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Intraoperative ST-Segment Changes Consistent with Myocardial Ischemia in the Neonate: A Report of Three Cases

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It is commonly believed that monitoring for intraoperative myocardial ischemia is unnecessary in neonates. Whereas electrocardiography (ECG) lead systems for adults are concerned with the detection of ischemia as well as dysrhythmias, neonatal ECG monitoring has focused on dysrhythmia recognition alone. Recent studies, however, suggest that the neonatal heart is more susceptible to ischemia than is the adult heart. The following cases illustrate the value of intraoperative calibrated ECG monitoring for myocardial ischemia in neonates.

CASE REPORTS

Case 1. A 790-g 1-month-old female (26 wk gestation) with a history of respiratory distress syndrome (RDS), intraventricular hemorrhage, and necrotizing enterocolitis presented for ligation of a patent ductus arteriosus (PDA). During awake laryngoscopy 3 mm ST-segment elevations occurred (lead II) without bradycardia and resolved after intubation of the trachea was completed (fig. 1). The anesthetic consisted of fentanyl (10 $\mu g/kg$), pancuronium (0.1 mg/kg), and an air/oxygen mixture (Fi_{O_2} = 1.0). During the procedure the lung was compressed without retracting the heart and a 2 mm ST-segment elevation again occurred in lead II with a decrease in heart rate from 180 to 160 beats/min. Both the heart rate and ST-segments returned to baseline when the lung was reexpanded. The patient's recovery was uneventful and postoperative ECG was normal.

Case 2. A 3.5-kg two-day-old full term male with transposition of the great vessels underwent an emergency Senning procedure (redirection of venous return *via* intraatrial baffle) after an atrial septostomy failed to provide adequate mixing and arterial oxygenation. Induction of general anesthesia with morphine (1 mg/kg) and pancuronium (0.2 mg/kg) was uneventful as was initiation of hypothermic circulatory arrest and cardioplegia. Thirty-five minutes after termination of cardiopulmonary bypass, during chest closure, 3 mm ST-segment elevations occurred in lead II (fig. 2). Increases in heart rate from 150 to 180 beats/min and blood pressure from 100/60 to 110/70 mmHg were noted, and an arterial blood gas demonstrated a *p*H of 7.24, P_{CO_2} 42 mmHg, P_{O_2} 72 mmHg (Fi_{O_2} = 1.0). Although ST-segments returned to baseline after the administration of dopamine (5 $\mu g \cdot kg^{-1} \cdot min^{-1}$), isoproterenol (0.3 $\mu g \cdot kg^{-1} \cdot min^{-1}$) and nitroglycerin (3 $\mu g \cdot kg^{-1} \cdot min^{-1}$), the patient continued to manifest progressive acidosis and hypotension, which led to electromechanical dissociation and cardiac arrest 12 h postoperatively.

Case 3. A 4-kg three-week-old full-term female presented for elective closure of a large subpulmonic ventricular septal defect (Q_p/Q_s = 5:1). Inhalation induction with halothane/O_2/N_2O and precardiopulmonary bypass maintenance of anesthesia with halothane/morphine/O_2/pancuronium were uneventful. The defect was repaired with a Dacron patch through a right ventricular incision, which was closed with a pericardial patch over the infundibulum. One hour after termination of cardiopulmonary bypass, the patient developed second-degree heart block with 1 mm ST-segment depression (lead II) and periods of ventricular tachycardia (fig. 3). Blood pressure decreased from 80/40 mmHg to 60/20 mmHg, but the ventricular rate remained at 150 beats/min. Arterial blood gas (Fi_{O_2} = 1.0) revealed *p*H 7.41, P_{CO_2} 32 mmHg, and P_{O_2} 397 mmHg. Blood pressure improved after calcium gluconate (100 mg/kg), dopamine (10 $\mu g \cdot kg^{-1} \cdot min^{-1}$), and temporary atrial–ventricular sequential pacing. Upon arrival in the Pediatric Intensive Care Unit, the patient no longer required cardiac pacing and the blood pressure and heart rate were stable at 80/40 mmHg and 150 beats/min, respectively. Postoperative ECG showed a normal sinus rhythm with 1 mm ST-segment depression in leads V_2–V_4, and echocardiogram demonstrated a dilated right ventricle, poor left ventricular function, septal and apical dyskinesis, and mild mitral

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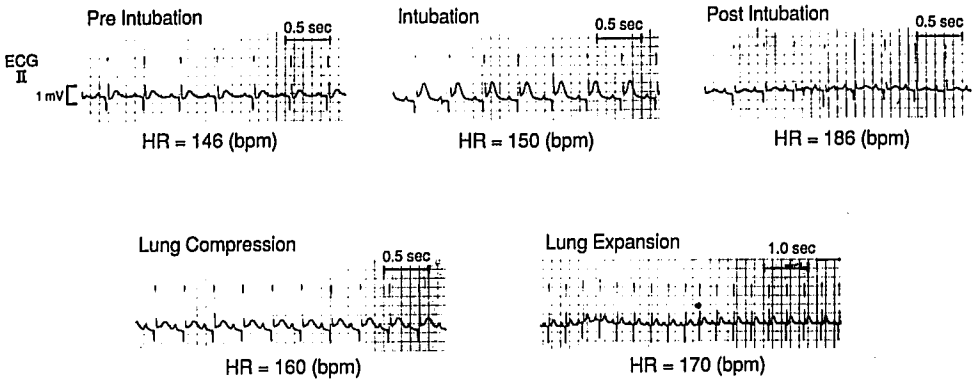

FIG. 1. ST-segment elevations noted on ECG (lead II) occurring during tracheal intubation and lung compression in a 1-month-old, 790-g female preterm infant undergoing closure of patent ductus arteriosus. Changes resolved after intubation and lung reexpansion without pharmacologic intervention.

and aortic insufficiency. With inotropic and ventilatory support, the patient improved and on the first postoperative day the ST-segments returned to baseline and echocardiogram showed improved septal and apical wall motion. By the fourth postoperative day both the ECG and echocardiogram were normal and the patient was withdrawn from ventilatory support and the trachea extubated without difficulty. She was discharged on the eighth postoperative day with a normal ECG and echocardiogram and requires daily digoxin (40 μg) and furosemide (4 mg).

DISCUSSION

The causes of ST-segment changes in pediatric patients are multiple.[1,2] Besides myocardial ischemia and infarction, ST changes may reflect left or right ventricular hypertrophy with strain, drug effects (particularly digitalis), myocarditis and pericarditis, and electrolyte aberrations, most notably hypokalemia. Calcium abnormalities may change the length of the ST-segment but do not usually cause elevation or depression. Likewise, increased catecholamines may shorten the QT interval, although the ST remains isoelectric. Marked alkalosis or hypernatremia may show ST depression similar to that seen with hypokalemia.

Nonpathologic ST changes may be seen classically with J-point depression or early repolarization. These changes are usually seen in the cardiograms of adolescents and black males. In addition, change in position with resultant vector reorientation may affect the ST-segment.

However, in the three cases presented, these nonischemic causes of ST-segment changes can most likely be excluded. Case 1 had ST elevation occurring during intubation and during lung retraction—both periods of hypoxia. Furthermore, the ST changes resolved with adequate oxygenation. The heart was not retracted or reoriented during either occurrence. Neither is it likely that transient electrolyte abnormalities occurred and self-corrected because electrolytes were normal preoperatively and postoperatively. More likely, ischemia secondary to hypoxia occurred in an infant more vulnerable because of RDS and heart failure.

In contrast, the last two cases demonstrate ischemia without hypoxia. The sudden presence of ST elevation during chest closure in case 2 could be confused with changes in vector orientation, although improvement with inotropic agents and nitroglycerin make this unlikely.

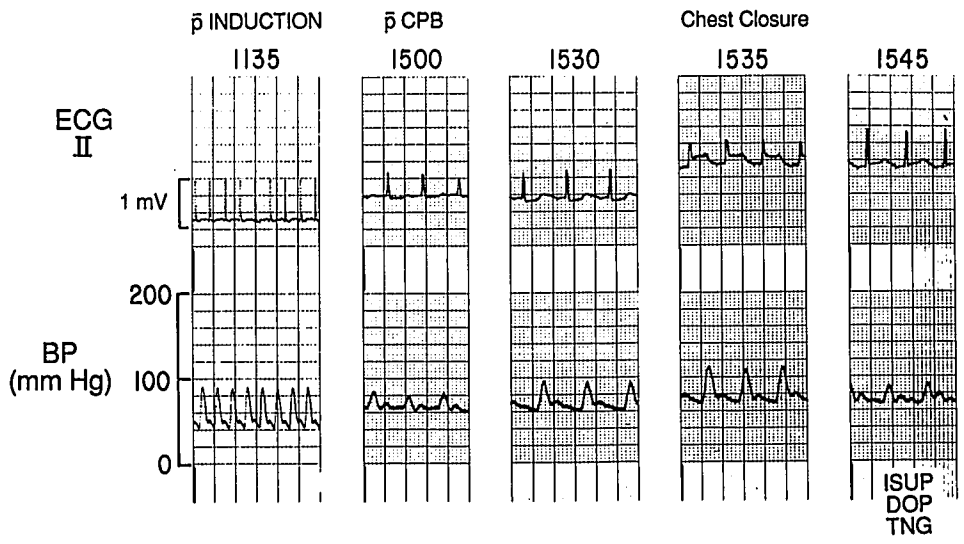

FIG. 2. Early ST-segment changes in lead II in a 2-day-old, 3.5-kg male undergoing Senning procedure first noted immediately postbypass (1500 h) and 30 min later (1530 h). Significant ST elevation during chest closure (1535 h) improved after administration of trinitroglycerine (TNG), isoproterenol (ISUP), and dopamine (DOP) at 1545 h.

Furthermore, progressive pump failure leading to the patient's death is consistent with the intraoperative diagnosis of ischemia. In case 3 ischemia and injury were confirmed by echocardiography.

The recent literature regarding neonatal vulnerability to ischemia is controversial. Several authors have suggested that the neonatal heart is less vulnerable to hypoxia but more vulnerable to ischemia than is the adult heart.[3–5] During global ischemia, the neonatal heart produces nearly twice the amount of lactate as that produced by the adult heart.[3] The subsequent lactate accumulation causes acidosis, production of oxygen free radicals, membrane phospholipid degeneration, and inhibition of glycolysis.[4–6] Conversely, others in separate laboratories have determined that neonatal myocardium is more tolerant of ischemic arrest than mature myocardium in the rabbit.[7,8] All of these studies were performed on normal hearts; vulnerability in pathologic hearts is not determined.

Myocardial hypertrophy and congestive heart failure seen with complex congenital heart disease increase the susceptibility of the myocardium to low perfusion injury, particularly with the decreased diastolic perfusion pressure that exists in severe left to right shunts (*e.g.*, PDA).[9] Direct cardiac compression with surgical retractors may decrease diastolic pressure and further jeopardize subendocardial blood flow in patients with right ventricular and pulmonary artery pressures equal to systemic blood pressure. Franciosi and Blanc reported a 38% incidence of infarction in infants with complete transposition of the great vessels.[10] Infarcts were localized to subendothelium and papillary muscle, suggesting that hypertrophy and relative coronary insufficiency contribute significantly to myocardial ischemia. This may indeed have been the case with the second patient.

As early as 1954, Enhorning and Westin reported the effects of prolonged asphyxia on the ECG of previable human fetuses.[11] Changes in the ST-segment and T-wave inversion did not occur until 45 min after the onset of hypoxia. However, ECG changes consistent with anterior infarction occurred within 30 s after injection of norepinephrine. Bor reviewed a series of 29 infants and children with autopsy-proven infarction and found only two patients without an ischemic pattern on ECG.[12] Although the presence or absence of Q-waves was a dubious predictor of infarction, T-wave and ST-segment changes reliably heralded ischemia in this series. And although the use of creatinine kinase isoenzymes and thallium imaging for the diagnosis of neonatal ischemia has been suggested,[13,14] these modalities are impractical for continuous monitoring in the operating room.

These cases illustrate the occurrence of myocardial ischemia complicating repair of congenital heart disease in neonates. ST-segment trend analysis and intraoperative

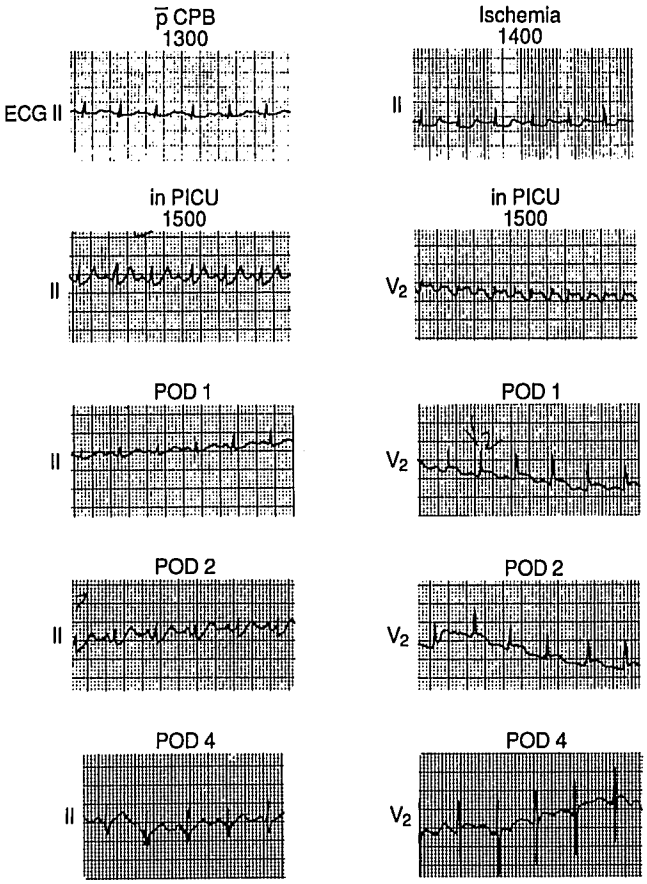

FIG. 3. Evidence of ischemic changes in leads II and V_2 occurring after repair of right ventriculotomy for VSD closure in a 3-wk-old infant. Postoperative echocardiogram revealed poor right and left ventricular function. Improvement in ventricular function paralleled return of ST-segments to baseline; 1 mV = 10 mm.

echocardiography in the adult cardiac surgical patient are being gradually introduced into clinical practice to enhance the traditional role of calibrated ECG. However, the same monitoring techniques are rarely considered in infants who may be at similar risk for ischemia. This clustering of patients with myocardial ischemia, potentially contributing to significant morbidity, demonstrates the importance of intraoperative calibrated ECG monitoring in neonates with congenital heart disease.

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Failure of Preoperative Echo Testing to Prevent Paradoxical Air Embolism: Report of Two Cases

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Much of the information about paradoxical air embolism (PAE) has been acquired because of the clinical significance of this problem in neuroanesthesia. In an attempt to reduce the risk of this complication, we and others[1] have suggested the possible value of detection of patent foramen ovale (PFO) prior to the occurrence at venous air embolism. The role of preoperative echo is not fully defined but has been advocated because it seems to be reasonable in helping to define the population at risk for PAE.

These case reports demonstrate that preoperative and intraoperative testing that fails to detect a PFO does not assure safety from PAE.

CASE REPORTS

Case 1. The patient was a 47-yr-old white male scheduled for suboccipital craniotomy for a pineal tumor. The patient had received radiation therapy for the lesion. It had continued to increase in size with an increase in symptoms. There was no history of cardiac or pulmonary disease.

Preoperative precordial echo was performed by an experienced echocardiographer (R.A.N.). A standard two-dimensional echocardiogram was initially done, viewing the heart from multiple tomographic positions, as previously described. Special attention was made to visualize the atrial septum, which appeared intact from the parasternal and subcostal windows. Agitated indocyanine green dye was then injected into the left brachial vein, followed by a flush injection of normal saline while imaging the heart from the apical four-chamber position during spontaneous respiration. This resulted in prompt opacification of the right atrium and right ventricle from the microbubbles in the indocyanine green dye solution with absence of microbubbles in the left sided chambers. Two more injections were performed during the release of a Valsalva maneuver. There was an increase in jugular venous pressure and a reflex tachycardia during the strain phase of the Valsalva maneuver indicating that the maneuver was done properly.[2] The injections were made during the end of the straining phase of the maneuver to assure maximal appearance of microbubbles during the release phase. There was no evidence of right to left shunting either during spontaneous respiration or following the Valsalva maneuver.

Intraoperative transesophageal echo (TEE) testing was also performed by a physician experienced with this technique (R.F.C.). A clear four-chamber view of the heart was obtained with a 3.5-MHz Diasonics esophageal echo probe mounted on a 5-mm bronchoscope body and connected to a 3400 Diasonics® echocardiographic machine. A multiorifice right atrial-superior vena caval catheter was inserted using electrocardiographic control. With the patient anesthetized with isoflurane 0.5% inspired and 50% nitrous oxide in oxygen partially paralyzed with vecuronium used for intubation, and in the upright position for surgery, 10 ml of agitated saline was injected at end-expiration without passage of the microbubbles into the left atrium. The injection was repeated twice with 20 cm H_2O positive end expiratory pressure (PEEP) and once with 30 cm H_2O PEEP with no evidence of left atrial microbubbles. The injection was performed just before the release of PEEP. The central venous pressure (CVP) remained at 8 mmHg just after the test until venous air embolism (VAE) occurred later.

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