pulmonary vasculature. This study demonstrates the accuracy of this technique.

Angiography by means of the Swan-Ganz catheter is safe when performed as described in the literature.5–9 Five to 10 ml of intravenous contrast medium should be injected by hand through the distal catheter port after the balloon is inflated and the correct pressure waveform verified. A chest roentgenogram then is taken after the injection and the balloon is deflated. The patient will commonly cough during the injection. There have been no reports of complications related to this technique. Complications may result from the Swan-Ganz catheter insertion and long-term maintenance. Allergic reactions to the radiographic contrast agent are also possible.

In our case, the Swan-Ganz catheter was inserted after thrombi had embozilized from the veins of the leg to the lung. Since the catheter is flow-directed, it will enter only a patent pulmonary artery. Thus, a false-negative diagnosis may be made if the catheter lodges in a segmental pulmonary vessel that does not contain an embolus. The tip of the Swan-Ganz catheter should be placed in as proximal a pulmonary artery as is possible to have the greatest chance of detecting an embolus. In this patient, standard pulmonary angiography demonstrated that the extent of the pulmonary emboli was much greater than visualized by bedside angiography. Pulmonary angiography using the Swan-Ganz catheter in this case substantiated the correct diagnosis so that appropriate treatment could be initiated and the patient stabilized prior to standard pulmonary angiography.

In summary, this case demonstrates the use of a Swan-Ganz catheter for diagnosing pulmonary thromboemboli which occurred during spinal anesthesia. The catheter was utilized for pressure measurements as well as pulmonary angiography. Bedside angiography using the Swan-Ganz catheter appears to be a safe, effective technique to diagnose pulmonary emboli.

REFERENCES

Anesthesiology 57:59–61, 1982

Nitrous Oxide Plays a Direct Role in the Development of Tension Pneumocephalus Intraoperatively

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While previous reports have implicated nitrous oxide in the genesis of tension pneumocephalus postcraniotomy,1–4 none provided direct evidence that nitrous oxide was involved. The following case provides direct evidence for a role of nitrous oxide in the genesis of tension pneumocephalus, and suggests that measuring CSF or intracranial pressure intraoperatively may contribute to early detection of this potentially hazardous problem.

REPORT OF A CASE

A 63-year-old woman was admitted for evaluation of hearing loss and dizzy spells. She had mild hypertension (140/85–170/95 mmHg) which was treated with amlodipine, one tablet orally each day. After a diagnosis of left acoustic neuroma was established, a craniotomy for tumor removal was scheduled to be performed in the seated position. Preoperative CT scan showed no intracranial air.

Anesthesia was induced with thiopental and maintained with fentanyl and enflurane 0.25–0.5 per cent and nitrous oxide 66 per cent in oxygen. Intraoperative monitors included a right atrial catheter, and

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Continuous measurements of ventricular cerebrospinal fluid (CSF) pressure via ventriculostomy, and precordial heart sounds via precordial Doppler probe. Additionally, expired concentrations of CO₂, nitrous oxide, and enfurane were continuously measured via mass spectrometer. CSF pressure and expired gas concentrations were recorded along with blood pressure (via radial artery catheter) and EKG on a strip chart recorder. The patient was placed in the sitting position and ventilation was controlled to maintain PaCO₂ at 26-28 mmHg. After the skull was opened, 25 g mannitol was administered intravenously, and 15 ml CSF was drained via the ventriculostomy.

Excision of the tumor was uneventful and at 5.5 hours after induction of anesthesia closure of the dura was completed. Mean CSF pressure at the time of closure was stable at 8 mmHg (zero reference at heart level). However, immediately following dural closure mean CSF pressure was observed to increase gradually over 5-10 min to 20 mmHg. The surgeons were informed and the patient was examined for causes of the CSF pressure increase. No extracranial cause was identified and we elected to remove additional CSF via ventriculostomy to decrease mean CSF pressure to 15 mmHg.

Several minutes later as the surgical incision was being closed, sounds suggestive of air embolism were heard on the precordial Doppler. Infused nitrous oxide was discontinued and 10 ml air aspirated from the right atrial catheter. Coincidentally, ventricular mean CSF pressure decreased from 15 mmHg to 2 mmHg (fig 1). No site for entry of air into the vascular system could be identified and surgical closure was continued. About 10 minutes after Doppler sounds had returned to normal, nitrous oxide was reintroduced into the inspired gases. Immediately a steady increase of ventricular CSF pressure was observed. We reasoned that nitrous oxide was increasing the volume of the pneumocephalus and when mean CSF pressure reached 15 mmHg nitrous oxide was discontinued. Immediately CSF pressure decreased to 6 mmHg. To confirm that a pneumocephalus was present and that nitrous oxide was responsible for the CSF pressure increase, nitrous oxide was again introduced into the inspired gases. Immediately a steady increase of CSF pressure to 19 mmHg was observed. Nitrous oxide was discontinued, CSF pressure decreased to 7 mmHg, and the surgeons were informed of our findings and presumptive diagnosis.

During completion of surgery, emergence from anesthesia, return to spontaneous ventilation, and transport to the neurosurgical intensive care unit (ICU) CSF pressure remained at 4-7 mmHg. A brow up lateral skull roentgenogram taken in the neurosurgical ICU revealed the expected large collection of intracranial air (fig. 2).

For the next 48 hours the patient was carefully observed and CSF pressure measured continuously. No lateralizing signs or decrease in level of consciousness were observed. CSF pressure reached a peak at 6 hours postcraniotomy of 9 mmHg and then decreased to 4 mmHg by the morning following surgery.

**DISCUSSION**

In previously reported cases where nitrous oxide was implicated in the genesis of tension pneumocephalus, it was proposed that the pneumocephalus began intraoperatively as room air was drawn into the subdural space. Air presumably entered through the surgical wound as the brain contracted during surgery. When the dura was closed, nitrous oxide that was dissolved in blood entered into the enclosed space more rapidly than nitrogen exited, increasing the volume of the pneumocephalus. However, in these cases tension pneumocephalus was only diagnosed postoperatively and CSF pressure was not measured at the time of closure of the dura.¹⁻⁴ Thus, in these reported cases there was no direct evidence that nitrous oxide contributed in any way to the subsequently diagnosed tension pneumocephalus. In each case the authors acknowledged that though a pneumocephalus was present, increased intracranial pressure may have resulted from other factors such as reaccumulation of CSF, reexpansion of the brain by rehydration, return to normal cerebral blood volume, and cerebral edema. My case
clearly illustrates that following closure of the dura, CSF pressure was directly and reversibly related to introduction and discontinuation of nitrous oxide from the inspired anesthetic gases. These observations strongly support the idea that nitrous oxide may indeed contribute to the genesis of tension pneumocephalus via the scenario outlined above.

The present case fails to support the proposal of Friedman et al.5 that "it may be advantageous to maintain anesthesia with high inspired concentrations of nitrous oxide until dural closure so that a pneumocephalus that formed intraoperatively would contain nitrous oxide that would then be reabsorbed rapidly when nitrous oxide was discontinued." As seen in the present case, administering high inspired concentrations of nitrous oxide may not provide a pneumocephalus containing high concentrations of nitrous oxide and little air because prior to closure of the dura, the subdural space communicates freely with the atmosphere and nitrous oxide entering it is rapidly vented. Thus, when the dura is closed the pneumocephalus will contain primarily air regardless of the inspired gases. In a patient breathing nitrous oxide subsequent to dural closure, the volume of the pneumocephalus will increase as in the present case. In a patient not breathing nitrous oxide subsequent to dural closure the size of the pneumocephalus will undergo little change.

In the present case the gas emboli detected following closure of the dura may have been produced by the tension pneumocephalus. The arachnoid granulations act as one-way valves to allow CSF to flow into the superior sagittal sinus when a critical opening pressure is reached. Saidman and Eger previously postulated that gas under pressure in the intracranial CSF space might escape via the arachnoid granulations into venous blood. Recently, Luce et al. confirmed by left pulmonary artery Doppler recording and expired gas analysis that venous gas emboli occurred when a mixture of 80 per cent helium and 20 per cent oxygen was introduced into the subdural space at 20 mmHg below mean arterial pressure.

In summary, the present case report provides direct evidence that nitrous oxide may play a role in the genesis of tension pneumocephalus and that measuring CSF or intracranial pressure intraoperatively may contribute to early detection of this potentially hazardous problem. Discontinuing nitrous oxide at the time of dural closure may prevent tension pneumocephalus by avoiding expansion of air which entered the subdural space prior to dural closure. In a patient breathing nitrous oxide after closure of the dura, increased CSF pressure may indicate the presence of a tension pneumocephalus and discontinuing nitrous oxide and reopening the dura to vent the pneumocephalus should be considered. Postcraniotomy, the diagnosis of tension pneumocephalus also should be considered, particularly after procedures in the sitting position, and a brow-up lateral skull roentgenogram should be ordered if tension pneumocephalus is suspected. If tension pneumocephalus is diagnosed, decompression via twist drill holes should be performed as indicated by neurologic examination or CSF or intracranial pressure measurement.

REFERENCES