

## An unusual case of repeated venous air embolism during awake bilateral deep brain stimulation surgery

WMN Wan Hassan<sup>a\*</sup>, BT Yeap<sup>a</sup>, L Ab Mukmin<sup>a</sup>, ARI Ghani<sup>b</sup> and RH Mohd Zaini<sup>a</sup>

<sup>a</sup>Department of Anaesthesiology and Intensive Care, School of Medical Sciences, Universiti Sains Malaysia, Kubang Kerian, Kelantan, Malaysia

<sup>b</sup>Department of Neurosciences, School of Medical Sciences, Universiti Sains Malaysia, Kubang Kerian, Kelantan, Malaysia

\*Corresponding author, email: [drnaza\\_anaest@yahoo.co.uk](mailto:drnaza_anaest@yahoo.co.uk)

Venous air embolism (VAE) is the entrainment of air either from a surgical site or from the environment into the venous or arterial vasculature, which can subsequently cause systemic effects. Many cases are subclinical but large volume and high rate of accumulation of air entrainment are potentially life-threatening. The relative risk is high in sitting position craniotomy and posterior fossa surgery but low in burr hole neurosurgery such as in deep brain stimulation (DBS) surgery. The authors report their experience of managing an unusual case of repeated VAE during both sides of burr-hole and electrode insertion in awake bilateral DBS surgery.

**Keywords:** awake craniotomy, burr hole, deep brain stimulation, neurosurgery, venous air embolism

### Introduction

Venous air embolism (VAE) is the entrainment of air from a surgical site or from the environment into the exposed venous or arterial vasculature, which can subsequently cause systemic effects. Many cases are subclinical but large volume and high rate of accumulation of air entrainment are potentially life-threatening. In neurosurgery under general anaesthesia, the relative risk of VAE is high in sitting position craniotomy and posterior fossa surgery with approximate expected reported incidences of > 25%.<sup>1</sup> Burr hole neurosurgery is classified as low relative risk with an approximate expected incidence of < 5%.<sup>1</sup>

Deep brain stimulation (DBS) surgery is a minimally invasive procedure where electrodes are placed in the deep brain structures for microelectrode recordings (MERs) and macrostimulation. After stimulation and clinical testing, the electrodes will subsequently be connected to an implanted pacemaker. It is indicated for the treatment of movement disorders such as Parkinson's disease (PD), essential tremors, dystonia and certain psychiatric disorders and commonly performed in awake and spontaneous ventilation patients.<sup>2,3</sup>

There have been a few reported cases of VAE in DBS surgery that raise a concern for extra monitoring and prompt management of this situation.<sup>4-6</sup> All previous reported cases occurred during the burr hole and insertion of the electrodes on only one side of the procedure. However, in our case the suspicious symptoms occurred repeatedly during both sides of the procedure. We report our experience of managing mild symptoms with low probability of VAE during the insertion of the first electrode, subsequently followed by more symptomatic and high probability of VAE during the surgery to implant the second electrode.

### Case report

A 70-year-old man, a known case of idiopathic PD for the past 10 years, presented with worsening resting tremors of both upper and lower limbs for the past year, and he was scheduled for elective bilateral placement of DBS electrodes. He was also known to have hypertension and dyslipidaemia for the past

20 years. On preoperative assessment, there was no preoperative history of respiratory symptoms and baseline electrocardiogram (ECG) was normal.

In the morning of surgery, a stereotactic frame was initially applied under local anaesthesia infiltration and computerised tomography (CT) of the brain was used for target localisation. In the operating theatre, the patient was placed supine in 30° head-up position. Intraoperative monitoring included invasive blood pressure (BP), oxygen saturation (SpO<sub>2</sub>) and ECG. He was given supplemental 3 L/ min of oxygen via a nasal prong, which also connected to capnography for end tidal carbon dioxide (E<sub>t</sub>CO<sub>2</sub>) monitoring. Peripheral venous access was established on both forearms.

Anaesthesia management was planned with a monitored anaesthesia care technique using intravenous (IV) dexmedetomidine infusion and target-controlled infusion (TCI) remifentanyl during the burr-hole and electrodes placement and then stopped during MERs and clinical assessment. A loading dose of 0.5 µg/kg of dexmedetomidine was initially given over 10 min and then maintained at the range of 0.2 to 0.5 µg/kg/hour. TCI remifentanyl was also started after the loading dose of dexmedetomidine and titrated between 0.1 and 2.0 ng/ml. The surgery started with the right burr hole and after the dura mater was opened, the patient had an intermittent and mild cough. At this point, we did not anticipate that the cough might be an early sign of VAE because the patient was relatively comfortable, with E<sub>t</sub>CO<sub>2</sub> maintained between 30 and 35 mm Hg and no episodes of desaturation. There was also minimal secretion upon suctioning of the mouth. The cough stopped as the surgery progressed and the procedure was successfully completed.

When the left burr hole was started, we restarted dexmedetomidine infusion at 0.5 µg/kg/hour and titrated TCI remifentanyl between 0.1 and 2.0 ng/ml. The edges of the burr hole were sealed with bone wax and then the dura mater was opened. The patient was initially comfortable but then developed a severe cough and became tachypnoeic. The oxygen saturation dropped subsequently to 50% and then the patient became

apnoeic.  $E_t\text{CO}_2$  was initially reduced to 25 mm Hg and the waveform was flat during apnoea. Heart rate was bradycardic until 39 beats/min but then reverted spontaneously to 70–80 beats/min without administration of any medications. BP was maintained at 130/50 mm Hg. TCI remifentanyl was withheld because of apnoea and assisted manual ventilation was commenced with the placement of an oral pharyngeal airway. Venous air embolism (VAE) was suspected and the surgery was immediately stopped. The head frames were removed and the patient was repositioned to supine. The surface of the surgical field was irrigated with copious amount of saline solution and at the same time bone wax was reapplied. A ProSeal™ Laryngeal Mask Airway (PLMA) (Teleflex Medical Europe Ltd, Athlone, Ireland) was immediately inserted to facilitate ventilation after boluses of IV propofol 1 mg/kg in titration and the restarting of TCI remifentanyl at 3 ng/ml. Using manual ventilation with 100% oxygen we managed to obtain good saturation on pulse oximetry. Maintenance of anaesthesia was initially maintained with sevoflurane at 5–6% with inspired oxygen of 100% and flow rate of 6 L/min. Auscultation of his chest revealed equal air entry and there were no rhonchi, crepitations or murmurs. Arterial blood gases (ABG) on 100% oxygen showed a pH of 7.34,  $p\text{O}_2$  446 mm Hg and  $p\text{CO}_2$  of 42 mmHg. He subsequently developed atrial fibrillation (AF) with the rate up to 130–140 beats/min but his BP was still stable in the range of 100/60–130/85 mm Hg. We started him on amiodarone infusion for the AF. He was subsequently stable and extubated 20 min after the heart rate reverted to sinus rhythm.

The surgery for the insertion of DBS on the left side was postponed and he was closely observed in the neurosurgical intensive care unit (ICU) for one day. The results after the procedure showed a normal value of creatinine kinase and troponin T level. Echocardiography by the cardiologist in ICU on the next day showed mild mitral regurgitation and trivial tricuspid regurgitation with an ejection fraction of 59%. Repeat computerised tomography (CT) scan of the brain and chest X-ray (CXR) were normal. The patient was discharged well after three days and the surgery for DBS was rescheduled to another date.

## Discussion

VAE is a potential life-threatening catastrophe that requires early detection and prevention to avoid fatal outcomes for the patient. It can occur whenever there is a gravitational pressure gradient between the atmospheric and venous pressure, which can trigger the entrainment of air into the breached venous access. An operative site more than 5 cm above the right atrium has been mentioned as one of the potential risk factors.<sup>6</sup> The mortality and morbidity of VAE can be affected by the rate and the volume of air entrainment, as well as the position of the patient.<sup>7</sup> In awake craniotomy as in DBS surgery, the other additional risk factors for VAE are awake condition, spontaneous ventilation and head elevation. All of these factors contribute to more negative intrathoracic pressure which is favourable for air entrainment into diploic venous channels of the porous skull bone or through the breached sinus system.<sup>8</sup>

There are clear differences in diagnosing the probability of VAE in awake patients and in fully anaesthetised patients. In awake patients, clinical symptoms are more important for early detection whereas fully anaesthetised patients are more dependent on the changes in monitoring parameters. The initial presentation of coughing, tachypnoea, chest pain, restlessness or agitation is more important in the suspicion of VAE in awake patients before being followed by dropped  $E_t\text{CO}_2$ , hypoxia,

hypotension or arrhythmias. However, the detection of VAE may be further delayed in patients undergoing awake procedures because VAE presentation is more atypical if it is compared with the more classical features of VAE in patients who are under general anaesthesia.<sup>5,6</sup> The severity of clinical presentation is determined by the volume of air, as well as the rate of air entrainment. An acute amount of air of more than 2 ml/kg is more likely to cause chest pain, right heart failure and cardiovascular collapse. In fully anaesthetised patients, other than routine monitoring, it is possible to use specific monitoring for early detection of VAE. Transoesophageal echocardiography is the most sensitive monitoring but it is invasive and inappropriate in awake patients. The other option for awake patients is precordial Doppler ultrasound, which is the most sensitive among non-invasive monitoring procedures for VAE. However, it is not available in our centre. Other high-sensitivity monitoring methods for VAE are pulmonary artery catheter and transcranial Doppler.<sup>1</sup> End tidal nitrogen and  $\text{ETCO}_2$  are classified under moderate sensitivity monitoring whereas the rest of monitoring such as oxygen saturation, oesophageal stethoscope and ECG are low in sensitivity.<sup>1</sup>

The presentation of acute cough is also an important predictor of VAE during DBS surgery. Hooper *et al.* did a retrospective review of DBS electrode placement to look for the incidence of VAE based on the presence of intraoperative cough and haemodynamic instability, which was subsequently followed by prospective evaluation using precordial Doppler ultrasound. The retrospective review of 286 DBS placement revealed a 3.2% incidence of cough per electrode and the prospective incidence was 4.5% per electrode. They concluded that patient positioning and the occurrence of cough are two important predictors to consider in VAE and highlighted the role of precordial Doppler for early detection.<sup>9</sup> Chang *et al.* reviewed a larger number of patients, 467 from consecutive DBS surgeries, and the results showed the incidence of clinically diagnosed VAE was only 1.3% based on the presentation of the most common symptoms such as coughing, oxygen desaturation and hypotension.<sup>10</sup> However, the pathophysiology of cough and tachypnoea during VAE is still not well understood. It has been suggested that the coughing encountered during VAE could be due to the cytokines and complement released during the lung's immune response to transient ischaemia.<sup>4</sup> The incidence of cough as a presenting sign of VAE in a patient under general anaesthesia is unknown and, even if it is present, it is difficult to differentiate between the signs of VAE and the signs of light anaesthesia. Most of the time, the detection of VAE during general anaesthesia is based more on the changes in monitoring parameters in a high-risk surgery group.

In our patient, the presentation was unique because it happened during both sides of the burr hole and electrode insertion. He presented initially with mild, intermittent and tolerable intraoperative cough during the right burr hole. However, there was no desaturation and the procedure was completed successfully. Acute intraoperative cough recurred during the left burr hole but this time it was more explosive and associated with desaturation, tachypnoea and then apnoea. It was only after the second event that we made a provisional diagnosis of VAE. Chang *et al.* in their retrospective review classified their patients into two groups: high probability and low probability of VAE. High probability was defined as a coughing episode plus either a desaturation event, hypotension and additional coughing episode within 3 min, development of chest pain, shortness of breath or a new heart murmur. Low-probability VAE was defined as an isolated coughing episode

without other signs and symptoms.<sup>10</sup> Our patient presented low probabilities during the first burr hole and high probabilities during the second burr hole. The differential diagnoses that can be considered for our patient were bronchospasm, aspiration pneumonia, acute myocardial infarction, opioid overdose, and venous thrombo-embolism. There were no symptoms of chest pain or ECG changes that might be suggestive of acute coronary syndrome except for the AF and transient bradycardia. The absence of crepitations and rhonchi on auscultation also ruled out the other respiratory causes. Therefore, based on the nature of the surgery and clinical presentation, the diagnosis of VAE was the most likely in our case.

Prompt recognition and management are critical to avoid further associated complications, and respiratory outcome of patients is generally good after several days of close observation.<sup>10</sup> In our case, the presentation of intermittent and mild cough during the first burr hole might be controlled with continuous saline irrigation and bone wax by the surgeon during the procedure. However, because of acute explosive cough associated with hypoxia and apnoea during the second burr hole, we had to back mask ventilation, followed by repositioning of the head and insertion of PLMA for assisted ventilation. All these measures were slightly challenging during the DBS surgery because its head frame reduced our access to the airway. We managed to extubate our patient in the operating theatre and he was discharged well after a few days of close observation in the neurology ICU.

### Conclusion

VAE is a potentially disastrous complication during DBS surgery and it can occur during both sides of a burr hole and electrode placement procedure. Early recognition of acute intraoperative cough as a suspicious symptom and prompt management of more critical presentations are crucial for the outcome of the patient.

### References

1. Mirski MA, Lele AV, Fitzsimmons L, et al. Diagnosis and treatment of vascular air embolism. *Anesthesiology*. 2007;106:164–77. <http://dx.doi.org/10.1097/0000542-200701000-00026>
2. Pereira EA, Green AL, Nandi D. Deep brain stimulation: indications and evidence. *Expert Rev Med Devices*. 2007;4:591–603. <http://dx.doi.org/10.1586/17434440.4.5.591>
3. Venkatraghavan L, Luciano M, Manninen P. Anesthesia management of patients undergoing deep brain stimulator insertion. *Anesth Analg*. 2010;110:1138–45.
4. Moitra V, Permut TA, Penn RM, et al. Venous air embolism in awake patient undergoing placement of deep brain stimulators. *J Neurosurg Anesthesiol*. 2004;16:321. <http://dx.doi.org/10.1097/00008506-200410000-00017>
5. Deogaonkar A, Avitsian R, Henderson JM, et al. Venous air embolism during deep brain stimulation surgery in an awake supine patient. *Stereotact Funct Neurosurg*. 2005;83:32–5. <http://dx.doi.org/10.1159/000085024>
6. Kumar R, Goyal V, Chauhan RS. Venous air embolism during microelectrode recording in deep brain stimulation surgery in an awake supine patient. *Br J Neurosurg*. 2009;23:446–8. <http://dx.doi.org/10.1080/02688690902775538>
7. Palmon SC, Moore LE, Lundberg J, et al. Venous air embolism: a review. *J Clin Anesth*. 1997;9:251–7. [http://dx.doi.org/10.1016/S0952-8180\(97\)00024-X](http://dx.doi.org/10.1016/S0952-8180(97)00024-X)
8. Balki M, Manninen PH, McGuire GP, et al. Venous air embolism during awake craniotomy in a supine patient. *Can J Anaesth*. 2003;50:835–8. <http://dx.doi.org/10.1007/BF03019383>
9. Hooper AK, Okun MS, Foote KD, et al. Venous air embolism in deep brain stimulation. *Stereotact Funct Neurosurg*. 2009;87:25–30.
10. Chang EF, Cheng JS, Richardson RM, et al. Incidence and management of venous air embolisms during awake deep brain stimulation surgery in a large clinical series. *Stereotact Funct Neurosurg*. 2011;89:76–82. <http://dx.doi.org/10.1159/000323335>

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