematocrit and RBC values, are derived from the hemoglobin concentration.1,4,20 If available, this information can further characterize the anemia and help narrow its etiology, primarily through the use of RDW and MCV. The MCV can help delineate anemia between microcytic, normocytic, and macrocytic classifications, thus narrowing etiologies (Table 2). Overlap can occur between these groups, especially with iron-deficiency anemia (IDA). Although MCV values may not be available in an austere setting, differential diagnoses based on these levels are discussed here for further consideration in the setting of the atraumatic anemia patient in an austere setting. Despite these expected limitations, the traditional differential for causes of anemia will be briefly discussed.

**TABLE 2 Differential for Anemia by Mean Corpuscular Volume**

<table>
<thead>
<tr>
<th>Microcytic (MCV &lt;80fL)</th>
<th>Normocytic (MCV = 80–100fL)</th>
<th>Macrocytic (MCV &gt;100fL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Iron deficiency anemia</td>
<td>Iron deficiency anemia</td>
<td>B12/folate deficiency</td>
</tr>
<tr>
<td>Anemia of chronic</td>
<td>Anemia Bleeding/hemorrhage</td>
<td>Alchoholism</td>
</tr>
<tr>
<td>disease</td>
<td>Anemia of chronic</td>
<td></td>
</tr>
<tr>
<td>disease</td>
<td>disease</td>
<td></td>
</tr>
<tr>
<td>Thalassemia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sideroblastic anemia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Zinc abnormalities</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Myelodysplastic</td>
<td>Chronic renal</td>
<td>Hypothyroidism</td>
</tr>
<tr>
<td>syndrome</td>
<td>insufficiency</td>
<td>Liver disease</td>
</tr>
<tr>
<td>MAHA</td>
<td>Sickle cell disease</td>
<td>Medications</td>
</tr>
<tr>
<td>Spherocytosis</td>
<td></td>
<td>(hydroxyurea, methotrexate)</td>
</tr>
</tbody>
</table>

MAHA, microangiopathic hemolytic anemia; MCV, mean corpuscular volume.

Microcytic anemia (MCV <80fL) is most commonly the result of IDA, which can result from poor dietary intake or chronic RBC loss and can be found among deployed service members.2,22,43 Low ferritin levels and transferrin saturation can support an IDA diagnosis.1 However, this value is not a prerequisite for IDA, and ferritin testing is likely unavailable in most forward-deployed settings, including small host-nation facilities.1,2,22,44 Increased RDW may be the initial sign of microcytic anemia caused by iron depletion, whatever the ferritin values.2,3 Regardless of supporting findings, a suspicion for IDA should prompt thorough evaluation for occult bleeding, including gastrointestinal.20,44 An increased reticulocyte count suggests thalassemia, whereas a low or normal reticulocyte count may indicate IDA, anemia of chronic disease (ACD), sideroblastic anemias, or other etiologies.

Normocytic anemia (MCV, 80–100fL) can be further differentiated through other laboratory values, including reticulocyte counts and RDW. Normocytic anemia with a normal RDW can prompt consideration for renal failure and ACD.1 Normocytic anemia may occur as a result of several emergent causes of hemolysis, such as HUS or DIC, as well as acute
hemorrhage without proper marrow response. Although unlikely in active-duty populations, renal causes can be suspected in nonmilitary personnel with known or suspected chronic kidney disease, especially when renal function testing reveals a glomerular filtration rate of <30mL/min. High reticulocyte counts in the setting of normocytic anemia should prompt a Coombs test for further evaluation because high counts may indicate acute microcytic hemolytic anemia resulting from RBC membrane defects.21

Macrocytic anemia (MCV >100fL) is generally classified into nonmegaloblastic and megaloblastic anemia, the latter describing an enlarged, oval erythroblast with an immature, “lacy” appearing nucleus.1,46,47 Most macrocytic anemias are megaloblastic, usually caused by vitamin deficiencies of B12 and/or folate.1,46,47 Megaloblastic anemia may be of initial concern with a patient history of nutritional imbalance, possibly with vegetarian regimens, or in cases of chronic alcohol use.46,48 Low laboratory values for B12 and folate can support the diagnosis, but measured vitamin levels can rebound quickly with acute dietary or supplemental changes and therefore should not be considered definitive in evaluation.1,46 Acute toxins or drugs (most commonly alcohol) are more likely to cause a nonmegaloblastic anemia; thus, further evaluation should include a detailed history of medications such as hydroxyurea (used in sickle cell anemia) and methotrexate (used in rheumatoid arthritis).47 Ancillary tests may help directly identify the source of anemia based on RBC morphology. This is most important for operational environments such as sub-Saharan Africa and parts of Asia, where malaria is endemic.49-53 BinaxNOW (Abbott Diagnostics) is a simple point-of-care test to identify malarial infection and distinguish between types of malarial parasites for targeted therapy.53 Thick and thin smears enable direct microscopic examination for malarial parasites or sickled RBCs; however, their clinical value has been debated.39,42,44,55 Nevertheless, this testing is still recommended to aid the evaluation of the undifferentiated sick and anemic patient.4,39,42

<1>Management

Initial management of anemia focuses on control of any active bleeding to halt preventable blood loss and improve tissue perfusion and hemodynamic status. This may include the use of supplemental oxygen when hypoxemia is present. Importantly, medics should continually assess for indications for blood product transfusion. Resuscitative fluids other than blood, such as crystalloids and colloids, should be restricted, given concerns for hemodilution and coagulopathy.26,29,30

Unstable patients with suspected or confirmed hemorrhage require immediate pressure on exposed sites, with tourniquet application on affected extremities. Whole blood (WB) transfusion is warranted in these cases, and if prestored supplies are lacking, a walking blood bank should be initiated.29,30 In hospital settings, emergency-release type O-negative is ideal for women of reproductive age (to prevent sensitization and future pregnancy complications) and O-positive in all other populations.1 Operational situations should consider the use of low-titer group O whole blood (LTOWB) or group-specific matching if LTOWB is unavailable.29 In suspected or visualized internal hemorrhage (e.g., hematemesis, hematochezia, melena), proper surgical consultation is needed in preparation for evacuation.

Blood transfusion goals center on the need to support tissue oxygenation through improved hemoglobin concentration. A single unit of packed red blood cells increases hemoglobin concentrations by approximately 1g/dL, whereas a unit of WB increases levels by 2g/dL and adds additional platelets and coagulation factors.1,26,56 Combat literature recommends assessing hemoglobin levels every hour in the hemorrhagic patient during transfusion efforts until a concentration of 8g/dL is reached, then every 6 hours thereafter.39

Although blood products can improve anemia and coagulopathy in the hemorrhagic patient, there are several limitations, particularly preservative mechanisms that bind serum calcium to prevent clotting during storage.29 Transfused WB lacks calcium, needed for the coagulation pathway and healthy cardiac function.29,57 In the hemorrhagic patient, medics should therefore give supplemental calcium, generally through calcium gluconate, with WB to further correct coagulopathy.29,57 Additionally, tranexamic acid (TXA) should be considered for additional coagulation assistance for those receiving care within 3 hours of the injury in the setting of trauma.29,30 TXA should also be considered in suspected or confirmed atraumatic hemorrhage, including gastrointestinal and reproductive (i.e., uterine) sources.58-60

Any infectious cause of anemia should be addressed immediately with specific therapies targeting the underlying cause. These include conditions that provoke acute hemolytic anemia, such as DIC, HUS, and TTP (Table 3). Additionally, when malaria is suspected (endemic region, presence of fever, preferably with confirmation from BinaxNOW and/or peripheral smears), antimalarial treatment should be initiated immediately. This includes atovaquone/proguanil (Malarone) or artesether/lumefantrine (Coartem).39 Blood transfusion should be considered in patients with hemoglobin concentrations of <7g/dL.

Patients without an obvious source of hemorrhage who are clinically stable (i.e., normal vital signs, non–ill appearing) despite anemia should be further investigated for a cause through a focused history and evaluation prior to intervention. Some anemic patients who are hemodynamically stable may not require further emergent evaluation or management.1,20 Historically, universal transfusion thresholds for the nonhemorrhagic patient held indications at a hemoglobin level of <10g/dL; however, these have shifted with growing literature for a more restrictive strategy.26,27,61 A hemoglobin threshold of 7g/dL is supported by multiple trials involving acute illnesses such as sepsis and gastrointestinal bleeding.62-65

Those who are incidentally anemic but otherwise stable may be managed with close follow-up when they are otherwise healthy and the hemoglobin level is >6g/dL.1,66 This will likely require medical evacuation from austere locations, and priority should be based on overall clinical presentation, including hemodynamic stability. When the medic is suspicious for IDA, daily supplementation with 300mg tabs of ferrous sulfate should be considered, along with vitamin C, which increases absorption.44,66 Generic multivitamins often contain iron, but not at these levels, and patients should not attempt to compensate by simply increasing the vitamin dosing regimen because doing so can cause toxicity from other ingredients. In hospital settings, intravenous iron formulations can markedly help with IDA, lifting hemoglobin levels 2 to 3g/dL within weeks of
**TABLE 3  Acute Hemolytic Anemia: Presentation and Management**

<table>
<thead>
<tr>
<th>Cause</th>
<th>Presentation</th>
<th>Supporting Evidence</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>DIC</td>
<td>History of underlying cause: sepsis, trauma, malignancy, heat stroke, liver disease, Fever, Petechiae/purpura, Bleeding</td>
<td>Normocytic anemia, Prolonged PT/aPTT, Elevated D-dimer, Thrombocytopenia, Low fibrinogen</td>
<td>Evaluate and treat underlying cause; Platelet transfusion (if &lt;50,000/mm³); Consider TXA if active bleeding; Consider FFP, cryoprecipitate transfusion</td>
</tr>
<tr>
<td>HUS</td>
<td>Bloody diarrhea, Abdominal pain, Oliguria</td>
<td>Elevated SCr level, Thrombocytopenia, Schistocytes on peripheral smear, Normal coagulation panel</td>
<td>Supportive care; Dialysis may be needed; Avoid antibiotics</td>
</tr>
<tr>
<td>TTP</td>
<td>History of ADAMTS13 deficiency, History of HIV, pregnancy, use of acyclovir, clopidogrel, quinine Fever, CNS abnormalities, Oliguria, GI bleed</td>
<td>Elevated SCr level, Thrombocytopenia, Schistocytes on peripheral smear, Normal coagulation panel</td>
<td>Plasma exchange; Consider FFP while awaiting plasma exchange</td>
</tr>
<tr>
<td>AIHA</td>
<td>Provoking medications: penicillin, cefalosporins, NSAIDs, hydrocortisone, isoniazid</td>
<td>Positive direct antiglobulin test, Spherocytes on peripheral smear, Normal coagulation panel</td>
<td>Corticosteroids: IV methylprednisolone 100-200mg divided over 24 h; Oral prednisone 60-100mg; Schistocytes causes</td>
</tr>
</tbody>
</table>

AIHA, autoimmune hemolytic anemia; APTT, activated partial thromboplastin time; CNS, central nervous system; DIC, disseminated intravascular coagulopathy; FFP, fresh-frozen plasma; GI, gastrointestinal; HIV, human immunodeficiency virus; HUS, hemolytic uremic syndrome; IV, intravenous; NSAIDs, nonsteroidal anti-inflammatory drugs; PT, prothrombin time; SCr, serum creatinine; TTP, thrombotic thrombocytopenic purpura; TXA, tranexamic acid.

administration, although their availability in local host-nation facilities may be limited.1,4,6,7,8 If intravenous iron is given, oral supplementation is still recommended for continued treatment of IDA.4,4

**Conclusion**

The prevalence of anemia in military populations and possible acute complications from trauma and disease necessitate the medic’s understanding to properly assess and treat causes. Despite their availability, the limitations of point-of-care testing devices to differentiate anemia beyond diagnostic hemoglobin concentrations force medics to understand anemia pathologies and initial stabilization and care.

**Disclaimer**

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**Authorship and Contributors Statement**

All authors conceived the review concept, wrote the first draft, and read, provided critical revisions for, and approved the final manuscript.

**Conflicts of Interest**

The authors have no conflicts of interest or relevant disclosures to report.

**Funding**

We received no funding for this research.

**References**


Inside this Issue:

- FEATURE ARTICLES: TCCC Maritime Scenario: Shipboard Missile Strike
- 20th SFG(A) Non-Trauma Module (NTM) Course
- Training Collaboration With a Medical School
- Assessing Body Composition Using Kinanthropometry
- CRITICAL CARE MEDICINE: The JSOM Critical Care Supplement
- Austere Crush Injury Management
- Analgesia and Sedation in the Prehospital Setting
- Prehospital Traumatic Brain Injury Management
- Shock and Vasopressors
- Prehospital Anemia Care
- Prehospital Treatment of Thrombocytopenia
- Prehospital Electrolyte Care
- Pathophysiology and Treatment of Burns
- Noninvasive Positive Pressure Ventilation
- Mechanical Ventilation
- Acute Lung Injury and ARDS
- Traumatic Coagulopathy: Prehospital Provider Review
- Prehospital Critical Care
- Pediatric Sepsis in the Austere Setting
- LETTER TO THE EDITOR: Arctic Tactical Combat Casualty Care
- ONGOING SERIES: Injury Prevention, Psychological Performance, There I Was, TCCC Updates, Book Review, and more!