

## Respiratory Pattern Changes during Repair of Posterior-fossa Arteriovenous Malformation

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Arteriovenous malformations in the posterior fossas of infants are rare anomalies that carry a high operative mortality rate. Death may follow profound hemodynamic changes that occur with the arteriovenous fistula, or death may be caused by mechanical encroachment on the vital brainstem structure. However, the circumstances surrounding many deaths are not clearly documented.

We report a case in which surgical correction of a posterior-fossa arteriovenous malformation was complicated by an unusual

respiratory pattern that culminated in cardiac arrest.

### REPORT OF A CASE

A 9-month-old, 9.5-kg, white male infant had first been seen at this institution at 2 days of age for evaluation of cyanosis. Roentgenograms of the chest at that time had demonstrated marked cardiomegaly and findings consistent with either increased pulmonary blood flow or aspiration pneumonia. The patient had been treated for the latter. At 1 month of age a systolic ejection murmur had been heard along the left sternal border, the liver had been enlarged, and an electrocardiogram was interpreted as biatrial enlargement and biventricular hypertrophy. The patient had responded well to treatment with digitalis.

At 6 months of age, increasing head size and cranial bruit alerted his physicians to the need for carotid and vertebral angiograms. A large arteriovenous malformation in the posterior fossa, supplied by three large branches of the posterior cerebellar artery, the superior cerebellar artery, and the anterior and posterior inferior cerebellar arteries was

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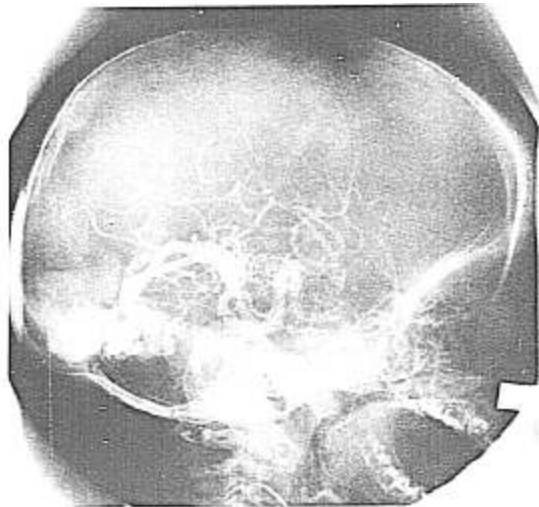


FIG. 1. Right carotid angiogram, showing large posterior-fossa arteriovenous malformation.

demonstrated (fig. 1). A cardiac catheterization demonstrated a left atrial pressure of 10 torr, a markedly increased left ventricular end-diastolic pressure of 22 torr, and a cardiac index that was two and a half times the predicted value. It was decided to treat the malformation surgically.

Preoperatively, the infant was alert but irritable. The pulse rate was 140/min, respiratory rate 40/min, and temperature 37.5 C. There was a grade II/VI systolic murmur over most of the precordium, and the lungs were clear to auscultation. A roentgenogram of the chest showed cardiomegaly with normal lung fields. An electrocardiogram showed incomplete bundle-branch block and biventricular hypertrophy. Hemoglobin was 11.5 g/100 ml, leukocyte count 10,100, and serum potassium was 5.0 mEq/l. A right atrial catheter was inserted the night before operation using fluoroscopic observation. The infant was premedicated with scopolamine, 0.1 mg, im.

Tracheal intubation was facilitated with succinylcholine. Anesthesia was induced with halothane-oxygen, using a high-flow nonrebreathing system, and maintained with 0.5 to 1.25 per cent halothane in 50 per cent nitrous oxide and oxygen. During the operation respiration was assisted. Circulation was monitored with an esophageal stethoscope, a Doppler blood-pressure apparatus, and continuous oscillographic display of the ECG. Respiratory excursion amplitude was monitored with a pneumograph<sup>§</sup> attached to the chest wall, recording continuously on a Grass polygraph. Exhaled CO<sub>2</sub> was monitored with an infrared analyzer. Temperature was monitored with a rectal thermistor probe. The infant was placed in the left lateral decubitus position with approximately 20-degree head-up tilt.

The operation proceeded uneventfully for about five hours, blood pressure, pulse rate, and respiratory pattern remaining unchanged. The procedure was interrupted for several minutes each time a vessel was clipped in order to observe vital signs and auscultate the lung fields. No change was observed. Expired CO<sub>2</sub> concentration remained between 5.2 and 5.8 per cent. About 10 minutes after the largest and last draining vein was clipped, the respiratory rate increased from 40/min to nearly 80/min without change in pulse rate or blood pressure. There was no air on aspirating from the atrial catheter. The lungs were clear to auscultation. Within 5 minutes the respiratory rate returned to 40/min. Suddenly the patient took one gasp and then assumed a bigeminal respiratory pattern, with alternating deep and shallow breaths. Respiratory excursions decreased progressively and all but ceased over a period of 15 minutes (fig. 2). Respiration was subsequently controlled. Blood pressure and pulse rate remained unchanged throughout this period and the lungs remained clear to auscultation.

Approximately 10 minutes following cessation of spontaneous respiration, sudden asystole occurred. No surgical maneuver had been performed since

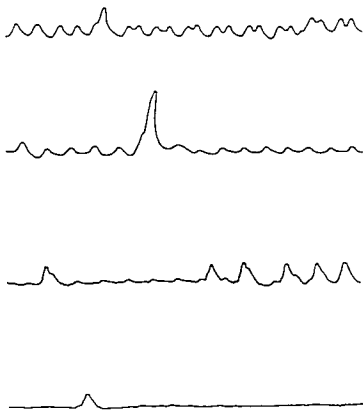


FIG. 2. Respiratory pattern change. What appears to be intermittent and incomplete apneusis was followed by gradual decline in respiratory excursion over a period of 15 minutes. Tall spikes in second through fourth tracings are due to artificial ventilation.

the clipping of the last draining vein. The patient was successfully resuscitated with closed-chest cardiac massage, 100 per cent oxygen, epinephrine and sodium bicarbonate. About 30 minutes after circulatory stabilization, while the dura was being closed, a sudden, profound bradycardia developed, progressing to asystole. The infant was again resuscitated successfully, and circulatory signs stabilized. He had two more such episodes during closure of the operative site. With each episode blood pressure became more difficult to maintain, necessitating isoproterenol infusion. There was no spontaneous respiratory effort. Five hours after operation, he had another episode of asystole and could not be resuscitated.

#### AUTOPSY FINDINGS

The heart was large, with slightly dilated chambers. The lungs were of normal weight, without evidence of edema. There were some areas of congestion and mucosal hemorrhage in the stomach, terminal ileum and colon.

Gross examination of the cerebral hemispheres showed slight, diffuse softening. Scattered in the distribution of both posterior cerebral arteries were areas of gray cortical discoloration, possibly indicating early infarction. Sections of the brainstem demonstrated

§ Harvard Apparatus, Inc., Millis, Mass.



FIG. 3. A section of the brainstem. Congested blood vessels and occasional slight perivascular collection of fresh blood.

congested penetrating blood vessels and occasional slight perivascular collections of fresh blood (fig. 3). There were a few fresh punctate hemorrhages in the folia of the cerebellum adjacent to the operative site. There was slight vascular congestion in the cerebellum.

#### DISCUSSION

Intracranial arteriovenous malformations are usually located supratentorially, with only 7 per cent found in the cerebellum and brainstem.<sup>1</sup> The manifestations are quite different in neonates than in older infants and children. In older children the symptoms are mainly neurologic, whereas neonates have cyanosis and congestive heart failure. It is established that an arteriovenous fistula increases cardiac output, resulting in ventricular hypertrophy, dilation and decompensation.

Large malformations in infants are associated with a high mortality rate. Gomez<sup>2</sup> reported four deaths in five neonates and Hope<sup>3</sup> reported five deaths in seven neonates not treated surgically. All died in intractable congestive heart failure. Levine *et al.*,<sup>4</sup> reporting the cases of three infants undergoing surgical correction, described one death from sudden congestive failure, which occurred four hours after operation. In a series of eight infants reported by Long *et al.*,<sup>5</sup> one died of congestive failure and another from sudden cardiac arrest, both occurring 12 hours postoperatively. A third infant had intraoperative cardiac arrest, with successful resuscitation.

Respiratory changes that occur during posterior-fossa craniotomy are thought to be the result of mechanical encroachment on the vital brainstem structure. In our patient, how-

ever, no surgical maneuver had been performed during the 40-minute period of abnormal respiratory pattern that ended in sudden cardiac standstill. There was no clinical or autopsy evidence of congestive failure. The congestion in the brainstem demonstrated at autopsy indicates that some of the surgically clipped venous channels might have served to drain vital centers to a significant extent. The resultant stagnant hypoxia would explain the rather gradual onset and slow progress of the respiratory changes and cardiac standstill. We consider that the insufficiency of the posterior cerebral artery found at autopsy did not contribute to this event, as this artery does not supply the brainstem structure.

Whether to control or to assist ventilation during posterior-fossa craniotomy is a matter of controversy. Recent literature<sup>6,7</sup> indicates that monitoring of respiration and monitoring of cardiac rhythm and rate appear to be equally sensitive in detecting the effects of torsion or traction on brainstem structures, and therefore respiration may be controlled safely provided the electrocardiogram is monitored continuously. Differential effects of brainstem ischemia on the respiratory and cardiovascular centers observed in our patient suggest, however, that monitoring respiration is more sensitive than that of cardiac activity in detecting slowly developing brainstem ischemia produced by venous stagnation during repair of posterior-fossa arteriovenous malformation.

There is some evidence that arrhythmias in patients with ischemic brainstem lesions are consistent with an increase in vagal tone. Atropine or isoproterenol could be used to prevent cardiac standstill that may follow changes in respiratory pattern in a hope that, given time, a new hemodynamic equilibrium may be obtained in the affected areas.

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