imity to the larger meningeal arteries.[3] Hence, local anesthetic scalp infiltration would be expected to block most of the afferent neural pathways of significance in the production of these hemodynamic responses.

The differences observed between the two groups may have been greater had not severe hypertension been treated with additional opiate, thiopental, or hydralazine in 11 patients in the saline group. No one in the bupivacaine group required this treatment.

Mixtures of local anesthetics and vasoconstrictor have long been injected into the scalp prior to craniotomy to promote hemostasis.[4–5] While these mixtures provided a readily available formulation of dilute vasoconstrictor, the effect of the local anesthetic content on the hemodynamic response to craniotomy under GA has not previously been systematically evaluated. We avoided the use of vasoconstrictor in our local anesthetic solution, as an accidental intravascular injection or systemic absorption may cause hypertension.[4] We chose bupivacaine because of its long duration of action and safety when used in the vascular tissues of the scalp.[6]

The narcotic/relaxant GA used is similar to that employed in many centers.[7,8] The results in our control group suggest that such a GA technique alone may not adequately control the hemodynamic responses to craniotomy in a large proportion of patients, at least without use of high doses of narcotics. Supplementation by local anesthetic scalp infiltration appears to overcome the problem without risk of unacceptable increases in intracranial pressure, as may occur when volatile anesthetic agents are used, including isoflurane.[9]

In summary, our results demonstrate that local anesthetic scalp infiltration significantly improves cardiovascular stability during craniotomy under narcotic/relaxant general anesthesia, suggesting that its routine use should be considered under such circumstances.

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Intraoperative Cardiac Dysrhythmias in a Patient With Bulimic Anorexia Nervosa

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Anorexia nervosa is a syndrome characterized by extreme weight loss, distorted body image, and a fear of becoming obese. Bulimia, a distinct syndrome, is characterized by binge-eating episodes followed by self-induced vomiting, fasting, and the use of diuretics and/or laxatives.[1]

Preoccupation with food is common to both syndromes. The potential for marked weight fluctuations exists with bulimia, and severe weight loss often occurs with anorexia nervosa. In addition, bulimic symptoms may be part of the anorexia nervosa syndrome. Anorexia and bulimia are estimated to effect 5–10% of adolescent girls and young women.[2] We report a case of cardiac dysrhythmias during general anesthesia in a patient with bulimic anorexia nervosa.

Case Report

A 16-yr-old girl presented in the ambulatory surgery unit for dental extractions. The patient had a 1-yr history of anorexia nervosa with

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Key words: Complications: dysrhythmias. Ions: potassium. Metabolism: anorexia nervosa; bulimia.

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bulimia and no other known medical problems. She had been followed by an adolescent medicine specialist and a psychotherapist for the past 6 months, and her condition was felt to be improved and clinically stable by both her family members and physicians. The patient had not previously undergone general anesthesia. The physical examination was significant only for her height (160 cm) and her weight (47 kg) (25th and 10th percentile, respectively). The most recent laboratory data had been obtained several months prior to the proposed operative procedure, and included a hemoglobin of 12.5 g/dl, serum sodium of 141 mmol/l, potassium of 3.7 mmol/l, chloride of 99 mmol/l, and total CO_2 of 35 mmol/l. At that time, blood urea nitrogen, creatinine, glucose, calcium, thyroxine and thyroid-stimulating hormone, bilirubin, and hepatic enzymes were all within normal limits.

Prior to the induction of general anesthesia, the right nares was anesthetized with a 4% cocaine solution on cotton-tipped swabs, while she received 2.5 mg of midazolam iv in divided doses. Anesthesia was induced with 275 mg of thiopental and 150 μg of fentanyl iv. Paralysis was achieved with 25 mg of atracurium iv. The trachea was intubated nasally with a 6.5-mm cuffed endotracheal tube. Anesthesia and paralysis were maintained with 70% nitrous oxide, 0–1% isoflurane, and atracurium. Ventilation was controlled. During the operation, the ECG was remarkable for P-R intervals of varying lengths and an intermittent junctional rhythm. The junctional rhythm was of no hemodynamic significance, and responded to decreasing the inspired isoflurane concentration (end-expired isoflurane concentration was not monitored). Following the extraction of four impacted third molars, the paralysis was reversed with 1.0 mg of atropine and 40 mg of edrophonium iv. Spontaneous ventilation was assisted. An abrupt change in cardiac rhythm from a sinus rate of 60 bpm to ventricular bigeminy was noted. This rhythm subsequently progressed to multifocal premature ventricular contractions with short, self-limited runs of ventricular tachycardia. Controlled ventilation with 100% oxygen was instituted, and the patient was given 50 mg of lidocaine iv. The rhythm then promptly reverted to sinus rhythm at a rate of 100 bpm. During the period of cardiac dysrhythmia, the atrial blood pressure remained stable (110–125 mmHg systolic), and arterial oxygen saturation (monitored by pulse oximetry) was 99–100%. End expired CO_2 was not monitored. When the patient awoke, her trachea was extubated, and she was transferred to the post-anesthesia recovery room. Her serum potassium was 2.3 mmol/l in the recovery room. Serum magnesium, calcium, and albumin concentrations were within normal limits. An ECG revealed a sinus rhythm of 100 bpm with a Q-T interval of 0.42 msec (upper limit of normal is 0.36 msec).[3] T-wave flattening was also noted. Arterial blood gases were not measured. The patient's body temperature was monitored intraoperatively and in the recovery room, and no abnormalities were noted.

When questioned in the recovery room, the patient admitted that, for approximately 3 days prior to surgery, she had been extremely frightened, and had experienced increasing anorexia, as well as an increase in the frequency of emesis. She was treated with iv potassium supplementation and was discharged home the next day with a serum potassium of 3.5 mmol/l, and without further evidence of cardiac dysrhythmia.

Medical and psychiatric follow-up have been performed by the patient's adolescent medicine specialist and psychotherapist. Three months after the reported incident, the patient continued to have difficulty with anorexia and frequent emesis; her serum potassium was 3.0 mmol/l, and an ECG revealed a sinus rhythm of 74 bpm, with a Q-T interval of .39 msec (upper limit of normal is 0.37 msec)[3] and T-wave flattening.

Discussion

With the attendant profound psychological and physiological consequences of anorexia and bulimia, morbidity and mortality for these eating disorders are among the highest for any psychiatric disorder.[2] Given the prevalence of these syndromes and their associated grave physiological changes, it is remarkable that there is such a paucity of published clinical experience in the anesthesia literature.

Cardiovascular changes in patients with eating disorders include reduced cardiac muscle mass with decreased cardiac chamber size, and impaired myocardial contractility.[4–6] These changes are associated with decreased cardiac output and relative hypotension. ECG changes are common, and include T-wave inversion or flattening, and S-T depression.[7–9] Sinus bradycardia and ventricular ectopy have been noted. Prolonged Q-T intervals and sudden death secondary to electrolyte disturbance and/or alterations in sympathetic-parasympathetic tone have been described in this population.[10] Incidence of mitral valve prolapse appears to be increased in patients with eating disorders, and the arrhythmogenic effects of mitral valve prolapse may present an additional risk factor for these patients.[9] Also of note in the bulimic population is a case report of fatal ipecac cardiomyopathy.[11]

Endocrine and metabolic disturbances include decreased or erratic vasopressin secretion[12] and abnormal temperature regulation.[13] 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 (*e.g.*, bradycardia).[2,9,10]

Renal abnormalities include decreased glomerular filtration rate, which occurs on the basis of dehydration. Starvation can cause a total body loss of sodium and potassium. Gastrointestinal abnormalities include increased gastric emptying time.[14] Transaminases and alkaline phosphatase levels may be elevated, reflecting hepatic dysfunction. Moderate anemia, leukopenia, and thrombocytopenia may occur.[2]

Bulimia, as an isolated syndrome or as part of the anorexia nervosa syndrome, causes another set of medical complications. Binge-eating and vomiting can cause acute gastric dilatation and rupture.[2] There is a risk of aspiration of gastric contents when consciousness is impaired by drugs or alcohol use. Hypokalemia and dehydration are risks of self-induced vomiting and of diuretic or cathartic abuse.

Common perioperative dysrhythmias have been described and the etiologic factors discussed by Katz and Bigger, who found that the incidence of perioperative dysrhythmias varies from 16–62%.[15] The significant ventricular ectopic activity observed in this patient occurred in a setting of severe hypokalemia and prolongation of the Q-T interval. Two potential contributing

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factors merit comment. Alterations in Pa_{CO_2} have a direct effect on serum potassium levels, potentially leading to dysrhythmias by either producing a reentrant mechanism or by altering phase 4 depolarization. With low serum and total body potassium concentrations, alterations in Pa_{CO_2} may have a more profound effect on serum potassium levels.[16] Also, a variety of dysrhythmias have been described in association with reversal of nondepolarizing neuromuscular blockade.[17]

The dysrhythmia observed in this patient appeared to occur primarily on the basis of physiologic changes attributable to her bulimic anorexia nervosa: low serum potassium (and probable low total body potassium) levels, prolonged Q-T interval, and possible autonomic imbalance. It is possible that reversal of neuromuscular blockade and ventilatory changes contributed to this patient's potential for cardiac dysrhythmia.

Anorexia nervosa may begin abruptly, or may be an insidious process, lasting months to years and manifesting itself as a fluctuating illness with exacerbations and remissions.[2] Furthermore, a patient with bulimic tendencies is often embarrassed by her symptoms and has difficulty telling others, including family, friends, and medical personnel.[2] Compensated anorexics and bulimics may be at risk for decompensating when faced with the stress of an impending hospitalization and operation. Even a careful preoperative interview may not reveal a recent symptom exacerbation, with the associated risk of acute physiological perturbation, as occurred in this case. In addition to the usual preoperative assessment, we recommend a determination of serum potassium and an ECG for patients with a history of an eating disorder who are to receive an anesthetic.

In summary, we have presented an otherwise healthy adolescent female with bulimic anorexia nervosa who developed significant cardiac dysrhythmia while anesthetized, attributable primarily to physiologic changes occurring on the basis of her eating disorder.

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