In this article, the author presents a review of pulmonary aspiration which encompasses the pathophysiology, treatment and prevention of this potential anesthesia complication.

One of the main complications associated with anesthesia is aspiration of gastric contents into the tracheobronchial tree. The morbidity and mortality will vary widely depending on the volume and, more particularly, on the chemical nature of the fluid aspirated.

Aspiration may be the result of either vomiting or regurgitation. Regurgitation is a passive process which is dependent on a pressure gradient between the stomach and oropharynx. Vomiting is an active reflex and is heralded by certain warning signs. Regurgitation is silent and hence more sinister in that there are no active signs initially to warn the clinician of the impending danger. Studies by Culver and associates and by Benson and Adriani suggest that a small percentage (16-27%) of anesthetized patients will, under ideal circumstances, show evidence of contaminant material in the tracheobronchial tree. This may occur as the result of channeling effects with a cuffed endotracheal tube in place but the incidence is low.

Normally, the epiglottis, vocal cords, and cough reflex all serve to protect the lungs from contamination by foreign material. These protective mechanisms are reduced in efficiency or made ineffective by numerous disease states or iatrogenically produced states. These include: anesthesia, drug overdose, coma, prolonged intubation, tracheostomy, hiatal hernia, increased intracranial pressure, intestinal obstruction, and gross obesity.

The aspirate

The nature of the aspirate can include gastric contents, blood, purulent material, fresh or salt water, and antacids. Aspirations are most often classified according to their pH. Mendelson was the first to stress the importance of acidity in determining the extent of pulmonary injury when he described the clinical course of 66 women who aspirated stomach contents during labor and delivery. He concluded that acid was the primary cause of the subsequent pulmonary injury. As a result of his work, the clinical syndrome of severe aspiration injury has been termed “Mendelson’s syndrome.”

Subsequent researchers have defined the critical level at which severe lung damage occurs—a pH value of less than 2.5 and a volume of 4ml/kg. Recently, Bond reported a case of gastric aspiration (pH 6.4) in which the sequelae of Mendelson’s syndrome occurred. The suspension material of an antacid was questioned as being the causative factor.

Pathology and physiology

After aspiration, the acid is rapidly distributed throughout the lungs, and damage occurs im-
mediately. Atelectasis can become extensive within three minutes. Pathologic examination within the first few hours reveals epithelial degeneration of the bronchi, pulmonary edema and hemorrhage. With electron microscopy, necrosis of type I alveolar cells and the presence of free lamellated inclusion bodies in the pulmonary transudate are noted.

Within four hours, there is an acute infiltration of polymorphonuclear cells, and fibrin can be seen in the alveolar space. Degeneration of type II alveolar cells leads to decreased surfactant and decreased pulmonary compliance. Glauser concluded that an aspirate with a pH of less than 2.5 increased alveolar epithelial permeability with concomitant exudation of intravascular liquid and protein into terminal air exchange units.

During the next 24-36 hours, marked polymorphonuclear infiltration occurs, resulting in alveolar consolidation. Damage to the airways in turn causes mucosal sloughing. After 48 hours, gross examination reveals a boggy, edematous, and hemorrhagic lung.

The severe chemical burn instigated by acid aspiration causes loss of alveolar capillary integrity and exudation of fluid and protein in the alveoli and bronchi. The accompanying loss of intravascular volume may cause severe hypotension.

Hypoxia can happen within minutes of acid aspiration and has multiple causes. One is that airway closure takes place as a result of acid aspiration. A decrease in surfactant activity occurs, leading to alveolar instability and atelectasis. The outpouring of fluid and protein causes interstitial and alveolar edema, resulting in further airway closure. Finally, alveolar hemorrhage and consolidation occur, followed by hyaline membrane formation. All these conditions contribute to the large alveolar-arterial oxygen difference (AVO₂) and increased physiological shunting.

Acid aspiration also causes pulmonary vascular changes. Initially, pulmonary artery pressure rises rapidly but then falls quickly due to the decreased cardiac output associated with the loss of intravascular volume. On the other hand, pulmonary vascular resistance is elevated by hypoxic vasoconstriction or anatomic obstruction.

In contrast to acid aspiration, the aspiration of large particles such as food can rapidly become fatal. Severity of reaction varies depending on the size of the particle and its composition. With aspiration of particulate matter, a continued pathologic response occurs. Within six hours, there is extensive hemorrhagic pneumonia, with erythrocytes, granulocytes, and macrophages in the alveoli and bronchi. Within 48 hours, a widespread granulomatous reaction with numerous macrophages and giant cells is present. Within another 24 hours, most of the reaction is mononuclear, and numerous granulomas are present. Within five days, food particles can be identified at the center of these granulomas.

A chest roentgenogram may show granuloma formation similar to that of miliary tuberculosis. The effects of foodstuff aspiration were once thought to be long term, but Wynne and associates discovered that the long term effects may be limited to approximately 21 days at which time the amount of fibrosis was minimal.

The physiologic changes that occur in foodstuff aspiration differ in several respects from acid aspiration. The shift of fluid from intravascular space to the lungs occurs much later and is not as great as in acid aspiration. Heavy bronchial transudation is usually not seen. Arterial PaCO₂ values are higher, indicating a more significant degree of hypoventilation.

In summary, acid aspirate is primarily hemorrhagic, granulocytic and necrotizing while foodstuff aspiration is mononuclear and granulomatous. Acid aspirates cause the greatest degree of damage, both anatomic and physiologic. However, determining whether the aspirate was acid or foodstuff may be technically difficult.

Chest roentgenograms are not reliable diagnostic tools as changes will not be noted until 24 hours post-aspiration and oftentimes later. However, roentgenogram findings do serve as valuable evaluation tools. Landay and his associates illustrated in 60 patients with pulmonary aspiration that roentgenogram findings were extremely variable and that there was no typical or characteristic appearance. Their study revealed that the distribution of the aspirate was most commonly bilateral and multicentric and usually favored perihilar or basal regions.

Patients with the most extensive radiographic abnormalities usually had the worst prognosis, but mild, early pulmonary infiltrates often developed into severe life-threatening complications. Radiographic changes often worsened over the course of several days, but improvement was generally manifested within the first week of aspiration. Worsening of infiltrates after initial improvements was most often associated with the development of bacterial pneumonia, adult respiratory distress syndrome (ARDS), and pulmonary embolism.

**Treatment of aspiration syndrome**

A positively diagnosed case of gastric acid
aspiration or food particle aspiration is generally treated as an occurrence of adult respiratory distress syndrome. In the event of foodstuff aspiration, an immediate bronchoscopy may be necessary to open up obstructed airways. A chest roentgenogram is necessary to assure an adequate data base. A gastric sample, via a nasogastric tube, should be obtained for pH measurement.

Mechanical ventilatory support must be considered if the patient’s PaO₂ is less than 70 torr on an FIO₂ of 21%, if the AV O₂ exceeds 140 torr on 40% oxygen, and if the physiologic shunt exceeds 15%. Positive end-expiratory pressure (PEEP) is indicated when adequate oxygenation cannot be maintained without the use of toxic levels of oxygen. PEEP can depress cardiac output by decreasing venous return; therefore, a level which allows maximum reduction in venous admixture with minimum reduction in cardiac output must be a primary objective. When higher levels of PEEP (15-50 cm/H₂O) are utilized, the insertion of a Swan-Ganz catheter to assess pulmonary and cardiac function is necessary in order to measure blood gas tensions in arterial and mixed venous blood samples. An indwelling arterial line is a basic necessity in the long-term management of these patients.

Proper fluid therapy is critically important with severe aspiration syndrome. Loss of intravascular volume can be significant and must be corrected quickly. Careful fluid monitoring of these patients is critical, as prolonged ventilatory support can cause accumulation of fluid.

Routine chest physiotherapy is a very useful adjunctive mode of treatment and should be performed on a daily basis. A daily complete blood count, blood cultures if temperature exceeds 39°C and sputum cultures twice weekly should also be considered.

The benefit of lavaging with large volumes of neutral or alkaline solutions, administering prophylactic antibiotics, and the use of corticosteroids has not been proven. Most studies have shown either no improvement or increased lung damage when large volumes of lavage were used.13, 14, 15, 16 Bynum and Pierce reported that initial treatment with antibiotics had no apparent effect on clinical outcome or subsequent development of infection in 54 cases of aspiration.17 In their series, when infection occurred it was usually due to gram-negative bacteria and was thought to be nosocomial in origin.

If prophylactic antibiotics are to be used in aspiration, coverage of all infecting organisms must be implemented. Completeness would require protection against all anaerobes, including Bacteroides fragilis, as well as broad spectrum coverage for aerobes. Wynne approached the problem by initially withholding all antibiotics, monitoring the patient clinically for evidence of infection, and basing the treatment on the results from well collected smear and culture specimens.

The use of steroids has come under increasing scrutiny in the last five years. Wolfe and his associates illustrated that the use of steroids results in a greater hemoglobin loss, and the more frequent occurrence of gram negative pneumonia.19 Wynne and his colleagues established that the pneumonitis associated with the aspiration of food particles actually represented a housekeeping effort on the part of the lung to ingest, eliminate or isolate foreign materials.11 Steroids were discovered to interfere with this clearing process and actually prolonged the state of acute inflammation.

Optimum management of patients with severe aspiration syndrome should entail immediate endotracheal suctioning, bronchoscopy if large particle aspiration has occurred, aggressive ventilatory support to include PEEP if indicated by clinical assessment and blood gas measurements, and adequate fluid replacement. Lavaging with large volumes of neutral or alkaline solutions, administering antibiotics, and the use of steroids are of unproven benefit.

**Prevention**

"That which cannot be easily treated had better be prevented," J. Alfred Lee, Synopsis of Anesthesia.

Prevention is probably the best treatment for aspiration. Proper positioning of patients in the high risk category can be very helpful. The lateral "tonsil position," with the patient’s head slightly lowered will promote the drainage of gastric aspirate out of the oropharynx should it occur. The use of antacids has recently come under criticism as a preventive measure in increasing the pH of gastric fluid. Separate studies by Bond and Gibbs established that the aspiration of antacids causes a more severe reaction in the lung than if gastric acid itself was aspirated.5, 18 The causative factor is thought to be the suspension material itself or the preservatives present in such antacids.

Cimetidine, an H₂ receptor blocking agent, has been found to be very effective in raising gastric pH values above the critical level of 2.5 as well as in decreasing volume. An intramuscular dose of 300 mg is effective within 60-90 minutes, with an intravenous dose requiring 30 minutes. Once an effective blood level is attained, an oral
dose schedule can be instituted, 300 mg every 6-8 hours.

The monitoring of cuff pressures is another preventive measure in reducing the incidence of aspiration as well as preventing tracheal ischemia. Careful tube feeding and monitoring for signs of increasing intragastric pressure is a necessity. Maintaining the patency of nasogastric tubes is critical.

To minimize the risk during anesthesia, either a rapid sequence intubation with cricoid pressure or an awake intubation must be considered. The passage of a nasogastric tube prior to intubation will remove gastric acid but will not remove food particles. The patient should not be extubated until he is fully awake and the glottic reflexes have completely returned. Ideally, a regional anesthetic technique should be utilized if possible.

There has been much controversy concerning the position the patient should assume during the induction phase of anesthesia so as to minimize the risk of aspiration. Some authorities recommend a sitting position using the effect of gravity to discourage the migration of gastric contents. In this position, however, it may be very difficult to visualize the larynx and hence, to intubate the patient. On the other hand, the head down position is advocated because if vomiting does occur it will run out of the oropharynx.

Criticism of this position has evolved around the fact that gravity itself promotes the migration of stomach contents, and that the clinician is increasing the risk of that happening. The ideal position is probably a semi-recumbent one, with the patient’s head elevated 25-30°. Gravity will keep the stomach contents down, however, the ability to visualize and intubate will not be severely impaired.

REFERENCES

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