

## Venous air embolism during scalp dissection in a case of cerebellar haemangioblastoma

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Sir,

Venous air embolism (VAE) is the entrainment of air from the operative field or other communication with the environment into the venous vasculature producing systemic effects.<sup>[1]</sup> We report a case of VAE during scalp dissection in a patient undergoing craniotomy in sitting position for cerebellar haemangioblastoma. A 17-year-old, 40 kg, American Society of Anaesthesiologists physical status I, male patient presented with gait instability for 1 year and headache in occipital region for 9 months. The patient was diagnosed to have cerebellar haemangioblastoma [Figure 1] and was posted for excision in sitting position. There was no history of any respiratory or cardiovascular abnormality. His routine blood investigations, electrocardiogram, and chest X-ray were within normal limits. The patient was premedicated with intramuscular glycopyrrolate 0.2 mg 1 hour before induction of anaesthesia. A general endotracheal



**Figure 1:** Contrast enhanced computed tomographic scan of the head showing tumour mass in the posterior fossa (cerebellar haemangioblastoma)

anaesthesia with fentanyl, propofol, sevoflurane, vecuronium and nitrous oxide and oxygen (2:1 ratio) was used. Central venous cannulation (right subclavian vein) and intraarterial cannulation was performed following induction of anaesthesia. The patient was preloaded with 1 litre of crystalloid solution to ensure a central venous pressure of 8 mmHg, before the patient was made seated. The vital parameters of the patient remained unremarkable after sitting position. But soon after the surgeons started dissection of the scalp and pericranium, a sudden fall in end tidal carbon dioxide (EtCO<sub>2</sub>) from 34 mmHg-20 mmHg and blood pressure from

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120/73 mmHg-80/43 mmHg was observed. The rapidity of the event leads us to assume VAE to be the most probable cause of the event. Immediately, the patient's lung was ventilated with 100% oxygen. The surgery was briefly interrupted and the operative field was covered with normal saline. In view of hypotension 500 ml of hydroxyl ethyl starch and 10 mg of mephenteramine (in 5 mg increments) were administered; simultaneously, approximately 40 ml of air was aspirated through the central venous catheter. Within a period of 15 minutes, the patient's haemodynamic parameters and EtCO<sub>2</sub> returned back to normal. Rest of the surgery was uneventful.

The prerequisites for the entry of gas into the venous system are incision of non-collapsible veins such as diploic veins, emissary veins or dural venous sinuses in presence of sub-atmospheric pressure (e.g., during surgeries performed with patient in sitting position).<sup>[1]</sup> EtCO<sub>2</sub> is a semi-quantitative and non-invasive monitor of intermediate sensitivity for the detection of VAE.<sup>[2]</sup> However, decrement of EtCO<sub>2</sub> is not specific to VAE. Acute hypotension and pulmonary embolism due to any cause can also lead to a decrease in EtCO<sub>2</sub>.<sup>[3]</sup> We ruled out pulmonary embolism as to be the cause of the event as there were no predisposing factors associated in our patient. The other possibility was acute hypotension due to sudden blood loss, which may happen after rupture of venous sinuses during the initial part of the surgery. Acute hypotension can cause fall in EtCO<sub>2</sub> but unlike

VAE the fall is usually gradual.<sup>[3,4]</sup> So we considered the aetiology of the above event to be VAE and managed as per the standard management protocol.<sup>[2]</sup> With this case report, we want to emphasise that VAE can occur through occipital diploic veins even during dissection of scalp and pericranium in posterior fossa surgery with patient in sitting position. In the event of sudden and sustained fall in EtCO<sub>2</sub> during surgery in sitting position, VAE should be the first diagnosis even at the initial stages of surgery.

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Quick Response Code:	Website: www.jnaccjournal.org
	DOI: 10.4103/2348-0548.124853