on chronic diuretic therapy, the patient did not have any electrolyte disorder, which may predispose to neurologic dysfunction.

The patient was on chronic dexamethasone and oestradiol therapy; and chronic steroid therapy may be associated with neuropathy [6]. Although difficult to prove, this may have been a contributory factor to the myoclonus in this case report. Another possible contributory factor to the myoclonus in this patient is the vitamin $B_{12}$ deficiency that was reported. Vitamin $B_{12}$ deficiency is associated with myelopathy and neuropathy [7]. However, the degree of deficiency in our patient was mild and the patient did not require vitamin $B_{12}$ therapy for permanent resolution of myoclonus. Lower limb neuropathy after spinal anaesthesia has been reported in a patient with thiamine deficiency [8], but our patient had a normal thiamine level.

The treatment of spinal myoclonus includes detection of the aetiology, abolition or minimization of the aetiology, and symptomatic treatment with benzodiazepines, baclofen or anticonvulsants [3,4,5]. Benzodiazepines are effective and the mainstay of treatment. Diazepam and clonazepam have been reportedly used successfully [3,5]. Midazolam was used for treatment in our patient because it was readily available and in ready-to-use injectable form. It is very potent, of rapid-onset, painless on injection and relatively short acting. Thus, we believe that midazolam is the benzodiazepine of choice for treating perioperative spinal myoclonus. Intrathecal baclofen is effective therapy for spinal myoclonus [2,3], but was not attractive to our team and our patient. Anticonvulsants such as carbamazepine and sodium valproate are also effective [5].

In conclusion, spinal myoclonus following spinal anaesthesia is a rare complication with unclear pathophysiology that can be treated effectively with midazolam. Anaesthesiologists and the perioperative team should be aware of this distressing complication, especially in patients with vitamin deficiency or neurological dysfunction. Long-term patient follow-up is important to rule out latent or evolving neuropathy.

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# A potentially fatal complication of postoperative vomiting: Boerhaave's syndrome

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EDITOR:
Herman Boerhaave, a Dutch physician, first described the spontaneous rupture of the oesophagus in 1724. He described a complete transmural laceration of the lower part of the oesophagus with the flow of gastric content into the mediastinum. Such patients may present following forceful protracted vomiting associated with sudden pain in the thorax and epigastrium, which may radiate to the neck or to the back together with progressive dyspnoea, tachypnoea, cyanosis and shock.

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Patients with Boerhaave's syndrome have a poor prognosis: one-third of patients die even when they undergo prompt surgical intervention. In those patients who do not undergo medical treatment, sepsis and shock are causes of death in the first 48 h (90% of patients) or within the next few days (10% of patients). We present a case of postoperative Boerhaave's syndrome with diagnostic delay, active conservative management and a good outcome.

A 62-yr-old female with an unremarkable previous medical history except for mild asthma underwent minimally invasive left total hip replacement for severe osteoarthritis at Pilgrim Hospital, Boston, under General Anaesthesia with propofol induction and tracheal intubation. Anaesthesia was maintained using oxygen, nitrous oxide, desflurane, fentanyl and atracurium. She received 4 mg of ondansetron perioperatively and also received cyclizine and ondansetron postoperatively as required. The patient remained haemodynamically stable throughout the procedure and there was no significant blood loss. Postoperatively she had prolonged periods of vomiting, which were treated with anti-emetics. The significance of this was not recognized at the time. The following day she developed shortness of breath. Electrocardiography showed atrial fibrillation for which she received intravenous digoxin. A chest X-ray was performed, which showed basal collapse of the left lung with pleural effusion, surgical emphysema and pneumomediastinum. A left-sided intercostal chest drain was inserted in the ward and 250 mL of pleural fluid was drained. The pleural fluid grew streptococcus viridans, which was treated with clarithromycin and ciprofloxacin.

She showed signs of deterioration and was admitted to the high dependency unit on the fourth postoperative day. After initial assessment and stabilization, a computer tomography (CT) scan of her chest was performed, which showed a large left-sided hydropneumothorax with the tip of the chest drain in the left chest. There was a smaller right-sided hydropneumothorax and evidence of a pneumomediastinum. Following this scan, a right-sided chest drain was inserted and the left chest drain was replaced.

She continued to drain fluid from both pleural cavities and required intermittent ventilatory support with non-invasive ventilation. A repeat CT scan was taken on the seventh postoperative day, which showed bilateral loculated pleural collections, which were more marked on the right side. The left-sided effusion had decreased in size and the left drain was in a good position. The right-sided effusion had remained the same size as in the previous scan. The right-sided chest drain was in a poor position for drainage of the effusion and was replaced. Unfortunately, the insertion of this drain resulted in a liver laceration for which the patient underwent an urgent laparotomy. A right-sided chest drain was inserted during the surgery.

Post laparotomy she remained intubated, ventilated and required inotropic support. The patient was on total parenteral nutrition from the sixth postoperative day. Upper gastrointestinal (GI) endoscopy and bronchoscopy were performed on the twelfth postoperative day and the findings were considered to be normal. Percutaneous tracheostomy was performed on day 12. She was slow to wean from ventilation. Thoracic ultrasound of the chest on the thirteenth day showed a multiloculated pleural effusion on the right side. Ultrasound-guided drainage was unsuccessful. CT scan on the fifteenth day showed bilateral pleural effusions, a good position of the right chest drain but the left-sided chest drain was seen to be crossing the midline behind the oesophagus. This was withdrawn by 5 cm.

On day 15 nasogastric feed was commenced. Almost immediately the feed was noted in the left chest drain, suggestive of an oesophagopleural fistula. Methylene blue solution was then given through the nasogastric tube and appeared in both of the chest drains. Expert cardiothoracic advice was sought and conservative management was recommended. The patient was weaned from respiratory support and her tracheostomy was removed. Two days later she was reintubated and ventilated after becoming acutely tachypnoeic and hypoxic following an episode of bronchospasm. She remained on respiratory support and had her tracheostomy tract recannulated. Free nasogastric drainage and parenteral nutrition was continued. She was then weaned from her ventilatory support. Confirmation of an oesophageal tear was achieved with a gastrografin swallow 1 month after the hip surgery.

The patient was referred to a tertiary centre for further management. After moving to the tertiary hospital, upper GI endoscopy and triple lumen nasogastric tube insertion was attempted but failed. Two days later, jejunostomy was performed through a left upper quadrant abdominal incision. The right-sided chest drain was removed during this procedure. Enteral feeding was started on the same day through the jejunostomy tube. A contrast swallow test was repeated 53 days after the hip surgery. This showed no further leakage and a barium meal ruled out gastric outlet obstruction. The next day, a chest X-ray was taken, which showed a small left pleural effusion that was drained under ultrasound guidance. Exactly 2 months after total hip replacement, jejunal feeding was stopped

and on day 62 after her hip replacement she was discharged home eating and drinking normally.

Boerhaave's syndrome is a rare but potentially life-threatening condition resulting from a spontaneous transmural oesophageal perforation usually after an effort of vomiting. In most cases, patients are men aged around 50, with a past medical history of alcohol intoxication or a neurological condition. The Mallory–Weiss syndrome, a similar condition, is differentiated from Boerhaave syndrome by nontransmural involvement. The pathophysiology of Boerhaave's syndrome involves barogenic injury to the oesophagus resulting from the huge increase in intramural pressure during vomiting against a closed cricopharyngeus. A disorganized vomiting reflex in which the upper oesophageal sphincter fails to relax during vomiting leads to an increase in oesophageal pressure. Other conditions such as straining, blunt trauma, seizures and asthma have also been reported as causes of oesophageal rupture.

The most common site of the tear in Boerhaave syndrome is the left posterior lateral wall of the lower third of the oesophagus with leakage into the left pleural cavity. This site is thought to be due to the lack of striated muscle and a vertical arrangement of longitudinal muscles in the lower oesophagus (anatomical zone of weakness). Though these tears are considered to be spontaneous, it is usually because of its frequent association with vomiting that helps in differentiating the iatrogenic, foreign body and traumatic perforations that are not included in Boerhaave's syndrome.

The diagnosis of Boerhaave's syndrome is often missed or delayed because of its similarity of presentation to many of the more common thoracic pathologies, e.g. pneumonia, myocardial infarction, pneumothorax, empyema and subphrenic abscess. The classic triad required for the diagnosis of Boerhaave's syndrome, i.e. vomiting, chest pain and subcutaneous emphysema (Mackler's triad), is seldom found. Usually, patients present with diverse signs and symptoms, frequently central chest pain, epigastric pain, vomiting, dyspnoea, hematemesis, respiratory distress, fever, subcutaneous emphysema and sepsis, all of which make early diagnosis difficult. In several patients with Boerhaave's syndrome, the diagnosis has been reached on the basis of elevated pleural fluid amylase, non-responsiveness of an empyema to standard treatment, a contrast study for dysphagia and even on postmortem findings.

In one series of 47 patients, only 21% were correctly diagnosed within 12 h of presentation [1]. It is manifestly important that an early diagnosis is made as immediate operative intervention is the treatment of first choice but the technique of bypassing the oesophagus via a feeding jejunostomy is recommended in patients who are diagnosed late. Cameron and colleagues [2] reported two cases of spontaneous oesophageal rupture managed successfully without surgery, but they are all reported to have had minimal symptoms. Pate and colleagues [3] reported four cases over a 30-yr period. All were managed conservatively, without surgery, and all patients died. To our knowledge, this is the first case of Boerhaave's syndrome with free pleural rupture, extensive pneumomediastinum and florid sepsis where the patient has survived without surgical intervention.

This case is relevant to anaesthetic practice because postoperative vomiting, while common after general anaesthesia, might rarely produce life-threatening conditions such as Boerhaave's syndrome. Awareness of this atypical presenting syndrome is important, as a failure to appreciate its significance could be fatal if not diagnosed early. Advocating prompt surgical intervention or delayed non-operative management should be individualized depending on the patient's overall clinical picture.

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