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## Reduction Cranioplasty and Severe Hypotension

DAVID PARSONS, M.D., F.R.C.P.(C.),\* STANLEY I. SAMUELS, M.B., F.F.A.R.C.S.,†  
GARY STEINBERG, M.D., PH.D.,‡ LAWRENCE SHUER, M.D., F.A.C.S.§

Reduction cranioplasty is an extremely rare operation performed in children when hydrocephalus has been neglected or ineffectually treated. We report the anesthesia problems encountered with removal of the cranium and the resultant loss of a massive amount of cerebrospinal fluid (CSF).

### CASE REPORT

A 5-yr-old, 18-kg woman with congenital hydrocephalus presented with a head circumference of 90 cm (normal 51 cm) and the inability to hold her head securely enough to ambulate. She had developed slowly but was able to communicate in two languages. She had undergone several prior ventricular peritoneal shunt revisions because of shunt malfunction, infection, and chronic subdural hematomas. At the time of surgery, she had a functioning shunt in place, was alert, and was without focal neurological signs. The surgical plan was to identify the sagittal sinus by digital subtraction angiography in order to avoid massive hemorrhage intraoperatively, and then to undertake a reduction cranioplasty.

Without premedication, anesthesia was induced in the radiology suite at 0730 h with nitrous oxide, oxygen, and halothane without incident. Once an intravenous line was established, endotracheal intubation was facilitated by the administration of pancuronium bromide

1.5 mg iv. Monitoring included ventilation-oxygen monitors, as well as electrocardiogram, precordial stethoscope, nasal temperature, automated blood pressure device, and pulse oximetry.

During the 3½-h radiological procedure, anesthesia was maintained with nitrous oxide, oxygen, and halothane, and pancuronium was used for muscle relaxation. Of note was a decrease in heart rate commensurate with the decrease in temperature from an initial 35.6° to 34.5° C. Following the radiological procedure, the child was transferred to the operating room, where a number of changes in anesthetic management became necessary. The patient's position was changed to semi-sitting and, thus, the nitrous oxide was discontinued and end-tidal carbon dioxide and nitrogen were monitored by mass spectrometry. A precordial Doppler was placed and tested with an aspirated saline bubble embolus. A left external jugular line was placed and passed centrally. A left radial arterial line was also inserted. A peripheral nerve stimulator and urinary catheter were also utilized. The anesthetic technique was as changed to a narcotic-based (morphine) one, with the anticipation of postoperative controlled ventilation.

The reduction in temperature was treated with warming lights, a warming blanket, and a heated humidifier placed in the circle system. Fluid management consisted of  $3.0 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}$  maintenance and  $4.0 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}$  for third space losses. Blood loss was replaced to account for measured and estimated losses.

Five and one-half hours later, as the cranial cap was being removed, there was a sudden and marked loss of CSF estimated at 1000-1500 ml. At the same time, the arterial blood pressure dropped progressively over a 1-2-min period from a mean of 70 mmHg to 35 mmHg. There was no change in ventilatory status, and no recent drug administration had occurred. The blood loss at that time was estimated to be 300 ml, which had been fully replaced.

Intravenous fluids were administered as blood and crystalloid. Vasopressors, including ephedrine and epinephrine, as well as calcium chloride, were used with little effect except for transient increases in arterial blood pressure. Additional lines for intravenous fluids were established and external cardiac compressions were begun when mean arterial pressures declined below 35 mmHg.

Continued low blood pressures mandated a change in position to Trendelenburg, but this action markedly accelerated the blood loss from sagittal sinus bleeding, making resuscitation more difficult. Blood loss following the change in position was estimated at 2000 ml during the resuscitation period. Resuscitation continued for 55 min

\* Resident in Anesthesia, Department of Anesthesia.

† Associate Professor of Anesthesia (Clinical), Department of Anesthesia.

‡ Assistant Professor of Neurosurgery, Division of Neurosurgery.

§ Assistant Professor of Neurosurgery (Clinical), Division of Neurosurgery.

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Address reprint requests to Dr. Parsons: The Vancouver General Hospital, Department of Anaesthesia, 855 West 12th, Vancouver, British Columbia, Canada V5Z 1M9.

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before a stable enough situation could be obtained to allow the surgery to continue.

The total of fluids administered were 3000 ml of crystalloid, 8 units of packed red blood cells, 1 unit of fresh frozen plasma, and 3 units of platelets. The persistent shock led to a moderate metabolic acidosis which was treated with intravenous bicarbonate. More aggressive warming was undertaken with an increase in body temperature from 34.5° C to 36.5° C. Following the successful resuscitation and the conclusion of surgery, the child was returned to the pediatric intensive care unit unconscious and with her trachea intubated. The vital signs were stable. Ventilation was continued overnight and tracheal extubation was accomplished the next morning without difficulty. The postoperative hematocrit was 42% and the weight was 20 kg.

For 2 weeks postoperatively, the patient was obtunded with abnormalities of eye movement and a left hemiparesis. Over the next 2 months, she gradually improved to her preoperative status, with resolution of her weakness and extraocular dysfunction. She was again able to communicate bilingually.

### DISCUSSION

Reduction cranioplasty is a rarely performed procedure, and only five cases are reported on the literature.<sup>1-5</sup> This particular surgical technique of removal of the cranial cap has not been reported previously. The frequent occurrence of neurological deficit following reduction cranioplasty in the literature attests to its high risk potential. As this child had suffered from recurrent sub-dural hemorrhages and was unable to ambulate due to her head size, this procedure was planned to allow her to function more normally with only mild mental impairment.

In this case, the issues raised are those of the cause of the acute hypotensive event and the refractory nature of its course. At the onset of the hypotension, the expected list of problems included an intravascular volume deficit, air embolus, and pneumothorax. The bilateral breath sounds and the normal values of the peripheral saturation and the end-tidal carbon dioxide/nitrogen were evidence against the latter two diagnoses, as were the unchanged Doppler tones. The ventricular-peritoneal shunt was clamped at the time of the hypotension. Consultation with the operative team revealed that the blood loss had been steady but slow up to this point, but that there had been the loss of 1000–1500 ml of CSF within the preceding 2 min.

At this point, the problem list was narrowed to hypovolemia and the more remote possibilities of either cardiac tamponade secondary to central line placement or sympatholysis secondary to midbrain manipulation. Despite this possibility of a "reflex" fall in arterial blood pressure, the clinical decision was to treat the far more common problem of hypovolemia. Vigorous resuscita-

tion with iv fluids over 55 min eventually resulted in a stable clinical state.

An alternate hypothesis to hypovolemia is an alteration in the sympathetic nervous system. As arterial blood pressure is controlled from the tonically active medullary vasomotor center (VMC),<sup>6</sup> ischemia from mild hypotension interacting with the mechanical stress of torsion from the surgery would have the result of reducing sympathetic tone and causing arterial venous vasodilation.

Further speculation as to the reason for recovery from this putative sympatholysis would center on the Trendelenburg position allowing adequate blood flow to return to blood vessels no longer contorted by abnormal brain positioning, and the adequate transfusion of volume to both fill up the larger intravascular space and perfuse the medulla.

This case report details the intraoperative events during a reduction cranioplasty. Recommendations for anesthetic management of a similar case in the future would include those that have been published previously for anesthesia for patients undergoing major facial osteotomies.<sup>7</sup> In addition, the potential for even greater perioperative anesthetic problems must be entertained and, thus, thought should be given to placement of a pulmonary arterial catheter to allow calculation of cardiac output values and, hence, systemic vascular resistances. Differentiation of hypovolemia from a loss of sympathetic tone would be greatly facilitated by this monitoring modality. Aggressive therapy with alpha-adrenergic agents would be the maneuver of choice if a low systemic vascular resistance was present.

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