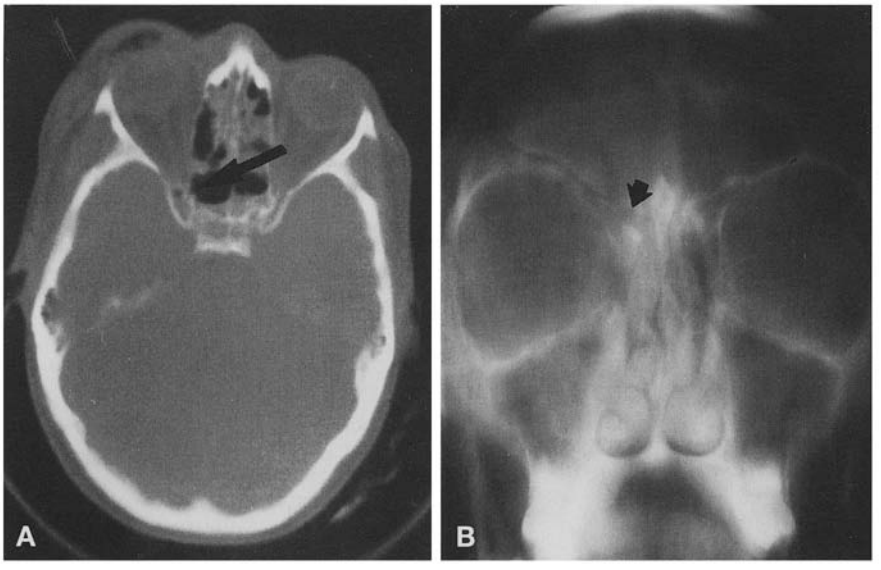

Fig. 1 **A** Initial computed tomography after the previous head injury shows the fracture of the sphenoid sinus *black arrow* and a small associated intrasinusoidal hematoma. **B** Conventional tomography demonstrates the large right sided skull fracture, particularly the fracture of the frontal sinus wall *black arrow*

The present case is of particular interest because of the spontaneous remission of the CSF rhinorrhea after SHT without the occurrence of meningitis over the following years. Delayed complications (meningitis or CSF rhinorrhea) from ethmoid or skull base fractures are known to occur months or years after the initial trauma, with an incidence of 9.3 to 36 % [2], but an onset after more than 10 years without any neurological complaints is extremely rare. The mechanism of reopening the fistula in the present case may have been due to the delivery with the transient elevated intracranial pressure and the consequent change in brain tissue or meningeal compliance [3]. Surgical occlusion of the frontobasal dural leak, if performed after the initially reported rhinorrhea several years before, could have prevented this patient from experiencing the delayed reopening of the fistula under elevated intracranial pressure.

With the availability of different noninvasive imaging techniques, which allow an accurate localization of the dural leak, such as coronal high-resolution computed tomography (HR-CT) [4], magnetic resonance imaging (MRI), and provoking positional MRI [5], the present case emphasizes the need for a standardized diagnostic protocol. For example, after spontaneous remission of CSF rhinorrhea the dural lesion should be identified, first by HR-CT, and, if negative, by MRI, and finally by provoking positional MRI. Detailed diagnosis combined with new methods in microsurgical techniques with their known low surgical mortality and morbidity may lower restraints against (neuro-) surgical procedures. Therefore, surgical occlusion of the dural leak should be considered even after spontaneous remission of the CSF rhinorrhea.

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Two identical episodes of acute quadriparesia in an intensive care unit patient

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Sir: Neuromuscular complications in intensive care patients [1] are mainly the critical illness polyneuropathy [2] and the acute myopathy that follows administration of neuromuscular blocking agents (NMBA) and corticosteroids (CS) [3].

We report a 48-year-old woman suffering from chronic obstructive pulmonary disease who was hospitalized twice for acute respiratory failure over an 18-month period. On the first admission, she received etomidate 20 mg, fentanyl 75 mg, and succinylcholine (SCh) 100 mg for initiation of mechanical ventilation. Sedation consisted of midazolam and morphine. Severe bronchoconstriction followed despite the administration of intravenous salbutamol up to 20 μg/min and prednisolone 300 mg/day (total dose 4800 mg) before extubation). Pancuronium was also administered (total dose 130 mg over 48 h). Mechanical ventilation was required for 19 days, during which there were no signs of renal failure, septic shock, or multiple organ dysfunction. Creatine kinase (CK) levels rose from normal values on admission to 1000 U/l (normal 25–140 U/l) during the first 48 h, then decreased to normal over 23 days. After extubation, symmetrical quadriparesia was present, without sensory deficit. Nerve conduction studies revealed normal sensory nerve conduction and a marked decrease in the amplitude of the compound muscle action potentials and electromyography showed diffuse fibrillation potentials. The patient recovered normal strength over a

3-month period. Eighteen months later, she was admitted for the same symptoms. Prednisolone (total dose 4725 mg before extubation), etomidate 20 mg, and SCh 150 mg were administered. Pancuronium was not given. A transient elevation of CK (280 U/l) was noted on the 3rd day. She was extubated after 11 days. Again, quadriparesia was present, associated with identical electrophysiologic findings. Needle muscles biopsies revealed fiber anisometry, atrophy, necrosis, and myophagism. The patient recovered normal strength over a 2-month period.

This patient presented two episodes of severe quadriparesia, increased CK, electrophysiologic signs of myopathy, and histologic myopathic changes. These findings are those of an acute intensive care myopathy that follows administration of NMBA and CS [3], different from those of polyneuropathy in critical illness [2]. Even if such polyneuropathy can present with pure motor axonal degeneration, recuperation was too rapid in this patient to be compatible with motor axonal degeneration. The first episode can be attributed to both pancuronium and CS. The second is possibly due to CS. However, the causative role of CS alone may be questioned. The first description of acute CS myopathy is attributed to MacFarlane and Rosenthal [4], but their patient, as most other cases reported in the literature, received both CS and pancuronium. Moreover, acute CS myopathy never follows high doses of CS given in other conditions, such as treatment of multiple sclerosis or spinal trauma. Thus, acute CS myopathy is probably extremely rare.

Alternative explanations for the second episode are: (1) a role for SCh alone, as suggested by Karoubi et al. [5], or in conjunction with CS, (2) sensitization of the muscle to the toxic effect of CS following the first episode, (3) another unknown factor in relation to respiratory failure, such as hypoxia or systemic inflammatory response.

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