Postoperative hydrocephalus may develop after posterior fossa tumor surgery due to obstruction of cerebrospinal fluid (CSF) pathways caused by the residual tumor or surrounding edema. Placement of a permanent shunt for CSF diversion remains debatable as the hydrocephalus may spontaneously regress over a short period. Alteration of consciousness, headache, vomiting, and ataxia are the common manifestations of hydrocephalus. In this report, we describe a patient in whom brainstem reflexes were impaired due to hydrocephalus following excision of a trigeminal Schwannoma, which resulted in delayed weaning from mechanical ventilation. The problem resolved with the placement of a ventriculoperitoneal shunt.

CASE REPORT
A 55-old man presented with a history of diminished hearing in the right ear for 20 years and right facial pain for three months. He had no history suggestive of raised intracranial pressure, or cranial nerve, motor, sensory or cerebellar dysfunction. His general examination, hematological and biochemical parameters were within normal limits. A magnetic resonance scan showed a Schwannoma in the region of Meckel’s cave extending into the cerebellopontine angle, and compressing the brainstem. There was no evidence of hydrocephalus. He underwent a right retromastoid suboccipital craniectomy and excision of the tumor; the tumor was noted to cause displacement of the VII, VIII, IX and X cranial nerves and the brainstem. Postoperatively, the patient developed pupillary asymmetry and worsening of the level of consciousness on the same day. A computed tomographic scan showed a haematoma in the tumor bed compressing on the midbrain and pons with evidence of obstructive hydrocephalus and blood in the occipital horn of the right lateral ventricle. The haematoma was evacuated by surgery and an external ventricular drain was instituted, following which, his level of consciousness improved significantly. On the third day after shunt, the patient was alert, could be fed orally with the tracheostomy in place. His cough reflex was good to expel his secretions and he could be ambulated with support. The patient could be successfully weaned off mechanical ventilation and the tracheostomy was closed uneventfully 10 days after the shunt. Five days after decannulation, the patient was feeding normally and had a good cough and gag reflex.

DISCUSSION
The striking attribute of the present patient is the dramatic improvement of the lower cranial nerve function following a CSF diversion procedure after a protracted clinical course marked by repeated failures at weaning from mechanical ventilation. Postoperative lower cranial nerve dysfunction may develop after posterior fossa tumor surgery due to intraoperative injury to the brainstem or the nerves themselves. Improvement of the function of these nerves may take several weeks and difficulties may be encountered in extubating the trachea in these patients. A Glasgow Coma Scale (GCS) score > 7 at time of intubation (odds ratio, 4.8;
95% confidence interval, 1.2-21.7) and absence of brainstem deficits (odds ratio, 4.3; 95% confidence interval, 1.3-16.7) are independently associated with successful extubation in patients undergoing surgery for infratentorial lesions.¹

It is difficult to explain the mechanism of rapid improvement of airway reflexes following a ventriculoperitoneal shunt after a protracted clinical course in our patient. The shunt might have relieved the stretch on the lower cranial nerves caused by tonsillar herniation secondary to hydrocephalus, a phenomenon that has been documented in patients with Arnold-Chiari malformation.² Alternatively, ventriculoperitoneal shunt placement possibly improved the perfusion pressure of the brainstem. Severe backward kinking of the brain-stem in the upper cervical region has been recorded in children dying with an acute swelling of the brain in a postmortem study.³ A similar mechanism is possible in our patient also who had hydrocephalus.

Association between swallowing function and supratentorial pathology has been reported in stroke subjects with involvement of internal capsule, primary somatosensory, motor, and motor supplementary areas, orbitofrontal cortex, putamen, caudate, basal ganglia.⁴ These manifestations seem to originate from the involvement of cortico-bulbar fibres. In our patient, raised ICP due to hydrocephalus might have caused functional impairment of any or some of these supratentorial structures, which was relieved by CSF diversion.

The forgoing publications indicate that supratentorial pathology (hydrocephalus in our patient) could rarely be associated with dysfunction of swallowing and airway reflexes. It may be postulated that an early shunt could have shortened the duration of mechanical ventilation and ventilatory weaning. But at present, there are no strong data to predict that such an improvement will occur with certainty. The risks of routine placement of shunt in all similar patients are by no means negligible.

In summary, hydrocephalus could be a possible cause of lower cranial nerve dysfunction in patients with cerebello-pontine angle tumors. CSF diversion might facilitate an early tracheal extubation. Future studies could investigate the relationship between postoperative hydrocephalus and impairment of airway reflexes, which is independent of injury to the lower cranial nerves.

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