Truncus Arteriosus: Recognition and Therapy of Intraoperative Cardiac Ischemia

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Truncus arteriosus and hemitruncus arteriosus are congenital cardiac anomalies that present with congestive heart failure during the early months of life. Surgical correction is required for prolonged survival. As a consequence of their pathophysiology, patients with these anomalies may be prone to develop myocardial ischemia during general anesthesia. The following cases demonstrate intraoperative electrocardiographic detection of myocardial ischemia prior to surgical repair, as well as successful intraoperative (prebypass) management.

REPORT OF FOUR CASES

Case 1. A 2.2-kg 6-week-old (34-week gestation) female with truncus arteriosus type I presented for repair. Her preoperative blood pressure was 85/50 mmHg. Induction of general anesthesia with fentanyl, midazolam, pancuronium, and an air/O₂ mixture (fractional inspired O₂ concentration [Fİ₂] = 0.3) was uneventful.

One hour later, 3-mm ST-segment depression occurred in leads II, III, and AVF (fig. 1). Her blood pressure was 55/37 mmHg and heart rate 148 beats per min. Her blood pressure was unresponsive to a fluid bolus of 5 ml/kg 5% albumin. Arterial blood gas (Fİ₂ = 0.3) revealed that pH = 7.48, CO₂ tension [PacO₂] = 38 mmHg, O₂ tension [PaO₂] = 104 mmHg, and base excess +2 mEq/l. Oxygen saturation (SOp₂) 92% by pulse oximetry. The ST-segment depression persisted, and the surgeons were notified. A temporary band was placed on the right pulmonary artery. The blood pressure immediately increased to 80/50 mmHg and the heart rate was unchanged. The ST-segment promptly returned to baseline and the surgical repair was performed after cardiopulmonary bypass was begun. Separation from cardiopulmonary bypass was as well as the postoperative course, was uneventful.

Case 2. A 5.3-kg 4-month-old female with right hemitruncus arteriosus and a hypoplastic left lung presented for repair. A murmur had been discovered at 2 months of age, and the patient now was exhibiting signs of congestive heart failure. Induction of general anesthesia with fentanyl (14 µg/kg), midazolam (0.2 mg/kg), and pancuronium (0.2 mg/kg), and an air/O₂ mixture (Fİ₂ = 0.3) was uneventful. Postinduction blood pressure was 90/50 mmHg and the heart rate was 130 beats per min. She received an additional 6 µg/kg fentanyl prior to skin incision.

Two hours later, before cannulation for cardiopulmonary bypass, 3-mm ST-segment depression occurred in leads I, II, III, AVF, and V5 (fig. 2). The blood pressure was 90/26 mmHg and the heart rate was 142 beats per min. The blood pressure was unresponsive to a bolus of 10 ml/kg 5% albumin. Arterial blood gas analysis (Fİ₂ = 0.3) revealed that pH = 7.33, PacO₂ 34 mmHg, and PaO₂ 134 mmHg. The Fİ₂ was decreased to 0.21 and the minute ventilation decreased to decrease the arterial O₂ saturation from 100 to 93% and to increase the end-tidal CO₂ tension (PETCO₂) from 34 to 44 mmHg.

The ST-segment depression persisted despite the above maneuvers, and therefore the surgeons were asked to place a temporary band on the right pulmonary artery. The blood pressure immediately increased to 105/36 mmHg, and the heart rate was unchanged. The ST-segment changes promptly resolved, and surgical repair was performed after cardiopulmonary bypass was begun. Separation from cardiopulmonary bypass was uneventful and the patient did well postoperatively without ischemic ECG changes.

Case 3. A 2.6-kg, 7-week-old term female with right hemitruncus arteriosus presented for repair. The patient had a history of progressively worsening congestive heart failure. Cardiac catheterization revealed a left ventricular end-diastolic pressure (LVEDP) of 20 mmHg. Induction of general anesthesia with fentanyl (20 µg/kg), midazolam (0.2 mg/kg), pancuronium (0.2 mg/kg), and air (Fİ₂ = 0.21) was uneventful. Baseline blood pressure was 85/45 mmHg and heart rate 150 beats per min.

Ninety minutes later, 4-mm ST-segment depression occurred in leads II, III, and AVF (fig. 3). The blood pressure was 66/26 mmHg and the heart rate 130 beats per min. The blood pressure was unresponsive to a bolus of 8 ml/kg 5% albumin. Arterial blood gas analysis (Fİ₂ = 0.21) revealed that pH = 7.45, PacO₂ 45, and PaO₂ 87. A temporary band was placed around the right pulmonary artery. The blood pressure immediately increased to 74/37 mmHg, and the heart rate decreased to 115 beats per min. The ST-segments improved to a 2-mm depression by the time of cardiopulmonary bypass 5 min later. Separation from cardiopulmonary bypass was uneventful, and the patient did well postoperatively. There were no postoperative ischemic ECG changes.

Case 4. A 3.7-kg 6-week-old term female with truncus arteriosus type I and congenital hypothyroidism presented for repair. Her preoperative blood pressure was 96/58 mmHg, and her LVEDP at the time of cardiac catheterization was 18 mmHg. On arrival in the operating room, her blood pressure was 90/30 mmHg. Induction of anesthesia was with fentanyl (10 µg/kg), midazolam (0.2 mg/kg), pancuronium, and an air/O₂ mixture (Fİ₂ = 0.66). Isoflurane was begun at 0.25%.

While a surgical cutoff was being done for placement of an arterial catheter, marked ST-segment depression was noted in several ECG leads. Isoflurane was discontinued, the Fİ₂ was immediately decreased to 0.21 and minute ventilation was decreased. Sop₂ at this time by pulse oximetry remained in the low 90% range. The arterial blood pressure...
PATIENT 1

![ECG waveforms](image)

**Fig. 1.** ECG evidence of ischemia in patient 1. Leads II, III, and aVF are shown. There is significant ST-segment depression at the time of ischemia in all three leads; it resolves after pulmonary arterial banding. The postband tracing was recorded 7 min after the ischemia tracing and approximately 2 min after placement of the band. All ECG recordings were done at the same gain (1 mV per 10 mm in the original) and in the “diagnostic mode” of the ECG.

at this time was 80/19 mmHg. Soon after, the infant developed two episodes of ventricular fibrillation, which were successfully electrically converted. FiO₂ was increased to 1.0. She then developed bradycardia, which responded to an epinephrine infusion. A midline sternotomy was performed rapidly, and both pulmonary arteries were partially snared. The arterial blood pressure increased to 85/40 mmHg with return of the ST-segments toward normal by the time of cardiopulmonary bypass several minutes later.

**DISCUSSION**

As described by Bell et al., a number of conditions can cause ST-segment changes that are exclusive of myocardial ischemia. The onset of the ST-segment changes in our patients occurred preceding or quite a while after sternotomy. Changes in cardiothoracic dimensions upon sternal spreading as a cause of the ST-segment changes can thus be excluded. In addition, the hearts were free of mechanical retraction or other manipulation during the ischemic period.

It was believed that the etiology of the ST-segment changes was myocardial ischemia secondary to inadequate coronary perfusion. Inadequate coronary perfusion was suspected because the ST-segment changes were coincident with or immediately followed the decrease in the systemic diastolic blood pressure in all four patients. The patient of case 3 demonstrated an LVEDP of 20 mmHg during cardiac catheterization. This patient had a systemic diastolic pressure of 26 mmHg during the proposed ischemic episode. If this patient had an intraoperative LVEDP that was similar to the one obtained during catheterization, then she may have had a coronary perfusion pressure of only 6 mmHg during the ST-segment changes. Similarly, the patient of case 4 had a potential coronary perfusion pressure approaching 0 mmHg.

Efforts to increase the systemic diastolic blood pressure with volume administration were ineffective. Within reasonable limits, efforts to increase pulmonary vascular resistance by ventilator manipulations (decrease in FiO₂ and increase in PaCO₂), thereby decreasing the aortic diastolic
runoff and increasing systemic arterial diastolic pressure, were uniformly unsuccessful in improving the ST-segment changes in all four patients. This maneuver was unsuccessful despite its recommendation in the management of other total mixing lesions with excessive pulmonary blood flow, such as hypoplastic left heart in neonates.  

The difference in the current cases may be due at least in part to the greater age of the patients with truncus arteriosus and to the already existing decreases in their pulmonary vascular resistance (which is why they are in heart failure) with partial regression of pulmonary arterial musculature. Even though FiO2 was decreased to 0.21, due to the excessive pulmonary blood flow SpO2 was never less than the low 90% range, and there was no apparent worsening of ischemia with this maneuver. Lastly, surgical placement of a temporary pulmonary artery band was immediately effective in increasing the systemic diastolic blood pressure and in resolving or ameliorating the ST-segment changes in all four patients. Use of a temporary pulmonary artery band has been suggested by others for use with pulmonary steal and inadequate systemic flow.  

In the congenital cardiac anomalies of the current cases, there is an unobstructed direct communication between the systemic and pulmonary circulations. The result is that these two circulations exist in parallel instead of in the normal series circulation. In accordance with the hemodynamic considerations for a parallel circuit, the much lower pulmonary vascular resistance contributes to a low resistance for the total circuit. The result is ventricular volume overload from an excessive amount of pulmonary blood flow, and a relatively low systemic diastolic blood pressure. This excessively large pulmonary blood flow was evidenced in all four patients by very high SpO2 or PaO2.

In patients with common mixing lesions such as truncus arteriosus, a systemic PaO2 of 90% represents a pulmonary: systemic flow ratio that may be in excess of 4:1. Ventricular dilatation leads to increased myocardial wall tension as described by the law of Laplace (T = Pr/2h where T = wall tension; P = pressure; r = intraventricular radius; and h = wall thickness). This increased wall tension necessarily means an overall higher myocardial O2 consumption. Confounding this increased myocardial O2 consumption is a low systemic diastolic pressure. The ventricle depends on an adequate coronary perfusion pressure for coronary perfusion. As discussed, in the patients of cases 3 and 4, ST-segment changes occurred when the coronary perfusion pressure may have been less than 6 mmHg. It is reasonable to assume that a high myocardial O2 consumption and a deleterious condition for coronary perfusion are favorable for the development of ischemia.

In three patients, ischemia did not develop until more than 1 hour after induction of anesthesia. It may be that the increased pulmonary blood flow and consequent decreased diastolic blood pressure as a result of anesthetic or surgical manipulation or both can be tolerated briefly, but with time the heart decompensates and dilates. It is possible that a more rapid institution of cardiopulmonary bypass after induction of anesthesia may have prevented this decompensation and the development of ischemia.

The detection and prompt treatment of myocardial ischemia before the actual surgical repair may have implications on the eventual outcome of the surgical procedure. Continued, untreated myocardial ischemia may lead to cardiac arrest in the prebypass period (as in the patient of case 4) or to cardiac failure in the postbypass period. In a report by Tawes et al., a significant number of infants who expired after placement of a Blalock-Taussig shunt or ligation of a patent ductus arteriosus had histologic evidence of myocardial ischemia/necrosis on postmortem examination. These authors attributed their findings to inadequate coronary perfusion. Our conclusions are simpler since in each case the ST-segment changes were accompanied by a low systemic diastolic pressure and responded favorably to an increase in this pressure.

Although we specifically concerned ourselves with myocardial ischemia, it is not unreasonable to suspect that this physiology results in inadequate perfusion of a variety of organs.

ADDENDUM

Since the submission of this manuscript, we have anesthetized two additional infants with truncus arteriosus and physiology similar to the above cases. Both patients developed ST-segment depressions consistent with myocardial ischemia that did not resolve with boluses of intravenous fluid or ventilatory maneuvers, also similar to our experience in the four patients above. In addition, one of these two patients was given several boluses of phenylephrine in doses of up to 4 μg/kg per dose. There was only a slight increase in systolic pressure and no increase in diastolic pressure or resolution of the ST changes. Ischemic ECG changes resolved in both patients after temporary banding of the pulmonary artery. One patient had a readily reproducible association of pulmonary artery banding, SpO2, and ischemic changes. With tightening of the band, SpO2 decreased and ST changes normalized. With loosening of the band, SpO2 increased and ischemic ST-segment changes returned, changes that demonstrate again the association of myocardial ischemia and increased pulmonary blood flow.

REFERENCES