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Repetitive acute shock following tracheal extubations after neurosurgery for a cerebellar tumor

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To the Editor: We present a case of repetitive acute shock following tracheal extubations after neurosurgery for a cerebellar tumor; there was no severe hypotensive episode during the surgery and the patient was quite alert before the extubations.

A 32-year-old man was operated for excision of a cerebellar astrocytoma. Anesthesia was maintained with nitrous oxide, propofol, and fentanyl. There were no episodes of hypotension (>30% decrease or <90 mmHg systolic blood pressure) or bradycardia (>30% decrease in heart rate within 5 min or <60 beats·min^{-1}) during the operation. The cerebellar tumor, adjacent to the dorsal pons and the rostral dorsal medulla, was resected. Spontaneous respiration resumed 20 min after the termination of the operation, when the oxygen saturation of the peripheral artery (S_{PO_2}) was 100% (when the fraction of inspiratory oxygen [$F_{I_{O_2}}$] was 0.3) and the partial pressure of end-tidal carbon dioxide ($P_{ET_{CO_2}}$) was 40 mmHg. The patient spontaneously opened his eyes and gripped and opened his hand according to verbal command. Ulnar nerve stimulation on his hand showed a train-of-four ratio of 1.0. He coughed strongly when a suction catheter was inserted into the trachea. The tracheal tube was removed. After the extubation, his respiratory pattern showed tracheal tag. The S_{PO_2} was above 98% during the first 3 min after the extubation, and decreased to 77% in the following 2 min. The systolic blood pressure rapidly dropped from 140 mmHg to 40 mmHg before the S_{PO_2} decreased below 90%. His heart rate did not change significantly, until ephedrine was administered. There were no abnormalities in the monitored ECG. Tracheal intubation was performed 5 min after the extubation. The initial $P_{ET_{CO_2}}$ after the re-intubation was 60 mmHg. After positive pressure ventilation for 3 min, sufficient spontaneous respiration resumed, when he spontaneously opened his eyes and gripped and opened his hand according to verbal command. He was transferred to the intensive care unit (ICU). On the third postoperative day, a similar hypotensive episode occurred after tracheal extubation.

Further neurological examination revealed that the patient had incomplete bulbar palsy; this was determined from the observation of motor weakness in protrusion of his tongue and in swallowing. A head-up tilt test on the seventh postoperative day revealed that his cardiovascular sympathetic activity was disturbed (Fig. 1). Tracheotomy was performed on the eighth postoperative day. He did not show any episodes of severe hypotension thereafter and was discharged from the ICU on postoperative day 12.

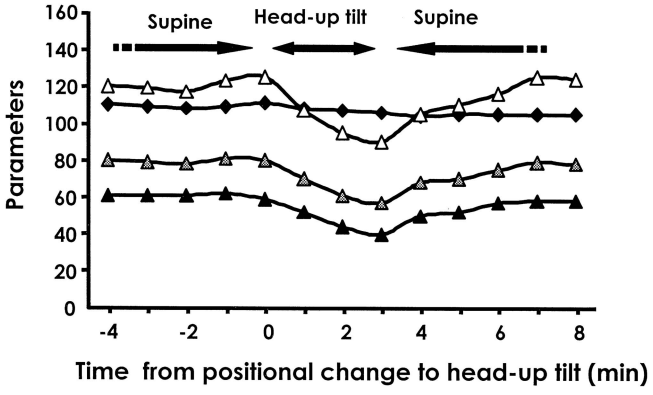

Fig. 1. Head-up tilt test on the seventh postoperative day. Reverse Trendelenburg position of 30° caused a significant fall of the arterial blood pressure (systolic blood pressure decrease >30 mmHg and diastolic blood pressure decrease >15 mmHg), with little change in the heart rate (<10 beats·min^{-1}), which suggested neurogenic orthostatic hypotension. Fluid balance before the test was +70 ml per day and the patient's central venous pressure before the test was 6 mmHg. The patient received low-dose dopamine (1 μg·kg^{-1}·min^{-1}) before and during the test. He did not receive any other vasoactive agents or any sedative agents. *Closed diamonds* show heart rate (beat·min^{-1}); *open triangles* show systolic arterial blood pressure (mmHg); *closed triangles* show diastolic arterial blood pressure (mmHg); and *gray stippled triangles* show mean arterial blood pressure (mmHg)

The brainstem plays a key role in controlling cardiovascular sympathetic activity [1]. Thus, postoperative edema of the brainstem can cause postoperative hypotension [2]. Cerebellar swelling in the posterior fossa, which could be exaggerated with hypercapnia, could also lead to compression of the brainstem. The head-up tilt test in the present patient suggests that modest sympathetic dysfunction, which was caused by surgical damage, enhanced the hypotension. There was another possibility: that hypoxemia and hypercapnia might show a local vasodilatory effect and a direct inhibitory effect on myocardial contractility [3,4].

Whatever the cause of the hypotension in the present patient, his postoperative respiratory pattern suggests that the hypoxia and the hypercapnia were caused by upper airway obstruction, due to bulbar palsy, which was caused by surgical damage. Indeed, no episodes of respiratory failure or severe hypotension were seen after the tracheotomy. It is important for management after neurosurgery in the posterior fossa to evaluate bulbar palsies and autonomic functions. Tracheotomy was very effective to prevent severe hypotensive episodes in this patient.

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