There is abundant evidence implicating CSF leak at the dural puncture site as the cause of PDPH.[1] It generally is believed that this CSF leak causes a decrease in CSF pressure and subsequent traction on pain-sensitive intracranial structures. Supporting this hypothesis are studies showing an increase in the incidence of PDPH with increasing needle size, the presence of decreased CSF pressure in patients with PDPH, and direct visualization of the CSF leak at subsequent laminectomy.[9] An epidural blood patch is believed to act by forming a gelatinous plug over the dural leak and promoting fibroblast activity and collagen deposition over the clot to permanently seal the dural tear.[7] This mechanism, along with the proven effectiveness of epidural blood patch,[10] provides additional evidence for CSF leak as the cause for prolonged PDPH.

We have presented a case of a postmyelogram headache with an unusually delayed onset. We propose that obstruction of CSF flow, and its subsequent restoration with treatment, led to delayed CSF leak and headache. Epidural blood patch yielded complete relief. This proposed mechanism further substantiates CSF leak as the cause of PDPH and the effectiveness of blood patch in this situation.

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External Compression of the Abdominal Aorta Reversing Tetralogy of Fallot Cyanotic Crisis

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Cyanotic spells are not rare during anesthesia in children with Tetralogy of Fallot (TOF). Classically, the treatment of this life-threatening crisis is pharmacologic.[1–3] During open-chest operations, surgeons may briefly clamp the thoracic aorta to reverse hypoxic spells.[4] We describe how a similar maneuver consisting of manual external compression of the abdominal aorta quickly interrupted severe cyanotic episodes.

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Case Reports

Case 1. This 6.9-kg, 6-month-old boy with TOF experienced profound hypoxic episodes with increasing frequency. Total repair was scheduled. He was given a bottle of water with sugar and 1 mg oral propranolol at 3:00 AM. At 7:00 AM, 1 mg morphine hydrochloride and 0.2 mg atropine sulfate were injected intramuscularly. The child was calm on arrival to the operating room. Peripheral hemoglobin oxygen saturation (Sp_{O_2}) noted from a pulse oximeter probe (Oscar, Datex, Finland) placed at the left big toe was 98%. Anesthesia was induced smoothly with up to 2.5% halothane in oxygen. A saphenous vein was cannulated and nasotracheal intubation performed. A sidestream capnograph was placed using a special pediatric connector (Oscar, Datex). Halothane was discontinued, and 1 mg diazepam, 50 μg fentanyl, 1 mg pancuronium bromide, and 20 ml of a 5% albumin solution then were given intravenously (iv). Mechanical ventilation was begun. A femoral artery and the right internal jugular vein were cannulated. Seventy five μg fentanyl was added. Central venous pressure was 10 mmHg, heart rate 120 beats per min, and Sp_{O_2} > 96%, and blood pressure had decreased slightly, from 92/65 to 85/47 mmHg. Twenty milliliters 5% albumin solution had been given to compensate for blood losses due to percutaneous catheter insertions.

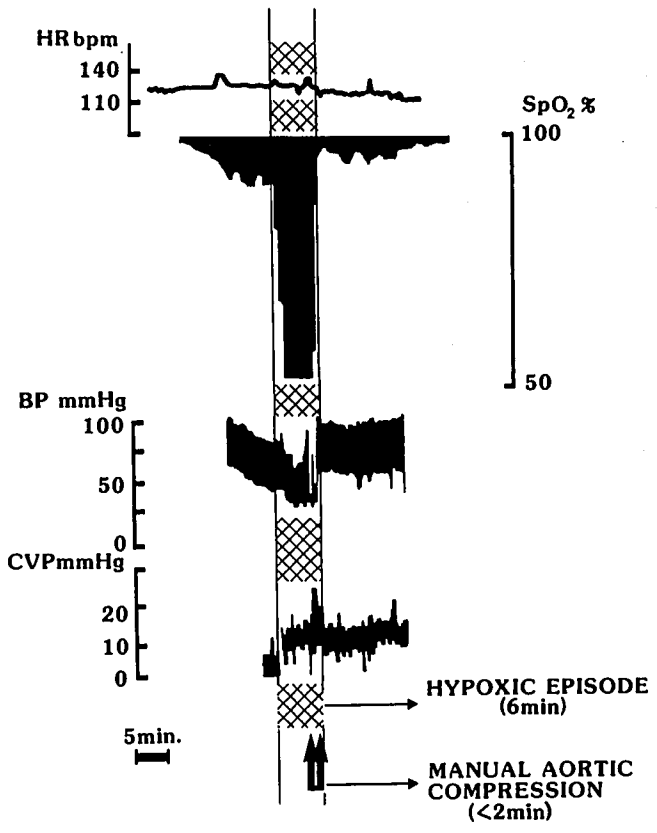

FIG. 1. Sequence of events described in the case report. The cross-hatched area corresponds to the clinical perception of ongoing cyanotic crisis, which lasted about 6 min. Note that values of Spo_2 lower than 50% could not be retrieved from the monitoring memory. External aortic compression lasted less than 2 min (between arrows) and was briefly released after about 1 min, as shown by a spike in the arterial pressure trace. HR = heart rate (beats per minute); BP = blood pressure tracing; CVP = central venous pressure tracing; Spo_2 = pulse oximeter readings.

A 7-cm roll was put under the shoulders, and the head was turned for left jugular puncture. The child's condition deteriorated rapidly after this change of position: he became pale and his lips turned gray. Spo_2 decreased to 65%. Auscultation of the lungs was normal. Blood pressure was 60/33 mmHg and pulse rate 125 beats per min (fig. 1, shaded area). Rapid infusion of 40 ml albumin solution transiently increased arterial and central venous pressures, but Spo_2 decreased further to 35% and end-tidal CO_2 pressure decreased to as low as 14 mmHg.

The abdominal aorta of the child then was progressively manually compressed, just enough to obtain disappearance of the femoral arterial tracing (first arrow, fig. 1), and compression was maintained for 45 s (fig. 2). Releasing it for a few seconds revealed an Spo_2 of 65%. Aortic compression was applied again for 30 s, after which Spo_2 was 97%, blood pressure 95/58 mmHg, central venous pressure 12 mmHg, and end-tidal CO_2 pressure 24 mmHg. No vasopressor nor any other drug was given, and the hemodynamics remained stable thereafter.

Case 2. This 4.8-kg 2-month-old boy presented a combination of tricuspid atresia and atrial septal defect with a patent right Blalock-Taussig shunt. He was anesthetized with 2.5% halothane in oxygen for an inguinal hernia repair. An abrupt hypoxic episode occurred shortly after tracheal intubation: Spo_2 decreased from 99 to 87% while the absence of the left radial and humeral pulses was noted. Manual external compression of the abdominal aorta was applied for 20 s: Spo_2 immediately increased to 94% and peripheral arterial pulses reappeared. A caudal block was performed and surgery proceeded uneventfully.

Case 3. This 3.1-kg, 6-week-old girl with TOF and severe cyanosis at rest (Spo_2 = 60%) was scheduled for a Blalock-Taussig shunt. Although Spo_2 increased toward normal levels (> 90%) during induction of anesthesia using halothane in oxygen, it slowly decreased back to 60% after intubation, when halothane was discontinued and replaced by fentanyl and pancuronium bromide. Twenty micrograms of phenylephrine given iv over 5 min increased Spo_2 to 80%. Manual external aortic compression resulted in a further increase of Spo_2, to 94%. Desaturation occurred again during surgery despite continuous iv phenylephrine and lasted until the shunt was opened to circulation.

Case 4. This 5.4 kg, 6.5-month-old girl with TOF was scheduled for a Blalock-Taussig shunt. Her surgery was delayed from 10 AM until 5 PM, and during the waiting period she received propranolol but neither

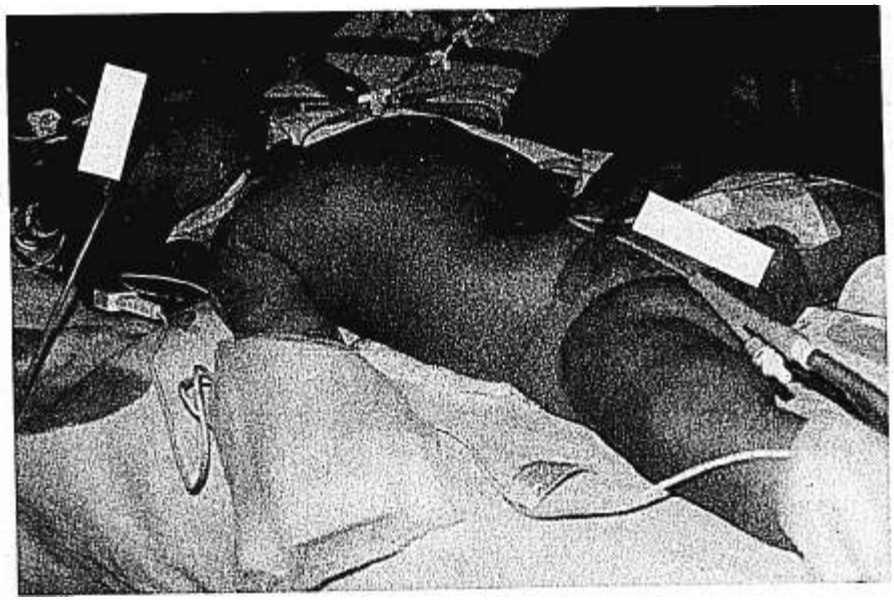

FIG. 2. External manual pressure is progressively applied, compressing the abdominal aorta between the physician's fingers and the child's vertebrae. Disappearance of femoral pulse indicates complete aortic occlusion, which may be monitored invasively, clinically, or by pulse oximetry of plethysmography.

feeding nor iv hydration. She arrived dehydrated and agitated in the operating room, and Sp_{O_2} was 88% on room air but increased to 97% when breathing oxygen by mask. Induction of anesthesia, performed with halothane in oxygen and the infusion of 40 ml of polygeline (Haemaccel®) to compensate the preoperative fluid deficit, was uneventful. While anesthesia was maintained with 1% halothane in oxygen, the child's Sp_{O_2} decreased to 75% when she was uncovered for surgery: an additional 20 ml 5% albumin solution was given, but the Sp_{O_2} decreased to as low as 39%. Manual external compression of the aorta resulted in an increase in the Sp_{O_2} to 95%. Fifteen minutes later, another episode of desaturation occurred (with Sp_{O_2} = 65%); this time, compression of the aorta was not able to keep Sp_{O_2} greater than 80% for more than a few seconds. A bolus of 10 μg phenylephrine was then injected, resulting in a Sp_{O_2} of 90%.

Discussion

Cyanotic episodes in TOF ("tet spells") usually are related to exercise but may also occur at rest and during anesthesia. Recently, intraoperative epicardial echography[5] elegantly confirmed that the mechanism responsible for cyanosis is intracardiac shunting of blood from right to left and showed how shunt reversal after treatment corrected hypoxemia.

The reasons for reversal of shunting are not yet fully understood. The following events have been cited as contributing to the cyanotic episodes[6,7]: blood pooling in the legs while standing, return of acid and desaturated venous blood from exercising muscles, hyperventilation, increased sympathetic tone with subsequent infundibular "spasm,"[2] and reduction of right ventricular diastolic volume by tachycardia. Decreased ventricular ejection through the congenitally narrowed right outflow tract is the final common mechanism precipitating right-to-left shunting, inadequate pulmonary blood flow, and the crisis.

Children with TOF spontaneously adopt a knees-to-chest position, known as "squatting," to control hypoxic episodes. This may act by ending physical activity and thereby decreasing oxygen consumption, and by reducing minute ventilation. It also has direct effects on both the venous and arterial sides of the systemic circulation.[2,6–8] Squatting is followed by a transient increase in blood flow through the inferior vena cava, which could increase right ventricular volume and counteract infundibular closure.[7] After this initial increase, flow through the inferior vena cava consistently decreases. This reduces venous return from exercised muscles in the legs and therefore improves the Sa_{O_2} of blood reaching the great circulation *via* the right ventricle. The venous effects depend on gravity and are almost nonexistent when squatting is performed while supine or underwater.[8] Passive squatting during anesthesia therefore is rarely efficient. Squatting also reduces the arterial blood flow to the legs, without interrupting it.[7] Any resulting increase in systemic resistance may contribute to restoring blood flow toward the lungs[9] because the resistance of the pulmonary circulation is fixed by the narrowed right ventricle infundibulum: with increased resistances, the left ventricle is forced to eject through the septal defect, reversing the direction of shunting. Additional evidence of a salutary effect of increased systemic resistance comes from the observation that children with the combination of coarctation of the aorta and TOF are seldom hypoxic.[10]

Nolan *et al.*[4] described how brief surgical clamping of the thoracic aorta consistently and totally reverted hypoxic spells in open-chest cases. When needed these authors extended surgical dissection to gain access to the aorta.

Current anesthesia practice[1–3] favors pharmacologic means to treat intraoperative hypoxic spells. Bicarbonate is used to overcome acidosis, propranolol to reduce pulmonary outflow hypercontractility, and vasopressors to increase systemic vascular resistances. Intravenous boluses of propranolol or phenylephrine both improve the Sa_{O_2} and reverse the shunt.[5,9] Oral propranolol is well established as chronic palliative therapy before surgical correction,[11] and continuous infusions of phenylephrine have treated hypoxic spells and prevented their recurrence during surgery.[12,13]

Our current observations show that external manual compression of the abdominal aorta can be as effective as surgical clamping or vasopressors. Pulse oximetry and capnography showed clear signs of improving pulmonary blood flow, and, in case 1, femoral arterial pressure monitoring documented complete aortic occlusion between the physician's fingers and the child's vertebrae. The maneuver most certainly acted by producing sudden increase in systemic resistance. In a similar way, in case 2, this action probably enhanced blood flow through the artificial shunt. Exclusion of the legs from the circulation may have decreased inferior vena cava blood flow, but right heart filling probably remained unaffected, because central venous pressure increased during compression, as documented in case 1. This may result from direct pressure transmission from the abdominal cavity, from increased diastolic pressure in the right ventricle due to shunt reversal, or from increased venous return from compressed abdominal parenchymas. Direct compression of the inferior vena cava was unlikely in the described cases because it was known to be normally located on the right side of the vertebral bodies. Moreover, none had abdominal situs inversus or was known to have abnormal intraabdominal anatomy.

External abdominal aortic compression offers several advantages over the conventional pharmacologic approach. It is simple. It can be performed without delay and is easy in small children. It needs no venous access. It can be done at any time, even during surgery, provided there is access to the abdomen. It avoids the use of drugs and the need to change the depth of anesthesia, both of which have long-lasting effects. When successful, its effects are immediate and can be monitored noninvasively with

pulse oximetry and capnometry. Moreover, since it takes little time to assess its efficacy, it does not delay other measures, should they be necessary. Finally, it may result in complete reversal of cyanosis and return to a stable and normal hemodynamic situation. This maneuver has the same physiologic impact as surgical aortic cross-clamping and therefore should not be unnecessarily prolonged.

We propose external manual compression of the abdominal aorta as the first therapeutic step in treating intraoperative cyanotic spells associated with TOF. Whether it also can be attempted in other circumstances (such as out of the hospital) is not known.

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Transesophageal Echocardiographic Diagnosis of Aortic Dissection during Cardiac Surgery

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Aortic dissection during cardiac surgery is a rare complication that is associated with significant morbidity and mortality. Early recognition and management improves patient outcome.[1] The intraoperative diagnosis of ascending aortic dissection is made when inspection of the proximal aorta reveals bleeding, intramural hematoma, or increasing aortic diameter. Visual inspection without aortotomy provides little information regarding the severity and extent of dissection. Dissection of the aortic arch and descending aorta during cardiac surgery is not readily apparent.

The proximity of the esophagus to the thoracic aorta and the high-resolution images produced by two-dimensional ultrasound have established transesophageal echocardiography (TEE) as an accepted standard outside of the operating room in the detection and evaluation of thoracic aortic dissection.[2–7] We report three cases that illustrate the utility of TEE in the diagnosis and intraoperative monitoring of acute aortic dissection during cardiac surgery. The patients were monitored with a 5.0-MHz TEE probe (Sonos 500, Hewlett-Packard, Andover, MA).

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Key words: Monitoring: transesophageal echocardiography. Surgery, cardiac: aortic dissection.

Case Reports

Case 1. A 61-yr-old, ASA physical status 4, 55-kg, 157-cm woman presented for repeat aortic and mitral valve replacements and tricuspid valvuloplasty. Past medical history was remarkable for incompetent bioprosthetic aortic and mitral valves leading to severe aortic and mitral regurgitation, congestive heart failure, and pulmonary hypertension.

The TEE probe was inserted after anesthetic induction and tracheal