Perioperative Implications

Preoperative Preparation

- Initiate prompt treatments with diphtheria antitoxin and antimicrobial therapy.
- Assessment of respiratory distress/airway compromise.
- Assessment of cardiac toxicity (for early detection of rhythm abnormalities. Initiate electrical pacing for clinically significant conduction disturbance and provide pharmacologic intervention for arrhythmias or for heart failure).
- Assessment of neurologic toxicity.
- Assessment of immunization status of exposed healthcare workers.

Monitoring

- Maintain close monitoring of cardiac activity for early detection of rhythm abnormalities.
- Provide two large-bore IVs for pts with a toxic appearance; provide invasive monitoring and aggressive resuscitation for pts with septicemia.
- Initiate electrical pacing for clinically significant conduction disturbance and provide pharmacologic intervention for arrhythmias or for heart failure.
- Consider PA cath/noninvasive cardiac output monitoring or transesophageal echocardiography to assess degree of myocardial involvement.

Airway

- Secure definite airway for pts with impending respiratory compromise or the presence of laryngeal membrane (careful manipulation as membrane will bleed if manipulated).
- Early airway management allows access for mechanical removal of tracheobronchial membranes and prevents the risk of sudden asphyxia through aspiration.

Induction and Maintenance

- Compensate for problems of exotoxin shock and possible CHF, as well as cardiac arrhythmia.

Extubation

- Early: May need prolonged ventilation.
- Late: Cardiogenic shock/extensive polyneuritis may necessitate prolonged ventilatory support.

Adjuvants

- Cardiac pacemaker for arrhythmia control/complete heart block.
- Minimize use of sedative-hypnotics because development of respiratory difficulties may be obscured.

Postoperative Period

- Careful observation for respiratory, cardiac, and neurologic compromises

Anticipated Problems/Concerns

- Airway obstruction requiring tracheostomy/intubation
- Myocardial conduction problems that may necessitate electrical pacing
- Cardiogenic shock/CHF
- Neuritis that can present as a Guillain-Barré-like syndrome

Disseminated Intravascular Coagulation

Adrian Hendrickse

Risk

- Most common coagulopathy in the ICU.
- 1% of all hospital admissions.
- Evidence of a coagulopathy in the DIC spectrum approaches 90% in cases of severe sepsis.
- The most important initiator of DIC is sepsis, along with trauma (hypovolemic shock, extensive tissue damage, fat embolism, head injury); surgery (neurosurgery, CPB); obstetric emergencies (hemorrhage, preeclampsia, retained products, amniotic fluid embolism); malignancy (acute promyelocytic leukemia, disseminated metastases); and severe liver disease. Vascular abnormalities, immunologic reactions, toxins, and drugs can also cause DIC.
- Mortality: Dependent on the underlying condition and the severity of the coagulopathy.

Perioperative Risks

- Existing coagulopathy
- Organ failure

Worry About

- Excessive bleeding from surgical and anesthetic access sites
- Organ dysfunction and the need for supportive measures
- Coordinating the management of the coagulopathy

Overview

- DIC is a syndrome characterized by the pathologic imbalance of the coagulation, anticoagulation, and fibrinolytic processes, leading to systemic intravascular thrombosis and the deposition of fibrin in the microcirculation. DIC exists as a spectrum of clotting disorders, the two ends being acute (life-threatening) and chronic (subclinical).
- Acute DIC exists when there is a rapid activation of the coagulation system resulting in the consumption of platelets and the depletion of clotting factors at a rate greater than the body can compensate for, which can lead to excessive hemorrhage. Chronic DIC is a slower affair where the rate of consumption of platelets and clotting factors can be compensated for and where the clinical picture is generally that of microvascular thrombosis.
- Dx: There are no specific laboratory tests for DIC. DIC can be diagnosed clinically on the basis of the presence of a suitable risk factor, along with a selection of laboratory findings: a rapidly falling platelet count or a count $<100{,}000/mm^3$; prolongation of clotting times (APTT, PTT, INR); the presence of FDPs; a reduction in plasma concentration of coagulation inhibitors (ATIII, protein C); TEG analysis of clot formation and lysis.
- Serial testing showing temporal trends are invaluable.

Etiology

- DIC is initiated in one of two ways:
  - Systemic inflammatory response resulting in the activation of the complement pathway and the release of cytokines leading to systemic coagulation.
  - Activation of the extrinsic pathway of coagulation by the presence of increased concentrations of tissue factor.
- An impairment of fibrinolysis, which normally keeps coagulation localized, also plays its part in the progression of the syndrome.

Usual Treatment

- Primary goal is to treat the underlying condition.
- Mechanical ventilation, invasive monitoring, and hemodynamic support are often required.
- Surgery intended to remove the cause of DIC should not be delayed.
- Early involvement of a hematologist, serial coagulation testing, and communication with the transfusion laboratory to guide the use of blood products is recommended.
- Blood products:
  - PRBCs for significant hemorrhage.
  - FFP for clotting factor deficiencies.
  - Cryoprecipitate infusions to maintain fibrinogen >100 mg/dL.
  - Platelet infusions to keep level $>20{,}000/mm^3$ (in the absence of hemorrhage) or $>50{,}000/mm^3$ (with active bleeding or prior to surgery).
- Pharmacologic agents (limited, mixed, or poor evidence):
  - Heparin may be of benefit in cases in which thrombosis predominates but should be used in the critical care environment for venous thromboembolism prophylaxis .
  - ATIII, activated protein C, and thrombomodulin (if available) have all been used in the care of specific subgroups of hematologic and septic DIC cases with some success.
  - Antifibrinolytic agents (ε-aminocaproic acid, tranexamic acid, aprotinin) are generally not recommended but may be considered in pts with DIC who continue to bleed.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
HEENT	Bleeding		Bleeding from minor sites of trauma	
CV	Sepsis Hypovolemic shock Microthrombi		Hypotension Signs of decreased organ perfusion	ECG PAC ECHO
RESP	Bleeding Microthrombi	Dyspnea Hemoptysis	Tachypnea	CXR ABGs
GI	Bleeding Microthrombi	Hematemesis		NG suctioning Stool sample, LFTs, clotting studies
GU	Bleeding Microthrombi	Hematuria PU/PV bleeding		Urine output, BUN, Cr
HEME	Bleeding Consumption of factors and platelets	Hemorrhage		Hb, Plt count, clotting studies, TEG, fibrinogen, D-dimer, ATIII, protein C, blood film
CNS	Bleeding Microthrombi		Neurologic deficits	CT
MS	Bleeding Microthrombi		Extremity infarcts	

Key References: Hunt BJ: Bleeding and coagulopathies in critical care, *N Engl J Med* 370(9):847–859, 2014; Levi M: Diagnosis and treatment of disseminated intravascular coagulation, *Int J Lab Hematol* 36(3):228–236, 2014.

Perioperative Implications

Preoperative Preparation

- Optimize the management of the precipitating cause.
- Correct coagulopathy.
- Liaise with laboratory to ensure blood product availability.

Monitoring

- Routine
- Invasive where indicated by severity
- Serial CBC, coagulation studies, and TEG

Airway

- Careful intubation to avoid trauma

Induction

- Be prepared for CV instability in sick pts.

Maintenance

- Use invasive monitoring and laboratory tests to guide interventions.

Extubation

- Organ dysfunction and/or failure may necessitate a protracted period of mechanical ventilation in an ICU.

Adjuvants

- Hepatic and/or renal failure increases the duration of action of most muscle relaxants.

Anticipated Problems/Concerns

- Periop management is best conducted in a critical care environment.
- Hemorrhage may continue into the postop period.
- Organ support may be prolonged.

Diverticulosis

Nancy C. Wilkes

Risk

- More prevalent in developed countries; common in the UK and other parts of northern Europe, North America, Australia, and New Zealand, but uncommon in southern Africa, the Middle East, the Far East, and the Pacific Islands.
- Prevalence in developing countries between 5–45%, depending on age of population and method of diagnosis; African and Asian countries with prevalence approximately 0.2%.
- Prevalence increases with age. In USA, seen in less than 5% of pts younger than 40 y; Approximately 30% by age 60 y and 65% by age 85 y.
- Low-fiber diet is the highest risk factor. High-fat and/or meat diets are high risk.
- Under age 50 y more common in men; over 50 y more common in women.
- Colonic motility disorders contribute.

Perioperative Risks

- Pts who present with diffuse peritonitis or fail nonoperative management of acute diverticulitis may require emergency surgery.
- Risks may include full stomach, obstruction, sepsis, and bleeding.

Worry About

- 15–25% of pts with diverticulosis will develop diverticulitis.
- Acute diverticulitis may be complicated by abscess, fistula, obstruction, or perforation.
- 15% of individuals with diverticular disease will develop acute GI bleeding. Of those, one-third will develop massive bleeding.
- Mortality rates of 22–39% reported for perforation and resultant fecal peritonitis.

Overview

- Multiple saclike herniations through weak points in the intestinal wall. Typically does not contain all layers of the wall but is a herniation of the mucosa and submucosa through the muscle layer.
- Vast majority (>90%) found in the sigmoid colon. Limited to the sigmoid in 65%, approximately 25% involving sigmoid and other segments.
- Of pts with significant diverticulosis, 70% remain asymptomatic and without related complications.

Etiology

- Not completely understood but thought to be related to low-residue diet with long transit time, as opposed to diets with high-fiber content with shorter transit time.
- Abnormalities of peristalsis and intestinal dyskinesis may contribute.
- With long transit times, intraluminal pressure increases, colon becomes distended, followed by acute and then chronic inflammation of diverticula.

Usual Treatment

- Dietary modification, high-fiber emphasis long term for diverticulosis.
- With the development of simple diverticulitis, 75% of cases are not associated with complications. Most are initially treated conservatively with medical therapy (low-residue diet and antibiotics); 85% respond quickly; 15% will require surgery.
- Severe abdominal pain, fever, and clinical signs of peritonitis and/or pelvic abscess require initial resuscitation, parenteral antibiotics, and operative intervention.