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Pediatric Oncologic Emergencies

Although the diagnosis of cancer in childhood is relatively rare, with an annual incidence of 165 cases per million,[1] it remains the leading cause of death by disease in children, accounting for approximately 10% of all childhood fatalities.[2] Since 1970, childhood cancer survival rates have increased from 45% to more than 80%.[3] This dramatic increase in survival has been accomplished due to the availability of new therapies and therapeutic strategies, as well as to prompt diagnosis and enhanced supportive care, including that provided in the emergency department setting.

The pediatric oncology patient may present with a variety of life-threatening situations, including those resulting from structural or functional compromise of the cardiopulmonary or neurologic systems, hematologic abnormalities, or a compromised immune system. Emergencies in these patients may occur as a result of the disease itself or as a consequence of treatment. The emergency department is often the first point of contact for newly diagnosed pediatric oncology patients, and they may be quite ill on initial presentation. Children with known malignancies often are immunocompromised secondary to their treatment and, as a result, pose unique diagnostic and therapeutic challenges.

This article will highlight six of the most commonly encountered oncologic emergencies.

— Ann Dietrich, MD, FAAP, FACEP, Editor

Emergencies Related to Structural Compromise

Malignancies of the Central Nervous System (CNS). *Brain Tumors.* Brain tumors are the most common solid tumors in childhood and the second most common type of childhood cancer overall.[4] Patients with CNS lesions are at risk for rapid deterioration as a result of increased intracranial pressure (ICP). Consequently, rapid diagnosis may be life-saving in this population. However, children and adolescents with brain tumors often present with non-specific complaints and, as a result, pediatric patients with CNS masses often make several visits to their primary care physician or emergency department prior to obtaining a diagnosis. Careful attention to the history and physical exam can assist the practitioner in determining which patients warrant imaging studies to evaluate for an intracranial mass.

Headache is perhaps the most common presenting complaint for a CNS mass. In a study of 3,291 subjects in the Childhood Brain Tumor Consortium Databank, 62% of children with brain tumors experienced chronic or frequent headaches prior to their first hospitalization.[5] Headache is a common complaint in the pediatric population, and accounts for a significant number of visits to emergency departments and pediatrician offices. Most patients presenting with such a complaint do not require further radiologic investigation. However, specific features of the headache and associated signs and symptoms should increase the practitioner's suspicion that this may be the result of a more serious condition. The practitioner should evaluate for signs of increased ICP or

Executive Summary

- More than 97% of patients with a brain tumor, presenting with a headache, will have a documentable neurologic abnormality found on physical exam.
- Localized or radicular back pain occurs in 80% of children with spinal cord compression.
- Orthopnea, upper body edema, and dyspnea at rest are all associated with increased anesthesia risk in a child with superior mediastinal syndrome.
- Tumor lysis syndrome is characterized by the triad of hyperkalemia, hyperuricemia, and hyperphosphatemia and is often complicated by secondary renal failure and symptomatic hypocalcemia.

localizing signs or symptoms. Table 1 lists symptoms that should raise suspicion that the headache may be secondary to a brain tumor.

A thorough physical examination, including a complete neurological and fundoscopic exam, is essential in making the diagnosis of a brain tumor and evaluating for increased ICP. More than 97% of patients with a brain tumor, presenting with a headache, will have a documentable neurologic abnormality found on physical exam.[5,6] Macrocephaly and splitting of sutures may be present in infants. Limitation in eye movement, visual field defects, seizures, and gait disturbance are suggestive of significant CNS pathology.

A computed tomography (CT) scan should be obtained for gross brain structural evaluation and signs of increased ICP if magnetic resonance imaging (MRI) cannot be performed. CT imaging must be reviewed with caution because it is not very accurate for evaluating brain tumors, especially posterior fossa tumors. MRI is the preferred test to evaluate brain structure, but may not be feasible in the emergency setting. Lumbar puncture should be approached with caution, and preferably deferred if increased ICP is suspected, as this can lead to herniation.

Emergent treatment of increased ICP consists of elevation of the head of the bed, intravenous dexamethasone at a loading dose of 0.5-2 mg/kg, followed by 0.25-0.5 mg/kg IV every 6 hours. Mannitol 20% solution and hypertonic saline solution should also be considered. Intravenous fluids of normal saline at a rate of 75% maintenance may be started. Endotracheal intubation should be considered in severe cases to control airway and partial pressure of carbon dioxide. Immediate pediatric neurosurgical consultation should be obtained. Oncology and radiation oncology consultations should be obtained.

Spinal Cord Compression (SCC). SCC occurs in approximately 3-5% of all pediatric oncology patients.[7] If not detected and treated in a timely manner, SCC may lead to irreversible neurologic damage, including permanent paralysis. SCC is the most common cause of lower limb paralysis in children.[8] Early recognition and treatment of cord compression is essential to decrease long-term morbidity. Appropriate, definitive management is best determined through multidisciplinary consultation with pediatric oncology, neurosurgery, and radiation oncology. SCC may result from virtually any malignancy and can present either at the time of diagnosis or as the disease progresses.

SCC may occur as a result of an infiltrative paraspinous process, tumors originating from the vertebral process, intrinsic spinal cord tumors, or infiltrative lesions. In the pediatric patient, cord compression most frequently results from a paravertebral tumor extending through the intervertebral foramina (e.g., soft-tissue sarcomas, tumors of neurogenic origin).[8] Tumors growing through the intervertebral foramina initially cause mechanical nerve root compression. If growth continues, transverse myelitis (inflammation of gray and white matter of the spinal cord) ultimately may occur. Also, the venous plexus may be compressed at the intravertebral level, resulting in ischemic injury to the cord.[10] Intradural spinal metastasis from an intracranial process (e.g., medulloblastoma) also may lead to cord compromise. These lesions tend to occur in the lumbosacral region. Primary spinal cord tumors, such as an intramedullary astrocytoma, also may be the cause of pain and neurologic symptoms in children. More rarely, leukemic chloromas may be a cause of SCC. Chloromas are more commonly due to acute myelogenous leukemia (AML) as opposed to acute lymphoblastic leukemia (ALL).[11] Leukemic infiltration of the spinal cord has a predilection for the cauda equina or conus medullaris,[12] and patients should be screened for symptoms of cord involvement.[13]

SCC should be suspected in any pediatric oncology patient presenting with back pain or suggestive neurologic findings. Localized or radicular back pain occurs in 80% of children with SCC.[7] Back pain in any child with a malignancy is highly suspicious for SCC and deserves further investigation. Patients with SCC also may present with complaints

Table 1. Symptoms Suggestive of a Brain Tumor as the Etiology of Headache

- **Occipital location of headache**
- **Worsening symptoms**
- **Awakens patient at night**
- **Associated with focal symptoms**
- **Emesis**

of motor or sensory abnormalities. Depending on the rate of tumor growth, the pain and/or deficits may be long-standing (present for weeks to months) and slowly progressive, or more acute in nature. History should elicit the time course and include questioning about changes in gait and bowel or bladder habits. Tenderness to palpation tends to localize to the site of the lesion. A thorough neurologic examination should be performed, with attention to motor strength, reflexes, gait abnormalities, sensation, and sphincter tone. The diagnosis of SCC is particularly challenging in the infant or young child who cannot verbalize back pain; regression in motor milestones or refusal to ambulate may be initial symptoms.

Imaging studies should be obtained as soon as possible. Plain radiographs are insufficiently sensitive. The optimal study in a patient with potential SCC is an MRI. In the rare case where an MRI cannot be performed in a timely manner, CT myelography is an acceptable alternative.

If SCC is suspected, dexamethasone should be initiated without delay. Corticosteroid therapy may decrease cord edema and preserve neurologic function while plans for more definitive therapy are underway. Optimal dosing of dexamethasone is not known; a loading dose of 1-2 mg/kg IV, followed by 0.25-0.5 mg/kg every 6 hours, has been suggested. If lymphoma or a leukemic infiltrate is the cause of the SCC, dexamethasone has the potential to be cytolytic and may lead to tumor lysis syndrome.

Definitive therapeutic options include surgical resection, radiation therapy, and/or chemotherapy. The optimal choice of treatment is best determined by a multidisciplinary team. Treatment choice will be influenced by whether a tissue diagnosis has been made, radio- and chemosensitivity of the tumor, extent of disease, and the degree of neurologic compromise present. Studies have suggested that the degree of neurologic deficit at the time of presentation and the duration of symptoms prior to initiating therapy both correlate with the ultimate functional outcome, regardless of the treatment method employed. When planning definitive therapy, both acute relief of SCC as well as long-term sequelae of the treatment modality used should be considered. Radiation therapy and surgical resection, while offering more immediate benefit in terms of local tumor control, may lead to long-term sequelae in terms of growth problems and scoliosis. For this reason, chemotherapy is generally the treatment of choice in a child with a chemosensitive process such as lymphoma, leukemia, or disseminated neuroblastoma and who has minimal symptoms of SCC.[14] If SCC occurs in the setting of progressive refractory disease, palliative therapy for SCC should not be withheld, as it may have a substantial positive impact on the quality of life.

Mediastinal Mass, Superior Mediastinal/Superior Vena Caval Syndrome. Masses within the mediastinum are fairly common in children, and the differential diagnosis is quite different than one would consider for an adult patient.[15] In the young infant, congenital anomalies should be considered. Children may present with a mass in the posterior mediastinum, often representing a neurogenic tumor such as neuroblastoma. The differential diagnosis of a mediastinal mass in a pediatric patient depends on the age of the child, the rapidity with which symptoms develop, and the mediastinal compartment that contains the mass. The majority of children or adolescents presenting with a mediastinal mass who are experiencing respiratory compromise ultimately will be diagnosed with a malignancy, most often lymphoma. Table 2 shows the differential diagnosis of a mediastinal mass by location within the mediastinum.

Children presenting with an anterior mediastinal mass often have symptoms suggestive of a respiratory infection, such as cough, fever, and wheezing. It is not uncommon for a child with a mediastinal mass to present to a medical facility and receive a diagnosis of a respiratory tract infection or asthma exacerbation on more than one occasion prior to receiving imaging studies and detection of the mass. Therefore, it is critical that the practitioner keep broad differential diagnoses in mind and perform a thorough physical exam and detailed history, especially in the child with wheezing or dyspnea with no previous history of asthma.

Masses in the anterior mediastinum have the potential to create life-threatening compromise of the airway or cardiovascular system. Superior mediastinal syndrome (SMS) refers to compression of the trachea or mainstem bronchi by a mediastinal mass. SMS is closely related to superior vena caval syndrome (SVCS), which refers to compression or obstruction of the vena cava. These two entities often coexist. In children, the trachea and mainstem bronchi are very compliant and vulnerable to collapse

Table 2. Differential Diagnosis of a Mediastinal Mass by Location Within the Mediastinum

Anterior
• Lymphoma
• Leukemia
• Malignant germ-cell tumor
• Benign teratoma
• Thymic lesion (thymic hyperplasia, thymoma, thymic cyst)
• Substernal thyroid
Middle
• Lymphoma
• Tuberculosis
• Histiocytosis
• Sarcoidosis
• Anomalies of the great vessels
Posterior
• Neuroblastoma
• Ganglioneuroblastoma
• Sarcoma

Table 3. Signs and Symptoms of SVCS

- Facial swelling
- Upper body edema
- Cyanosis of the face or upper body
- Plethora
- Conjunctival edema or suffusion
- Headache
- Tachycardia
- Elevated venous pressure
- Vocal cord paralysis, hoarseness
- Dyspnea
- Cough
- Decreased mentation
- Horner's syndrome

from external compression. The superior vena cava is a thin-walled vessel with low intraluminal pressure. Compression by a mediastinal mass or obstruction by a tumor or thrombosis leads to venous stasis and diminished blood return to the heart. (*See Table 3.*) It should be noted that a child with an indwelling central venous catheter may develop SVCS secondary to thrombosis of the catheter. SMS and SVCS generally result from compression from a mass in the anterior compartment of the mediastinum, with lymphoma being the most common cause. Children with leukemia also may present with a mediastinal mass; therefore, it is important that all newly diagnosed leukemia patients have a chest X-ray performed as part of their initial evaluation.

A thorough history and physical exam should be performed on any child or adolescent with a suspected mediastinal mass, with particular attention to signs and symptoms of SMS/SVCS.

Children with SMS and/or SVCS may be asymptomatic at the time of presentation, or they may come to medical attention just prior to cardiopulmonary collapse. In some cases, the airway may be so tenuous that total airway loss may be precipitated by simply placing the child in a supine or flexed position. Once there is suspicion of a mediastinal process, care must be taken to protect the airway. When performing the physical exam or while obtaining imaging studies, it is important that the child not be forced to lie in a position that may precipitate airway collapse. Diagnosis and initiation of therapy should occur in the most expeditious and least invasive manner possible.

Children with evidence of SMS/SVCS must be managed with care to avoid complete loss of the airway or cardiovascular collapse. In particular, the use of procedural sedation or general anesthesia should be avoided until a thorough evaluation of the child's anesthesia risk has been completed. Several clinical factors have been associated with an increased risk for life-threatening anesthesia-related complications in the context of SMS. Orthopnea, upper body edema, and dyspnea at rest have all been associated with increased anesthesia risk.[16] Orthopnea most closely correlates with the level of risk assumed, and questioning for this symptom should occur directly, as parents may not give such information unless it is specifically asked. Although these elements of the physical exam and history should be taken into consideration, it should be noted that the degree of symptoms does not always correlate with the degree of airway compromise,[17] since anesthesia-related deaths have been reported in asymptomatic children with anterior mediastinal masses.[18-20] Therefore, in a child with a known mediastinal mass, a CT scan and pulmonary function testing should be performed prior to subjecting the child to anesthetics. A CT scan can assess the tracheal cross-sectional area (TCA), which can then be compared to well-established norms for age and gender.[21] Pulmonary function testing to determine the peak expiratory flow rate (PEFR) may be done fairly easily at the child's bedside with a handheld device, and readings should be done in both the sitting and supine position. Studies have shown that children with a TCA and PEFR of > 50% for age can be safely given general anesthesia or procedural sedation if needed.[22] Table 4 shows findings for relative contraindications for anesthesia in a child with a mediastinal mass.

In severe cases of SMS, empiric therapy may need to be employed prior to making a tissue diagnosis. In most cases, empiric corticosteroid therapy will provide sufficient tumor reduction.[23] However, administration of steroids may result in tumor lysis syndrome and may impair the ability to make a diagnosis. Steroids, therefore, should be given only in an immediately life-threatening situation after consultation with a pediatric oncologist. Even more rarely, the mass may not be sensitive to corticosteroids, and emergent radiation therapy may be considered.[24] However, in most cases, a tissue diagnosis can be made prior to instituting therapy. If general anesthesia or sedation must be avoided, the use of local anesthesia with the child in a seated position should be considered.

Hematologic Emergencies

Hyperleukocytosis. Leukemia is the most common type of childhood malignancy. Approximately 3500 children are diagnosed with acute leukemia in the United States each year, accounting for approximately one-third of childhood cancers.[25] The presentation of leukemia varies widely, from children presenting with only mild symptoms, such as fatigue, to those presenting in extremis. Likewise, the degree of hematologic derangement in a newly diagnosed pediatric patient ranges from a normal peripheral blood count to life-threatening hyperleukocytosis, defined as a white blood cell (WBC) count of greater than 100,000 per μL.

Hyperleukocytosis is present at the time of diagnosis in approximately 10% of cases of ALL.[26] Among ALL patients, those with infant leukemia, hypodiploid chromosome blasts, and those with T-cell ALL are at

Table 4. Critical Airway: Relative Contraindications to Anesthesia in a Child with a Mediastinal Mass

- Orthopnea
- Upper body edema
- Dyspnea
- Clinical findings of impending respiratory failure
- Tracheal cross-sectional area < 50% normal for age and sex
- Severe compression of one or both mainstem bronchi
- Peak expiratory flow rate of < 50% predicted (performed in sitting and supine position)

particular risk for hyperleukocytosis.[26] The clinician should be certain to obtain a chest X-ray early in the initial management of the ALL patient with hyperleukocytosis, as the presence of a mediastinal mass in these patients is not uncommon.[26] Five percent to 20% of pediatric AML cases will present with hyperleukocytosis, and virtually all patients with chronic myelogenous leukemia (CML) will have hyperleukocytosis at diagnosis.[27,28]

Hyperleukocytosis results in hyperviscosity and leukostasis. Aggregation of leukemic blasts occurs in the microvasculature, which may result in multisystem organ dysfunction. End-organ damage results from interaction between aggregates of leukemic blasts and injured epithelium. This interaction leads to tissue hypoxia, with resultant release of inflammatory cytokines.[29] Myeloblasts are more apt to cause damage due to their larger size and increased adhesiveness.[30] Leukostasis may cause devastating dysfunction in any organ system. In the central nervous system, cerebrovascular accidents, both thrombotic and hemorrhagic, may occur spontaneously. This is most common in the setting of AML.[27] From a cardiovascular standpoint, fatal pericardial hemorrhages secondary to hyperleukocytosis have been reported. Pulmonary complications also may occur, including hypoxia, pulmonary hemorrhage, and respiratory failure. Intra-abdominal complications, including gastrointestinal bleeds, are also possible.[27] Splenic rupture is most commonly a risk in the patient with extreme hyperleukocytosis and CML. In addition to the damage caused by leukostasis, children with hyperleukocytosis from acute leukemia are at risk for the metabolic complications from tumor lysis syndrome, including renal failure, hypocalcemia, hyperphosphatemia, and hyperuricemia. Life-threatening metabolic abnormalities occur more commonly in the setting of hyperleukocytosis in ALL.[27] This topic is covered more thoroughly in the tumor lysis section. Disseminated intravascular coagulation (DIC) is most common in high white-blood-cell count AML patients. All patients with hyperleukocytosis should be screened for a coagulopathy, as correction of these abnormalities may decrease the incidence of bleeding complications.[27]

Patients with hyperleukocytosis should be closely monitored for signs and symptoms of end-organ damage. Neurologic symptoms may include headache, seizures, and altered mental status. On examination, papilledema or retinal vascular distention may be detected. Pulse oximetry should be employed to detect hypoxia, and the patient should be screened for other signs and symptoms of respiratory compromise. Priapism, clitoral enlargement, and dactylitis, all secondary to leukostasis, may be present on physical examination.

Children with hyperleukocytosis are at increased risk of death early in their course of treatment. The threshold at which hyperleukocytosis becomes clinically significant varies by leukemia type. This is most likely due to differences in adhesiveness of the leukemic blasts in different types of leukemia. The risk of fatal complications is highest in patients with AML with hyperleukocytosis. In patients with hyperleukocytosis, early death occurs in approximately 20% of patients with AML and 5% of patients with ALL.[27] Studies have suggested that life-threatening viscosity becomes increasingly prevalent at WBC greater than 200,000/µL in AML and greater than 300,000/µL in ALL and CML.[23]

Aggressive supportive care and prompt consultation with a pediatric oncologist for initiation of cytoreductive chemotherapy reduces the risk of early death in these patients. Supportive care is directed at correction and prevention of metabolic abnormalities and coagulopathies. Table 5 outlines the suggested initial studies and management of the patient with hyperleukocytosis. Hydration should be instituted. Supplemental potassium should not be given, even in the setting of hypokalemia, as hyperkalemia may result from tumor lysis even prior to the initiation of therapy. The patient should be monitored very closely for the development of metabolic abnormalities. Efforts should be made to correct coagulopathies, including the transfusion of platelets if the platelet count is < 25,000 per µL. For the anemic patient, red blood cell transfusion should be avoided if the patient is hemodynamically stable, as this will further increase the blood viscosity. Emergent cranial radiation, which has been used in the past in an effort to prevent intracranial bleeds, is of no proven benefit and is no longer routinely employed or recommended.[27] Leukapheresis and exchange transfusion may be considered as temporizing measures or to reduce the leukemic burden prior to initiation of cytoreductive therapy; however, this remains controversial.[26,31,32] It should be noted that the blast count will rise again quickly after these procedures are completed. Prompt initiation of effective cytoreductive therapy remains critical.

Tumor Lysis Syndrome. Tumor

Table 5. Initial Supportive Therapy in the Setting of Hyperleukocytosis

- Hydration: D5 1/2 NS at 3000 mL/m^2/day. No supplemental potassium should be given. Consider alkalinized fluids if allopurinol is being used and patient is not hyperphosphatemic.
- Treatment of hyperuricemia
  - Allopurinol 300 mg/m^2 divided TID
  - Consider administration of rasburicase if patient is hyperuricemic and not G6PD deficient.
- Transfuse platelets if platelet count is < 25,000/μL.
- Correct other significant coagulopathies.
- Avoid transfusion of packed red blood cells if hemodynamically stable.

NOTE: Management of hyperuricemia and tumor lysis covered more thoroughly in tumor lysis section.

Table 6. Laboratory Studies and Clinical Monitoring

- CBC, calcium, phosphate, magnesium, uric acid, urea nitrogen, creatinine, lactate dehydrogenase at presentation and then every 6-12 hours depending on risk
- Sequential vital signs
- Strict assessment of intake and output
- Body weight once to twice daily
- Cardiorespiratory monitor with multi-lead ECG as needed for hyperkalemia (e.g., if K > 6 mEq/L, look for wide QRS and peaked T waves)
- Close clinical evaluation for signs of hypocalcemia or renal failure

lysis syndrome (TLS) is an oncologic emergency characterized by a triad of hyperkalemia, hyperuricemia, and hyperphosphatemia, and it is often complicated by secondary renal failure and symptomatic hypocalcemia. TLS may occur prior to the initiation of cytoreductive therapy and up to one week after initiation of therapy. When tumor cells lyse, large amounts of intracellular chemicals are released into the circulation and may cause metabolic derangements, which may lead to renal dysfunction and cardiac dysrhythmia.

TLS may occur with any malignancy with rapid cell turnover and high tumor burden. It most commonly occurs in the setting of leukemia or high-grade lymphoma. Children with hyperuricemia or renal insufficiency at presentation are at particularly high risk. Evidence of tumor lysis may be present prior to the initiation of the chemotherapy or after the child receives corticosteroids.

Conditions at highest risk for TLS include:

- ALL or AML (WBC >100,000/μL)
- Leukemia with massive lymphadenopathy and/or organomegaly
- T-cell ALL
- Infant leukemia
- Burkitt's lymphoma
- Large cell lymphoma.

Laboratory monitoring should include CBC and blood chemistries, with specific attention paid to serum potassium, calcium, magnesium, phosphate, uric acid, creatinine, urea nitrogen, and lactate dehydrogenase. Initially, these studies should be done every 6 hours, and then spread out as the tumor lysis improves. Fluid input and output should be monitored closely, and body weight documented twice daily. Sequential vital signs should be performed and a cardiopulmonary monitor with multi-lead ECG should be used to monitor for effects of hyperkalemia. (*See Table 6.*)

Interventions should be initiated to prevent TLS in tumors with a risk of TLS, and aggressive treatments should be started if there is evidence of TLS. These include hydration and alkalinization. Urine alkalinization helps with excretion of uric acid but should be performed with care because a urine pH > 7.5 may lead to precipitation of CaPO4 and xanthine crystals. Hyperkalemia should be treated with hydration, sodium polystyrene sulfonate, calcium gluconate, or insulin and dextrose as needed. Dialysis may be needed. Hyperphosphatemia can be treated with aluminium hydroxide, and dialysis may be necessary.

Hyperuricemia is treated with allopurinol and, when possible, should be started prior to initiation of cytolytic therapy. For severe hyperuricemia, rasburicase may be used. Table 7 outlines suggested intervention for the management of TLS.

Infections in the Immunocompromised Child

Febrile Neutropenia. The advances made in the overall survival rates for children over the past few decades have been due, in part, to an increased intensity of chemotherapy given to pediatric patients. Cancer-directed therapy for this population is often quite cytotoxic to rapidly growing cells, often resulting in hematologic and immunologic suppression and breakdown of normal mucosal barriers. Selected patients may also be receiving radiation therapy, which may result in further tissue injury and impairment of the body's innate immunologic

Table 7. Prevention and Treatment of TLS

Delay and/or titrate cytolytic therapy (if possible) until prophylactic measures can be implemented (see "hydration therapy," "urinary alkalinization," "hyperuricemia management" below). AVOID intravenous radiologic contrast agents that might precipitate renal failure.	
Hydration therapy	• Administer IV fluid: D5 0.2% NaCl + $NaHCO_3$ 40 mEq/L at 3000 mL/m^2/day • Urine output goal: ≥ 100 mL/m^2/hour OR 3 mL/kg/hour (if body weight is < 10 kg) • Urine specific gravity goal ≤ 1.010 • Diuretics may be required (furosemide or mannitol)
Urinary alkalinization (see IV fluid above)	• Urine pH goal 6.5-7.5 to aid in excretion of uric acid Note urine pH > 7.5 may increase precipitation of CaPO4 and xanthine crystals • Adjust amount of $NaHCO_3$ in IV fluid to achieve urine pH and serum [sodium] goals
Hyperkalemia	• Moderate and asymptomatic (≥ 6.0 mEql/L): sodium polystyrene sulfonate orally • Severe and/or symptomatic (> 7.0 mEq/L): –Calcium gluconate 100-200 mg/kg IV –D25 (2 mL/kg) IV + regular insulin 0.1 units/kg IV –Dialysis may be necessary
Hyperphosphatemia	• Aluminum hydroxide 50-150 mg/kg/day divided in 4–6 doses (or 30–40 mL 6 to 8 hours) • Dialysis or continuous renal replacement therapy may be necessary
Hyperuricemia (uric acid crystals form in renal tubules and distal collecting system and cause oliguria)	• Allopurinol 300 mg/m^2 PO divided TID: Decreases production of uric acid by inhibiting xanthine oxidase If possible, start 12-24 hours before induction of cytolytic therapy **OR** • Urate oxidase (rasburicase): Metabolizes uric acid Should be considered in high-risk situations; contraindicated in G6PD deficiency, pregnancy, and lactation

barriers. Additionally, most pediatric cancer patients have indwelling central venous access, representing a further breakdown in the body's natural defenses against infection. As a result, children receiving therapy for cancer are at high risk for life-threatening infections, which are known to occur, especially in the setting of neutropenia. Although fever in the general pediatric patient is a fairly common, often benign presenting complaint, fever in the potentially neutropenic pediatric cancer patient is a medical emergency and needs to be recognized quickly as such, with prompt initiation of treatment.

The generally accepted definition of fever in the neutropenic patient is a single temperature of greater than 38.3°C (101° F) or two consecutive temperatures greater than 38.0°C (100.4°F) taken orally in a 12-hour period and lasting at least 1 hour.[33] It should be noted that axillary temperature is, on average, about 0.6°C lower than oral temperature. Rectal temperature should never be performed in children with neutropenia. Note that the absence of fever in a neutropenic patient with localizing signs or symptoms does not exclude infection. This is particularly true of the child receiving steroid therapy as a part of treatment. Such patients may present with non-specific complaints as the only sign of bacteremia. Clinically significant neutropenia is defined as an absolute neutrophil count (ANC) < 500/μL or < 1,000/μL and expected to decline due to recent chemotherapy administration.

Certain risk factors place the febrile neutropenic patient at particularly high risk. Patients with severe neutropenia, prolonged neutropenia, and those who developed neutropenia over a short period of time are at highest risk for the development of septicemia.[34] Individuals with an ANC of less than 200 are particularly vulnerable, as are those patients who have been neutropenic for more than 10 days.

A thorough history and physical examination should be performed, with particular attention to potential sites of infection (oral lesions, perirectal tenderness, and central venous access sites). Rectal temperatures should be avoided due to the risk of bacteremia from colorectal organisms. Neutropenic patients do not have the ability to mount the same degree of inflammatory response as an immunocompetent host. As a result, the localizing signs and symptoms may be subtle (e.g., pneumonia

Table 8. Frequently Chosen Antibiotics for Febrile Neutropenia

Coverage	Antibiotic
Broad spectrum	Ceftazidime; cefepime; piperacillin-tazobactam; meropenem
Additional Gram-negative coverage	Amikacin; gentamicin; tobramycin
Additional Gram-positive coverage	Vancomycin
Additional anaerobic coverage	Clindamycin; metronidazole
Atypical bacterial coverage	Azithromycin; clarithromycin

may present with mild respiratory symptoms and may not have characteristic findings on examination or CXR). Any reported areas of discomfort should be pursued as potential sites of infection.

The initial laboratory work-up of a febrile neutropenic pediatric oncology patient should include, at a minimum, a CBC, electrolytes, BUN/creatinine, and blood cultures for bacteria and fungi from all lumens of the central venous access device. Peripheral blood cultures are not routinely needed. Urinalysis and urine culture should be obtained. A chest X-ray should be considered and certainly obtained if hypoxia, tachypnea, physical exam findings suggestive of a respiratory infection, or respiratory complaints are present.[35] Stool studies and wound cultures should be considered if the history warrants. Meningitis is fairly rare in the febrile neutropenic patient; however, a lumbar puncture should be considered if there is alteration of mental status or neck stiffness.

Neutropenic patients are at risk for both common and opportunistic infections; therefore, the differential diagnosis of the febrile neutropenic patient is broad. Bacterial infections are the most common type of infection in this patient population and they present the most immediate risk to life. Broad-spectrum antimicrobials should be instituted within 30 minutes of presentation to a medical facility. Administration of broad-spectrum antimicrobials should not be delayed while awaiting laboratory results or imaging studies. The optimal choice of empiric antibiotic therapy is guided by institutional susceptibility and resistance patterns and history and exam findings unique to the individual patient. At a minimum, treatment should include a broad-spectrum beta-lactam antibiotic with antipseudomonal coverage. Potential agents for monotherapy include piperacillin/tazobactam, third- and fourth-generation cephalosporins (ceftazidime, cefepime), or carbapenems (imipenem, meropenem).[36-40]

In certain circumstances, monotherapy may be inadequate as empiric coverage. In the case of clinical sepsis, triple therapy with vancomycin and an aminoglycoside should be considered. Recent data suggest a shift in the overall trend of positive cultures toward increased infection with Gram-positive organisms in the febrile neutropenic patient.[41] There are several circumstances under which Gram-positive coverage should be broadened, including severe mucositis, potential line infections, and concern for cellulitis. In addition, recent treatment with high-dose cytarabine raises the risk for Gram-positive infection, particularly *Streptococcus viridans.*[42] If any of these risk factors are present, there should be a low threshold for adding vancomycin. Abdominal pain may be a particularly ominous symptom in the neutropenic patient, as it may indicate the onset of neutropenic enterocolitis (typhlitis). In such a setting, or in the case of a perirectal infection, coverage should include a broad-spectrum beta-lactam antibiotic with antipseudomonal coverage, vancomycin, and additional anaerobic coverage such as metronidazole. If respiratory symptoms are present, coverage for atypical organisms, and perhaps vancomycin, should be considered. Table 8 shows the suggested initial antibiotic management of the febrile neutropenic patient, and Table 9 shows additional antibiotic coverage for specific indications.

In addition to bacterial infections, oncology patients with prolonged severe neutropenia are at risk for opportunistic infections. Pediatric oncology patients routinely receive prophylaxis against *Pneumocystis jirovecii* (*carinii*), but there are instances of noncompliance, and breakthrough infections may occur even in the patient receiving prophylaxis. Significant hypoxia and diffuse infiltrates on chest X-ray should raise concern for *Pneumocystis jirovecii* pneumonia (formerly PCP). Fungal infections are possible causes of fever in the neutropenic patient. Antifungal coverage should be considered after consultation with the patient's oncologist. Viral infections such as HSV, varicella, RSV, and influenza may be life-threatening in the neutropenic pediatric patient and should be considered in the differential diagnosis. When symptoms and community infection patterns are suggestive, rapid RSV or influenza testing should be employed, and practitioners should have a low threshold for administering antiviral agents to such patients when the situation warrants.

Other Common Oncologic Emergencies

Pleural and Pericardial Effusions. Pleural or pericardial effusions may result from the primary malignancy, from complications of treatment such as radiation therapy, or from infectious complications. Dyspnea and cough are common presenting symptoms. Evaluation should include a chest

Table 9. Indications for Specific Antimicrobial Therapy in Addition to Broad-spectrum Antibiotic

Indication	Antimicrobial Therapy
Clinical sepsis	Broad-spectrum antibiotic + vancomycin + aminoglycoside
High-dose cytarabine chemotherapy or mucositis or cellulitis or concern for CVC infection	Broad-spectrum antibiotic + vancomycin
Pulmonary infiltrates	Broad-spectrum antibiotic + vancomycin + atypical antibacterial agent; if infiltrates are diffuse and hypoxia is present, consider treatment for PJP (PCP)
Abdominal symptoms	Broad-spectrum antibiotic + vancomycin + aminoglycoside + metronidazole or clindamycin
Perirectal lesions	Broad-spectrum antibiotic + vancomycin + metronidazole or clindamycin

radiograph, ECG, echocardiography, and monitoring of central venous pressure. Echocardiography is the most useful test in the confirmation of pericardial effusions. If there is significant respiratory distress or circulatory compromise, thoracentesis and pericardiocentesis for pleural effusion and pericardial effusions, respectively, should be considered in addition to routine resuscitative measures. Steroids should be considered for steroid-responsive tumors.

Bowel Obstruction or Perforation. This may result from the tumor process, such as Burkitt's lymphoma, or as a complication of treatment, such as steroids. Vincristine often leads to constipation and, if not treated, may cause obstipation. Whenever an acute abdomen is suspected in an oncologic patient, the patient should be placed on NPO status and surgical consultation should be obtained.

Syndrome of Inappropriate Antidiuretic Hormone (SIADH). Several agents may cause SIADH with resultant hyponatremia, which may result in mental status changes and seizures. Fluid restriction is usually sufficient in mild cases. Administration of hypertonic solution should be considered in severe cases. Furosemide to promote free water excretion may be helpful. Electrolytes, especially sodium, should be monitored closely.

Pancreatitis. This frequently occurs as a complication of chemotherapy and may present with abdominal pain, nausea and vomiting, anorexia, and fever. Evaluation should include complete blood count, complete metabolic panel, and lipase and amylase enzyme levels. Treatment should include pain management, NPO status, intravenous fluids, and hospital admission. A nasogastric tube should be placed in patients with ongoing emesis, and antibiotics should be administered to febrile or septic patients.

Coagulopathy. This can result either in bleeding or deep venous thrombosis. A complete physical exam, including a careful neurologic exam, should be performed. Laboratory studies should include a complete blood count, PT, APTT, D-dimer, and fibrinogen. Imaging of suspected deep vein thrombosis (DVT) sites should be performed. Treatment with fresh frozen plasma and cryoprecipitate should be considered.[44] Careful anticoagulation should be initiated in patients with DVT with no evidence of bleeding.

Summary

Cancer is the leading medical cause of death in children. Morbidity and mortality are either directly secondary to the cancer or to complications of treatment. The significant improvements in childhood cancer survival that have been witnessed in the past 25 years are due to improvements in chemotherapy, radiation therapy, surgical techniques, supportive care, and the recognition and treatment of oncologic emergencies. Prompt recognition and appropriate management of oncologic emergencies can decrease patient agony, decrease mortality, and minimize long-term sequelae. A multidisciplinary approach involving the emergency department physician, the oncologist, and radiation oncologist is essential in achieving optimal outcomes.

Brain tumors and other cancers that spread to the CNS can cause increased intracranial pressure, the symptoms of which can range from subtle to obvious. Management ranges from observation to administration of corticosteroids, mannitol, cytoreductive chemotherapy, to emergent radiation therapy. Spinal cord compression can result from intradural or extradural malignancies. If not treated promptly, it can result in paralysis and other sequelae. Management includes observation, cytoreductive chemotherapy, corticosteroids, intrathecal chemotherapy, radiation therapy, and surgical decompression.

Mediastinal tumors can result in airway compromise or vascular compromise. If airway compromise is suspected, care should be taken to avoid placing the patient in positions that may worsen airway compromise. Sedation and anesthesia should be

considered with care. Emergent cytoreductive therapy with steroids and/or radiation therapy may be necessary.

Rapid proliferating and high tumor-burden cancers can lead to tumor lysis syndrome. Tumor lysis may occur even prior to the initiation of cytoreductive chemotherapy. Hydration, alkalinization, and allopurinol may be necessary. Rasburicase should be considered in severe hyperuricemia, provided the patient is not G6PD deficient. Severe tumor lysis syndrome may necessitate dialysis.

Fever in the neutropenic patient is an emergency, and prompt evaluation and administration of broad-spectrum antibiotics are needed. Blood culture from indwelling catheters should be collected, preferably prior to initiation of antibiotics. However, the administration of antibiotics should not be delayed for diagnostic studies.

The awareness and vigilance of emergency care providers and other physicians for possible oncologic emergencies are critical in maintaining and further improving good outcomes for children with malignancies.

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Physician CME Questions

43. Cancer is the leading nontraumatic cause of death in children.
    A. true
    B. false

44. Which of the following is an oncologic emergency?
    A. spinal cord compression
    B. tumor lysis syndrome
    C. neutropenic fever
    D. superior mediastinal syndrome
    E. all of the above

45. Which of the following is a sign of increased intracranial pressure?
    A. headache
    B. papilledema
    C. emesis
    D. splitting of sutures
    E. all of the above

46. Steroids are contraindicated in spinal cord compression.
    A. true
    B. false

47. Which of the following cancers is *not* likely to present with a mediastinal mass?
    A. T-cell ALL
    B. Hodgkin lymphoma
    C. neuroblastoma
    D. Wilms' tumor

48. When a child with signs of respiratory distress secondary to a mediastinal mass presents, the following measure should be taken immediately:
    A. intubation
    B. emergent radiation therapy
    C. place in supine position
    D. allow the child to choose preferred posture

49. Which of the following findings is *not* suggestive of tumor lysis syndrome?
    A. hyperkalemia
    B. hyperuricemia
    C. hypercalcemia
    D. hyperphosphatemia

50. Which of the following is *not* part of the management of tumor lysis syndrome?
    A. hydration
    B. alkalinization
    C. fluid restriction
    D. allopurinol
    E. rasburicase

51. Which of the following is a possible complication of hyperleukocytosis?
    A. mental status changes
    B. respiratory distress
    C. thromboembolism
    D. tumor lysis syndrome
    E. all of the above

52. It is important to cover for Pseudomonas in a child presenting with neutropenic fever.
    A. true
    B. false

Answers: 43. A; 44. E; 45. E; 46. B; 47. D; 48. D; 49. C; 50. C; 51. E; 52. A

Pediatric Emergency Medicine Reports

CME Objectives

Upon completion of this educational activity, participants should be able to:

- recognize specific conditions in pediatric patients presenting to the emergency department;
- describe the epidemiology, etiology, pathophysiology, historical and examination findings associated with conditions in pediatric patients presenting to the emergency department;
- formulate a differential diagnosis and perform necessary diagnostic tests;
- apply up-to-date therapeutic techniques to address conditions discussed in the publication;
- discuss any discharge or follow-up instructions with patients.

CME Instructions

Physicians participate in this continuing medical education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to test their knowledge.

To clarify confusion surrounding any questions answered incorrectly, please consult the source material. After completing this activity, you must complete the evaluation form that will be provided at the end of the semester and return it in the reply envelope provided to receive a credit letter. When your evaluation is received, a credit letter will be mailed to you.

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Instructions: Fill in the appropriate answers. Please write in answers to the open-ended questions in the space provided. Return the questionnaire in the enclosed postage-paid envelope by **July 1, 2011.**

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- O A. Always
- O B. Most of the time
- O C. Some of the time
- O D. Rarely
- O E. Never

2. How would you describe your satisfaction with your subscription to *PEMR*?

- O A. Very satisfied
- O B. Somewhat satisfied
- O C. Somewhat dissatisfied
- O D. Very dissatisfied

Questions 3-14 ask about articles appearing in *Pediatric Emergency Medicine Reports*. Please mark your answers in the following manner:

A. very useful B. fairly useful C. not very useful D. not useful at all

3. Trauma Evaluation (June 2010)	O A	O B	O C	O D
4. Orthopedic Injuries Pt. 1 (July 2010)	O A	O B	O C	O D
5. Orthopedic Injuries Pt. 2 (Aug. 2010)	O A	O B	O C	O D
6. Traumatic Brain Injury (Sept. 2010)	O A	O B	O C	O D
7. Critical Child (Oct. 2010)	O A	O B	O C	O D
8. Streptococcal Syndromes (Nov. 2010)	O A	O B	O C	O D
9. Burn Management (Dec. 2010)	O A	O B	O C	O D
10. Fever in Infants < 3 Mos. (Jan. 2011)	O A	O B	O C	O D
11. Minor Head Trauma (Feb. 2011)	O A	O B	O C	O D
12. Whooping Cough (March 2011)	O A	O B	O C	O D
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14. Oncologic Emergencies (May 2011)	O A	O B	O C	O D

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Please rate your level of satisfaction with the following items.
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26. Length of newsletter	○ A	○ B	○ C	○ D
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Pediatric Emergency Medicine Reports

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Pediatric Oncologic Emergencies

Initial Supportive Therapy in the Setting of Hyperleukocytosis

- Hydration: D5 1/2 NS at 3000 mL/m^2/day. No supplemental potassium should be given. Consider alkalinized fluids if allopurinol is being used and patient is not hyperphosphatemic.
- Treatment of hyperuricemia
  - Allopurinol 300 mg/m^2 divided TID
  - Consider administration of rasburicase if patient is hyperuricemic and not G6PD deficient.
- Transfuse platelets if platelet count is < 25,000/μL.
- Correct other significant coagulopathies.
- Avoid transfusion of packed red blood cells if hemodynamically stable.

NOTE: Management of hyperuricemia and tumor lysis covered more thoroughly in tumor lysis section.

Critical Airway: Relative Contraindications to Anesthesia in a Child with a Mediastinal Mass

- Orthopnea
- Upper body edema
- Dyspnea
- Clinical findings of impending respiratory failure
- Tracheal cross-sectional area < 50% normal for age and sex
- Severe compression of one or both mainstem bronchi
- Peak expiratory flow rate of < 50% predicted (performed in sitting and supine position)

Laboratory Studies and Clinical Monitoring

- CBC, calcium, phosphate, magnesium, uric acid, urea nitrogen, creatinine, lactate dehydrogenase at presentation and then every 6-12 hours depending on risk
- Sequential vital signs
- Strict assessment of intake and output
- Body weight once to twice daily
- Cardiorespiratory monitor with multi-lead ECG as needed for hyperkalemia (e.g., if K > 6 mEq/L, look for wide QRS and peaked T waves)
- Close clinical evaluation for signs of hypocalcemia or renal failure

Differential Diagnosis of a Mediastinal Mass by Location Within the Mediastinum

Anterior

- Lymphoma
- Leukemia
- Malignant germ-cell tumor
- Benign teratoma
- Thymic lesion (thymic hyperplasia, thymoma, thymic cyst)
- Substernal thyroid

Middle

- Lymphoma
- Tuberculosis
- Histiocytosis
- Sarcoidosis
- Anomalies of the great vessels

Posterior

- Neuroblastoma
- Ganglioneuroblastoma
- Sarcoma

Signs and Symptoms of SVCS

- Facial swelling
- Upper body edema
- Cyanosis of the face or upper body
- Plethora
- Conjunctival edema or suffusion
- Headache
- Tachycardia
- Elevated venous pressure
- Vocal cord paralysis, hoarseness
- Dyspnea
- Cough
- Decreased mentation
- Horner's syndrome

Symptoms Suggestive of a Brain Tumor as the Etiology of Headache

- Occipital location of headache
- Worsening symptoms
- Awakens patient at night
- Associated with focal symptoms
- Emesis

Indications for Specific Antimicrobial Therapy in Addition to Broad-spectrum Antibiotic

Indication	Antimicrobial Therapy
Clinical sepsis	Broad-spectrum antibiotic + vancomycin + aminoglycoside
High-dose cytarabine chemotherapy or mucositis or cellulitis or concern for CVC infection	Broad-spectrum antibiotic + vancomycin
Pulmonary infiltrates	Broad-spectrum antibiotic + vancomycin + atypical antibacterial agent; if infiltrates are diffuse and hypoxia is present, consider treatment for PJP (PCP)
Abdominal symptoms	Broad-spectrum antibiotic + vancomycin + aminoglycoside + metronidazole or clindamycin
Perirectal lesions	Broad-spectrum antibiotic + vancomycin + metronidazole or clindamycin

Prevention and Treatment of TLS

Delay and/or titrate cytolytic therapy (if possible) until prophylactic measures can be implemented (see "hydration therapy," "urinary alkalinization," "hyperuricemia management" below). AVOID intravenous radiologic contrast agents that might precipitate renal failure.	
Hydration therapy	• Administer IV fluid: D5 0.2% NaCl + $NaHCO_3$ 40 mEq/L at 3000 mL/m^2/day • Urine output goal: ≥ 100 mL/m^2/hour OR 3 mL/kg/hour (if body weight is < 10 kg) • Urine specific gravity goal ≤ 1.010 • Diuretics may be required (furosemide or mannitol)
Urinary alkalinization (see IV fluid above)	• Urine pH goal 6.5-7.5 to aid in excretion of uric acid Note urine pH > 7.5 may increase precipitation of CaPO4 and xanthine crystals • Adjust amount of $NaHCO_3$ in IV fluid to achieve urine pH and serum [sodium] goals
Hyperkalemia	• Moderate and asymptomatic (≥ 6.0 mEql/L): sodium polystyrene sulfonate orally • Severe and/or symptomatic (> 7.0 mEq/L): –Calcium gluconate 100-200 mg/kg IV –D25 (2 mL/kg) IV + regular insulin 0.1 units/kg IV –Dialysis may be necessary
Hyperphosphatemia	• Aluminum hydroxide 50-150 mg/kg/day divided in 4–6 doses (or 30–40 mL 6 to 8 hours) • Dialysis or continuous renal replacement therapy may be necessary
Hyperuricemia (uric acid crystals form in renal tubules and distal collecting system and cause oliguria)	• Allopurinol 300 mg/m^2 PO divided TID: Decreases production of uric acid by inhibiting xanthine oxidase If possible, start 12-24 hours before induction of cytolytic therapy **OR** • Urate oxidase (rasburicase): Metabolizes uric acid Should be considered in high-risk situations; contraindicated in G6PD deficiency, pregnancy, and lactation

Frequently Chosen Antibiotics for Febrile Neutropenia

Coverage	Antibiotic
Broad spectrum	Ceftazidime; cefepime; piperacillin-tazobactam; meropenem
Additional Gram-negative coverage	Amikacin; gentamicin; tobramycin
Additional Gram-positive coverage	Vancomycin
Additional anaerobic coverage	Clindamycin; metronidazole
Atypical bacterial coverage	Azithromycin; clarithromycin

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Trauma Reports

EVIDENCE-BASED MEDICINE FOR THE ED

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AHC Media

Trauma in Pregnancy

Resuscitation in the pregnant patient is an uncommon occurrence, estimated at 1 in 30,000 deliveries,[1] yet it is unique in its potential to save not one, but two lives. Trauma is estimated to occur in approximately 5% of pregnant patients,[2,3] and it is the leading cause of nonobstetric mortality in this population.[3,4] The physiologic changes of pregnancy, and the need to balance the care of mother and fetus, make the care of a critically injured pregnant patient a challenge for any physician. This article reviews physiologic changes of pregnancy and injuries unique to pregnancy, and discusses the assessment and management priorities of the pregnant trauma patient.

—The Editor

Physiologic Changes of Pregnancy

To effectively manage the injured pregnant patient, it is essential to understand a number of predictable changes in maternal physiology. These changes have the potential to impact vital signs, the physical examination, and results of laboratory studies. Most importantly, physiological alterations associated with a normal pregnancy have a significant effect on cardiopulmonary reserve and injury tolerance.

Respiratory. During pregnancy, the resting oxygen requirement and minute ventilation increase to meet increasing metabolic demands. At the same time, the functional residual capacity decreases by 20%,[5,6] due to elevation of the diaphragm by the gravid uterus. With greater oxygen demand, and less physical space for expansion of the lungs, the pregnant patient functions with a significantly decreased oxygen reserve.

While the pregnant patient will desaturate quickly, the fetus is even more vulnerable to hypoxia. The reason for this difference is that the umbilical vein and artery have a much lower partial pressure of oxygen than the maternal circulatory system. Maternal oxygenation is important for fetal well-being, as fetal oxygenation remains constant until maternal paO_2 drops below 60 mmHg.[7] As a general rule, the fetus has roughly two minutes of oxygen reserve. Animal studies reveal that significant maternal hypoxia results in a 30% reduction in uterine blood flow, further compromising fetal outcome.[8]

The pregnant patient is also at significant risk for aspiration. During pregnancy, progesterone acts to decrease gastrointestinal motility[6] and increase laxity of the lower gastroesophageal sphincter.[9] This effect, in concert with the anatomical compression of the stomach by the uterus, renders the pregnant patient more prone to aspiration. Increased and more frequent oral intake further increases the chance of aspiration.

Pregnancy is an edematous state. These changes affect the entire body, including the tongue and supraglottic soft tissues.[1,10] Capillary engorgement due to increased blood volume and decreased plasma oncotic pressure causes swelling of the respiratory tract mucosa and easy bleeding that may complicate intubation. (*See Table 1.*)

Cardiovascular. Several cardiovascular changes of pregnancy should be considered when caring for the pregnant trauma patient. Beginning in the eighth

Executive Summary

- Tachycardia and hypotension should be viewed as late signs of severe hemorrhage in the pregnant patient.
- Placental abruption can also occur after a minor mechanism of injury, with a rate as high as 5%. The absence of clinical findings in this setting does not reliably exclude the diagnosis.
- As little as 0.001 mL of Rh-positive fetal blood can cause maternal sensitization in the Rh-negative mother.
- Missed abdominal injuries are especially common in: patients with neurological impairment due to brain injury or alcohol; patients with multiple coincident injuries; and patients with severe or "distracting" injuries.

week of pregnancy, increasing progesterone causes smooth muscle relaxation and a significant decrease in total peripheral resistance. By week 12, blood pressure starts to gradually decline to a nadir around week 28, with a total systolic and diastolic blood pressure drop of 5 to 15 mmHg.[6,9] These effects are seen in central venous pressure as it drops 9 mmHg to around 4 mmHg in the third trimester.[11] Due to an increase in alpha receptors stimulated by estrogen within the myometrium, heart rate increases 10 to 15 beats per minute, and cardiac output increases to 30-50% above normal during the second trimester.[11] As gestation advances, maternal blood volume increases steadily, peaking at 40% above pre-pregnancy levels at term.

These changes, which help the pregnant patient tolerate the increasing metabolic demand of the fetus and prepare her for the expected hemorrhage of childbirth,[12] may easily conceal the presence of shock. Hemodynamic changes are often not apparent until 35% total blood volume loss.[11] Therefore, tachycardia and hypotension should be viewed as late signs of severe hemorrhage in the pregnant patient. To preserve maternal circulation during hemorrhage, blood is shunted away from the uterus and fetus via uteroplacental vasoconstriction,[6,11] making fetal distress, such as decelerations or low variability of the fetal heart rate (the fifth vital sign in obstetrics[6]), a subtle sign of compensated shock in the mother.

Table 1. Changes in Respiratory Physiology

Alteration	Implication	Action
• Upper airway edema	• Potentially difficult laryngoscope	• Protect the airway early • Preparation is essential
• Increased gastric pressure	• Increased aspiration risk	• Be cautious during RSI
• Elevation of the diaphragm	• Decreased FRC • Decreased respiratory reserve • Chest tube misplacement into the abdominal cavity	• Aggressively manage blunt and penetrating chest injury • Probe the hole with your finger before inserting a chest tube

When a pregnant patient is in a supine position, uterine compression of the great vessels (inferior vena cava, abdominal aorta, and iliac arteries) can cause a significant decrease in venous return. This aortocaval compression, or "supine hypotension syndrome," can result in a 30% decrease in cardiac output.[9] It also should be noted that the increased venous pressure caused by the gravid uterus may contribute to dangerous hemorrhage from lower-extremity wounds.[11] (*See Table 2.*)

Gastrointestinal. The anatomic changes that develop within the pregnant abdomen may hide significant injury despite a reassuring abdominal exam. As the uterus gradually enlarges, the stretched peritoneal cavity becomes less sensitive to irritation, and the attenuated rectus muscles may prevent guarding.[2,13] The spleen becomes engorged and is at greater risk of rupture. The small bowel is displaced cephalad, increasing the risk of bowel injury after penetrating trauma.[13,14] (*See Table 3.*)

Genitourinary. The most significant genitourinary change during pregnancy is the gradually enlarging uterus. During the first trimester, it remains encased and protected by the bony pelvis. By the 12th week of gestation, the uterus rises above the pelvic brim, becoming an abdominal organ. The fetus is small and well-cushioned by a large amount of amniotic fluid throughout the second trimester. In the third trimester, however, the uterus is larger and thin-walled, making it more

Table 2. Changes in Cardiovascular Physiology

Alteration	Implication	Action
• Decreased resting blood pressure • Increased resting heart rate	• Misinterpretation of vital signs • Delayed recognition of shock	• Monitor carefully • Obtain objective measures of perfusion (e.g., serum lactate)
• Uterine vasoconstriction during shock states	• Increased risk of fetal hypoxemia	• Early fetal monitoring • Fetal distress equals maternal hemorrhage until proven otherwise
• Compression of the inferior vena cava by the gravid uterus	Supine hypotension syndrome: • Decreased venous return • Impaired cardiac output	• Deflect the uterus to the left (or elevate one side of the back board) • Optimize resuscitation

Table 3. Changes in Gastrointestinal Physiology

Alteration	Implication	Action
• Peritoneal stretching	• Physical examination less reliable	• Bedside FAST • Lower threshold for imaging
• Splenic engorgement	• Increased risk of injury	• Bedside FAST • Lower threshold for imaging
• Viscera pushed cephalad by the enlarged uterus	• Increased risk of injury with penetrating upper abdominal wounds	• Lower threshold for imaging and consultation

susceptible to injury.[12,13] Further, the marked increase in uterine blood flow in late pregnancy potentiates rapid exsanguination following an injury to the uterus or uterine vessels.[14]

Toward the end of pregnancy, the fetal head drops into the pelvis and may be injured with maternal pelvic fractures.[9] The bladder is more susceptible to injury as the uterus pushes the bladder into the abdomen and out of the protective bony pelvic rim.

Pelvic radiographs may be difficult to interpret in later pregnancy as the pelvis changes in preparation for delivery. These changes include widening of the pubic symphysis and sacroiliac joint space. These areas, therefore, may appear abnormal on pelvic radiographs.

Renal changes include increased renal blood flow by 60% and increased glomerular filtration rate (GFR). As a result, serum creatinine decreases by half. Thus, a "normal" creatinine in a pregnant trauma patient is an important marker of renal impairment.[12] In addition, the increased GFR results in an increased urination frequency and renders urine output a poor indicator of shock.[13]

The elevated levels of progesterone in pregnancy stimulate the medullary respiratory center, increasing ventilatory drive. The physiologic effect is a decrease in pCO_2 to 25-30 mmHg, and a compensatory renal excretion of sodium bicarbonate to maintain a normal pH. As a result, maternal buffering capacity is impaired, placing the patient at greater risk for lactic acidosis following hemorrhage.[11] (*See Table 4.*)

Hematology. During pregnancy, there is a disproportionate increase in red blood cells by 20-30% and plasma volume by 50%, resulting in a "physiologic anemia of pregnancy." A normal hematocrit in pregnancy ranges from 31-34%.[11] Pregnancy also causes an increase in all coagulation factors and a decrease in fibrinolysis, creating a hypercoagulable state. This protects against hemorrhage but increases the risk of thromboembolism.[13] (*See Table 5.*)

Injuries Unique to Pregnancy

Placental Abruption. The uterus is made of elastic tissue that can respond to the acceleration and deceleration forces involved in blunt trauma. In contrast, the placenta does not contain elastic tissue and lacks the ability to expand and contract. As a result of the differing properties of these two adjacent structures, trauma may lead to a shearing force that causes a separation of the placenta from the uterus with bleeding into this space, known as placental abruption. Placental abruption is not uncommon and is noted in 40% of cases of severe maternal trauma.[6] In these cases, the rate of fetal demise can be as high as 60%,[6] making placental abruption the second most common cause of

Table 4. Changes in Genitourinary Physiology

Alteration	Implication	Action
• Increased uterine mass and blood flow	• Risk of exsanguination with major uterine injury	• Early sonographic assessment and OB/GYN consultation
• Increased GFR • Decreased BUN/Cr • Increased urine output	• A "normal" BUN/Cr indicates renal dysfunction • Urine output is not a reliable guide of tissue perfusion	• Understand laboratory changes • Use objective measures of resuscitation (e.g., serum lactate)
• Respiratory alkalosis and compensatory excretion of bicarbonate	• Limited serum buffering capacity	• Optimize resuscitation • Assess and treat metabolic acidosis
• Pelvic symphysis during late pregnancy	• Pelvic radiographs can be difficult to interpret	• Consider pelvic injury in the right clinical setting

Table 5. Changes in Hematologic Physiology

Alteration	Implication	Action
• 20-30% increase in red cell mass • 40-50% increase in plasma volume	• Physiologic anemia • Normal Hct 31-34%	• Understand laboratory changes
• Hypercoagulable state	• Increased risk of thromboembolic complications	• Employ DVT prophylaxis

fetal death in trauma after maternal injury.

Concern should be high for placental abruption in the patient with abdominal pain, uterine contractions, back pain, or vaginal bleeding. A rigid uterus or "large for dates" fundal height is highly suggestive of the diagnosis. When uterine abruption is strongly suspected, uterine sonography and cardiotocographic monitoring are recommended. Ultrasound is highly specific for placental abruption, but has a sensitivity of only 50%. This is especially true for small abruptions and those located along the posterior wall of the uterus.[3] Abnormalities on fetal monitoring are a highly sensitive marker of fetal distress in the setting of placental abruption, although these findings are nonspecific. The complementary use of these two tools is logical in the ED evaluation of placental abruption. (*See Figures 1 and 2.*)

Placental abruption can also occur after a minor mechanism of injury, with a rate as high as 5%.[3] The absence of clinical findings in this setting does not reliably exclude the diagnosis. For stable patients with a viable pregnancy suffering minor trauma, 4-6 hours of continuous fetal monitoring are recommended.

Uterine Rupture. During pregnancy, the uterus enlarges to accommodate the growing fetus. As this occurs, the uterus extends out of the pelvis and into the abdomen, where it is much more susceptible to trauma. Uterine rupture occurs in less than 1% of pregnant trauma patients.[10] While this is a rare event, it almost always results in fetal demise, and carries a 10% maternal mortality rate.[10] Uterine rupture most commonly occurs in patients who have had a previous cesarean section. Patients will typically present with severe abdominal pain, clinical signs of peritonitis, and hemodynamic instability. Often, fetal parts or uterine irregularity can be palpated on abdominal exam. The combination of shock and an acute abdomen in a pregnant trauma patient should prompt immediate evaluation for uterine rupture, in addition to the traumatic abdominal injuries seen in the general population.

An abdominal radiograph will typically show an uncoiled fetus, and this diagnosis may be confirmed by ultrasound. As time to diagnosis and definitive surgical treatment is critical, time-consuming diagnostic studies should be avoided. The diagnosis of uterine rupture should prompt immediate actions to stabilize the mother and obtain emergent obstetrical consultation.

Fetal-Maternal Hemorrhage. Fetal-maternal hemorrhage occurs when injury provokes the mixing of fetal and maternal blood. This becomes clinically relevant when Rh-positive fetal blood is introduced into the circulation of an Rh-negative mother. As little as 0.001 mL of Rh-positive fetal blood can cause maternal sensitization in the Rh-negative mother.[9] If

Figure 1. Marginal Abruption

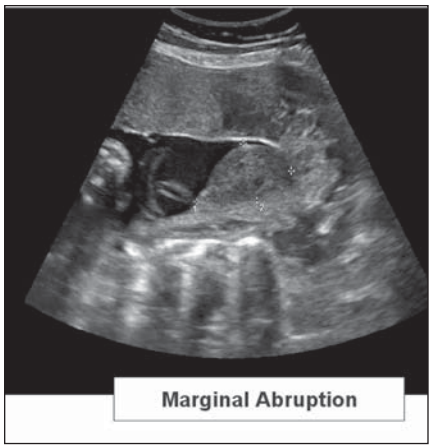
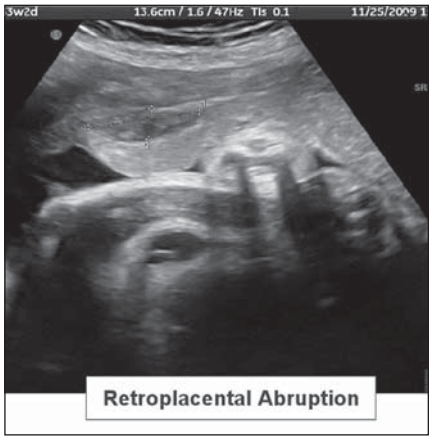


Figure 2. Retroplacental Abruption



sensitization occurs, future pregnancies could be complicated by erythroblastosis fetalis, resulting in fetal anemia, hypoxia, and even death. Rh status should be determined in all pregnant trauma patients unless the injury is isolated and remote from the uterus (for example, a penetrating injury to the extremity). Universal determination is particularly important, as injury severity is not related to the incidence of fetal-maternal hemorrhage.

Immune globulin should be administered to all Rh-negative trauma patients within 72 hours of injury. During the first trimester, a dose of 50 mcg may be used instead of the traditional dose of 300 mcg, which is used after 13 weeks gestation. If significant fetal-maternal hemorrhage (> 30 mL) is suspected, higher doses of immune globulin may be required. In these cases, a Kleihauer-Betke test may be useful to quantify the degree of transplacental hemorrhage. Recent data also suggest that a positive Kleihauer-Betke test is correlated with uterine contractions and with risk for preterm labor following trauma.[15] Patients with a positive Kleihauer-Betke test, therefore, may require more extensive monitoring following trauma.

Assessment and Management

Initial Assessment. The assessment of the injured pregnant patient should be nearly identical to that of the injured nonpregnant patient, with the following important exceptions:

1. The pregnancy must be immediately recognized. This is especially relevant in women who may not realize they are pregnant and in those unable to communicate due to head injury, intoxication, severe respiratory distress, or profound shock. When pregnancy is not clinically obvious, a urine pregnancy test should be done in 100% of injured women of childbearing age. When pregnancy is clinically obvious, the assessment of the fetus occurs during the secondary survey.
2. The normal changes in physiology resulting from the pregnancy must be interpreted in the context of injury.
3. The ability of the pregnant mother to tolerate and respond to specific injuries must be understood.
4. The team must not be distracted by an obviously gravid uterus when a focused assessment and critical interventions are needed to stabilize the mother.
5. Once the mother has been assessed, an appraisal of fetal viability should be performed.

Unique features of the primary assessment and the secondary survey are outlined below.

Airway. The physiologic and anatomic changes of pregnancy significantly affect the respiratory system and airway management. These changes necessitate aggressive airway management and preparation for a potentially difficult airway. All steps should be taken to maximally oxygenate mother and fetus. During advanced stages of pregnancy, the airway will be difficult for two reasons: the upward excursion of the uterus reduces lung volumes, functional residual capacity, and respiratory reserve; and increased intragastric pressure increases the risk of regurgitation. When intubating the pregnant trauma patient, precautions for both aspiration and cervical spine injury should be taken.

Rapid sequence intubation (RSI) is the preferred method for intubation. Traditional RSI medications are safe in pregnancy in the absence of other contraindications. Both depolarizing and non-depolarizing paralytic agents cross the placenta and may result in a flaccid, apneic infant if immediate delivery were to ensue.

Once paralyzed, rapid desaturation is the rule rather than the exception. This is worsened in the presence of acute chest injury. As such, all trauma airways in the pregnant patient should be considered high-risk. To decrease this risk of aspiration, cricoid pressure is vital during airway management.[9] Keep in mind that a smaller endotracheal tube than usual may be necessary for successful intubation in the setting of edematous periglottic tissue. Once intubation is accomplished, gastric decompression should be performed to decrease the risk of aspiration.

Breathing. For the reasons outlined above, significant trauma in the second or third trimester of pregnancy is associated with a potentially compromised respiratory status. With limited respiratory reserve, blunt or penetrating chest injuries are not well tolerated. The goal of the physical examination is to identify clinical findings suggestive of chest injury, i.e., tachypnea or respiratory distress, external chest trauma, and clinical signs of rib fractures, flail segments, pneumothorax, or hemothorax. The gradual 4 cm elevation of the diaphragm by the growing uterus is important to remember when placing a chest tube. It should

be inserted one to two intercostal spaces higher than standard to avoid diaphragmatic, liver, or spleen injury. Prior to insertion, the chest-tube tunnel should be digitally palpated to ensure proper placement.[7]

Circulation. In the first trimester, the assessment of circulation is unchanged. In the second and third trimesters, predictable increases in blood volume will mask the typical response to hemorrhage, i.e., hypotension and tachycardia. In essence, the maternal "tank" is over-filled; thus, proportionally larger volumes of blood loss will be required to mount a detectable cardiovascular response. It is equally important to understand that while maternal heart rate climbs in the third trimester, tachycardia (a heart rate ≥ 100 bpm) is not a normal finding.

Large-bore IV access should be obtained as in any trauma patient, and aggressive resuscitation with crystalloid should be initiated if shock is either evident or suspected. If hemodynamic compromise continues despite this, transfusion of type-specific blood is recommended. If time does not allow for this, however, type O, Rh-negative blood should be used.

Vasopressor therapy should be avoided whenever possible. The disadvantage of vasopressor therapy in the pregnant patient is that this compromises perfusion to the uteroplacental unit and, therefore, the fetus.[10] If aggressive volume replacement and blood products are ineffective, vasopressor therapy should be initiated at the lowest possible dose to maintain maternal perfusion. Ephedrine and mephentermine offer a theoretical advantage, as these agents do not compromise uterine perfusion to the same extent that epinephrine and norepinepherine do.[10]

When hypotension is noted in a patient who is greater than 20 weeks gestation, attempts should be made to displace the uterus from the great vessels and restore cardiac output. This is typically achieved by placing the patient in the left lateral decubitus position. In circumstances where spinal precautions must be maintained, displacement of the gravid uterus from the vena cava may be achieved with manual displacement or by raising the backboard to a 15- to 30-degree angle.[11] Manual displacement is achieved by manually lifting the uterus and displacing it to the left and toward the patient's head.

If CPR is required, chest compressions should be modified in the pregnant patient. These patients require deeper compressions (1.5-2 inches) with more force than the general population. This is due to decreased chest-wall compliance caused by elevation of the diaphragm. The position of chest compressions also should be shifted from the traditional mid-sternum to slightly above the mid-sternum. Standard ACLS protocols for pharmacologic agents and defibrillation should be followed in the pregnant patient. Electrical therapy with standard joule delivery has never been found to have injurious effects on the fetus.[1,16]

When obtaining central venous access, lower-extremity sites should be avoided if possible. Drugs administered to sites below the uterus will have difficulty reaching the central circulation due to aortocaval compression.

The Secondary Survey. The secondary survey should be done in the same order and with the same cadence as in any other trauma patient. A methodical head-to-toe examination will help identify injuries posing a threat to both patients.

The examination of the abdomen is especially relevant. Severe tenderness may reflect an injury to maternal viscera or to the enlarged uterus. A large-for-dates uterus may occur following uterine abruption and retroplacental hemorrhage. (*See Figure 2.*) Palpation of fetal parts outside the uterus reflects uterine rupture.

In the pregnant patient suffering severe multisystem trauma, the pelvic examination should not be overlooked. The exam may reveal vaginal bleeding, rupture of membranes, or vaginal lacerations resulting from pelvic-ring fractures.

It is important to recognize that the physical assessment of the trauma patient has limitations. For example, large studies describe a 5% to 10% rate of "occult" abdominal injuries when patients are evaluated with physical examination alone.[17-19] Missed abdominal injuries are especially common in: patients with neurological impairment due to brain injury or alcohol; patients with multiple coincident injuries; and patients with severe or "distracting" injuries. It is logical that the same would apply to the pregnant trauma patient.

Fetal Assessment. Fetal assessment is initiated during the secondary survey of the pregnant trauma patient. Initially, the presence of fetal cardiac activity should be confirmed

Figure 3. Fetal Monitoring Strip

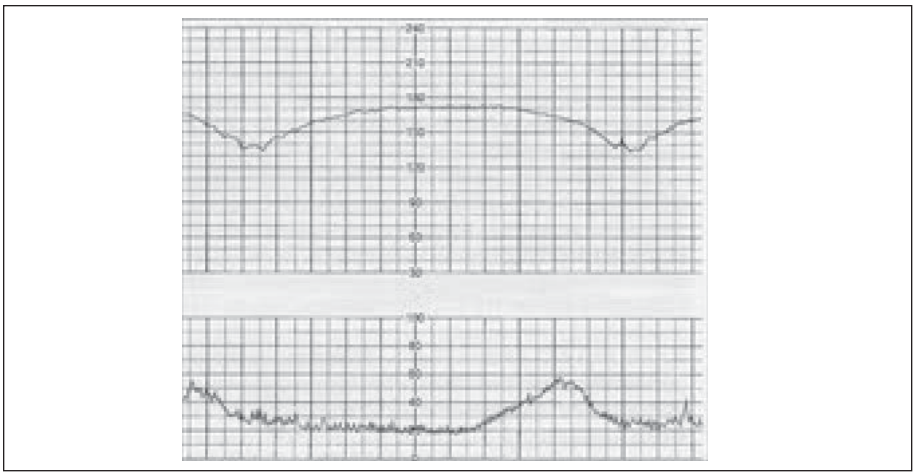


Figure 4. Fetal Monitoring Strip

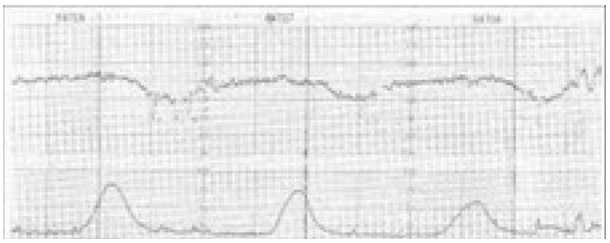


Table 6. Medications[20,21]

	Resuscitative Medications	Analgesics	Sedatives/ Paralytics	Other
USED				
	Magnesium (B)	Acetaminophen (B)	Rocuronium (B)	Ondansetron (B)
	Atropine (C)	Oxycodone (B)	Vecuronium (B)	Cefazolin (B)
	Epinephrine (C)	Fentanyl (C)	Propofol (B)	Promethazine (C)
	Etomidate (C)	Morphine (C)	Succinylcholine (C)	
	Lidocaine (C)	Hydromorphone (C)		
	Bicarbonate (C)			
	Dopamine (C)			
	Dobutamine (C)			
	Adenosine (C)			
	Bretylium (C)			
AVOIDED				
	Ketamine (D)			

by Doppler ultrasound. Once this has been established, if a fetus is viable (> 23 weeks gestation), continuous external fetal monitoring should be initiated. Fetal monitoring can be useful for both fetal and maternal assessment, as fetal distress is often an early marker of impending maternal hemodynamic compromise. When significant hemorrhage or hypotension occurs, blood is first shunted away from the uteroplacental unit,[1] and fetal distress may be seen before maternal vital signs become abnormal. It is for these reasons that the fetal heart rate is often considered the "fifth vital sign" in the pregnant patient.[10]

Normal fetal heart rate varies between 120 and 160 beats per minute. Fetal distress can be manifested by bradycardia, tachycardia, loss of beat-to-beat variability, or heart-rate decelerations. A fetal monitoring strip showing a late deceleration is shown in Figures 3 and 4.

Assessment of Gestational Age. During the secondary survey, palpation of the fundal height provides a crude assessment of whether the fetus is viable outside of the uterus (≥ 20 weeks gestation) or unlikely to survive outside the uterus (≤ 20 weeks gestation; "pre-viable"). A fundal height at or above the umbilicus identifies a fetus at greater than or equal to 20 weeks gestation. Bedside sonography with measurement of crown-rump length and/or biparietal diameter is a useful adjunct when making this determination.

Pharmacology

Acknowledging limited data, most medications used in the trauma setting have not been proven to be harmful in the human fetus. The pharmacologic strategy used during the resuscitation of the pregnant patient should follow standard American Heart Association guidelines. Successful resuscitation of the mother provides the greatest potential for fetal survival. The following are the safety categories in pregnancy:

A: Controlled studies in pregnant women fail to demonstrate a risk to the fetus in the first trimester with no evidence of risk in later trimesters. The possibility of harm appears remote.

B: Animal studies show no risk or adverse fetal effects, but controlled, human, first-trimester studies are not available; no evidence of second- or third-trimester risk; fetal harm possible but unlikely.

C: Animal studies show adverse fetal effects, but no controlled human studies OR no animal or human studies; weigh possible fetal risk vs. maternal benefit.

D: Positive evidence of human fetal risk; maternal benefit may outweigh fetal risk in serious or life-threatening situations. (*See Table 6.*)

Imaging in Pregnancy

Choosing appropriate imaging modalities for the pregnant patient can be anxiety-provoking for the emergency physician (EP) due to concerns about fetal radiation exposure. The imaging strategy should carefully balance the need to rapidly detect and prioritize injuries with the radiation dose required to achieve this goal. Because trauma is the leading non-obstetric cause of maternal death, fast, accurate diagnostics are imperative for both maternal and

fetal well-being. Ultimately, injury detection takes priority, and concerns about radiation exposure should neither deter nor delay radiographic imaging in the pregnant trauma patient.[22,23]

Effects of Ionizing Radiation Exposure to the Fetus. The effects of ionizing radiation exposure to the fetus depend on gestational age and radiation dose. Most effects are seen at doses that far exceed those typically used for diagnostic imaging. See Table 7 for an overview of the estimated mean fetal-absorbed dose from various radiologic studies. Before implantation (0-2 weeks after conception), there is an all-or-none risk of either death of the embryo or no consequence at all at threshold doses 50-100 mGy.[22,24] There is some variable evidence to suggest increased risk of childhood leukemia by a factor of two with a single pelvic CT scan within the first two weeks after conception, but the increase in absolute risk is very low.[25] During organogenesis (2-8 weeks after conception), congenital anomalies are seen at 200 mGy and growth retardation at 200-250 mGy. At 8-15 weeks, the risk of severe mental retardation is seen in doses of 60-310 mGy, and at 16-25 weeks in doses of 250-280 mGy.[22]

Computed Tomography. Computed tomography (CT) is often the imaging modality indicated in trauma evaluation, and this imaging should be pursued without hesitation, although with efforts to use the lowest dose possible to achieve necessary information. When the fetus is out of the imaging field, such as with CT of the head, cervical spine, chest, and extremities, the radiation exposure to the fetus is low, and these images can be safely obtained during any trimester of pregnancy. Because the fetus is in the direct line of radiation for CT of the abdomen and pelvis, the theoretic risk to the fetus is greater, and the EP should work with the radiologist to minimize the radiation dose.[23,24] When possible, studies such as ultrasonography and magnetic resonance imaging (MRI) that do not have ionizing radiation should be used for the pregnant patient.[22] Shielding of the fetus should be performed for all radiographs, with the exception of pelvic X-rays. Careful attention should be paid to avoid radiographic redundancy.[25]

Table 7. Imaging and Fetal Exposure Levels[22,23,26,27]

Modality	Fetal Exposure
Chest X-ray	0.0002-0.0007 mGy
Abdominal X-ray	1 mGy
Pelvis X-ray	2 mGy
CT Scan Head or Chest	< 10 mGy
CT Scan Abdomen or Pelvis	35 mGy

Focused Assessment with Sonography for Trauma (FAST). Ultrasound is not associated with known adverse fetal effects.[23] Both the technique and the areas of fluid accumulation are the same. The sensitivity and specificity for detection of intra-abdominal injury in the pregnant patient range from 61-83% and 94-100%, respectively.[22] Given the possibility of false negatives, a CT scan is often needed for further examination.[28]

Concluding recommendations from the 2010 EAST practice management guidelines work group as published in *The Journal of Trauma* are as follows:

"Level II: clinical studies in which data were collected prospectively and retrospective analyses that were based on clearly reliable data. Types of studies so classified include observational studies, cohort studies, prevalence studies, and case-control studies.

1. Concern about possible effects of high-dose ionizing radiation exposure should not prevent medically indicated maternal diagnostic X-rays whenever possible.

2. Exposure to < 5 rad has not been associated with an increase in fetal anomalies or pregnancy loss and is herein deemed to be safe at any point during the entirety of gestation.

3. Ultrasonography and magnetic resonance imaging are not associated with known adverse fetal effects. However, until more information is available, magnetic resonance imaging is not recommended for use in the first trimester.

4. Consultation with radiology should be considered for the purposes of calculating estimated fetal dose when multiple diagnostic radiographs are performed."[25]

Special Considerations

Perimortem Cesarean Section. A perimortem cesarean section should be performed in cases of traumatic arrest if there is a potentially viable fetus (> 23 weeks). It should be performed by a provider trained and credentialed to perform the procedure. If gestational age is unknown, a rapid method to estimate gestational age is to assume viability if the fundus is palpable above the level of the umbilicus. A bedside ultrasound also may be used to rapidly assess for fetal cardiac activity if immediately available; however, this should not delay the procedure. A study assessing fetal outcome in 33 infants following perimortem cesarean section found that there were survivors as early as 26 weeks gestation, and that none of the 13 infants delivered despite absent fetal heart tones survived.[29] This suggests that a rapid assessment for gestational age and fetal heart tones can help the physician determine if a patient is appropriate for perimortem cesarean section.

A perimortem cesarean not only allows for further resuscitation of the infant, but it also relieves uterocaval compression; this increases maternal venous return and cardiac output. In this situation, return of maternal

circulation may also occur. CPR and ACLS protocols should be continued during the procedure. Ideally, perimortem cesarean section should begin within 4 minutes of maternal cardiac arrest, with a goal of delivery within 5 minutes of maternal cardiac arrest. This timing is recommended because improved fetal outcome has been demonstrated when delivery is within 5 minutes of maternal cardiac arrest.[30,31] Data from Katz's study demonstrate that 70% of infants who survived perimortem cesarean section were delivered within 5 minutes of maternal arrest. However, there have been case reports of return of spontaneous maternal circulation and normal fetal neurologic outcome after more than 15 minutes following maternal arrest, so arrest time greater than 5 minutes is not a contraindication to perimortem cesarean delivery.[32]

Domestic Violence During Pregnancy. When caring for the pregnant trauma patient, careful consideration should be made for the possibility of physical abuse. Domestic violence rates are increased during pregnancy, as this is frequently a time of both emotional and financial strain. The rate of violence during pregnancy is estimated at 10-15%.[3] Furthermore, the vast majority of physical assaults on pregnant patients are perpetrated by boyfriends and spouses, and are likely to be significantly underreported. Assaults on pregnant patients tend to recur and to increase in severity throughout the pregnancy. Common areas of injury during pregnancy include the abdomen, breasts, and genitals.[32] Other warning signs include frequent office or emergency department visits, depression, substance abuse, and a history inconsistent with injury. It is recommended that all pregnant trauma patients be screened for domestic violence. If possible, screening should be performed without the presence of partners or family members.

Burns. Approximately 7% of women seen for the treatment of burns in the United States are pregnant. Most of these events occur in the workplace.[26] Fetal outcomes are closely linked to burn severity. As the total body surface area (TBSA) of the burn increases, so does the risk of poor fetal outcomes and fetal death. In general, uncomplicated burns of less than 20% TBSA will have little effect on fetal well-being. Burns greater than 30% can lead to fetal distress and premature labor. Fetal survival is uncommon when burns exceed 60% TBSA.[34]

There are insufficient data in the literature to develop specific guidelines for the management of burns in the pregnant patient. Most recommendations are based on small case series or are extrapolated from nonpregnant patients. With that in mind, several recommendations can be made with regard to airway management and fluid resuscitation.

In the patient suffering major burns and/or smoke inhalation, careful and early attention to airway management is of paramount importance. In the third trimester, tidal volume and minute ventilation both increase by 30% to 50%. As such, impaired ventilation and gas exchange due to upper airway edema, inhalation injury, or concomitant chest injury can profoundly impair maternal and fetal physiology.

RSI is the technique of choice when intubation is required. Because gravid patients desaturate rapidly following paralysis, careful preparation and planning are fundamental. It is important to recall that acute burns are not a contraindication to the use of succinylcholine as the preferred neuromuscular blocking agent. Conversely, succinylcholine should be avoided in subacute burns (i.e., ≥ 72 hours old) to avoid drug-induced hyperkalemia. When upper-airway burn edema threatens the success of laryngoscopy, alternative techniques should be considered as the primary approach, or immediately available as part of a "double set-up."

Use of the Parkland formula (*see Table 8*), which has not been validated in the pregnant patient, is controversial. Pacheco and colleagues[35] argue that predictable physiologic changes occurring during pregnancy create an inherent risk of underresuscitation if the Parkland formula is used: pregnancy is a hyperdynamic state with an increase in cardiac output, a drop in systemic vascular resistance, and increased overall metabolic demands; intravascular volume requirements increase by 50% at term; decreases in colloid osmotic pressure increase tissue fluid extravasation; and total body surface area is increased, promoting burn-surface fluid loss. Without outcome data or guidance to the contrary, the Parkland formula seems like a logical starting point. To ensure adequate resuscitation, ongoing monitoring of vital signs, urine output, cardiotocographic monitoring, and objective measures of tissue perfusion (e.g., serial serum lactate or base deficit) is important.

Burn cleansing and debridement should be done in standard fashion. Topical antibiotics, such as bacitracin and silver sulfadiazine, and biosynthetic dressings (e.g., Biobrane®, TransCyte®, Aquacel®) are not associated with fetal malformations and are considered safe in pregnancy.[36]

Victims of major burns, and all those burned in an enclosed space, should be assessed for carbon monoxide (CO) poisoning. Because fetal hemoglobin avidly binds CO, hyperbaric oxygen is recommended for all symptomatic pregnant patients and asymptomatic patients with a venous CO level greater than 15%.[37]

Electrical Injury. Traditional teaching emphasizes that even low-voltage electrical injuries are

Table 8. Parkland Formula[26,34]

- **Fluid requirement = TBSA burned (%) × Weight (kg) × 4 mL**
- **Half given in the first 8 hours and half over the next 16 hours**
- **Normal saline or lactated Ringer's solution**

associated with a significant risk of poor fetal outcomes and fetal demise.[38-40] Pathophysiologically, this is attributable to the high conductivity of amniotic fluid, putting the fetus at risk when the offending current "crosses" the uterus. There is no debate that major electrical injuries, lightning strikes, and Taser-gun injuries pose a major risk.[41,42]

A review of 31 patients by Einarson and colleagues points out that most electric injuries in pregnancy are due to 110 V household current, with no proven difference in outcomes.[43] Given the lack of certainty on this issue, it seems reasonable to assess fetal well-being immediately in all pregnant patients suffering electric injury. In the absence of fetal distress and maternal indications for admission (loss of consciousness, persistent neurological symptoms, history of cardiac disease, abnormal maternal ECG), a 4-hour period of fetal monitoring is reasonable. With most significant maternal injuries or any signs of fetal distress, immediate obstetric consultation and a longer period of maternal and fetal monitoring are indicated.

Conclusion

Management of the injured pregnant patient presents unique challenges to the emergency physician. A thorough knowledge of the differences between a pregnant and a nonpregnant trauma patient is imperative to adequately care for the pregnant trauma patient. Ultimately, maternal stabilization should be the primary focus to ensure the best possible outcome for both mother and fetus.

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CME/CNE Questions

1. Which findings on fetal monitoring indicate fetal distress?
   A. bradycardia (HR < 120)
   B. tachycardia (HR > 160)
   C. reduced variability
   D. all of the above
2. Which of the following is one of the physiologic changes of pregnancy?
   A. decreased heart rate
   B. increased cardiac output

C. decreased respiratory rate
D. increased lung capacity

3. Which of the following statements is true regarding Rh immune globulin (RhoGAM)?
   A. It must be administered within 24 hours of trauma.
   B. It should be administered after even minor abdominal trauma.
   C. It is effective up to 72 hours following trauma.
   D. B and C are true.

4. Which medication should be avoided during resuscitation in the pregnant patient?
   A. rocuronium
   B. morphine
   C. ketamine
   D. etomidate

5. A 33-year-old G1P0 pregnant patient at 30 weeks gestation presents as a belted driver in a low-speed motor vehicle crash. After assessment of the patient reveals only a knee contusion, a bedside ultrasound confirms a fetal heart rate of 143. Which of the following is correct regarding patient disposition?
   A. The patient can be safely discharged home.
   B. Further ultrasound imaging is required to evaluate for placental abruption.
   C. Continuous fetal monitoring is required for a period of 4-6 hours.
   D. The patient should be admitted to the OB service for 24-hour observation.

6. A 24-year-old G2P1 with a pregnancy of unknown gestational age presents to the emergency department intubated for respiratory distress after a motor vehicle crash. What is the quickest way to estimate gestational age in this setting?
   A. bedside ultrasound for crown-rump length
   B. calculation of biparietal diameter
   C. palpation of the fundal height
   D. rapid retrieval of records from the obstetrician

7. Which of the following should be considered when ordering imaging tests in the pregnant patient?
   A. Consult with a radiologist to ensure lowest radiation dose.
   B. High-dose ionizing radiation should not be used despite the need for maternal diagnostics.
   C. The FAST exam can rule out intra-abdominal injury.
   D. Exposure to < 5 rad has been associated with fetal anomalies.

8. A 29-year-old G3P2 at 30 weeks gestation presents to the emergency department with complaint of falling down the stairs and injuring her left arm. Which of the following should prompt concern for domestic abuse?
   A. frequent office or emergency department visits
   B. history of depression or substance abuse
   C. a history inconsistent with the injury
   D. all of the above

9. Which of the following are necessary changes of ACLS for a pregnant patient?
   A. Medication dosages must be adjusted.
   B. Defibrillation joules should be decreased.
   C. Chest compressions should be deeper and more cephalad.
   D. All of the above are necessary.

10. Which of the following is true regarding electrical injuries in pregnant patients?
    A. Most electrical injuries in pregnant patients are due to 110 V household current.
    B. Amniotic fluid has low conductivity of current.
    C. Lightning strikes to the mother pose minimal risk to the fetus.
    D. All of the above.

Answers: 1. D; 2. B; 3. D; 4. C; 5. C; 6. C; 7. A; 8. D; 9. C; 10. A

CNE/CME Objectives

Upon completing this program, the participants will be able to:

- discuss conditions that should increase suspicion for traumatic injuries;
- describe the various modalities used to identify different traumatic conditions;
- cite methods of quickly stabilizing and managing patients; and
- identify possible complications that may occur with traumatic injuries.

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Physicians and nurses participate in this continuing medical education/continuing education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to evaluate their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. *After completing this activity, you must complete the evaluation form that will be provided at the end of the semester and return it in the reply envelope provided to receive a credit letter.* When your evaluation is received, a credit letter will be mailed to you.

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