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Anesthetic Management of Hemodynamic Changes during Vein of Galen Aneurysm Clipping

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The Vein of Galen malformation is a midline intracranial arteriovenous fistula with aneurysmal dilatation of the Vein of Galen. This abnormality comprises less than 1% of all arteriovenous malformations,[1,2] the highest incidence being in the infant and young child. The lesion is responsible for a 91% cumulative mortality in neonates (both surgically treated and medically managed), with mortality dropping to 53% in surgically treated infants age 1–12 months, and to 25% in children who present for surgical repair after 1 yr of age.[1–3] Mortality is usually due to cardiac complications or uncontrollable hemorrhage at operation. In survivors, long-term central nervous system morbidity in the form of seizures, hemiparesis, blindness, and intellectual impairment remains high. Factors complicating anesthetic management include massive intraoperative hemorrhage, acute congestive heart failure from increased systemic vascular resistance when the aneurysm is clipped, and acute coronary insufficiency due to low diastolic arterial pressure.[2,4,5] We report successful outcome in a 9-week-old infant undergoing Vein of Galen aneurysm clipping, under high-dose narcotic anesthesia. Therapeutic intervention was guided by hemodynamic data obtained from a pulmonary artery catheter.

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CASE REPORT

A 9-week-old, 6-kg male infant presented with seizures and cardiomegaly. Diagnosis of Vein of Galen malformation was made by CAT Scan and cerebral angiogram (fig. 1). Preoperative echocardiogram demonstrated increased left atrial and left ventricular dimensions with good contractility of both right and left ventricles. On physical examination, heart rate ranged from 160 to 190 bpm and arterial blood pressure from 110/40 to 90/35 mmHg with bounding peripheral pulses, no gallop, cranial bruit, or hepatomegaly. Neurological examination was only remarkable for mild hypertonicity of the lower extremities. Blood volume was estimated by Cr^{51} labeled red blood cell study to be 780 ml, 150% of the predicted value. Twelve hours preoperatively, ketamine 1 mg/kg was given iv to facilitate placement of a right femoral pulmonary artery catheter and ulnar arterial line. Initial cardiac index (CI) was 7.0 $l \cdot min^{-1} \cdot m^2$, central venous pressure (CVP) was zero, pulmonary artery diastolic (PAD) pressure was 4 mmHg; pulmonary wedge pressure was 3 mmHg; and systemic vascular resistance index (SVRI) was 680 dynes $\cdot s^{-1} \cdot cm^5$. Pulmonary artery diastolic filling pressures correlated with the wedge pressure and were followed as a guide to left ventricular filling, due to the concern that repeated balloon inflation might obstruct right heart output. Due to low filling pressures and preoperative anemia (hct 24%), the infant was transfused to a hematocrit of 35% the evening prior to surgery. The normocytic hypochromic anemia was attributed to chronic disease and hemodilution with no evidence of hemolysis. Maintenance fluids were given iv to prevent a preoperative fluid deficit.

Intraoperative monitoring consisted of a V_5 ECG lead, precordial stethoscope, precordial doppler, automatic oscillometric BP cuff, continuous ECG monitor (PSA-I), mass spectrometer for measurement of end-tidal CO_2 and inspired oxygen concentrations, and pulse oximeter. A V_5 lead was used for detection of myocardial ischemia. Anesthesia was induced with iv fentanyl, 10 μg/kg, and neuromuscular blockade was provided by pancuronium bromide, 0.1 mg/kg iv. A nasal route was chosen for tracheal intubation to provide for postoperative stability of the tracheal tube.

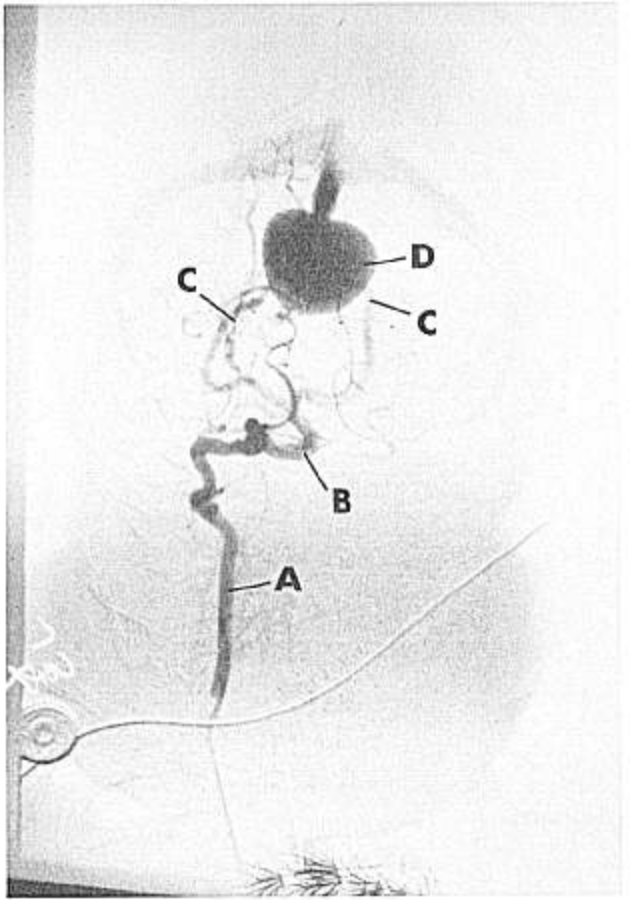

FIG. 1. Angiogram with dye injection in left vertebral artery showing giant Vein of Galen malformation. A = Left vertebral artery; B = posterior cerebral artery; C = enlarged aberrant vessel feeding the arterovenous malformation; D = Vein of Galen Aneurysm.

Pressure pre-set controlled ventilation was provided with a Bourns BP200 ventilator, and the patient placed in the sitting position. The sitting position was chosen by the neurosurgeon for better exposure of the major feeding vessels that were located posteriorly. At this time, CVP rose to 16–18 mmHg and PA pressures increased to 50/12 mmHg without a change in cardiac index (fig. 2). This abrupt increase in cardiac filling pressures was attributed to an auto-transfusion of blood from the venous portion of the aneurysm due to the gradient produced by the sitting position.

Anesthesia was maintained with fentanyl 110 μg/kg iv during the 7-h procedure. Prior to aneurysm clipping, an adequate circulating blood volume was maintained by replacement of blood and fluid losses based on filling pressures, cardiac index, measured blood loss, and calculated maintenance needs. With surgical exposure of the aneurysm, furosemide 1 mg/kg was given iv to reduce the intravascular volume prior to clipping in an attempt to prevent acute cardiac failure from an abrupt increase in afterload. Placement of the first aneurysm clip reduced cardiac index by 30% with minimal change in right heart pressures. However, heart rate acutely decreased by 15%. Clips applied to other feeding vessels produced little subsequent change. The estimated total blood loss was 150 ml. The power spectrum analysis of EEG initially showed significant differences in power output of the right *versus* left frontal regions. This discrepancy equalized after aneurysm clipping, suggesting that the preoperative intracranial steal syndrome confirmed by angiographic (decreased flow to anterior cerebral arteries, particularly on the right) and clinical examination (frontal lobe focus for seizures, hypertonicity) had improved with treatment of the aneurysm. The infant's postoperative course was complicated by an increase in seizure activity on postoperative day three, which resolved with adjustment of anticonvulsant therapy. When follow-up examination was performed 8 months postoperatively, the infant had met his developmental milestones, and objective tests of hearing and vision were normal. The infant has been seizure free since the third postoperative day while receiving maintenance doses of phenobarbital.

DISCUSSION

Morbidity and mortality remain high in infants undergoing surgical treatment for Vein of Galen aneurysms, despite various recommendations for optimal perioperative management. Cardiopulmonary bypass with profound hypothermia has been suggested by one group of authors as a means of reducing intraoperative blood loss and improving neurological outcome.[7] However, in neonates and young infants, this technique has resulted in massive blood loss and intraoperative death, as well as a profound left hemiparesis in the infant originally reported.[4,7] It does not appear that this method of intraoperative management offers any advantages over the patient's own pulsatile circulation.

Another approach has been the use of inhaled anes-

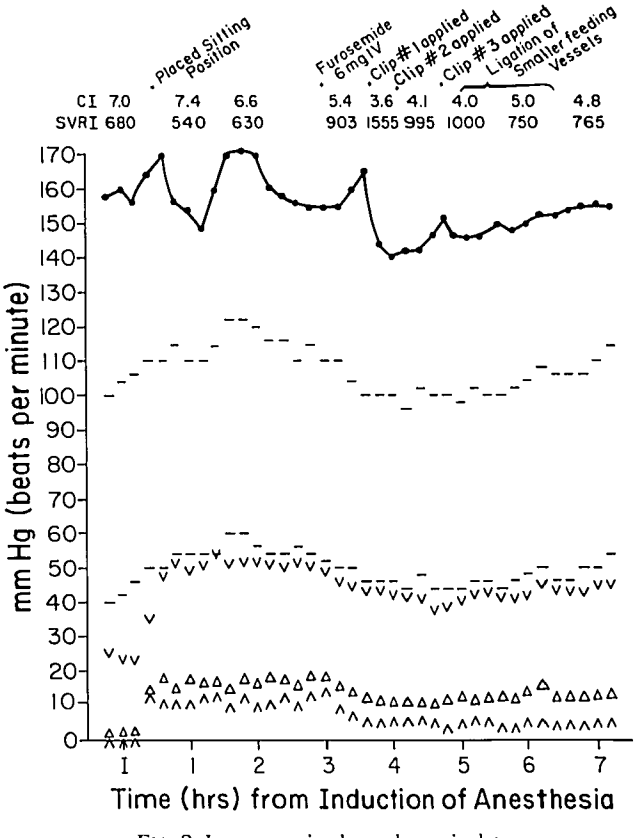

FIG. 2. Intraoperative hemodynamic data.

thetics or other hypotensive agents to minimize blood loss and to prevent sudden increases in afterload associated with aneurysm clipping.[5-7] However, this technique has also resulted in subendocardial ischemia, acute myocardial infarction, severe residual neurological deficit, and death.[2-4,8] All volatile anesthetics produce some degree of myocardial depression and reduction in systemic vascular resistance, which may not be well tolerated by the already stressed cardiovascular system. The use of hypotensive anesthesia may also produce adverse effects on the central nervous system. When mean arterial pressure is reduced to prevent excessive blood loss, cerebral perfusion pressure may be drastically reduced or vascular steal may be accentuated. However, inhaled anesthetics may be useful in selected cases to reduce afterload following aneurysm clipping in patients with adequate myocardial reserve.

Evidence of chronic subendocardial ischemia and acute myocardial infarction has been noted in both newborn and older infants dying intraoperatively from either massive blood loss or acute intraoperative cardiac failure.[4] For this reason, "normal" intravascular volume as evidenced by cardiac filling pressures and index was maintained until the time of vessel clipping. Vasodilator infusions of nitroglycerin and nitroprusside, as well as cardiac inotropic infusions, were prepared should cardiovascular decompensation occur with vessel occlusion.

Another consideration in these infants is their elevated myocardial oxygen consumption from a hyperdynamic circulation; therefore, the evening before surgery, the child was slowly transfused from a hematocrit of 24–35% to increase oxygen-carrying capacity. We avoided the use of nitrous oxide in our patient for three reasons: 1) there is an increased risk for air embolus in the sitting position and nitrous oxide has been shown to exacerbate symptoms produced by air in the circulation;[11] 2) nitrous oxide may cause an elevation in pulmonary artery pressures,[12] which, in our infant, were already increased to 49/11 mmHg; and 3) myocardial depression may result from adding nitrous oxide to a high-dose iv narcotic anesthesia.[12]

In summary, operative treatment of infants with Vein of Galen aneurysm has a high associated morbidity and mortality. Because myocardial complications are the leading cause of perioperative death, preoperative evaluation of the cardiovascular system is imperative. A major goal of intraoperative management is meticulous replacement of blood and insensible fluid losses and maintenance of optimal filling pressures to prevent acute myocardial decompensation secondary to hypoperfusion from low diastolic arterial pressure. Due to the poorly compliant myocardial muscle and the relatively small intravascular volume of these patients, frequent communication by the neurosurgeon with the anesthesiologist of ongoing blood loss is crucial. In our patient, preoperative placement of the PA catheter allowed baseline hemodynamic values to be obtained, which were then used as a guide for intraoperative fluid replacement. Preparations must also be made for the increase in afterload that can occur with aneurysmal clipping, which can also result in cardiac ischemia or failure. Continuous monitoring of arterial blood pressure can be used as a guide for maintenance of adequate myocardial and cerebral perfusion pressures. Devices that allow for continuous EEG monitoring, such as the PSA-1® monitor in our patient, may also alert the anesthesiologist to unfavorable changes in hemispheric cerebral blood flow. Earlier detection of these discrepancies might allow for intraoperative changes in technique or procedures that could lower postoperative neurological morbidity.

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