Both EMG activity and radiofrequency interference from electrical equipment in the operating theatre influence the BIS algorithm. Under normal conditions, the EEG is of sufficient power to override these other electrical sources and is thus weighted appropriately by the algorithm. In circumstances of extreme EEG suppression, as during deep hypothermic circulatory arrest, it is possible that electrical interference from either EMG or radiofrequency noise may be interpreted by the algorithm as EEG activity and assigned a high BIS value. When EEG activity resumes, as during rewarming, the monitor then re-interprets it appropriately, discounting the EMG and radiofrequency activity in the algorithm (personal communication, David Zaratek, Aspect Corporation). This appears to have happened in our case, as there was no demonstrable evidence of neurologic insult, such as seizure activity or choreoathetosis, seen postoperatively.

In conclusion, we present a case where electrical interference was misinterpreted as EEG activity by an Aspect A-2000 BIS monitor during a period of intense EEG suppression. This misinterpretation was displayed as a high BIS value with a low suppression ratio. Observation of the real-time EEG waveform suggested that this was an artefact, but was not absolutely conclusive. During periods of significant EEG suppression, BIS monitors may misinterpret electrical interference as EEG and could possibly lead to unnecessary therapeutic interventions. Observation of the real-time EEG waveform may aid in the diagnosis of this artefact, which, in our experience, occurs infrequently. Further studies are necessary to determine if monitoring suppression ratio and BIS during deep hypothermic circulatory arrest is beneficial and cost effective.

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References
1 Sigl JC, Chamoun NC. An introduction to bispectral index analysis for the EEG. J Clin Mon 1994; 10: 392–404


Perioperative management of a patient requiring surgery for pituitary apoplexy and severe angina pectoris

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We describe the management of a 71-yr-old man with pituitary apoplexy and severe angina pectoris who underwent treatment of an intra-cranial haemorrhage and open-heart surgery requiring anticoagulant therapy within a very short period. Subtotal removal of the pituitary tumour was undertaken under stable cardiovascular conditions. But ventricular fibrillation occurred after the neurosurgery in the intensive care unit. After the patient was defibrillated, intra-aortic balloon pumping was necessary to assist coronary artery blood flow. Twenty hours after neurosurgery, oozing from the surgical wound stopped and coronary artery bypass grafting with full heparinization was performed uneventfully.

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Pituitary apoplexy is a clinical syndrome of sudden haemorrhagic necrosis of the pituitary gland. Subsequently, tumour expansion causes headache, visual field deficit and ophthalmoplegia. Recovery of the patient depends on the duration and severity of symptoms. Surgical treatment of intracranial haemorrhage is recommended immediately after appearance of the neurological symptoms.

We experienced a rare case of a patient with pituitary apoplexy coexisting with severe angina pectoris, which required urgent coronary artery revascularization. However, anticoagulant therapy for cardiopulmonary bypass (CPB) might have induced intra-cranial haemorrhage. Pituitary apoplexy has been reported to occur following coronary artery bypass grafting. Sequential management of the intracranial haemorrhage and open-heart surgery requiring anticoagulation present a challenge to the anaesthetist.

Case report

A 71-yr-old man (weight 54 kg) was referred to our hospital for surgical treatment of severe triple-vessel coronary artery disease. On admission, he was suffering frequent episodes of angina pectoris at rest requiring treatment with nitroglycerine (0.3 \( \mu \text{g kg}^{-1} \text{min}^{-1} \)), diltiazem (0.06 mg kg\(^{-1} \text{h}^{-1} \)) and lidocaine (1 mg kg\(^{-1} \text{h}^{-1} \)) i.v. He had a past medical history of hypertension and a myocardial infarction.

A pituitary tumour had been diagnosed by cranial computed tomography (CT) 2 days before admission. He had had a slight headache but no other neurological deficits. In hospital awaiting cardiac surgery, he became comatose and was found to have a Glasgow Coma Score (GCS) of 6. Neurological examination revealed dilated bilateral pupils, a visual field deficit, ophthalmoplegia and proptosis. Review of the CT showed a fairly large, iso-dense and high-density sellar mass with haemorrhage. The tumour extended into the suprasellar cistern with compression of the optic nerves and cavernous sinus on both sides. There was haemorrhage into the ventricle and brain tissue.

He underwent emergency neurosurgery before cardiac surgery. In the operating room, invasive blood pressure, central venous pressure, pulse oximetry, end-tidal carbon dioxide and the electrocardiogram were monitored. Anaesthesia was induced with midazolam 2 mg and fentanyl 200 \( \mu \text{g} \) and pancuronium 8 mg was administered to facilitate tracheal intubation. Anaesthesia was maintained with 0.5–2% sevoflurane and an i.v. fentanyl (2 \( \mu \text{g kg}^{-1} \text{h}^{-1} \)) infusion, and muscle relaxation was maintained with intermittent boluses of pancuronium. Mechanical ventilation with a tidal volume of 10 ml kg\(^{-1} \) and respiratory rate of 12 min\(^{-1} \) was adjusted to maintain normocapnia. Dopamine 3–7 \( \mu \text{g kg}^{-1} \text{min}^{-1} \), nitroglycerine 0.15–0.7 \( \mu \text{g kg}^{-1} \text{min}^{-1} \), diltiazem 0.06–0.1 mg kg\(^{-1} \text{h}^{-1} \) and lidocaine 1 mg kg\(^{-1} \text{h}^{-1} \) were administered continuously throughout surgery. After bifrontal craniotomy, the neurosurgeon approached the tumour inter-hemispherically and performed subtotal tumour resection. The 10-h surgery was completed without complication and with a blood loss of only 300 ml. Examination of the surgical specimens revealed haemorrhagic and necrotic pituitary tissue. After surgery, the patient was haemodynamically stable and was transferred to an intensive care unit, where he was ventilated mechanically and sedated with midazolam. When suctioning was performed through the tracheal tube, ventricular fibrillation suddenly occurred. The patient was cardioverted, additional lidocaine (1 mg kg\(^{-1} \)) was administered and sinus rhythm returned. However, premature ventricle complexes occurred frequently and systolic blood pressure was below 90 mm Hg. An intra-aortic balloon pump was placed through the left femoral artery to assist coronary artery blood flow. We did not use any anticoagulant to prevent bleeding from the surgical site. We delayed cardiac surgery for about 20 h after neurosurgery until oozing from the subcutaneous drainage site stopped. The CABG procedure was then undertaken. Upon arrival in the operating room, a pulmonary artery catheter was placed via the internal jugular vein. Anaesthesia was induced with midazolam 2 mg and fentanyl 100 \( \mu \text{g} \), then maintained with propofol 2 mg kg\(^{-1} \text{h}^{-1} \) and fentanyl 2 \( \mu \text{g kg}^{-1} \text{h}^{-1} \) intravenously. Muscle relaxation was obtained with pancuronium 6 mg and maintained with intermittent boluses. The patient continued to receive dopamine, nitroglycerine, diltiazem and lidocaine at the same doses until starting CPB. The duration of aortic clamping was 60 min and that of CPB was 100 min. The patient was weaned from CPB uneventfully, but balloon pump was continued. Mechanical ventilatory support was continued in the intensive care unit. On the second day postoperatively, CT brain revealed no haemorrhage. The patient regained airway reflexes and spontaneous ventilation. The trachea was extubated. A week after neurosurgery, diabetes insipidus developed and was treated with vasopressin 10 u. day\(^{-1} \) intravenously. For about a month after the CABG procedure, his conscious state fluctuated between somnolence and delirium. When delirious, the patient was sedated by a midazolam 1–2 mg h\(^{-1} \) infusion.
Within a month, he recovered and had a GCS of 14. Visual field deficit and ophthalmoplegia gradually improved.

Discussion

Pituitary apoplexy is due to an intra-cranial tumour, into which haemorrhage produces an expanding mass, causing visual and neurological impairment. The patient requires neurosurgical treatment immediately. Severe unstable angina with the necessity for an urgent CABG procedure coexisted with the pituitary expansion in this patient. It is often suggested that non-cardiac surgery and CABG should be performed simultaneously. In this case, however, anticoagulant therapy with CPB would have induced intracranial haemorrhage and the neurological prognosis could have been worse. Confronted with such conflicting situations, we had a dilemma regarding the best surgical management.

In patients with severe angina pectoris and a history of myocardial infarction, the risk of perioperative cardiac complications including death is increased. The American Heart Association reported that the frequency of myocardial infarction and death during simultaneous carotid endarterectomy and a CABG procedure is lower than in a staged operation. Allie and colleagues recommended that a rapid staged procedure (an interval between endarterectomy and CABG procedure of less than 24 h) with intra-aortic balloon pumping was safe and effective in the very high risk patient population with coronary artery disease. But the optimal strategies for the management of patients with carotid and coronary artery disease have not been established. In this patient, the neurological prognosis depended on immediate neurosurgical decompression. We, therefore, performed neurosurgery before the CABG procedure as a staged operation.

The point at which intra-cranial haemostasis is achieved after neurosurgery remains uncertain. Wijdicks reported that restarting anticoagulation therapy in a patient with mechanical heart valves is safe 1–2 weeks after intracranial haemorrhage. However, in this patient, we could not delay the CABG procedure because the patient had ventricular fibrillation and required mechanical support using intra-aortic balloon pumping. When the oozing from the neurosurgical wound stopped, we immediately performed the cardiac surgery using anticoagulant therapy 20 h after neurosurgery.

Endocrine failure is a problem in a staged operation. If acute adrenal insufficiency, hypothyroidism and diabetes insipidus occur during the perioperative period of cardiac surgery, it might worsen the angina pectoris. For example, administration of vasopressin induces coronary vasoconstriction. It is, therefore, important to perform cardiac surgery before any uncontrollable endocrine failure occurs. This is one of the reasons why we performed the neurosurgery before the cardiac surgery.

In conclusion, we managed a patient with severe angina pectoris and pituitary apoplexy. We have described a two staged operation with sequential management of intra-cranial haemorrhage and open-heart surgery requiring anticoagulant therapy. Our case illustrates the importance of coronary care during the time between the two operators.

References