Case Report

Severe bradycardia and hypotension after connecting negative pressure to the subgaleal drain during craniotomy closure

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Negative pressure drainage systems are often used after craniotomy for evacuation of potential bleeding. There are several reports of haemodynamic disturbances with epidural negative pressure drainage, but such reports are very few for subgaleal drains placed over the bone flap. We report a case in which a patient developed severe cardiovascular disturbances after the vacuum drainage was connected to a subgaleal drain after craniotomy for aneurysm clipping. The patient had no significant cardiac history, had an uneventful intra-operative course and yet developed bradycardia and hypotension, which were reproducible and severe enough to require atropine administration. Anaesthetists must be aware of these effects, so that they can anticipate and treat such complications.

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Epidural or subgaleal drains are commonly placed and are connected to a vacuum system towards the end of cranial surgery to prevent accumulation of intracranial and extracranial blood. Although, bradycardia and arterial hypotension have been shown to occur with epidural negative pressure drainage,1,2 such reports are uncommon with subgaleal drains placed over the bone flap.3,4 We encountered a case in which the patient developed three episodes of bradycardia and hypotension, probably caused by two different mechanisms, after connecting vacuum drainage to the subgaleal drain after an uneventful cerebral aneurysm surgery.

Case report

A 65-yr-old, 56 kg female patient, who had had an episode of transient loss of consciousness 1 month previously followed by left hemiparesis, was listed for clipping of right middle cerebral artery bifurcation aneurysm. She had also had an intermittent headache for 2–3 months. Apart from the aneurysm, magnetic resonance angiography demonstrated infarcts in frontal and occipital cortex and in basal ganglia, on the right side. She had essential hypertension, which was controlled on enalpril 25 mg once daily and was also taking aspirin 100 mg, which was stopped 7 days before the day of surgery. All her preoperative investigations were normal.

After an overnight fast the patient was premedicated 1 h before surgery with oral diazepam 5.0 mg, enalpril 25 mg and i.m. glycopyrrolate 0.2 mg. In the operating theatre, noninvasive monitoring (5-lead ECG, noninvasive automated blood pressure and pulse oximetry) was established. A 16 gauge cannula was inserted in the dorsum of her left hand. Her baseline heart rate was 100 beats min

After pterional craniotomy, the aneurysm was clipped after temporary clipping of the feeding vessel for 1 min 35 s. The dura mater was closed in a watertight manner
and the bone flap was secured with Craniofix® (B. Braun Aesculap AG, Germany). A subgaleal drain was placed before scalp closure. After skin closure suction was applied to the drain to evacuate any blood collection. There was a sudden decrease in heart rate from 80 to 38 beats min⁻¹ and in blood pressure from 128/60 to 85/40 mm Hg. There was no alteration in the morphology of the ECG. Suction was immediately removed and the heart rate and blood pressure returned to normal.

After surgical wound dressing, the drain was connected to a Romovac® (Romsons Scientific & Surgical Industries (P) Ltd, India) 800 cm³ capacity vacuum bottle with moderate negative pressure. Again there was a decrease in heart rate from 58 to 20 beats min⁻¹ and in blood pressure from 128/60 to 85/40 mm Hg. Atropine sulphate 0.6 mg was administered i.v. The heart rate increased to 120 beats min⁻¹. The Romovac® was disconnected and reapplied without negative pressure. The drain was then allowed to drain under gravity. Again, a slight decrease in heart rate from 118 to 96 beats min⁻¹ was noted but there was only a small decrease in blood pressure from 146/84 to 138/82 mm Hg. The heart rate returned to normal as the drain was lifted up. There was about 20–30 ml of sero-sanguinous fluid in the drain. The patient made an uneventful recovery after the neurointensive care unit after extubation. Her postoperative neurointensive care stay was uneventful and she was discharged on the fifth postoperative day without any new neurological deficit.

Discussion

Development of acute intracranial hypotension leading to severe haemodynamic instability has been described in connection with negative suction pressure to epidural, epicranial or subgaleal drains towards the end of cranial surgery.

Our patient developed three episodes of bradycardia and hypotension after the application of negative pressure to the subgaleal drain at the end of the surgery. The intra-operative course till the development of these episodes was uneventful. The only significant event that preceded these episodes was the application of negative pressure to the subgaleal drain. Hernandez and colleagues demonstrated significant decreases in heart rate and intracranial pressure with the application of negative pressure through the epidural drainage connected to a vacuum system, but they did not find a decrease in blood pressure.

Van Roost and colleagues have recently reported hypoxic brain lesions on CT/MRI in patients with the use of subgaleal negative pressure drainage leading to non-awakening and dramatic neurological deterioration after uneventful neurosurgical procedures. As hypoxaemia was ruled out as being responsible for these changes, they termed this condition ‘pseudo-hypoxic brain swelling (PHBS)’. In 2 of 17 patients analysed, severe bradycardia and arterial hypotension on connecting negative pressure to a subgaleal drain were reported. Our patient also had bradycardia and hypotension, but she did not have delayed neurological recovery or other neurological complications. The drain volume collected was not great. The event in our case was probably a milder form of the same phenomenon. It could have been part of a continuum of effects that can occur because of the rapid development of severe intracranial hypotension. As it was detected early and the cause was promptly removed, the stage of PHBS was not reached.

Thus, the initial two episodes of bradycardia and hypotension in our case can be attributed to a sudden and severe decrease in intracranial pressure associated with subgaleal negative pressure application. The third episode may be attributable to some different mechanism as after the second episode subgaleal drain was connected to the collection unit (Romovac) without any negative pressure. Traction on the scalp nerve endings on hanging the drain under gravity may have triggered the trigeminocardiac reflex (TCR). The TCR has also been described during ocular (oculocardiac reflex), maxillo-facial and intracranial surgery. Afferent impulses travel through the sensory branches of the trigeminal nerve, to the main sensory nucleus of trigeminal nerve under the floor of the fourth ventricle. Short inter-nuclear fibres connect with the motor nucleus of the vagus nerve. In our case scalp traction might have stimulated all three divisions of the trigeminal nerve (ophthalmic through the supraorbital nerve, maxillary through the zygomaticotemporal nerve and mandibular through the auriculotemporal nerve). Stopping further stimulation is the first step in management of TCR. Light anaesthesia, hypoxia, hypercarbia and acidosis can potentiate the response; hence these should be corrected, if present. Often, the reflex fatigues with repetitive stimulation, but if bradycardia and or arterial hypotension continue or recur, atropine or glycopyrrolate should be given. Although, atropine had been administered after the second episode of bradycardia, a slight decrease in heart rate was still noted as the third episode. Anticholinergic drugs are not recommended prophylactically as tachycardia is not protective and they can cause refractory arrhythmias. The efficacy of anticholinergic drugs in preventing further episodes of bradycardia and hypotension is dose-dependent and though the number of such episodes can be reduced, they are not totally prevented.

In summary, we propose two different mechanisms for the haemodynamic instability that we observed; the development of severe intracranial hypotension through negative pressure drainage and trigeminocardiac reflex. Immediate return of heart rate to baseline on removal of negative pressure and release of traction further supports our belief.

Suction should be applied gradually to drains after craniotomy and the anaesthetist should be particularly vigilant. Also, traction through the drain to the scalp should be avoided. Should unexplained bradycardia develop during
craniotomy closure, prompt action should be taken to identify and remove the cause. It is possible that haemodynamic alterations because of a decrease in ICP on application of negative subgaleal drainage (if, ignored) may be a harbinger of the much more sinister PHBS.

References


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