

Cardiac Arrest Associated with Tension Pneumocephalus

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Tension pneumocephalus is an intracranial collection of gas under pressure which is a complication of posterior fossa surgery performed in the sitting position.¹⁻³ Characteristically, this syndrome is suspected because of delayed return to consciousness and neurologic deterioration in the postoperative period. Diagnosis can be confirmed by computerized tomographic (CT) scan. Hemodynamic instability progressing to cardiac arrest due to entrapped gas has not been reported. We describe a case involving a 5-year-old child who sustained a sudden cardiac arrest apparently caused by tension pneumocephalus.

REPORT OF A CASE

A 5-year-old 20-kg boy presented with headaches and diplopia of recent onset. On examination, palsy of the sixth cranial nerve on the right side and papilledema was noted. CT scan demonstrated a left cerebellar cystic mass with a presumptive diagnosis of astrocytoma. A posterior fossa exploration was scheduled with the child in the sitting position.

Preanesthetic evaluation revealed an otherwise healthy child with no cardiac or respiratory abnormalities. All laboratory data were within normal limits. He was treated for four days before surgery with 2 mg dexamethasone q.i.d. to decrease peritumor edema. Immediately prior to induction of anesthesia, 2 g mannitol was administered intravenously over a 45-min period. A central venous cannula had been inserted through the right subclavian vein and its position within the midatrium ascertained by a radiograph the previous evening.

Anesthesia was induced with 100 mg thiopental intravenously. The trachea was intubated after administration of 40 mg succinylcholine intravenously. Anesthesia was maintained with 50 per cent nitrous oxide and after establishment of hyperventilation, 0.5-1 per cent halothane was introduced. Throughout surgery, core temperature, central venous pressure, arterial pressure, blood gases, and precordial Doppler sounds were monitored continuously. Ventilation was controlled to maintain P_{aCO_2} levels of 30-33 mmHg.

A posterior fossa craniotomy was performed with the child in the sitting position. A cystic astrocytoma situated in the left cerebellar hemisphere was isolated and excised. The tumor did not involve the

brain stem or vital centers. A ventricular drain was placed for intracranial pressure monitoring postoperatively. After 5 h of surgery, tumor excision was complete and hemostasis secured. Estimated blood loss was 150 ml and this had been replaced with packed erythrocytes, 100 ml. Intraoperative fluid infusion was replaced at the rate of 4 ml/kg/hour to a total of 400 ml of lactated Ringer's solution. No preoperative fluid deficit was assumed, as fluids had been infused through the central cannula during the night. Urinary output was 800 ml which included the diuresis following mannitol administration. All other vital signs were stable with arterial blood pressure ranging between 120-110/70-65 mmHg and heart rate of 85-95 beats per minute. Nitrous oxide was discontinued approximately 5 min before the dural flaps were approximated and closed. As the dural edges were sutured, severe hypotension and bradycardia suddenly occurred. Cardiac arrest as evidenced by a straight line on the oscilloscope and absence of detectable heart sounds through the esophageal stethoscope followed within 2 min. Although no changes had been heard on the Doppler monitor, an unsuccessful attempt was made to aspirate air through the central venous catheter. The child was immediately returned to the supine position, and resuscitation, including external cardiac massage, ventilation with 100 per cent oxygen and appropriate pharmacotherapy including epinephrine, sodium bicarbonate atropine, and calcium gluconate administered. Within 3 min, sinus tachycardia was re-established with systolic blood pressure of 120 mmHg. The wound was promptly closed and the child taken for CT scan which showed bifrontal subdural and ventricular gas (fig. 1). Downward displacement of the brain also was demonstrated. During this time the cardiovascular status remained stable, although apnea and unresponsiveness with equal, fixed and non-reactive pupils persisted. The child was returned to the operating room where frontal burr holes were performed and approximately 100 ml of air immediately aspirated. Saline was flushed through two ventricular drains in a further attempt to displace the gas within the ventricles. Almost immediately spontaneous respiration returned and the pupils reacted to light. Repeat radiograph showed considerable improvement.

Over the next two 2 h, the child regained consciousness and the trachea was extubated. Twenty-four hours later, transient diabetes insipidus developed which responded promptly to pitressin. The remainder of the postoperative course was uneventful and the child was discharged neurologically intact.

CT scans were repeated daily. A considerable amount of air could still be detected within the cranium after 7 days but without neurologic changes or deficit (fig. 2).

DISCUSSION

Surgical procedures performed in the posterior fossa or neck with the patient in the sitting position under general anesthesia are associated with many complications.⁴⁻⁹ The early diagnosis and management of air embolism have been reviewed extensively and with careful monitoring, major morbidity associated with infused air should be preventable.^{4,5} Tension pneumocephalus was first reported by Kitahata and Katz.¹ Gravitational effect

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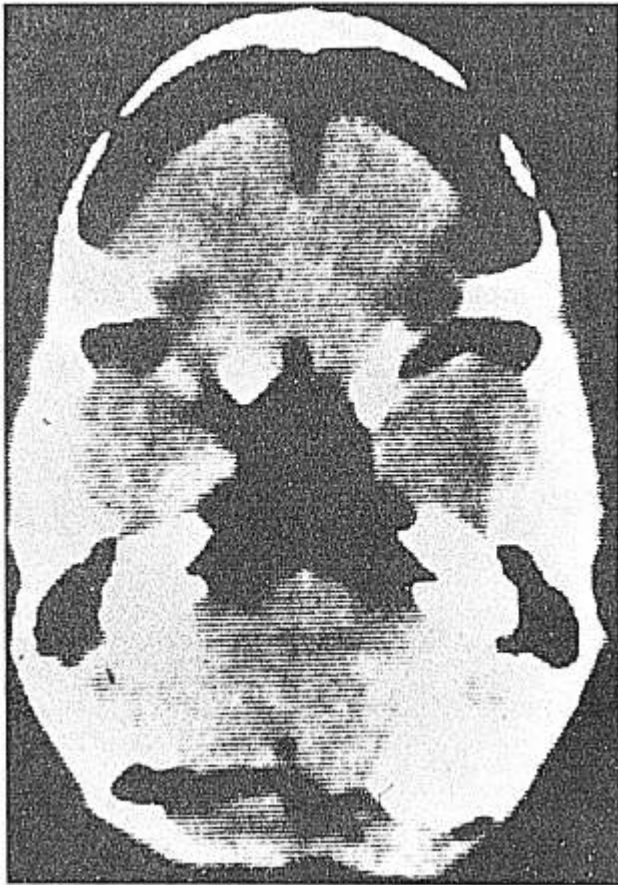


FIG. 1. Large amounts of gas are seen in the frontal areas and within the ventricular system.

in the sitting position and decrease in brain size caused by hyperventilation, diuretics, and possibly steroids causes the cerebral hemispheres to settle into the lower cranial vault. Intraoperative drainage of cerebrospinal fluid, either through a ventriculostomy, ventricular incision, or by subarachnoid drainage result in further ventricular collapse. As the cerebral cortices recede, a pocket is formed between the arachnoid and dura mater and air becomes trapped in the frontal areas.

In this case, sudden cessation of cardiac action resulted from the obliteration of an escape site for gas combined with forced downward movement of the brain caused by approximating the dural edges. Restoration of heart action was achieved quickly when the supine position was assumed and gas and cerebrospinal fluid were redistributed from the ventricular to the spinal subarachnoid system which also probably caused some upward increased pressure on the brain stem.

Although diffusion of nitrous oxide into intracranial air pockets has been cited as a cause of further increase in size because of the high solubility of the gas compared

to nitrogen, baseline levels for intracranial pressure are re-established within 10 min of discontinuing its use.¹⁰ Thus, although nitrous oxide was discontinued rather late, it is unlikely that use of this agent caused the cardiac arrest. Moreover, Friedman *et al.*¹¹ have described a situation in which, despite discontinuation of nitrous oxide 1.5 h prior to the conclusion of a case, considerable postoperative difficulty with intracranial gas was encountered. We have seen similar situations in which re-expansion of the brain during return to the normocarbic state, rehydration, or cerebral edema increased intracranial pressure and a comatose state with other neurologic abnormalities (mainly decreased pupillary responses and hypopnea or frank apnea) persisted for many hours postoperatively.

Aids to prevent tension pneumocephalus include flushing the subdural space with saline to displace as much gas as possible. Ventriculostomy drains should be left open during dural closure. We have placed small rubber tubes as drains at the upper and lower limits of the dural incision until dural flaps are approximated. Fluid is then

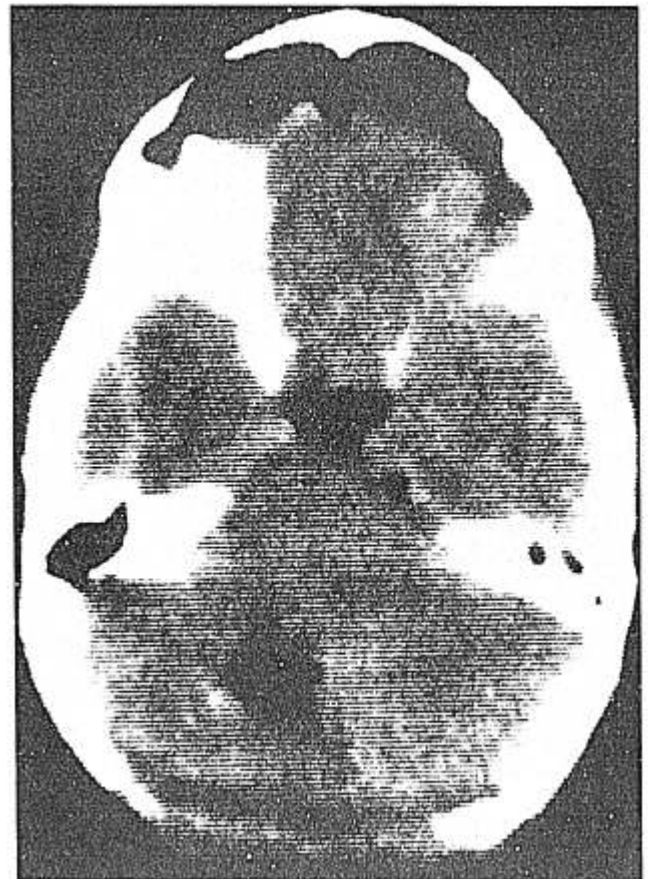


FIG. 2. CT scan taken 7 days after procedure shows air persisting in the frontal region. A small amount of ventricular gas remains.

irrigated through the tubes before they are withdrawn in a further attempt to displace gas. Nitrous oxide should probably be discontinued 15 min before dural closure although it has been suggested that if anesthesia is maintained with nitrous oxide, an intraoperative pneumocephalus would be more quickly reabsorbed.¹¹ If at all possible, hyperventilation should be decreased to allow brain expansion as the dura is closed. However, ventilation must be controlled again when bone and muscle are manipulated to prevent the gasp response of air embolism.¹²

As the effects of pneumocephalus may be obscured by prolonged anesthesia, skull radiographs or CT scans should be performed early in the postoperative period. Frequently, following major surgical procedures performed within the posterior fossa, neurosurgical technique advises placement of a ventriculostomy cannula for drainage. This cannula also may serve as a convenient means to measure intracranial pressure. We advise use of this monitoring technique whenever it is available in the early recovery phase after prolonged surgery in the sitting position. Finally, as air is absorbed only very slowly from the intracranial compartments (fig. 2), nitrous oxide should probably be avoided if surgical re-exploration becomes necessary during the following 1 to 2 weeks.

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