

# EMERGENCY CARE ISSUES IN HEMOPHILIA

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Treatment of Hemophilia Monographs  
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# Emergency Care Issues in Hemophilia

W. Keith Hoots

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## Introduction

Hemophilia-associated hemorrhage is preventable if clotting factor replacement therapy is infused to achieve and maintain normal physiologic levels of factor VIII (FVIII) or factor IX (FIX) respectively for hemophilia A or B. However, such dosing is achieved rarely – essentially only in perioperative hemostatic management to avoid bleeding following surgery. Even the well-established practice of infusing FVIII or FIX prophylactically to prevent spontaneous hemarthrosis uses dosing strategies insufficient to prevent all potential trauma-induced hemorrhages. Therefore, all individuals with hemophilia are, under very adverse circumstances, at risk for severe bleeding that may quickly evolve to life- or limb-threatening circumstances requiring emergency care. This monograph discusses a number of potential emergent hemorrhagic events, focusing on therapeutic strategies to reduce the risk of adverse outcomes, such as neurological dysfunction or even death.

## Central nervous system hemorrhage

Possibly the most frequently encountered emergent hemorrhagic event in hemophilia management is central nervous system (CNS) bleeding. Most of these events, which involve bleeding inside the skull or spinal canal, are caused by trauma. However, since patients with hemophilia can experience bleeding even weeks after a minor head injury, a history of head trauma may be hard to determine, particularly in children [1, 2]. Spontaneous CNS bleeding in individuals with hemophilia is rare except when there has been a recent antecedent CNS hemorrhage (i.e., a recurrent bleed at a previously injured site) or when there is an associated anatomic lesion that predisposes acute hemorrhage (e.g., aneurysm or arterio-venous malformation). Data from the Universal Data Collection Project of the U.S. Centers for Disease Control and Prevention indicate that predisposing risk factors for intracranial hemorrhage include HIV infection, presence of inhibitor, and age less than five years or over 51 years [3]. The risk of traumatic events affects the entire spectrum of

hemophilia phenotypes. For people with mild hemophilia, risk of intracranial hemorrhage is at least 50% that of those with severe disease.

## Intracranial hemorrhage

Bleeding inside either the **calvarium** or the spinal canal is acutely harmful to the tissues of the nervous system: the expanding hematoma quickly compresses and damages the tissues. This “space-occupying effect” impinging on the nervous system can occur even when the total volume of the hemorrhage is small because there is no space to accommodate the blood. It is therefore absolutely essential to immediately raise the clotting factor level, even before the bleed is documented via brain imaging studies, such as a computed tomography (CT) scan.

The site of intracranial CNS bleeding can be subdural, epidural, or **intraparenchymal**. All are capable of causing rapidly deteriorating CNS brain function, associated brain swelling, and in the most extreme circumstances, herniation of the brainstem and rapid death. If the bleeding is stopped with rapid clotting factor concentrate (CFC) replacement, adverse clinical effects can be avoided. However, with intraparenchymal hemorrhage, even small hemorrhages can induce permanent structural and/or neurologic sequelae (in particular, if the anatomic site of the bleed is essential for routine brain function) [4-8]. Because the degree of injury is both volume and duration dependent, at times the blood at surgically approachable sites (in particular subdural hematomas) should be evacuated through neurosurgical intervention [9]. This requires providing adequate CFC replacement both to stop the bleeding and for hemostatic coverage for the procedure itself. The strategy for hemostatic replacement during such scenarios will be discussed below. In addition to CFC replacement and neurosurgery, aggressive airway management is essential for comatose or incapacitated patients and may also be required as adjunctive management for injury-associated increased intracranial pressure [10].

Neonatal intracranial hemorrhage is most commonly due to birth trauma [11]. Difficult vaginal deliveries

(often requiring the application of forceps or vacuum extraction) are predisposing factors for intracranial hemorrhage in hemophilic newborns [12]. A large review of neonatal intracranial hemorrhage indicates an incidence of 3.58% [6]. Given that nearly one-third of new cases of severe hemophilia A represent new mutations (with no family history), all male newborns presenting with intracranial hemorrhage should have an activated partial thromboplastin time (aPTT) performed immediately and FVIII and FIX assays performed as soon as possible [11]. Suspicion of intracranial hemorrhage is call for an immediate ultrasound of the brain through the **fontanelle**.

### Spinal hematoma

Decompression surgery by **laminectomy** may be required at any age to treat hematomas compressing the spinal cord. However, early recognition of spinal hematomas and CFC replacement to normal physiologic levels (as with intracranial hemorrhage) may prevent the need for neurosurgery [13]. Furthermore, it helps avoid later spinal deformities in children [14]. Failure to relieve pressure during the first critical hours following a bleed will require surgery to reduce risk of paralysis of the nerves at or below the site of compression. As with intracranial bleeding, rapid replacement with CFC immediately after the bleed is essential to minimizing the volume of bleeding. Replacement therapy should be continued for a minimum of 10 to 14 days, even when laminectomy is not required [13]. Confirmation with imaging studies should await the initial infusion of high dose CFC. Once confirmed, any individual with neurological symptoms should be admitted to hospital, preferably to a critical care unit. Even when symptoms are not present, non-critical care hospitalization is necessary if imaging confirms a CNS hemorrhage.

### Dosing strategies for CNS hemorrhages

CFC dosing strategy to treat CNS hemorrhage in hemophilia A and B is based on three important principles:

1. Infuse enough to ensure a normal physiologic level initially (at least 100% within the normal range).
2. Infuse frequently enough to ensure that the FVIII or FIX level never falls to a non-physiologic level (i.e., a minimum of 50% with a 10-20% "cushion" for confidence).

3. Monitor FVIII or FIX levels as frequently as feasible to ensure that these physiologic levels are being achieved.

### FVIII and FIX dosing

For hemophilia A, this generally implies an initial acute dose of FVIII of approximately 50 IU/kg followed by repeated bolus dosing every 8-12 hours. The exact frequency depends on FVIII measurements. If neurosurgery is required, the second dose (and perhaps subsequent doses as well) will likely be needed much sooner because of high FVIII clearance during surgery. Correspondingly, increased FVIII monitoring will be needed for such clinical scenarios.

Factor IX dosing for hemophilia B likely needs to be product-type specific. For recombinant FIX (rFIX), a minimum initial dosing of 120 IU/kg with recurrent infusion every 18-24 hours adjusted as needed by FIX monitoring is a reasonable strategy. For high-purity plasma-derived FIX concentrates (monoclonal and others), 100 IU/kg per dose with similar frequency of administration (based on FIX plasma levels) is a reasonable regimen. For plasma-derived prothrombin complex concentrates (PCCs), which should be used in this setting only if rFIX or high purity plasma concentrates are not available, an initial dose of 100 IU/kg is reasonable—however, subsequent dosing will need to be significantly less because of the risk of acute thrombosis and **disseminated intravascular coagulation** associated with PCCs. Lower doses that target FIX levels below physiologic levels (i.e., below 50%) may be required to balance bleeding versus thrombotic risks. Laboratory monitoring of FIX levels, if available, is particularly important when PCCs are used and should include markers for disseminated intravascular coagulation and thrombosis (e.g., platelet count, prothrombin time, aPTT, fibrinogen, fibrin split products, D-dimers, etc.).

### Management of inhibitors

Other strategies for CNS hemorrhage in patients with hemophilia A or B, such as continuous infusion for replacement therapy, are discussed in the medical literature and provide viable management options [15]. Discussion of continuous infusion methodologies for dosing and monitoring are beyond the scope of this monograph. In addition, these CFC infusion recommendations make the critical assumption that no FVIII or FIX inhibitor is present. It is worth noting that initial venous access for CFC infusion in a patient

with a presumed CNS hemorrhage could well be used to draw blood for a FVIII or FIX inhibitor screen. In circumstances in which observed hemostatic effect is less than expected, having confirmation about inhibitor status can be most opportune.

When an inhibitor is known to be present in an individual with an acute CNS bleed (or any other life- or limb-threatening hemorrhage) alternative hemostatic replacement strategies will be needed. This can entail infusion of higher doses of FVIII or FIX or of so-called “bypassing agents” such as activated prothrombin concentrates (aPCCs) or recombinant factor VIIa (rFVIIa). An in-depth discussion of the appropriate use of these or other strategies to treat patients with CNS bleeding and a clinically significant inhibitor is not possible here. In many cases the literature on inhibitors in CNS hemorrhages consists of small series or case reports. Even so, the availability of aPCCs and rFVIIa has dramatically improved the potential for a good clinical outcome in inhibitor patients with a CNS hemorrhage. Aggressive hemostatic replacement using any or, in rare cases, all of these options appear justified since the morbidity and mortality secondary to uncontrolled CNS bleeding is very high.

#### *Duration of therapy*

The duration of CFC replacement therapy following CNS bleeding is less well-defined than dosing. The risk of recurrent bleeding in the days or early weeks following such a hemorrhage appears to be quite high, particularly when lower than normal physiologic levels of FVIII or FIX are permitted. Many investigators attempt to maintain circulating factor levels above 50% at all times up to three weeks following a CNS bleed in order to minimize the re-bleed risk. A longer duration may be needed if an extensive neurosurgical procedure was required to treat the patient. Furthermore, even after maintaining physiologic levels in the normal range for an extensive period, consideration needs to be given to slowly weaning the CFC replacement therapy. This may include a period of secondary prophylaxis if the patient is not being returned to a primary prophylaxis regimen. The duration of any secondary prophylaxis regimen will depend on many factors such as the availability of CFCs, severity of injury and associated conditions, patient willingness to adhere to the regimen, rehabilitative regimens, etc.

## Other emergent hemorrhagic events

### Tracheal hemorrhage

While CNS hemorrhage is likely the most frequently observed severe hemophilic trauma (due to falls in infants, injuries related to sports and activities in children and adolescents, and motor-vehicle injuries in adults), the injury state that may require the most rapid clinical therapeutic response is bleeding that is compressing or compromising the airway [16]. The time from the injury until the trachea is compressed may be long, sometimes many hours. However, once the compression is sufficient to cause difficulty breathing, the window for infusion to stop the bleeding may be very narrow. Failure to stop the bleeding at this point may necessitate a **tracheostomy** in order to prevent asphyxiation as compression well below the level of the larynx may not permit effective intubation, even in the most skilled hands. As such, bleed recognition is critical in these instances [17].

An acute neck injury or a **retropharyngeal** hemorrhage induced by dental or oral surgical instrumentation can lead to a dissecting facial plane hematoma. This in turn can sometimes lead to compression and acute airway compromise. Rarely, the antecedent bleeding may manifest as facial swelling [18]. Similarly, tongue swelling (e.g., caused by a hematoma of the lingual artery) [19] may signal potential airway compromise even when no obvious antecedent trauma has occurred. For **oral maxillofacial surgery**, adequate preoperative CFC replacement therapy is the key to preventing this complication [20]. Also the avoidance of such at-risk manipulations such as blocking the **inferior alveolar nerve** will forestall this dangerous outcome. However, for trauma to the neck, an expanding hematoma (usually from **cephalad to caudad**) may occur even with adequate preoperative CFC replacement—requiring immediate infusion to 100% normal plasma concentrations (1 IU/mL). In addition, immediate expert **otolaryngology** consultation should be arranged, with anesthesiology consultation if airway compromise is imminent. For stable individuals whose imaging studies indicate a potentially expanding hematoma, hospitalization in a critical care unit is prudent. If the hematoma causes compression and respiratory compromise, specialists should be consulted about surgical intervention.

Following the initial infusion of CFCs, repeat or continuous infusions are necessary until the neck swelling has resolved and any surgical manipulation is partially healed. The total duration will depend on individual circumstances. For any obviously dissecting hematoma, high intensity infusions maintaining physiologic levels initially with gradual weaning (as appropriate based on plasma levels, clinical findings, and imaging studies) will likely require hospitalization for a minimum of seven days. Follow-up CFC infusion after hospital discharge is prudent.

### Gastrointestinal hemorrhage

There is a wide spectrum of esophageal and gastrointestinal (GI) bleeding. A review of 41 episodes of gastrointestinal bleeding in one institution over 10 years implicated duodenal ulcer (22%) and gastritis (14%) as the most common source [21]. In 22%, no source was identified. **Mallory-Weiss Syndrome** has also been cited as a cause for upper GI bleeding in hemophilia patients [22].

For bleeding originating from the lower GI tract, mild **hematochezia** or **melena** typically necessitates a routine CFC replacement regimen followed by appropriate diagnostic work-up (typically endoscopy or colonoscopy) if the bleeding recurs after replacement therapy is withdrawn. This rarely gives rise to an emergent hemorrhagic scenario when the CFC replacement is commensurate with the severity and duration of the bleeding [21]. By contrast, profound melena or hematochezia sufficient to cause an acute drop in hemoglobin level is a hemostatic emergency. Rarely, melena and/or hematochezia in the newborn period can be the *de novo* presentation of hemophilia [23]. At the other end of the age spectrum, the adult hemophilia patient treated in the past before viral inactivation of CFCs may sometimes present with an emergent GI hemorrhage from esophageal varices associated with chronic hepatitis C with or without HIV co-infection [22, 24].

Since variceal bleeding is of itself not of coagulopathic origin, but rather vascular, in this case the hemostatic management is complementary to gastrointestinal intervention (e.g., **transjugular intrahepatic portosystemic shunt**) [24]. Nonetheless, CFC replacement is needed to ensure that instrumentation does not induce new bleeding sites in the hemophilia patient, and that once pressure issues related to the varices are addressed, post-instrumentation oozing is controlled.

Further, patients with hepatic cirrhosis and varices may also have coagulopathies caused by impaired synthesis of non-hemophilia-related protease deficiencies (e.g., deficiencies of factors VII, II, X). Together, these causes may lead to a very high hemorrhagic risk. Accordingly, replacement therapy with fresh-frozen plasma may have to accompany CFC replacement to achieve an adequate hemostatic outcome once the variceal bleeding is controlled.

For lower GI bleeding not related to varices, massive melena rarely has hemophilia as the primary etiology. Bleeding **telangiectasias**, ruptured abdominal arteriovenous malformations, or other aberrant vasculature may be an initiating event [25]. Just as with varice-related bleeding, replacement therapy with FVIII or FIX permits appropriate diagnostic studies to be done and, if required, necessary therapeutic procedures to be initiated. CFC replacement therapy is needed both pre- and post procedure. Duration of CFC infusion will be very dependent on the therapeutic regimen required and whether abdominal surgery is performed requiring either **laparoscopy** or **laparotomy**.

### Ruptured organ or capsular hematoma of an abdominal viscus

Blunt abdominal trauma is a frequently cited cause of an evolving **intraperitoneal** hemorrhage with the risk for massive blood loss and even **exsanguination**. However, there are cases reported of bleeding within a **viscus** evolving to organ rupture without history of trauma [26, 27]. Having hemophilia (particularly severe hemophilia) appears to increase the likelihood that physical impact—even of modest energy to the spleen, liver, kidneys, bowel wall, or pancreas—can induce peri-capsular hemorrhage that evolves into an expanding hematoma because of impaired hemostasis. In the instance of an evolving hematoma of the bowel wall, there may be the additional risk for gut obstruction [28].

Initially, abdominal pain (or abdominal distension in a newborn with hemophilia) [29] may be the only indication that such an evolving hemorrhage exists. A history of any abdominal trauma and new onset abdominal pain in a hemophilia patient should arouse suspicion. Failure to recognize an expanding hematoma and immediately and intensively initiate CFC replacement may result in acute rupture of the hematoma [25]. Such hematomas rarely occur in the absence of obvious trauma [30].



Immediate exsanguination is a very real risk when a large capsular hematoma of an abdominal viscus ruptures. Volume loss may be so severe that replacement with red blood cell concentrates and volume expanders may not be sufficient to maintain adequate circulation and CNS perfusion. In this latter scenario, immediate laparotomy is required to surgically remove the bleeding organ (spleen) [31, 32] or to repair it (liver, kidney, or bowel wall) [33]. On the other hand, if the infusion of CFCs sufficient to achieve physiologic levels is initiated before the hematoma expands, risks for intra- or **retroperitoneal** hemorrhage may be markedly diminished [33].

On occasion, retroperitoneal hemorrhage into the **psoas muscle** or **obturator muscle** can be severe enough to cause a significant drop in hemoglobin. As opposed to rupture of an abdominal organ or capsule, the bleeding will **tamponade** before life-threatening hemorrhage occurs. However, extreme pain and substantive impingement on the **sacral plexus** nerves can cause significant morbidity [34]. CFC replacement until symptoms decrease is the recommended therapy. Other interventions are rarely required.

A ruptured appendix in an individual with hemophilia may occur, as opposed to rupture of other abdominal viscera, without antecedent trauma [25]. (A preceding small bowel hematoma may even create “pseudo appendicitis” although modern imaging techniques should confirm this finding, avoiding the need for an unnecessary laparotomy [35].) Nonetheless, the need for preemptive replacement therapy is paramount, regardless of whether abdominal surgery is required when an acute abdominal event presents itself [36].

### **Compartment syndrome and nerve compression**

Just as CNS bleeding predisposes significant clinical neuronal damage from the space-occupying effect of blood in a confined volume, permanent debilitation can occur when the vascular and/or neural bundles that supply and drain an extremity (e.g., arm or leg) are extremely compressed by blood. Such nerve compression due to a closed space hemorrhage, known as “compartment syndrome,” can compromise limb function. Predisposing injuries include blunt trauma, **arterial cannulation** with **iatrogenic** bleeding from the puncture, and, particularly in neonates and young infants, perforation of large veins during venipuncture attempts. Initial swelling and

engorgement will, if bleeding persists, result in loss of pulse and cold extremities. In addition, **paresthesia** or **paresis** often accompany nerve compression.

Early recognition of significant bleeding based on swelling in an extremity compartment (e.g. forearm, wrist, or calf) is essential so that CFC infusion can be initiated or accelerated. In most instances, bleeding that is recognized early can be stopped before pulse and nerve function (indicated by peripheral paresthesia) are impaired. However, once the distal part of the extremity becomes paler and cooler than the opposite corresponding distal extremity, surgical intervention with **fasciotomy** may be the only way to preserve function of the extremity. Fasciotomies are difficult in these circumstances, and the associated perioperative bleeding may be severe even after “adequate” CFCs have been administered. Early recognition and intervention with appropriate doses (usually levels to achieve 1 IU/mL of plasma or 100% initially) will often forestall the need for fasciotomy.

### **Ophthalmologic emergencies**

An individual with blood in the eye’s anterior chamber or **vitreous humour** has an emergent ophthalmologic situation. In the context of hemophilia, the propensity not to clot increases the likelihood that any bleeding into the closed spaces of the eye will impair vision. This is similarly true for bleeding associated with retinal detachment, where the prospects for visual loss are likely increased in individuals with hemophilia. For any of these clinical ophthalmologic conditions, including **hyphema**, vitreous hemorrhage or even hemorrhagic glaucoma (whether diagnosed or suspected based on ophthalmoscopic exam), immediate infusion to 100% activity (1 IU/mL of plasma) is essential followed by emergency assessment by an ophthalmologist [37-39]. Whether or not surgery is recommended, hospitalization is indicated until the clinical situation is deemed stable enough for outpatient management by both the ophthalmologist and the hematologist [40].

### **Rare clinical emergencies**


Any bleeding situation that may require emergency management in an individual without a bleeding tendency is clearly compounded if the individual has hemophilia. An example is pericardial bleeding, which in people with hemophilia predisposes tamponade or pulmonary hemorrhage. Since surgical intervention is likely required, the patient with

hemophilia needs to be managed hematologically as though the intervention were a scheduled major surgery. After the initial CFC infusion immediately following diagnosis, subsequent infusions post-operatively should be similar to those described above for neurosurgical or laparotomy events. As in those situations, maintaining the patient in a state of physiologic clotting (normal FVIII or FIX levels) is essential until healing is well along.

### **Pseudotumour rupture**

While pseudotumours in patients with hemophilia are in themselves not hemophilic emergencies, an acute rupture of a large pseudotumour in the pelvis or thigh may lead to acute hypotension from blood loss. When such an event occurs, an immediate correction of the FVIII or FIX level is indicated, while keeping in mind that the presence of chronic clots with fibrinolysis in the pseudotumour may also predispose to further hemorrhage [41]. Laboratory assessment for systemic hyperfibrinolysis is typically indicated. Antifibrinolytic therapy with epsilon aminocaproic acid or tranexamic acid may be indicated to supplement CFC therapy if this complication is present.

### **Conclusion**

Emergent bleeding events, while uncommon among people with hemophilia, require recognition and immediate intervention with high-dose CFC infusion. Other care, such as surgery, may also need to be undertaken urgently. However, replacement with FVIII or FIX must occur first or in parallel with any intervention in a patient with hemophilia who is critically ill. Only removal of an acutely injurious agent or cardiopulmonary resuscitation supersedes factor replacement in a critically ill patient with hemophilia. Not only must enough CFC be infused initially to reduce or stop bleeding, it must be given as often as necessary to permit healing from the injury. Ideally measurement of the *in vivo* factor level on a frequent and ongoing basis is performed to ensure that the desired circulating plasma levels are maintained. Special management considerations relevant to the anatomic bleeding site may exist and contributions from experts in the respective medical field are essential for optimal clinical outcomes. 

## Appendices

**Table 1: High-risk hemorrhagic events for acute morbidity or mortality in hemophilia A and B**

**Central nervous system hemorrhage**

- Intracranial hemorrhage
- Spinal hematoma

**Soft-tissue hemorrhage predisposing to airway impingement**

- Retropharyngeal hemorrhage following mandibular molar extraction leading to hemorrhage along facial planes
- Neck hematoma associated with dissection
- Tracheal hemorrhage following airway instrumentation
- Large tongue hematoma

**Gastrointestinal bleeding**

- **Hematemesis** from esophageal injury or ulceration of gastric or duodenal mucosa
- Hemorrhage from ruptured esophageal varices
- Hematochezia or melena from bleeding telangiectasia polyps, etc.

**Ruptured abdominal organ or capsular hematoma of abdominal viscus**

- Splenic rupture, kidney capsular rupture, liver laceration
- Hematoma of bowel wall
- Ruptured appendix
- Ruptured pelvic or abdominal pseudotumour

**Acute compartment syndrome**

- Hematoma impingement of nerves, vasculature of extremities

**Hemorrhage in or around the eye**

- Hyphema
- Vitreous hemorrhage
- Hematoma following orbital fracture

**Table 2: Guidelines for acute management of severe hemorrhage in hemophilia A and B**

1. Assure adequate airway, breathing, and circulation by assessing respiration, pulse, and blood pressure (basic cardiopulmonary resuscitation guidelines).
2. Attain venous access as quickly as possible.
3. Infuse appropriate FVIII (hemophilia A) or FIX (hemophilia B) at a dose to achieve physiologic levels immediately (50 IU/kg body weight FVIII or 100-120 IU/kg high purity FIX respectively; 70-80 IU/kg of prothrombin complex concentrate if high purity FIX is unavailable).
4. Obtain CT scan, ultrasound, or other imaging studies as indicated to ascertain bleeding site/source.
5. Request consultation from appropriate physician consultant for bleeding site (e.g., ophthalmologist for bleeding in/around the eye).
6. Hospitalize.
7. Monitor FVIII/FIX levels respectively on a frequent basis to maintain level in the mid physiologic range.
8. Continue with frequent bolus or continuous clotting factor infusions adjusted according to measured FVIII or FIX plasma levels until the acute bleeding event has resolved. Dosing may be adjusted downward as the risk for further bleeding is substantially reduced.
9. Examine the patient following hospitalization to ensure any sequelae receive appropriate long-term care.

## Glossary

**Arterial cannulation:** The insertion of a tube into a vein, primarily for the administration of intravenous fluids and medicines, and used during major operations and in critical care areas to measure beat-to-beat blood pressure and to draw repeated blood samples.

**Calvarium:** Upper skull, roof of the skull.

**Cephalad to caudad:** From head to torso to lower extremities.

**Disseminated intravascular coagulation:** A process where the blood starts to coagulate throughout the whole body, depleting the body of its platelets and coagulation factors, with a paradoxically increased risk of hemorrhage.

**Exsanguination:** The fatal process of total blood loss, most commonly known as “bleeding to death.”

**Fasciotomy:** A surgical procedure where the fascia (soft part of connective tissue) is cut to relieve tension or pressure causing loss of circulation to an area of tissue or muscle. Fasciotomy is a limb-saving procedure when used to treat acute compartment syndrome.

**Fontanelle:** The “soft spot” between an infant’s incompletely formed cranial bones.

**Hematemesis:** The vomiting of blood, generally from the upper gastrointestinal tract.

**Hematochezia:** The passage of bright red, bloody stools from the rectum. It is commonly associated with lower gastrointestinal bleeding.

**Hyphema:** The collection of blood in the anterior chamber of the eye.

**Iatrogenic:** Brought on unintentionally by something that a doctor does or says.

**Inferior alveolar nerve:** A branch of the mandibular nerve.

**Intraparenchymal:** Within the functional parts of an organ. Intraparenchymal hemorrhage can be caused by brain trauma, or it can occur spontaneously in hemorrhagic stroke.

**Intraperitoneal:** Within the peritoneal cavity. The peritoneum is the serous membrane that forms the lining of the abdominal cavity.

**Laminectomy:** Surgical removal of part of the posterior arch of a vertebra to provide access to the spinal canal and relieve pressure on the spinal cord.

**Laparoscopy:** Minimally invasive surgery involving small incisions in the abdomen.

**Laparotomy:** A surgical procedure involving an incision through the abdominal wall to gain access into the abdominal cavity.

**Mallory-Weiss Syndrome:** Esophageal mucosal injury, with bleeding from tears in the mucosa at the junction of the stomach and esophagus, usually caused by severe retching, coughing, or vomiting.

**Melena:** Stool with blood that has been altered by the gut flora and appears black or tarry, associated with gastrointestinal hemorrhage.

**Obturator muscle:** The muscle extending from the pelvis to the femur.

**Oral maxillofacial surgery:** Surgery to correct injuries and defects in the head, neck, face, jaws, and the hard and soft tissues of the oral and maxillofacial region.

**Otolaryngology:** The branch of medicine that specializes in the diagnosis and treatment of ear, nose, throat, and head and neck disorders.

**Paresthesia:** Abnormal sensations of the skin such as numbness, tingling, or prickling.

**Paresis:** Impaired or partial loss of movement.

**Psoas muscle:** The muscle at the hip and groin, extending from the sides of the spinal column to the upper end of the femur.

**Retropharyngeal:** Behind the pharynx.

**Retroperitoneal:** Outside the peritoneum, behind the abdominal cavity.

**Sacral plexus:** A nerve plexus emerging from the sacral vertebrae that provides nerves for the pelvis and lower limbs.

**Tamponade:** Pathological compression of an organ, a condition of blood flow stoppage either by constriction of the vessel or by an outside force.

**Telangiectasias:** Small blood vessels near the skin surface.

**Tracheostomy:** A surgical procedure performed on the neck to open a direct airway through an incision in the trachea (windpipe).

**Transjugular intrahepatic portosystemic shunt:** An artificial channel in the liver from the portal vein to a hepatic vein (for blood), created by physicians via the jugular vein.

**Varices:** Distended veins.

**Viscus:** An internal organ, in particular of the thorax or abdomen.

**Vitreous humour:** The clear gel that fills the space between the lens and the retina of the eyeball.

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