# **Venous Air Embolism during Neuroendoscopy**

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## **Abstract**

Sir,

Among the various problems associated with neuroendoscopic surgery, venous air embolism (VAE) has not been described earlier. We report here the development of this complication during endoscopic removal of a third ventricular (TV) craniopharyngioma cyst in an adult male patient and hypothesise the probable causative mechanism. This patient was subjected to our routine anaesthesia & monitoring protocol and neuroendoscopic technique. Before the start of surgery, the patient's heart rate (HR) was 86 beats/min, invasive arterial blood pressure (ABP) was 114/72 mmHg, end-tidal carbon dioxide value (EtCO<sub>2</sub>) was 33 mmHg and pulse oximetry value (SpO<sub>2</sub>) was 99%. Excision of the TV cyst was begun with a rigid neuroendoscope aided by a slow infusion of the irrigating fluid. During surgery, profuse bleeding occurred from a torn septal vein, obscuring the surgical field. The rate of irrigation was stepped up, the egress port of the endoscope was occluded to tamponade the bleeding and cauterization was attempted. Suddenly, the patient's HR and ABP increased to 154 beats/min & 177/94 mmHg respectively, followed immediately by an abrupt fall in his EtCO<sub>2</sub> to 20 mmHg and development of ventricular bigeminy. This alerted us to the possibility of VAE as a cause for these changes. N<sub>2</sub>O was discontinued and the patient was administered Xylocard 80 mg intravenously. Some air could also be aspirated from the ventricular system. Soon, the EtCO<sub>2</sub> rose to 30 mmHg and the haemodynamic values normalized. The remaining procedure was uneventful; however, the patient did not wake up at the end and was put on postoperative ventilation and continuous external ventricular drainage. He eventually died on the 4th postoperative day.

In this patient, the abrupt fall in EtCO2 values and

development of bigeminy is very suggestive of VAE, a known complication of intracranial surgeries. VAE usually occurs from entrapment of atmospheric air into an open vein with negative pressures. Conversely, if the pressures surrounding the vein are higher, air could be driven inside, through the following proposed mechanism. Endoscopic manipulations can inadvertently injure the many blood vessels in the TV area, necessitating an increase in the irrigation rate to improve visibility (1). Forceful irrigation, especially against a closed egress port is known to produce intraoperative tachycardia and hypertension (seen in our patient too) for which, one of the reasons suggested is an abrupt increase in TV pressures reflecting raised intracranial pressures (2). Some air invariably enters the ventricular system through the burr hole or via the endoscope sheath during endoscope insertion and the air volume is likely to expand with the use of N<sub>2</sub>O. This air could have been forced into the injured blood vessel because of the high TV pressures. A possibly low CVP due to preoperative use of mannitol and limited fluid intake in this patient could have further facilitated air entrapment. Thus, one needs to be alert to the occurrence of VAE during endoscopic manipulations and irrigation inside the TV cavity. N<sub>2</sub>O is best avoided (3) and though CVP monitoring is not routinely practiced during neuroendoscopy, it can prove useful in aspirating air if VAE is suspected.

### References

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