Cardiovascular disturbances caused by extradural negative pressure drainage systems after intracranial surgery


Summary
Extradural drainage systems connected to a vacuum device for preventing postoperative haematoma formation are often used in neurosurgical practice. Cardiovascular complications, including bradycardia or low arterial pressure caused by intracranial hypotension, have been described associated with their use. We have investigated the relationship between the negative pressure applied to extradural drainage systems and intracranial pressure (ICP), and analysed the effects of negative pressure of the drains on systolic (SAP), diastolic (DAP) and mean (MAP) arterial pressures and on heart rate (HR). We studied prospectively 15 patients undergoing neurosurgery for supratentorial tumours or aneurysms. Transient decreases in ICP (P<0.001) and HR (P<0.001), with no clinical effects, were observed after connecting the vacuum device to the drain. There were no significant changes in SAP, DAP or MAP. (Br. J. Anaesth. 1998; 80: 599–601)

Keywords: equipment, extradural drainage systems; heart, heart rate; surgery, neurological; cardiovascular system, effects

Increased intracranial pressure (ICP) can cause bradycardia and hypertension as a result of the stimulus of pressure or stretch, or both, of the brainstem, referred to as Cushing’s response. However, haemodynamic changes associated with intracranial hypotension have seldom been described. Most reports are based on clinical observations. Many neurosurgical procedures, such as surgery for brain tumours and haematomas, require the use of extradural or epicranial drains, which are frequently connected to a vacuum device for preventing haematoma formation or drain obstruction. Severe cardiovascular disorders (bradycardia, asystole or arterial hypotension) related to their use have been published.

The aim of our study was to investigate the effect on ICP of the negative pressure achieved by an extradural drainage system and its consequences on heart rate (HR), systolic (SAP), diastolic (DAP) and mean (MAP) arterial pressures.

Patients and methods
We studied 15 consecutive patients, aged 28–65 yr, ASA I–II, undergoing elective supratentorial tumour and aneurysm surgery. The study was approved by the Institutional Human Ethics Committee and patient consent was obtained.

On arrival in the operating room, patients were premedicated with midazolam 0.02–0.03 mg kg⁻¹ i.v. Anaesthesia was induced with thiopental 4–6 mg kg⁻¹, fentanyl 3–4 μg kg⁻¹ and lidocaine 1.5 mg kg⁻¹. Vecuronium (up to 0.2 mg kg⁻¹) was administered to aid tracheal intubation. Anaesthesia was maintained with isoflurane (0.2–0.6 % end-tidal concentration) and 60–70% nitrous oxide in oxygen, and fentanyl 1–2 μg kg⁻¹ h⁻¹. Neuromuscular block was produced with vecuronium 0.08–0.1 mg kg⁻¹ h⁻¹. Controlled mechanical ventilation (Servo 900 C ventilator) was adjusted to maintain end-tidal carbon dioxide partial pressure (P CO₂ ) at 3.7–4.3 kPa.

Before induction of anaesthesia, we monitored invasively radial arterial pressure, HR, electrocardiogram (leads II and V5) and arterial haemoglobin oxygen saturation (S O₂ ) using a Siemens Sirecust 730 monitor. P CO₂ was measured with an Engström Eliza carbon dioxide analyser, and neuromuscular function was monitored with a Microstin Plus peripheral nerve stimulator. After induction of anaesthesia, we inserted a nasopharyngeal thermometer, oesophageal stethoscope and central venous catheter.

The skin was infiltrated with 0.5% bupivacaine before the start of surgery. Mannitol 0.5 g kg⁻¹, sometimes associated with a loop diuretic, was administered. MAP was 80–110 mm Hg during surgery.

After tumour resection or aneurysm clipping, and before totally closing the dura mater, the neurosurgeon placed a subdural fiberoptic transducer attached to a Camino monitor (Camino 420 OLM) for continuous recording of ICP. ICP recording was started at the end of surgery.

In all patients the dura mater was sutured in a watertight manner, and when there remained a space between the brain and dura mater, it was filled with saline solution infused without applying pressure. Before bone flap replacement, an extradural drain was positioned in the extradural space, as far as...
Results

Patient data (age, sex, weight), duration of surgery, duration of anaesthesia, negative pressure values in the vacuum system and type of surgical procedure are presented in Table 1.

A decrease in SAP, MAP and an increase in DAP with regard to baseline values was observed immediately after application of the vacuum to the drainage system, but these differences were not significant (Table 2). There were significant decreases in HR and ICP (P<0.001) after applying negative pressure.

Discussion

In this study, placement of an extradural drain connected to a vacuum system at the end of intracranial surgery produced a sudden decrease in ICP that reached subatmospheric values in all patients. This sudden reduction in ICP was caused by transmission of negative pressure from the area (extradural or epicranial space) where the drain was located to the subdural space. Severe decreases in ICP may cause a rostral shift of the brain which is reversed when ICP returns to its previous values. Physical properties of brain tissues may preclude this shift, even during suction.

In our study, a significant decrease in HR without changes in arterial pressure was observed immediately after connecting the vacuum system and before reaching the peak decrease in ICP. However, there was a rapid return to pre-suction values. These data are in agreement with those of Gilsanz and colleagues who observed a significant decrease in HR 3 min after connecting the vacuum system to the epicranial drain. However, in our study, the effect of the negative pressure on ICP and HR was more immediate.

Several reports have described severe bradycardia or arterial hypotension, or both, after connection of negative suction pressure to extradural or epicranial drains after craniotomy. These haemodynamic changes were similar to those observed when ICP suddenly decreased after decompression of hydrocephalic ventricles. Rapid removal of cerebrospinal fluid (CSF) in patients with obstructive hydrocephalus or intracranial application of negative pressure via an extradural or epicranial drain, may lead to a sudden state of intracranial hypotension resulting in a rostral movement of the brain or brainstem nuclei responsible for the changes in cardiac rhythm or arterial hypotension, or both. Reduction in HR secondary to a sudden decrease in ICP may be caused by a mechanism similar to Cushing’s response to intracranial hypertension. Several authors have demonstrated that the haemodynamic features of Cushing’s response can be produced by electrical stimulation or distension of tissues within a thin strip of brain along the floor of the fourth ventricle in the rostral medulla and caudal pons, the efferent limb of the bradycardic response being the vagus nerve. The observed cardiovascular disorders reverse on releasing the negative pressure or by filling the ventricles with saline solution after decompression in hydrocephalus. In our study, we observed no
correlation between HR and ICP values, which would indicate the contribution of other factors in the decrease in HR. Application of a strong negative pressure to the drainage system, reduction of the brain bulk at the end of craniotomy (provoked by the use of mannitol, hyperventilation, CSF leakage during surgery and tumour resection), administration of β blockers and stretching on the dura mater may lead to important haemodynamic disturbances with subsequent clinical consequences.5 10 14 15

In our study, the use of an extradural drain connected to a vacuum system after craniotomy had no clinically significant adverse effects. However, there exists the possibility of an important decrease in ICP which could provoke severe cardiovascular consequences. Therefore, haemodynamic variables must be monitored closely and controlled at the time of applying negative pressure to the drain. If severe bradycardia occurs, it should be managed promptly with anticholinergic agents and closure of the drainage system.

References

1. Hoff JT, Reis DJ. Localization of regions mediating the Cush-}
2. Doba N, Reis D. Localization within the lower brain stem of a recep-
     tive area mediating the pressor response to increased intracranial pressure (the Cush-}
3. Korwica Z, Brzezinski J. Chronic subdural haematoma treated
     by burr holes and closed system drainage: personal experience
4. Toledo E, Eynan N, Shalit M. Intracranial hypotension—An
     iatrogenic complication of vacuum drainage systems. Acta Neu-
5. McCulloch GAJ, Pattinson WJ. Circulatory changes caused
     by a closed, negative pressure drainage system after crani-
6. Wasnick JD, Lien CA, Rubin LA, Fraser RAR. Unexplained
     bradycardia during craniotomy closure: the role of intracranial
7. Gilsanz FJ, Vaquero J, Lora-Tamayo JJ, Álvarez J, Martínez R. Drene-
8. Hernández-Palázón J, Sánchez-Ortega JL, Tortosa JA, Moya-
     Solera J, López F. Hipotensión intracraneal y drenaje epidural
9. Jacka M, Wood G. Bradycardia related to sudden decreases in
     intracranial pressure during craniotomy. Anesthesia and Anal-
10. Laurendon VG, MacFarlane M, Davis FM. Negative pressure
11. Bahl C, Wadwa S. Cardiovascular collapse after rapid
12. Alfery DD, Saphiro HM, Gagnon RL. Cardiac arrest follow-
     ing rapid drainage of cerebrospinal fluid in a patient with
13. Smith McK, Ray CT. Cardiac arrhythmias, increased
     intracranial pressure, and autonomic nervous system. Chest
     1972; 61: 125–133.
15. Hopkins CS. Bradycardia during neurosurgery—a new reflex?