Onset of NEC is typically during the first several weeks after birth when feeds have been started, and the age of onset inversely related to gestational age at birth. Early in the disease process, neonates can exhibit signs of feeding intolerance with vomiting, increased gastric aspirates, bile-tinged (green) gastric aspirates, or decreased bowel sounds with abdominal distention and tenderness. Gross or occult blood can be present in stools indicating mucosal injury. Many of these signs are non-specific and can occur with other disorders. Progression of NEC results in systemic signs such as lethargy, long pauses in breathing called apnea, temperature instability, and poor perfusion (pumping of fluid through an organ or tissue.) Ultimately this can lead to respiratory failure and cardiovascular collapse requiring mechanical ventilation and vasopressors. A palpable mass and erythema (abnormal redness of the skin due to capillary congestion, as in inflammation) of the abdominal wall is indicative of a more advanced disease process. NEC affects 5 to 10% of premature infants born weighing less than 1500 g. Among the risk factors defined for NEC, prematurity and birth weight remain inversely related to risk for NEC. Term infants who develop NEC usually have specific risk factors such as congenital heart disease, sepsis, and low blood pressure. Many other gastrointestinal diseases can mimic NEC, so when evaluating a patient it is important to consider and treat alternative etiologies. Sepsis can cause an ileus with abdominal distention and feeding intolerance. Premature infants, especially those on indomethacin or steroids can also present with spontaneous intestinal perforation (SIP). SIP is characterized by isolated perforation of the distal small bowel. Other causes of severe abdominal distention in neonates include bowel obstruction from Hirshsprung's disease, small bowel atresia, meconium ileus, and malrotation with volvulus. NEC can also be mistaken for allergic enterocolitis secondary to cow's milk protein allergy. NEC is diagnosed clinically and radiographically. Once clinical suspicion arises, an abdominal X-ray is performed as an initial evaluation. This is repeated serially depending on acuity and clinical course to assess disease progression. Characteristic findings on NEC process on abdominal radiographs include pneumatosis intestinalis (air in the intestinal wall), abnormal persistent dilated loops, thickened bowel wall, pneumoperitoneum and portal vein gas. Pneumoperitoneum defined as abdominal free air is a surgical emergency indicating bowel perforation and usually requires intervention. Abdominal ultrasonography can also be used to evaluate for free fluid in the abdominal cavity or abscess formation. Addition laboratory studies to evaluate severity of NEC include a blood culture, coagulation studies and complete blood count with manual differential to assess for leukocytosis with bandemia, neutropenia, anemia and thrombocytopenia. Blood gases are checked serially to assess severity of acidosis and need for respiratory support or to assist with fluid management.