Anesthetic considerations with anorexia nervosa

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Anorexia nervosa is a chronic illness characterized by a severe loss of weight. It occurs in children and young adults and is seen more frequently in females. Some anorexic patients refuse to eat totally, while others may practice “binge eating.” Patients who binge eat and employ postprandial purging are referred to as “bulimic.” Anorexia nervosa and bulimia can produce multisystem abnormalities.

Dysfunction of organ systems in anorexia is the result of self-induced vomiting, laxative abuse, and severe malnutrition. Diseases of the skeletal, gastrointestinal, pulmonary, endocrine, and cardiovascular systems may have an impact on the plan of anesthesia care. A thorough anesthetic interview and evaluation to assess the debilitation of each patient is essential so that appropriate preparation can be made for comprehensive anesthetic care.

This paper focuses on the pathophysiology of anorexia nervosa, discusses a thorough preanesthetic assessment, and guides the appropriate anesthetic management of the anorexic patient. Anorexia bulimia is briefly mentioned, since it is an eating disorder with physiological derangements similar to anorexia nervosa.

Key words: Anorexia bulimia, anorexia nervosa.

Introduction

Anorexia nervosa is a personality disorder characterized by an extreme aversion to food. An understanding of this eating disorder is needed so that the anesthetist can consider appropriate anesthetic care. The etiology of the condition is complex and thought to be a symptom of underlying mental illness. It occurs most commonly in females between the ages of 12 and 21. A distortion of the patient's body image and a fear of becoming obese are associated with this syndrome. There is a progressive, severe loss of weight, with amenorrhea occurring in women and impotence in men. Severe caloric restriction in the anorexic patient may result in weight loss amounting to as much as 50% of normal body weight and profound deficiencies in essential vitamins and minerals. The patient may have to be fed parenterally. Psychiatric therapy is usually required if the patient totally refuses to eat, especially when he or she is emaciated and denies that anything is wrong.

Bulimia is a disorder in which the patient will binge eat enormous numbers of calories, is not hungry, carefully plans his/her binges, and is usually of normal body weight. Bulimic anorexic patients reveal characteristics of both anorexia and bulimia. They combine binge eating with postprandial purg-
ing, accomplished by self-induced emesis and the abuse of cathartic and diuretic drugs. The added stressors of surgery and anesthesia in both of these extremely compromised types of patients increase their risk of decompensating.

**Multisystem pathophysiological effects**

The pathophysiology associated with anorexia nervosa produces multisystem abnormalities. For example, esophageal complications can occur from mechanical and chemical injury to the esophagus as a result of repeated vomiting. The complications include esophagitis, Mallory-Weiss tears, and esophageal rupture. Esophagitis caused by gastric acid bathing the esophageal mucosa may result in stricture formation. Retching stresses the esophagus and can cause rents, known as Mallory-Weiss tears, in the lining. These tears can bleed massively, but do not require surgical repair. Perforation or rupture can also occur at the lower end of the esophagus. An esophageal rupture is life-threatening and requires immediate surgery.

Gastrointestinal complications of anorexia include gastritis, acute gastric dilatation, and delayed gastric emptying. Total acid secretion in the stomach is significantly less in patients with anorexia than in normal subjects. This decrease persists even after patients gain weight.

Colonic complications commonly occur from laxative abuse. Laxatives, although ineffective in significantly reducing caloric absorption, are often used in enormous quantities. In extreme cases it may be necessary to give nutrients by nasogastric tube or parenterally in order to sustain life.

Abnormalities in heart size and rhythm also occur in these patients. A decrease in cardiac dimensions has been demonstrated by echocardiography, including reduced cardiac muscle mass with decreased cardiac chamber size and impaired myocardial contractility. These changes are associated with decreased cardiac output and a comparatively decreased blood pressure.

Electrolyte abnormalities cause many cardiac dysrhythmias. The intrinsic conduction defects that have been found, along with the sudden deaths which occur, are attributed to hypokalemia. Presumably, these changes are in response to malnutrition. Electrocardiographic changes include T wave inversion of flattening, ST depression and prolonged QT intervals. Sinus bradycardia and ventricular ectopy also have been noted. The incidence of mitral valve prolapse may present an additional risk factor for these patients. Ipecac abuse has been linked to several deaths of patients with eating disorders. Emetine, the active ingredient of ipecac syrup, has marked cardiomyotoxicity and myopathy.

Pulmonary complications may arise because of self-induced vomiting. There remains the possible decrease of lung elasticity from the dehydration and malnutrition which would manifest themselves as decreased pulmonary compliance during the intraoperative period, as well as the occurrence of aspiration pneumonia and spontaneous pneumomediastinum.

Endocrine and metabolic abnormalities are secondary to malnutrition, since there is no evidence of primary pituitary, gonadal, thyroid, or adrenal dysfunction. Approximately 20% of patients are known to develop amenorrhea. Gonadotropin release from the hypothalamus is impaired, resembling a prepubertal pattern. In male anorexics, serum testosterone levels are low, which may explain the impotence and low libido in these patients. Growth hormone levels are often elevated, probably as a result of decreased somatomedin C levels.

Although anorexic patients have signs and symptoms suggestive of hypothyroidism, they do not exhibit a true low-thyroid state. The metabolic activity of the thyroid is impaired, and elevated T4 and T3 levels are often found in these patients. There is abnormal thermoregulation in response to heat and cold and a lack of a shivering response to hypothermia. Carbohydrate deprivation, hypoglycemia, or both may alter hypothalamic function directly. Hypothermia is found in patients who are hypoglycemic.

Adrenal metabolism may be compromised, resulting in altered levels of active androgens. Plasma cortisol levels may be normal or elevated, with elevations secondary to decreased metabolism of cortisol or to increased cortisol production. Dexamethasone suppression tests are abnormal.

Alterations of the autonomic nervous system also occur. Decreased norepinephrine synthesis occurs during fasting, although in anorexics the concept of heightened sympathetic tone has been evoked to explain some manifestations of the syndrome (e.g., increased cutaneous vasoreactivity), while hypervagal states have been implicated in other manifestations, for example, bradycardia.

Renal abnormalities include a decreased glomerular filtration rate, which occurs on the basis of dehydration. Transaminase and alkaline phosphatase levels may be elevated, reflecting hepatic dysfunction.

Low white blood cell counts, decreased white blood cell function, low platelet counts, and mild
anemia may occur. Bone marrow is often hypocellular, with decreased fat content. The degree of abnormalities does not correlate with the amount or duration of weight loss. Immunocompetency is preserved in anorexia until weight loss is as much as 50% of the normal body weight.3

Anorexia nervosa's effect on the musculoskeletal system is evidenced by osteoporosis secondary to low calcium intake and estrogen deficiency. Muscle abnormalities may be secondary to electrolyte depletion. Chronic ipecac syrup ingestion also can cause toxic peripheral muscle weakness.4

Few neurological complications have been described in the literature. Hypoglycemia and hyperglycemic comas are probably the most serious complications. Peripheral nerve palsies can occur secondary to nerve compression from marked cachexia and loss of cushioning subcutaneous tissue.4

Anorexics who self-induce vomiting and purging may exhibit dermatologic complications. Phenolphthalein, an ingredient in most over-the-counter laxatives, can cause a drug reaction. Skin lesions recur at the same site upon repeated exposure to phenolphthalein and may develop brownish-gray hyperpigmentation over time. Subconjunctival hemorrhages of the blood vessels in the eye may result from excessive and forceful vomiting.4

**Preanesthetic assessment**

The most obvious goal of the preanesthetic visit is the acquisition of factual information about the patient. This information can come from several sources, including a chart review, a careful health history, and a thorough physical and psychological assessment. Thus, a baseline of data is established for comparing intraoperative or postoperative abnormalities.10 Past and present hospital records should be reviewed, with attention focused on prior anesthetic experiences and the physiologic alterations brought about by disease.11

Laboratory tests are of value primarily to aid in the exclusion of systemic diseases such as carcinomatosis, diabetes mellitus, and intestinal malabsorption, which may account for extreme weight loss.3 Severe malnutrition, whatever the cause, can result in anemia, low white cell and platelet numbers, and relative lymphocytosis. Two laboratory abnormalities seen are elevated beta-carotene levels and high cholesterol levels in the blood. There is no explanation for either finding.4

Blood glucose concentrations are monitored when the anorexic is receiving nutritional supplementation delivered by a nasogastric or orogastric tube. Complications of enteral feeding can include hyperglycemia, leading to osmotic diuresis and hypovolemia. Exogenous insulin is administered when glucose levels exceed 250 mg/dL. The high osmolarity of elemental diets often causes diarrhea.12

Total parenteral nutrition is indicated when the gastrointestinal tract is not functioning. Associated potential complications include catheter-related sepsis, hyperglycemia, nonketotic hyperosmolar hyperglycemic coma, hepatic dysfunction, hypomagnesemia, and hyperchloremic metabolic acidosis. These complications result from the liberation of hydrochloric acid during the metabolism of amino acids present in the parenteral nutrition solutions.

Hypophosphatemia from the administration of phosphate-depleted solutions can result in a shift in the oxyhemoglobin dissociation curve to the left and decreased release of oxygen from hemoglobin to tissues. One of the main reasons for slowing or discontinuing infusion of parenteral nutrition solutions before the induction of anesthesia is to avoid intraoperative hyperosmolarity secondary to rapid infusion of the solution. However, abrupt discontinuation should be avoided, since the persistence of increased circulating levels of endogenous insulin could contribute to hypoglycemia. In this situation it is important to measure blood concentrations of glucose as well as phosphate and potassium preoperatively. Increased production of carbon dioxide, resulting from metabolism of large quantities of glucose, may result in the need to initiate artificial ventilation of the lungs or in failure to wean patients from long-term ventilatory support.12 Along with a complete analysis of serum electrolytes, an electrocardiogram is also recommended for all anorexic patients who are to receive an anesthetic.1

Attention should be given to the other pharmacologic agents which are currently being prescribed or which the patient has taken in the past. These drugs are to be considered in the development of the anesthetic care plan because of their possible interactions with anesthetic agents.10 The ability to tolerate the adverse effects of anesthesia and surgery depends largely on the normality of respiration and circulation as well as on the hemostatic functions of the liver, kidneys, and endocrine and central nervous systems. Anesthetists should be able to predict the effect of the surgical position and operation on the physiologic process.11

**Anesthetic considerations**

The anorexic patient may decompensate when faced with the stress of an impending hospitaliza-
tion. Even a careful preanesthetic interview may not reveal a recent exacerbation of symptoms, with the associated risk of acute physiological disturbance. Indeed, in the debilitated or anesthetized patient, natural compensatory mechanisms are further reduced. Therefore, the negative effects of positioning become more severe. Anatomical complications which occur from improper positioning include nerve palsies and fractures. Nerve palsies can be prevented by proper positioning and by adequate padding of bony prominences and pressure areas.

As evidenced by the cardiovascular effects presented, the electrocardiogram is a vital monitoring device for the anorexic. The electrocardiogram is a practical method of monitoring the balance between myocardial oxygen requirements and myocardial oxygen delivery in unconscious patients. When this is unfavorably altered, myocardial ischemia occurs, as evidenced by at least a 1 mm downsloping of the ST segment from the baseline. A precordial V₅ lead is a useful choice for detecting ST segment changes characteristic of myocardial ischemia of the left ventricle during anesthesia. The appearance of signs of myocardial ischemia on the electrocardiogram supports the aggressive treatment of adverse changes in heart rate and/or blood pressure.

Arrhythmias are best determined in lead II as it parallels the P wave vector, thereby differentiating ventricular from supraventricular arrhythmias and the presence or absence of the hemodynamic effect of the atrial kick. Both leads should be monitored.

The delayed emptying which occurs with anorexia increases the possibility of gastroesophageal reflux and the potential for aspiration of stomach contents. Preoperative regimens to increase gastric pH and emptying time should be considered. The insertion of a nasogastric tube preoperatively can be an effective measure to deflate the stomach and remove any residual gastric juice. The anorexic could be considered a full-stomach patient.

It is essential to maintain adequate body temperature in the anorexic intraoperatively because of an abnormal thermoregulation and shivering response. Techniques include a warming blanket, a heat and moisture exchanger applied to the patient's circuit, and for general anesthesia, the warming and humidifying of inspired gases, warming intravenous fluids, and covering areas not involved in surgery. The temperature should be monitored regardless of the type of surgery.

Local and regional anesthetic techniques may not be recommended for the anorexic who presents with severe skin lesions. The anesthetist should carefully inspect the intended puncture site before a determination is made. As with the use of regional techniques, extreme caution regarding the sympathetic effects of anesthetics is recommended, and adequate intravenous hydration should be given prior to their use.

Because of the continuing controversy over halothane-associated hepatic dysfunction, as well as with the fluoride-induced nephrotoxicity of enflurane, isoflurane may be the least compromising volatile anesthetic agent for the patient with anorexia nervosa. The administration of halothane also could be associated with an increase of ventricular dysrhythmias, because of the anorexic's decreased cardiac dysrhythmic threshold for epinephrine.

Isoflurane produces the least dose-dependent reduction in cardiac output compared to the other inhalation agents. Although the coronary artery steal syndrome has been reported to be associated with the use of isoflurane, autoregulation of coronary blood flow seems to be maintained during its administration, so long as the anesthetist avoids drug-induced events that adversely alter myocardial oxygen supply (hypotension) or myocardial oxygen demand (tachycardia).

Prolongation of neuromuscular blocking agents can manifest in the anorexic with chronic ipecac syrup ingestion because of the potential for peripheral muscle weakness. If muscle relaxation is indicated, a nondepolarizing neuromuscular blocker is recommended. The decision to reverse a neuromuscular blocking agent is based on the patient's overall clinical picture. Allowing neuromuscular blockade to wear off in the postanesthesia care unit before extubation could be the preferred alternative, especially since a variety of dysrhythmias have been described in association with the reversal of nondepolarizing neuromuscular blockade.

Conclusion

The review of the literature on anorexia nervosa documents a patient who is ready to decompensate when faced with the stress of an impending hospitalization and surgery. The dysfunction of organ systems affected by anorexia nervosa is the result of self-induced vomiting, laxative abuse, and severe malnutrition. A thorough anesthetic interview and evaluation, with special emphasis on assessing the debilitation of each patient, is essential so that appropriate preparation can be made for comprehensive anesthetic care.

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