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Peripheral Tapia's syndrome after cardiac surgery

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Editor,

Tapia's syndrome, first described by the Spanish otorhinolaryngologist A.G. Tapia in 1904, includes two distinct conditions, differing in clinical manifestation and pathophysiological mechanism. The central Tapia's syndrome is a typical crossed brainstem syndrome caused by a lesion of nucleus ambiguus, nucleus of hypoglossal nerve and the pyramidal tract.[1] The peripheral Tapia's syndrome is due to an extracranial lesion of the hypoglossal nerve and recurrent laryngeal branch of the vagal nerve that can be provoked by tumours, head and neck operations, radiation therapy, meningitis, infectious mononucleosis and carotid artery dissection. The latter has been described, in a few cases, as a complication of oropharyngeal manipulation during anaesthetic airway management. Indeed, it has been demonstrated that the anterior displacement of the tongue during laryngoscopy may cause a substantial strain on the hypoglossal nerve. Moreover, reclination of the head, as performed in the 'sniffing position' during laryngoscopy, may affect distension of the nerve as well.[2] Up to now, only one case of peripheral Tapia's syndrome following oropharyngeal manipulation during cardiac surgery has been reported, in particular after coronary artery bypass.[3]

We now describe another case of peripheral Tapia's syndrome with unilateral hypoglossal and recurrent laryngeal nerve palsy occurring after uncomplicated orotracheal intubation during cardiac surgery.

A 72-year-old man, with arterial hypertension, ulcerative rectocolitis and aortic valvular disease, was scheduled for aortic valve replacement. He was on angiotensin-converting enzyme (ACE) inhibitors, β-blockers and calcium antagonists.

Anaesthesia was induced using a combination of thiopentone 375 mg intravenously (i.v.) and sufentanil 25 μg i.v., and muscle relaxation was facilitated with vecuronium 8 mg i.v. Laryngoscopy was performed by means of a stainless steel Macintosh blade size 4 (Medicon; Tuttlingen, Germany), and transoral intubation was carried out with a 9 mm endotracheal tube. No complications were encountered during laryngoscopy and intubation. The cuff of the endotracheal tube was inflated to a pressure of 20 cmH_2O. The tube was positioned in the right side of the mouth. A nasogastric tube (PVC, Levin duodenal tube, size Ch.16, Rüsch) was placed through the left nostril. General anaesthesia was maintained with sevoflurane in oxygen–air and sufentanil. After midline sternotomy, before the beginning of the extracorporeal circulation, i.v. heparin (3 $mg\,kg^{-1}$ at a total dose of 210 mg) was administered. An uncomplicated operation was performed, and the patient was then transferred to the ICU and extubated after 25 h. After 72 h in the ICU, he was transferred to the cardiac surgery ward in good general condition.

In the next few hours, the patient complained of dyspnoea and dysphagia. Neurological examination revealed paralysis of the muscles of the left part of the tongue causing left-sided deviation with protrusion, consequent dysphagia, and dysphonia and hoarseness, suggesting the occurrence of Tapia's syndrome. Methyl-prednisolone 200 mg was given i.v., and the patient was then switched to oral therapy for 10 days. Laryngoscopy showed severe paresis of the left hemilarynx, with saliva stagnancy in the piriform sinus and normal motility of the right hemilarynx. Moreover, a haematoma of the left posterior side of the rhinopharynx was observed. A computed tomography (CT) and MRI scan did not reveal any lesion involving the brain or the neck. The patient was able to breathe adequately, but he needed a restricted diet in order to reduce the risk of aspiration. One week later, on otorhinolaryngological examination, the size of the haematoma was decreased and the movement of the left vocal cord was starting to recover. A needle electromyography (EMG) performed 4 weeks after the onset of symptoms showed fibrillation and positive sharp waves in the muscles of the left side of the tongue, which appeared hypotrophic. Three months later, the patient was re-evaluated: the neurological examination showed a marked improvement of signs and symptoms, with the disappearance of dysphonia; a minimal left-sided deviation of the tongue with protrusion was still observed. The needle EMG revealed sporadic fibrillation in the muscles of the left part of the tongue, with a voluntary pattern recruitment characterized by polyphasic, low-amplitude regenerant motor unit potentials.

The peripheral Tapia's syndrome is a rare complication of surgery, the pathophysiological mechanism of which is often a consequence of orotracheal intubation. In the case reported here, unilateral paralysis of the muscles of the

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tongue and of the ipsilateral vocal cord was due to a lesion of the 10th and 12th cranial nerves occurring after oropharyngeal manipulation during general anaesthesia for aortic valve replacement surgery. It has been proposed that, depending on the extent of the exerted strain, the nerve function may be temporarily impaired due to stretching (i.e. neuroapraxia) or the nerve may be permanently damaged due to extraction of its fibers (i.e. axonotmesis or neurotmesis). We assume that, in this case, a lesion of the nerves was probably caused by the rhinopharyngeal haematoma due to the placement of the nasogastric tube and worsened by heparin therapy. The haematoma was located in the pharynx along the nasogastric tube just at the crossing of the vagus and the hypoglossal nerve (which run on the lateral prominence of the anterior surface of the transverse process of C1).[4] Additionally, reclination of the head, as performed in the sniffing position during general anaesthesia, might have affected distension of the nerve as well. To our knowledge, there are only four reports on peripheral unilateral Tapia's syndrome after transoral intubation for general anaesthesia and only one after cardiac surgery. Yavuzer *et al.*[5] described a case of Tapia's syndrome after septorhinoplasty as a consequence of the compression of the nerve after inflation of the cuff within the larynx. Tesei *et al.*[4] presented a unilateral Tapia's syndrome following endotracheal intubation after rhinoplasty under general anaesthesia. They believed that compression by the endotracheal tube just at the crossing of the vagus and hypoglossal nerve and the hyperextension of the neck were able to cause a pressure neuropathy.[4] Boisseau *et al.*[6] reported a case of Tapia's syndrome after general anaesthesia following shoulder surgery. The syndrome in their case was believed to be due to a compression by the endotracheal tube, caused by the displacement of the head.[6] Johnson and Moore[7] showed a case of paralysis of the 10th and 12th cranial nerves' after interscalene brachial plexus block. Tapia's syndrome has also been described in cardiac surgery by Sotiriou *et al.*[3] as a postoperative complication of coronary artery bypass grafting (CABG) surgery. They observed a 52-year-old woman who developed airway obstruction each time extubation was attempted. A neurological examination revealed bilateral hypoglossal and recurrent laryngeal nerve palsy. The patient was unable to move the tongue and had pooled secretions in the oropharynx. Electromyographic examination showed denervation of the tongue. The authors hyphothesized that inadvertent hyperextension and lateral flexion of the neck during sternotomy and endotracheal tube malposition had occurred, leading to compression at the crossing point of the vagal and hypoglossal nerve.[3]

Although all the above-mentioned mechanisms proposed for Tapia's syndrome cases in the literature have to be considered, in our experience nasogastric tube placement played a central role by causing the rhinopharyngeal haematoma. In our opinion, heparin therapy, as a main step in the management of patients undergoing cardiac surgery, could have reasonably exacerbated the haematoma, leading to delayed symptoms. In conclusion, we describe here another cause of Tapia's syndrome related to airway management procedures and recommend careful manipulation of nasogastric tubes in anticoagulated patients.

References

1 Krasnianski M, Neudecker S, Schluter A, *et al.* Central Tapia's syndrome ('matador's disease') caused by metastatic hemangiosarcoma. *Neurology* 2003; **61**:868–869.

2 Dziewas R, Ludemann P. Hypoglossal nerve palsy as complication of oral intubation, bronchoscopy and use of the laryngeal mask airway. *Eur Neurol* 2002; **47**:239–243.

3 Sotiriou K, Balanika M, Anagnostopoulou S, *et al.* Postoperative airway obstruction due to Tapia's syndrome after coronary artery bypass grafting surgery. *Eur J Anaesthesiol* 2007; **24**:377–386.

4 Tesei F, Povera LM, Strali W, *et al.* Unilateral laryngeal and hypoglossal paralysis (Tapia's syndrome) following rhinoplasty in general anaesthesia: case report and review of the literature. *Acta Otorhinolaryngol Ital* 2006; **26**:219–221.

5 Yavuzer R, Basterizi Y, Ozkose Z, *et al.* Tapia's syndrome following septorhinoplasty. *Aesthetic Plast Surg* 2004; **28**:208–211.

6 Boisseau N, Rabarijaona H, Grimaud D, Raucoules-Aimė M. Tapia's syndrome following shoulder surgery. *Br J Anaesth* 2002; **88**:869–870.

7 Johnson T, Moore H. Cranial nerve X and XII paralysis (Tapia's syndrome) after an interscalene brachial plexus block for a left shoulder Mumford procedure. *Anesthesiology* 1999; **90**:311–312.

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Dissimulation in patients with myotonic dystrophy

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Editor,

Performing general anaesthesia in patients with myotonic dystrophy is still a challenge. Some reviews have been published that deal with anaesthetic problems and complications[1,2] and that discuss anaesthetic priorities and procedures.[3–5] Although there remain several open questions, for example the proper choice of a general anaesthetic, most authors agree with the statement made by Hannon *et al.*[6] that recognition of the disorder during the preoperative assessment is the key to avoiding perioperative complications. However, preoperative screening requires the cooperation of the patient. In the following, we report a patient with myotonic dystrophy, which complicated perioperative care because the patient had disregarded her disease.

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A 60-year-old woman presented in our anaesthesiology outpatient clinic for preoperative assessment for laparoscopic cholecystectomy. She reported a medical history of thyroidectomy, cataract surgery and hysterectomy without any complications. She mentioned that, after cataract surgery, she was thought to have myotonic dystrophy, although the diagnosis had never been confirmed. On explicit questioning by the anaesthesiologist, the patient stated that she had an unimpaired exercise tolerance. Thyronajodid was her sole long-term medication and she was classified as having American Society of Anesthesiologists II (ASA II) status. On the day of surgery, she was questioned by the anaesthesiologist and, again, she denied having myotonic dystrophy. Subsequently, anaesthesia was induced with sufentanil (25 μg), propofol (150 mg) and rocuronium (30 mg). During laryngoscopy, unusual jaw stiffness was observed. General anaesthesia was continued by total intravenous anaesthesia with propofol ($4\,mg^{-1}\,kg^{-1}\,h^{-1}$) and repeated boluses of sufentanil (35 μg). Twenty minutes before the end of the 90 min surgery period, the patient received 7.5 mg piritramide. Recovery from anaesthesia was prolonged, though relaxometry demonstrated a complete recovery from muscle relaxation. After 40 min, the trachea was extubated and the patient was taken to the recovery unit. There she appeared to be disorientated and agitated, making monitoring of her vital parameters and preventing her from falling out of bed almost impossible. Pulse oximetry revealed a desaturation of 80% and the ECG showed a new atrial fibrillation of approximately 130 beats min^{-1}. The analysis of heart enzymes, 12-channel ECG and chest radiograph demonstrated no pathological results. As desaturation persisted and the patient required noninvasive ventilatory support, she was transferred to the ICU for one night, where she recovered and from where she was discharged to the surgery ward the following day. Contacting her general practitioner and her husband, it emerged that the diagnosis of myotonic dystrophy had been confirmed by genetic analysis in 1998, with her father and uncle also being affected. The patient's first symptoms of muscle weakness occurred at the same time. In 2004, her exercise tolerance was limited to 500 m of walking and she had developed a severe mental depression, which had required neuropsychiatric rehabilitation. The patient was, in fact, mostly wheelchair-dependent, but she had completely disregarded her symptoms. Medical reports concerning her poor physical condition and history were not available. On being confronted with this information, the patient still denied any physical restrictions. Hence, after consultation with neurologists, the patient was recommended for further neuropsychiatric rehabilitation.

Myotonic dystrophies are dominantly inherited disorders characterized by muscle weakness and atrophy, myotonia and endocrine effects such as cataract, or hypogonadism, together with involvement of different organs, including the brain.[7] Anaesthetic problems in patients with myotonic dystrophy are frequent. The complication rate ranges between 52%[2] and 8%.[1] However, typical complications have been reported to be pulmonary, including acute ventilatory failure, atelectasis and pneumonia, as well as cardiological, such as first-degree heart block and other rhythm disturbances.[2] So far, the problems described in this case report seem to be typical in patients with myotonic dystrophy. Owing to the increased risk of perioperative pulmonary complications, careful monitoring during the postoperative period, chest physiotherapy and protection of the upper airways are recommended in patients with severe muscular disability or those who have undergone upper abdominal surgery.[1] The problem complicating perioperative care in our patient was that she had ignored the diagnosis and the progress of the disease. As there have been some reports describing perioperative complications unmasking the diagnosis of myotonic dystrophy,[1,2,6] we would like to raise the question of whether anaesthesiologists should anticipate that patients with myotonic dystrophy tend to conceal their disease deliberately. Recently published literature in neurology focused on the cerebral involvement in myotonic dystrophies. Meola and Sansone[7] reported that patients minimize their symptoms, do not keep outpatient appointments and seem unconcerned about their health. The authors characterized the clinical impression of patients with myotonic dystrophy to be one of apathy, decreased emotional participation and psychomotor delay. Neuropsychiatric interviews demonstrated an avoidant trait personality disorder.[8] Investigating psychopathological and emotional deficits in these patients, Bungener *et al.*[9] measured low scores for expressiveness and high scores for anhedonia. They hypothesized that this emotional deficit may be either an adaptive reaction to the disease or the effect of CNS lesions, which occur with myotonic dystrophy, or both.[9]

In summary, we want to point out that patients with myotonic dystrophy are not only compromised by perioperative pulmonary and cardiac complications, but also tend to disregard their physical impairment, thereby impeding adequate anaesthesia and postoperative care. Thus, anaesthesiologists should be very alert if there is any uncertainty in respect of the diagnosis or the progress of the disease, and should contact the patient's general practitioner or relatives rather than relying on the patient's statement.

References

1 Mathieu J, Allard P, Gobeil G, *et al.* Anesthetic and surgical complications in 219 cases of myotonic dystrophy. *Neurology* 1997; **49**:1646–1650.
2 Aldridge LM. Anaesthetic problems in myotonic dystrophy. A case report and review of the Aberdeen experience comprising 48 general anaesthetics in a further 16 patients. *Br J Anaesth* 1985; **57**:1119–1130.
3 Stevens RD. Neuromuscular disorders and anesthesia. *Curr Opin Anaesthesiol* 2001; **14**:693–698.

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4 Driessen JJ. Neuromuscular and mitochondrial disorders: what is relevant to the anaesthesiologist? *Curr Opin Anaesthesiol* 2008; **21**:350–355.
5 Catena V, Del Monte DD, Rubini A, *et al.* Anesthesia and myotonic dystrophy (Steinert's syndrome). The role of total intravenous anesthesia with propofol, cisatracurium and remifentanyl. Case report. *Minerva Anestesiol* 2007; **73**:475–479.
6 Hannon VM, Cunningham AJ, Hutchinson M, McNicholas W. Aspiration pneumonia and coma: an unusual presentation of dystrophic myotonia. *Can Anaesth Soc J* 1986; **33**:803–806.
7 Meola G, Sansone V. Cerebral involvement in myotonic dystrophies. *Muscle Nerve* 2007; **36**:294–306.
8 Meola G, Sansone V, Perani D, *et al.* Executive dysfunction and avoidant personality trait in myotonic dystrophy type 1 (DM-1) and in proximal myotonic myopathy (PROMM/DM-2). *Neuromuscul Disord* 2003; **13**:813–821.
9 Bungener C, Jouvent R, Delaporte C. Psychopathological and emotional deficits in myotonic dystrophy. *J Neurol Neurosurg Psychiatry* 1998; **65**:353–356.

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Spinal anaesthesia and neuromyelitis optica: cause or coincidence?

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Editor,

Subarachnoid anaesthesia is considered to be a well tolerated regional anaesthesia technique. Despite local anaesthetic neurotoxicity, mainly regarding concentrations above those used in clinical practice, previous lesions, such as demyelinating diseases, might lower the threshold for toxicity and show an increased sensitivity to local anaesthetics, which may unmask silent plaques.[1] Neuromyelitis optica (NMO) has, until recently, been considered a variant of multiple sclerosis (MS), but is now considered to be a separate disease with a specific marker (a serum antibody to aquaporin-4 water channels);[2] however, distinguishing between the two may be difficult because there is some overlap between features of NMO and MS. Recently, one case of NMO following subarachnoid anaesthesia was reported, giving rise to concern about a possible cause–effect relationship between the two.[3] In the present article, we report on a further patient who developed a NMO following subarachnoid anaesthesia, an event which raises again the matter of a possible causal relationship between the two.

A 39-year-old woman underwent subarachnoid anaesthesia for caesarean section 5 years before coming to our notice because of a claim. She had had a lesion of the conus medullaris during the postoperative period, followed 6 months later by relapsing NMO. As the patient reported left leg pain on spinal needle insertion and the postoperative MRI had shown a hyperintense lesion at the level of the conus medullaris, the anaesthetist was charged because of the spinal cord lesion and, then, for the NMO. However, a rigorous analysis of records showed the following facts:

(1) The patient's history included previous uneventful subarachnoid anaesthesia for caesarean section, at the age of 30 years.
(2) At the age of 34 years, the patient underwent a second caesarean section. The subarachnoid anaesthesia was performed with a 24-gauge spinal Sprotte needle (Braun, Melsungen, Germany), inserted at the L2–L3 level, and 2 ml of hyperbaric 0.5% bupivacaine was administered. During insertion of the needle, the patient felt a piercing lumbar pain, radiating to the medial side of the left thigh and an electric shock-like sensation in the left leg; the anaesthetist repositioned the spinal needle and completed the injection with no further problems.
(3) On the first postoperative day the patient complained of intense pain and motor impairment of the left leg. The consultant neurologist reported a strong contracture of the quadriceps with normal ankle jerk reflex and no sensory deficit and diagnosed an L3–L4 nerve root lesion.
(4) The clinical picture worsened during the following week. The patient's leg pain was associated with dysaesthesia, motor deficit, loss of tendon and plantar reflexes, deficit of all sensory modalities at the L4–S1 level and disturbed micturition. MRI showed a hyperintense spinal cord lesion, extending from T12 to L1.
(5) Later on, the patient only partially improved from the motor deficit, whereas pain, sensory and micturition deficits persisted. Six months after the operation, she had a left optical neuritis, associated with dysaesthesia and sensory-motor deficit in the right limbs; this was followed, 6 weeks later, by right optic neuritis. MRI disclosed scattered hyperintense areas in the periventricular white matter, the semioval centres and spinal cord from C2 to T2 (Fig. 1). No oligoclonal bands were present; the antibody to aquaporin-4 water channels were not checked, as this test was not available at the time of diagnosis.

The patient currently has bilateral blindness, severe tetraparesis along with tactile and thermal hypoaesthesia and neurogenic bladder.

Central blocks are to be regarded as well tolerated regional anaesthesia techniques, but an increasing number of reports dealing with severe neurological complications have been published.[4,5] The so-called atraumatic needles, with smaller and smaller diameter, have been introduced in an attempt to minimize the risk of postdural puncture headache (PDPH), rather than to reduce

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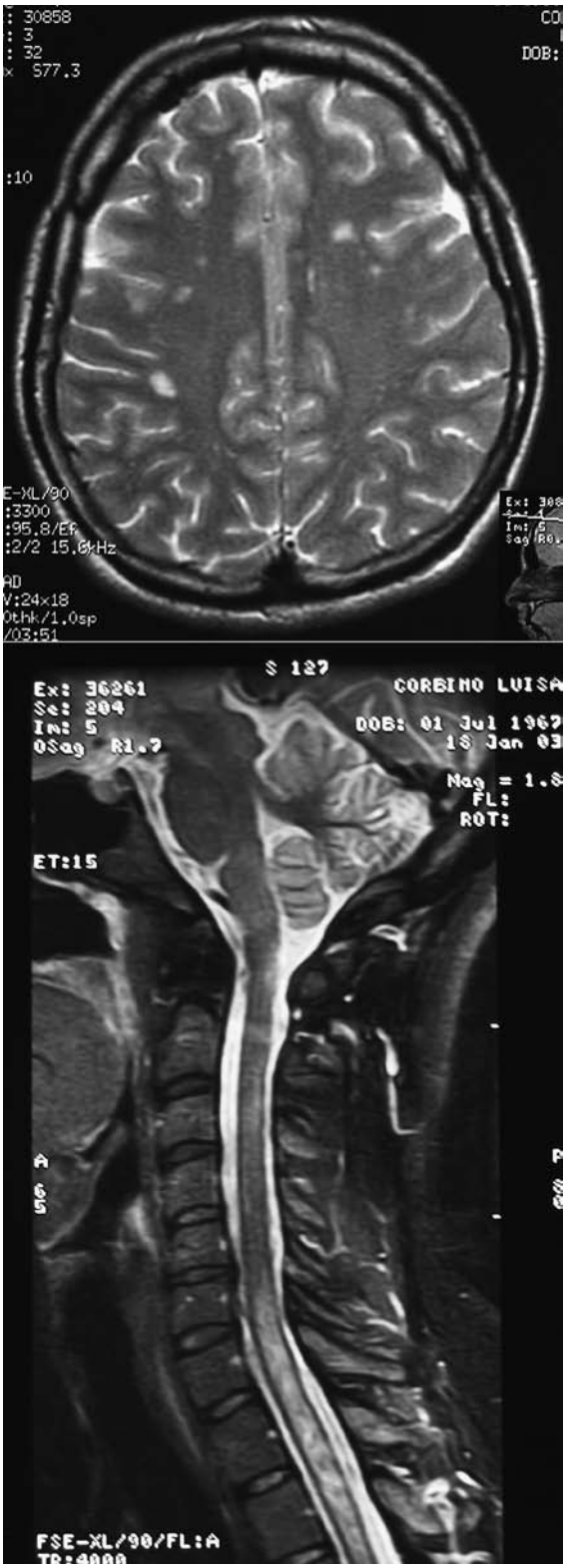
Fig. 1



MRI in a patient who developed a neuromyelitis optica 6 months after caesarean section. Scattered hyperintense areas in the white matter of the hemispheres (top) and in spinal cord from C2 to T2 (bottom) are present.

the risk of neurological complications. These needles have at least a 1 mm blind tip beyond the hole; also, the smaller the diameter, the higher the resistance to cerebrospinal fluid outflow. Both of these may lead to a deeper needle insertion than strictly necessary, increasing the risk of traumatic lesions. When the spinal interspace is a concern, the lack of reliability of the Tuffier's line is now well known.[6] The insertion of the needle at levels up to four times the one intended can occur even in the hands of skilled anaesthetists. Although the potential neurotoxicity of local anaesthetics is usually negligible at the concentrations used in clinical practice, this might give rise to some concern in patients with demyelinating diseases, who are more sensitive to these drugs. In fact, oligopeptides with Na-channel blocking activity have been found in the cerebrospinal fluid of patients with MS.[1] Only one case has been reported in the literature, in which the subarachnoid anaesthesia precipitated the appearance of a silent MS.[7] Moreover, one case of NMO following subarachnoid anaesthesia was reported and the authors considered local anaesthetic toxicity to be the cause.[3] A further aspect to be taken into account in obstetric patients is the postpartum period, which strongly increases the likelihood of spontaneous exacerbation of the disease.[8] Despite patients with MS and NMO showing a high sensitivity to local anaesthetics, regional anaesthesia is not contraindicated; the only suggestion is to carefully check the presence of mild neurological symptoms and prior neurological diseases before performing regional anaesthesia. In our patient, a clear cause–effect relationship between subarachnoid anaesthesia and a conus medullaris lesion was present, but the occurrence of NMO seems to be unrelated to subarachnoid anaesthesia:

(1) The conus medullaris lesion showed that the subarachnoid anaesthesia was performed at least two levels higher than believed.
(2) The neurological deficit progressively worsened during the puerperal period. Despite it being a clear consequence of a spinal cord lesion, it might also have been enhanced by a latent NMO.
(3) The NMO probably underwent a spontaneous worsening as a result of delivery and the puerperal period, leading to the first manifestation of symptoms and, then, to the progressive neurological damage, which finally led to the diagnosis of NMO.

Our patient is a further example of a possible cause–effect relationship between the two. Should a relationship between subarachnoid anaesthesia and NMO be claimed, it would lead to compensation for NMO developed following subarachnoid anaesthesia (as happened in our case), calling for a critical analysis of causes and coincidences. However, Hosseini *et al.*[3] missed the simplest explanation, that is, the unmasking of a latent neurological disease; this remains the most reasonable explanation of NMO developed by our patient. On the

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base of available knowledge it seems simply to be *consecutio temporum*, rather than a cause–effect relationship, in which the ability to unmask neurological deficits appears at the same time as a confounding factor and a tool for early diagnosis. Taking the greatest care to ask the patient for apparently irrelevant neurological symptoms, a full disclosure of risks as well as the correct interpretation of the sequence of events may help to avoid stressful consequences for both the anaesthetist and the patient, who cannot accept the idea of such severe iatrogenic damage; on the contrary, neither sentence nor compensation can be appropriate for spontaneous, latent, coexisting disease.

References

1 Hebl JR, Horlocker TT, Schroeder DR. Neuraxial anesthesia and analgesia in patients with preexisting central nervous system disorders. *Anesth Analg* 2006; **103**:223–228.

2 Weinshenker BG, Wingerchuk DM. Neuromyelitis optica: clinical syndrome and the NMO-IgG autoantibody marker. *Curr Top Microbiol Immunol* 2008; **318**:343–356.

3 Hosseini H, Brugieres P, Degos JD, Cesaro P. Neuromyelitis optica after a spinal anaesthesia with bupivacaine. *Mult Scler* 2003; **9**:526–528.

4 Moen V, Dahlgren N, Irestedt L. Severe neurological complications after central neuraxial blockades in Sweden 1990–1999. *Anesthesiology* 2004; **101**:950–959.

5 Serpell MG. Pencil point spinal needles and neurological damage. *Br J Anaesth* 2002; **89**:800–801.

6 Broadbent CR, Maxwell WB, Ferrie R, *et al.* Ability of anaesthetists to identify a marked lumbar interspace. *Anaesthesia* 2000; **55**:1122–1126.

7 Levesque P, Marsepoil T, Ho P, *et al.* Multiple sclerosis disclosed by spinal anesthesia. *Ann Fr Anesth Reanim* 1988; **7**:68–70.

8 Kuczkowski KM. Labor analgesia for the parturient with neurological disease: what does an obstetrician need to know? *Arch Gynecol Obstet* 2006; **274**:41–46.

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Implications of carbon dioxide levels in capnography during anaesthesia

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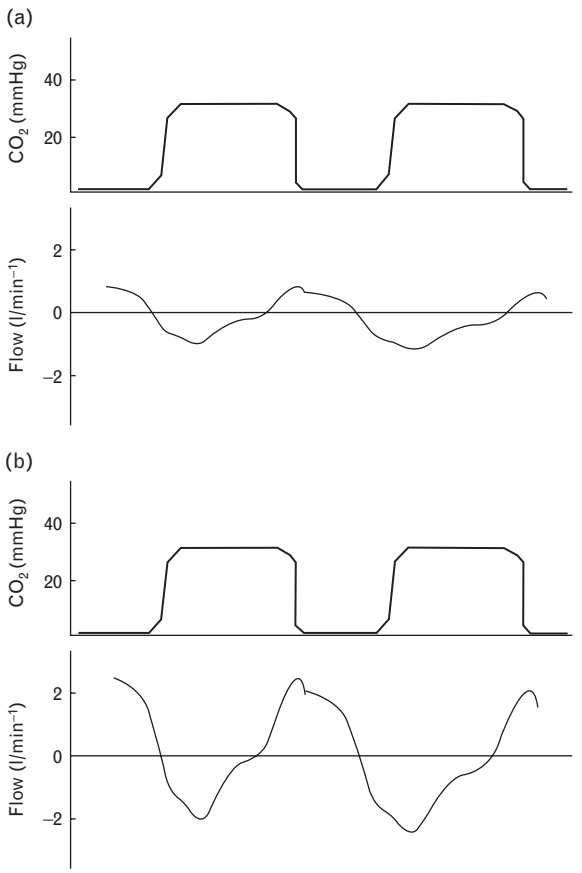
Editor,

Anaesthesiologists interpret the patient state by obtaining useful information from a monitor screen. In particular, measurements of carbon dioxide levels related to artificial respiration greatly contribute to the safety of the anaesthetized patient.[1,2] We present this report because special attention should be given to the interpretation of carbon dioxide waveforms on the monitor.

Carbon dioxide curves provide much information on the patient state. On the basis of this information, anaesthesiologists can identify the presence of both artificial and spontaneous respiration. However, when an irregular carbon dioxide curve at the early recovery stage of anaesthesia changes into a regular one as anaesthesia recovery progresses as in Fig. 1, care must be taken to accurately assess the patient state. When carbon dioxide concentration is low at the initial anaesthesia recovery, it is estimated that ventilation is minimal. However, while spontaneous respiration resumes after slight anaesthesia recovery, the carbon dioxide curves sometimes appear in regular patterns and the value is high as in Fig. 1. At that time, tidal volume, that is, the flow rate, is not related to carbon dioxide value. For this reason, tidal volume is either extremely low as in Fig. 1(a) or

Fig. 1



Capnography and flow-time curve in self-respiration during anaesthesia recovery. Because capnography reflects ratio of carbon dioxide in total measured gas, it does not reflect tidal volume or minute ventilation. These capnographies mean only that there is approximately 4% of carbon dioxide in expiratory gas. (a) Capnography shows regular breathing, though minute ventilation is relatively small. (b) Even though capnography shows the same curve pattern, tidal volume and minute ventilation is larger than that shown in (a).

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rather high as in Fig. 1(b). The discrepancy between the carbon dioxide curve and the flow rate may be explained by the fact that the carbon dioxide curve does not represent the absolute value, but the ratio of carbon dioxide to the total expiratory gas. The irregular carbon dioxide basically originates from abrupt initiation of inspiration and expiration during early recovery, which also cause abrupt change of the ratio of carbon dioxide to the total expiration gas. Although tidal volume is relatively small as in Fig. 1(a), it can be presented as such regular patterns and high value if the patient breathes regularly and the ratio of carbon dioxide to the total expiratory gas is periodically changed. Likewise, if carbon dioxide concentration does not increase despite the fact that tidal volume increases from the value in Fig. 1(a) to that in Fig. 1(b), the carbon dioxide curve will be unchanged. In Fig. 1, the carbon dioxide curve indicates that expiratory gas contains approximately 4% (=35/760) carbon dioxide, but it gives little information on tidal volume. Therefore, after anaesthesia recovery progresses to some extent, we should evaluate tidal volume by the movement of the bellows or the patient chest as well as the flow-time curve displayed on a spirometre in an anaesthesia machine instead of the carbon dioxide curve.

References

1 Goldman JM, Gravenstein JS, Paulus DA, Hamburger A. Capnography during anesthesia. In: Gravenstein JS, Jaffe MB, Paulus DA, editors. *Capnography*. New York: Cambridge University Press; 2004. pp. 47–58.
2 Ross WT. Monitoring the anesthesia machine and respiratory gases. In: Lake CL, Hines RL, Blitt CD, editors. *Clinical monitoring*. Philadelphia: W.B. Saunders; 2001, pp. 293–314.

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Tourniquet use during total knee replacement in a Jehovah's Witness with sickle cell trait: a case report

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Case presentation

An 82-year-old white woman was admitted for an elective right total knee replacement. She was a Jehovah's Witness with sickle cell trait. Her preoperative haemoglobin (Hb) was 10.4 g/dl and electrophoresis revealed sickle Hb (HbS) of 35%. She also had borderline low ferritin levels and was on parenteral iron therapy. She also had hypertension, hypertension-induced renal dysfunction and chronic pulmonary disease.

She refused any form of blood transfusion, including re-infusion of shed blood. After discussion with surgical team it was decided to use tourniquet intraoperatively.

Prior to induction 125 μg of fentanyl was given to obtund the hypertensive response to intubation. Rapid sequence induction of anaesthesia was conducted with propofol and suxamethonium as patient was obese and had a history of gastroesophageal reflux disease. The airway was secured uneventfully. Anaesthesia was maintained with air, oxygen and sevoflurane. Her end tidal was maintained between 3.9 and 4.2 kPa.

Femoral and obturator nerve blocks were performed (with 30 and 5 ml of 0.25% bupivacaine, respectively) using a nerve stimulator. Forty milligrams of atracurium was then given.

She had a 300-mmHg tourniquet pressure for nearly 90 min. Her blood pressure was elevated during surgery requiring intravenous labetalol.

Postoperatively she did not manifest any painful crisis and her Hb was 8.4 g/dl. She was discharged after 8 days in hospital.

Discussion

Sickle cell disease is a common blood disorder that has affected millions worldwide. It is caused by inappropriate substitution of valine for glutamine at the sixth position of the β-globin chain. This altered Hb has a low affinity for oxygen and polymerizes to form insoluble crystals which disrupts the structure of the erythrocytes. In the homozygous state (sickle cell anaemia, HbSS) both genes are abnormal whereas in the heterozygous state (sickle cell trait, HbAS) only one chromosome carries the gene.[1] Whereas sickle cell disease is associated with significant morbidity in terms of growth and development, renal disease, cerebral infarcts, splenic infarcts and haemolytic anaemia, sickle cell trait is usually asymptomatic and affected individuals have normal life expectancy.

When deoxygenated Hb (HbSS) polymerizes, it damages the red cell membrane. Although this is reversible, with repeated sickling this could lead to irreversible damage to red cell membrane.

Anaesthetic techniques for patients with sickle cell disease and trait have tended to focus mainly on preventing sickling by avoiding hypoxaemia, acidosis and maintaining body temperature and intravascular volume. The use of tourniquets in sickle cell disease and trait remains controversial, as they cause hypoxaemia, hypercapnoea and lactic acidosis in the isolated limb.[2] Therefore, some authorities regard both conditions as definite contraindications for tourniquet application,[3] although there are case reports revealing its successful use in patients with sickle cell disease[4] and a study carried out revealing no increased evidence of complications when torniquets applied in patients with sickle cell trait.[5] There is a

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case report that has suggested the initiation of sickle cell crisis in patients with sickle cell trait after application of tourniquets,[6] and as such their use should be balanced against the risks in sickle cell trait.[7]

Haematologists regard sickle cell trait as a benign disease with low morbidity. The incidence of sickle cell trait in African–Americans is one in 12. The occurrence under anaesthesia of cerebral thrombosis, and other mishaps including sudden death in these individuals may suggest cause-and-effect but in reality there is no proven association.[8] Therefore, from the haematological viewpoint the use of tourniquets in limb surgery is regarded as safe.

In our case we believe that nonapplication of tourniquet in her case could be fatal not only because of her refusal of transfusion but also because of increased bleeding risk secondary to hypertension. Furthermore as she was a carrier her risk of going into a crisis was very low.

Conclusion

Providing anaesthesia to sickle cell disease/trait was considered a challenge and there was currently no consensus in anaesthetics over the use of tourniquets. We believe that although the situation is less clear in sickle cell disease, sickle cell trait should not be considered a contraindication to tourniquet application.

References

1 Kumar, Clark. Clinical Medicine. Haematological disease. 7th ed. W.B. Saunders Publication; 2009. p. 409.
2 Wilgis EFS. Observations on the effects of tourniquet ischaemia. *J Bone Joint Surg* 1971; **53**:1343–1346.
3 Cavill G, Kerr K. *Preoperative management. Fundamentals of anaesthesia.* 2nd ed. Cambridge University Press; 2003. p. 18.
4 Abu-Gyamfi YA, Sankarankutty M, Marwa S. Use of a tourniquet in patients with sickle cell disease. *Can J Anaesth* 1993; **40**:24–27.
5 Stein RE, Urbaniak J. Use of the tourniquet during surgery in patients with sickle cell hemoglobinopathies. *Clin Orthop Relat Res* 1980; (151):231–233.
6 Martin WJ, Green DR, Dougherty N, *et al.* Tourniquet use in sickle cell disease patients. *J Am Podiatry Assoc* 1984; **74**:291–294.
7 Aziz ES. Tourniquet use in orthopaedic anaesthesia. *Curr Anaesth Crit Care* 2009; **20**:55–59.
8 Beutler E. *Williams Haematology*. The sickle cell diseases and related disorder. 6th ed. Mc-Graw Hill Publication; 2001. p. 594.

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Intralipid reverses coma associated with zopiclone and venlafaxine overdose

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Editor,

In the UK and Ireland, Intralipid (Kabivitrum Inc., Alameda, California, USA) has been accepted as a part of the recognized management of local anaesthetic cardiotoxicity, having been incorporated into national guidelines.[1] There are a growing number of case reports demonstrating the success of Intralipid in reversing the toxic effects of local anaesthetics not only on the cardiovascular system but also on the central nervous system (CNS). How it exerts this effect is at present unclear, but it is widely thought that this occurs by absorbing the lipophilic local anaesthetics, thereby preventing their action. This 'lipid sink' theory has led to the use of Intralipid in the treatment of other cases of lipid-soluble drug toxicity, with positive results. We present the use of Intralipid in the treatment of coma associated with a mixed overdose of venlafaxine and zopiclone.

Case report

A 55-year-old man was brought into hospital by ambulance with a reduced level of consciousness, having taken a deliberate overdose of zopiclone and the sustained release formulation of venlafaxine. He had a history of depression and had taken numerous overdoses in the past. A family member confirmed that he had taken 1.8 g of venlafaxine and an unknown quantity of zopiclone prior to admission. On arrival in the emergency department, his Glasgow coma score (GCS) was 10. He had a patent airway, a respiratory rate of 15 breaths min^{-1} and was achieving saturations of 96% on 4 l min^{-1} of O_2 via a face mask. He was in sinus rhythm with a heart rate of 69 beats min^{-1} and was hypotensive at 63/34 mmHg. This responded rapidly to a 500 ml bolus of colloid. Blood sugar was 4.6 mmol l^{-1}. His sodium was 141 mmol l^{-1}, potassium 3.7 mmol l^{-1}, urea 6.3 mmol l^{-1} and creatinine 117 mmol l^{-1}. Full blood count and liver function tests were normal. Arterial blood gas sampling revealed no abnormalities (pH 7.36, $paCO_2$ 6.0 kPa, paO_2 13.4 kPa, HCO_3^- 24.6 mmol l^{-1}, base excess 0.4 mmol l^{-1}, lactate 1.3 mmol l^{-1}). ECG was normal, showing no prolongation of the QRS complex or the QT interval. He also had evidence of self-sustained superficial cuts to his anterior chest wall. Paracetamol and salicylate levels were normal.

The opportunity to give activated charcoal had passed, being greater than 1 h following ingestion. He had a gag reflex and was protecting his own airway, so the decision was made to admit him to the hospital for overnight observation and cardiac monitoring.

Four hours later, the anaesthetic services were requested to re-assess him, as there had been a decline in his GCS to 3. Pupils were 4 mm and reactive bilaterally, and there was no focal neurology. All other observations were unchanged. It was discussed whether to intubate him for airway protection, which would have necessitated transferring him to another facility for further management, as there were no critical care beds available.

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On evaluating the potential risks, it was decided to give Intralipid, which had recently been reported to reverse the coma generated by similarly lipophilic drugs.[2]

He was transferred to an anaesthetic room and a nasopharyngeal airway inserted and 10 l min^{-1} of O_2 applied via a nonrebreath mask. He then received a 100 ml (1.5 ml kg^{-1}) bolus of Intralipid, followed by a 400 ml infusion over the next 40 min. Over the course of 30 min, his GCS improved to 11, negating the need for airway management. He was subsequently monitored in the postanaesthetic care unit for 3 h after completing the Intralipid infusion. During this time, observations were stable. His GCS had increased to and remained at 14. He was, therefore, transferred back to the medical assessment unit for ongoing care and neurological observations. He was discharged from hospital 2 days later following psychiatric review.

A toxicology screen of his urine taken at the time of admission revealed no evidence of opiates nor other sedatives but confirmed the presence of both zopiclone and venlafaxine.

Discussion

Venlafaxine is a serotonin–norepinephrine reuptake inhibitor (SNRI) used in the treatment of major depression and other mood disorders. In overdose, it is potentially fatal. It may cause prolongation of the QT interval and QRS complex, arrhythmias, hypotension or hypertension, serotonin syndrome, CNS depression and coma.[3] Zopiclone is a hypnotic with properties similar to benzodiazepines. It binds to gamma-aminobutyric acid receptors, thereby promoting inhibitory neurotransmission within the CNS. Toxicity can lead to drowsiness, confusion, ataxia, bradycardia, hypotension, respiratory depression and coma.[3]

Lipid emulsion has, over the last few years, become widely accepted as having a role in treatment of cardiac arrest due to local anaesthetics. Initial work in animals showed the beneficial effects of lipid emulsion.[4] This has been brought into the clinical domain by an increasing number of case reports, demonstrating its efficacy in reversing the cardiotoxic effects of local anaesthetics. This growing body of evidence has prompted its incorporation into recognized national protocols in the UK.[1] Its use in the treatment of local anaesthetic toxicity has been extended to the treatment of CNS complications.

The mechanism of action of lipid emulsion remains unclear. Whether it directly improves metabolism within the cardiac myocyte, acting as a positive intotrope, or has an indirect plasma-binding effect on local anaesthetics is still uncertain. Certainly, this latter theory has led to alternative applications for lipid emulsion. If it binds lipophilic local anaesthetics then it should act as a 'lipid sink' for other similarly lipophilic drugs.

Animal studies have shown its potential in improving outcome from toxicity related to a number of different lipophilic drugs. Acute cardiac toxicity due to verapamil, clomipramine and propanolol has all been improved by the infusion of lipid emulsion. Again, a number of case reports have provided evidence that lipid emulsion may also be an effective treatment for lipophilic drug-induced cardiac toxicity in humans.[5] Recently, lipid emulsion has been introduced into a number of guidelines for treatment of cardiotoxicity secondary to several lipophilic drugs in the United States.[6]

The use of lipid emulsion has been broadened to include the treatment of CNS depression due to deliberate self-poisoning with lipophilic drugs.[2] This latest development may have enormous implications in demands on acute hospital services. Avoiding intubation and subsequent high dependency care would have beneficial effects in this group of patients and inevitably allow greater provision elsewhere.

This case provides further evidence as to the potential applications of lipid emulsion. However, we must exercise caution; as reminded in a recent editorial, the decision to use it in the attempt to reverse lipophilic drug-induced coma must be balanced against any possible complications.[7] To date, there have been no documented complications of its use in the resuscitation setting, but the potential risks of allergy, fat embolism, pancreatitis and drug interaction do exist. Lipid emulsion will need further evaluation if its use is to become commonplace.

References

1 Association of Anaesthetists of Great Britain and Ireland. http://www.aagbi.org/publications/guidelines/docs/latoxicity07.pdf. [Accessed 20 October 2009]

2 Finn SDH, Uncles DR, Willers J, Sable N. Early treatment of a quetiapine and sertraline overdose with Intralipid. *Anaesthesia* 2009; **64**:191–194.

3 UK National Poisons Information Service. Poisons index A–Z. http://toxbase.u5e.com/Poisons-Index-A-Z/V-Products/Venlofaxine; http://toxbase.u5e.com/Poisons-Index-A-Z/Z-Products/Zopiclone. [Accessed 20 October 2009]

4 Weinberg G, Ripper R, Feinstein DL, Hoffman W. Lipid emulsion infusion rescues dogs from bupivacaine-induced cardiac toxicity. *Reg Anesth Pain Med* 2003; **28**:198–202.

5 Young AC, Velez LI, Kleinschmidt KC. Intravenous fat emulsion therapy for intentional sustained-release verapamil overdose. *Resuscitation* 2009; **80**:591–593.

6 Novel antidotes in calcium channel antagonist toxicity: chicken soup for the toxic heart. Official Newsletter of the California Poison Control System, Spring 2009. http://www.calpoison.org/hcp/2009/callusvol7no1.html. [Accessed 20 October 2009]

7 Picard J, Harrop-Griffiths W. Lipid emulsion to treat drug overdose: past present and future. *Anaesthesia* 2009; **64**:119–121.

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