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Mitral Valve Prolapse—Another Cause of Intraoperative Dysrhythmias in the Pediatric Patient

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Mitral valve prolapse is a condition that is being recognized with increasing frequency.[1] Means of its discovery may range from auscultation of a systolic click on a routine physical examination to autopsy findings associated with mital valve prolapse in a patient who expires unexpectedly.[2] Only a few reports exist in the anesthesia literature about the varying symptoms and the many different clinical presentations associated with mitral valve prolapse.[3] We describe intraoperative dysrhythmias that occurred during two separate operations in a pediatric patient before his cardiac abnormality was recognized.

REPORT OF A CASE

The patient, a 14-year-old boy with a history of moderate pectus excavatum, presented for a pectus repair with an internal strut. Physical examination revealed a well-developed boy with a blood pressure of 110/70 mmHg, heart rate of 60 bpm and regular, weight 57.2 kg, and height 170 cm. He had a sinus rhythm, normal first and second heart sounds, and no murmurs, gallops, or rubs. The remainder of the physical examination was normal except for the presence of a moderate pectus excavatum, with the left hemithorax being much larger in appearance than the right. His laboratory studies were normal. We provided preanesthetic sedation with oral diazepam (10 mg) and induced anesthesia with thiopental (300 mg) iv. Following succinylcholine (80 mg) iv, we inserted a #7 mm ID orotracheal tube. Anesthesia was maintained with halothane (1.0–1.2% inspired), nitrous oxide (70%), and oxygen (30%), supplemented by iv morphine (20 mg in divided doses). Metocurine (12 mg) was given iv for neuromuscular blockade. Estimated blood loss was 500 ml, and 1.0 l of 5% dextrose in lactated Ringer's solution and 1.4 l of lactated Ringer's solution were given iv. We monitored neuromuscular transmission with a nerve stimulator evaluating a tetanic stimulus; and at the end of surgery, mild fade to tetanic stimulation was present. We reduced halothane to 0.5% for 30 min before the end of surgery and then discontinued it. Antagonism of neuromuscular blockade then was accomplished with a mixture of neostigmine (2.5 mg) and atropine (1 mg) iv. Within 4 min of the administration of these drugs, ventricular bigeminy occurred at a rate of 100–110 bpm. Lidocaine (75 mg) was given iv and hyperventilation instituted. Normal sinus rhythm returned immediately; however, over the next 5 min, ventricular ectopy recurred in the form of multifocal premature ventricular contractions (PVCs). Then a run of 6 beats of ventricular tachycardia occurred. Additional lidocaine (75 mg iv) was given and hyperventilation continued. A normal sinus rhythm returned at a rate of 90 bpm. Over the next 5 min, occasional multifocal PVCs occurred that proceeded to 8 beats of ventricular tachycardia. This was treated with lidocaine (50 mg iv) and continued hyperventilation. The rhythm reverted to a normal sinus rhythm at a rate of 90 bpm. When spontaneous ventilation was allowed to return, mild paradoxic breathing was evident. Because we thought this might be caused by inadequate antagonism of metocurine, we administered edrophronium (30 mg iv) and observed improvement in his pattern of breathing. With an $F_{I_{O_2}}$ of 0.3, the Pa_{CO_2} was 65 mmHg; the Pa_{O_2}, 96 mmHg; the pH_a, 7.20; and the base deficit, −5.0 mEq/l. The hematocrit was 38%. A chest roentgenogram revealed a 50% pneumothorax on the right and a small (10%) pneumothorax on the left. We treated the right pneumothorax by needle aspiration. Arterial blood pressure throughout this interval was in the range of 120–140/70 mmHg. The halothane and N_2O had been discontinued for 1 h at this point. The patient then was taken to the recovery room, where he was responsive and moving his extremities. Ventilation appeared adequate, and the trachea was extubated. Over the next 5 min, the patient became difficult to arouse and his respirations became rapid, shallow, and appeared labored. He was given naloxone (0.2 mg iv and 0.2 mg im). He responded rapidly by awakening, and his ventilatory pattern became normal. Pa_{O_2} was 111 mmHg; Pa_{CO_2}, 46 mmHg; pH_a, 7.35, with an $F_{I_{O_2}}$ of 0.4. Electrolytes were normal, and the hematocrit was 37%. For the first 15 min in the recovery room, he occasionally had nodal rhythm but no other dysrhythmias. His recovery from this point on was completely uneventful. Questioning of his family revealed no previous difficulty with dysrhythmias, chest pain, or any other symptoms that might be related to the cardiovascular system.

He returned 6 months later for removal of the metal strut from the chest. We induced anesthesia with thiopental (300 mg iv) and maintained it with nitrous oxide (70%) and halothane (2%). Ventilation was assisted. Within 12 min of anesthetic induction, a ventricular bigeminy occurred. Because of the dysrhythmia, we changed the anesthetic from halothane to enflurane, and the rhythm reverted to normal. The operative time was 15 min. The patient was taken to the recovery room with a normal arterial blood pressure, heart rate, and respiratory rate and was discharged from the hospital shortly thereafter. He has remained asymptomatic. He was referred to a pediatric cardiologist who performed an echocardiogram that revealed the classic findings of mitral valve prolapse. The pediatric cardiologist heard a loud midsystolic click that was more apparent upon the rapid assumption of the upright posture. The ECG was within normal limits.

DISCUSSION

Various explanations were sought for the dysrhythmias that occurred following the first anesthetic. The halo-

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thane was reduced from a maintenance inspired concentration of 1.2% to 0.5% for the last 30 min of surgery. The dysrhythmias began within 5 min of the administration of atropine and neostigmine and continued for the next 1 h and 20 min. Hypercarbia in conjunction with halothane as an explanation of dysrhythmias seemed unlikely, since the halothane had been reduced and then discontinued several minutes before the dysrhythmias appeared. The pneumothorax also was considered to be a possibility, particularly if there was an associated hypoxia, but the Pa_{O_2} was 96 mmHg. We finally considered the possibility of catecholamine release secondary to hypercarbia. However, dysrhythmias continued after the Pa_{CO_2} was reported as normal.

Mitral valve prolapse is an extremely common entity occurring in 6–20%[1] of the adult population, with an incidence in children of 1.4%.[4] The natural course of the disease would suggest that the myxomatous degeneration is a progressive process and that, as a result, mitral valve prolapse would become clinically more apparent with aging. The other possibility is that there is a low level of awareness of the diagnosis in the pediatric age group as exemplified by this case report. The pathophysiologic alterations include structural alterations in the leaflets of the mitral valve resulting from myxomatous degeneration[5] and abnormal cardiovascular regulatory mechanisms[6] secondary to sympathetic nervous system imbalance.[7] Although patients with mitral valve prolapse have atypical chest pain, dyspnea, palpitation, fatigue, and dizziness, many, such as our patient, are entirely asymptomatic. Physical findings on auscultation include an isolated midsystolic click, a midsystolic click with late systolic murmur, or an isolated late systolic murmur that can be increased in the standing position. We heard a midsystolic click on auscultation of our patient postoperatively. The primordium of the mitral valve develops as the thoracic cage and vertebrae are initiating chondrification and ossification during the seventh week of fetal life. Thus, associated anomalies, such as pectus excavatum, straight back syndrome, or connective tissue disorders are often present.[8] A combination of M-mode, two dimensional, and Doppler echocardiography will provide a definitive diagnosis.[9]

The electrocardiogram may show low, initially inverted or totally inverted waves with or without depression in the inferior leads.[10] However, these changes may be evanescent, so that the normal ECG noted in this patient often will be seen.[11] Paroxysmal supraventricular tachycardia is a frequent dysrhythmia in these patients, but multifocal premature ventricular contractions occur, particularly during exercise.[12] In view of the diagnosis, this patient's dysrhythmias readily are explained. Any drug (atropine) or maneuver (pneumothorax) that tends to decrease ventricular volume also worsens prolapse of the mitral leaflets. The excessive traction on the chordae tendineae and papillary muscles of the prolapsing leaflets causes myocardial ischemia. The dysrhythmias of myocardial ischemia induced by the prolapsed leaflets may be enhanced further by the increased catecholamine levels and increased sensitivity to catecholamines[7] seen in patients with mitral valve prolapse, coupled with the sensitization of the myocardium to catecholamines by halothane. However, ventricular dysrhythmias were present for 85 min after discontinuation of halothane, making an interaction between halothane and catecholamines an unlikely explanation for the dysrhythmias.

There is no consensus about antibiotic prophylaxis against subacute bacterial endocarditis during surgical situations. However, the current American Heart Association recommendations for antibiotic prophylaxis for dental, gastrointestinal, and genitourinary surgery, or nasotracheal intubation probably should be followed.[13] Mitral valve prolapse accounted for one-third of the cases of subacute bacterial endocarditis patients in one series.[14] The endocarditis has been reported to occur with either regurgitation[6] or merely systolic click.[15]

Because of the prevalence of the mitral valve prolapse syndrome, the anesthesiologist must suspect its presence. When recognized, the perioperative management should include the following: 1) careful preoperative discussion with, and preparation of, the patient for the perioperative experiences—adequate sedation is essential since emotional upset may precipitate dysrhythmias and sudden death[16]; 2) avoidance of drugs known to produce tachycardia; 3) maintenance of intravascular volume to avoid an increase in prolapse; 4) iv propranolol for supraventricular tachycardia or ventricular ectopy, which may occur in the absence of other known causative factors; and 5) iv antibiotics for endocarditis prophylaxis. If unexpected and otherwise inexplicable ventricular dysrhythmias occur during surgery, the patient should be referred to a cardiologist for evaluation.

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Percutaneous Transtracheal Ventilation for Emergency Dental Appliance Removal

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High-frequency ventilation has been used via endotracheal and transtracheal routes for ventilation during surgery and emergency airway support.[1,2] Occasionally transtracheal ventilation has been used in cardiopulmonary resuscitation.[3] In this report a patient is described with partial upper airway obstruction from a hypopharyngeal foreign body with four sharp appendages. Notably percutaneous transtracheal ventilation was utilized successfully to provide an airway during removal of a foreign body, thus avoiding tracheostomy.

Report of a Case

The patient was a permanently institutionalized 65-year-old man with chronic schizophrenia and cerebral deficits from bilateral frontal lobotomy. Six hours prior to transfer he had swallowed his lower denture, lodging it in his hypopharynx. An attempt to remove the foreign body under iv diazepam sedation was abandoned at a local hospital due to his uncooperative nature and the attendant risk of airway compromise or aspiration pneumonia. History regarding his oral intake was unreliably documented. He also had atherosclerotic heart disease with complete heart block for which a permanent pacemaker was in place.

On examination the patient was alert but demented and uncooperative. He could not talk and was sitting forward, producing copious oropharyngeal secretions. His anxious respiration was interrupted by frequent coughing and intermittent inspiratory stridor. Respiratory rate was 18–20 breaths/min. Cyanosis was absent, as were auscultatory wheezes. Anteroposterior and lateral roentgenograms of the neck (fig. 1) revealed a denture in the hypopharynx. This denture had four molar teeth on either side and was normally attached to his lateral incisors by two pairs of curved sharp hooks. The denture was securely lodged in the hypopharynx with the dental surface anteriorly. A major concern was the contiguous relationship of the denture to the epiglottis and larynx, together with the two pairs of hooks impinging on the lateral hypopharyngeal wall.

Initially the patient was sedated heavily, using incremental doses of iv droperidol to 7.5 mg and thiopental 50 mg. The oropharynx was anesthetized topically with lidocaine 10%. Gentle laryngoscopic examination of the oropharynx revealed the dentures to be wedged securely in place. Further, the vocal cords were obscured completely by the denture. During subsequent discussions, concern was expressed that further forceful manipulation of the airway for either oral endotracheal intubation or removal of the appliance could lead to damage to the pharynx and larynx by the hooks on the denture. Tracheostomy under local anesthesia first was considered to secure the airway. Instead, a trial of transtracheal ventilation with high-frequency manual insufflation was elected, with emergency tracheostomy to be performed if that approach failed.

Transtracheal lidocaine 2%, 2 ml, was administered through the cricothyroid membrane. A 2½ inch 14-gauge percutaneous catheter then was inserted via the cricothyroid membrane. The patient tolerated this well without coughing. Ventilation then was instituted manually through the catheter with a 50 PSI Venturi-Saunders® insufflator at 60 cycles/min and $F_{I_{O_2}}$ 1.0. Excursion of the chest occurred, and patency of the upper airway was demonstrated. General anesthesia then was induced rapidly and maintained with iv thiopental boluses and succinylcholine by iv infusion. Muscle relaxation was monitored with a peripheral nerve stimulator. Arterial systolic

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