

- 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 Anesthesiol* 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

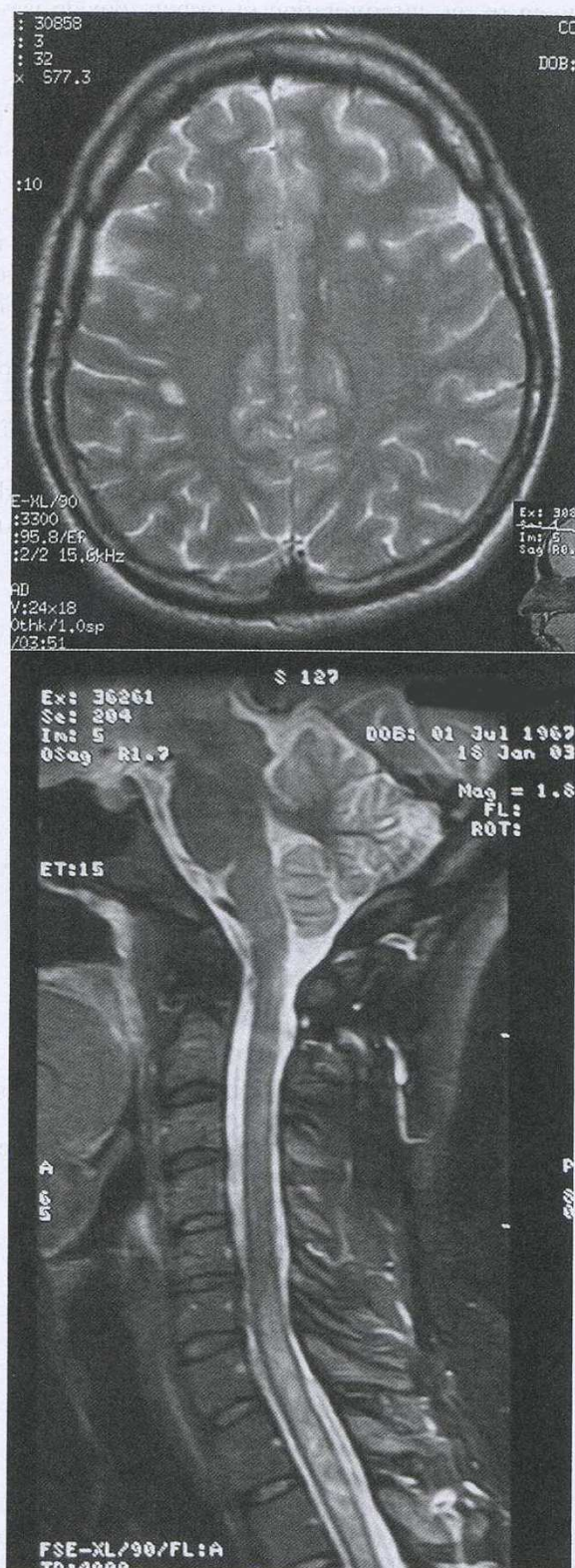
reported left leg pain on spinal needle insertion and the postoperative MRI had shown a hyperintense lesion at the level of the conus medullaris, the anaesthetist was charged because of the spinal cord lesion and, then, for the NMO. However, a rigorous analysis of records showed the following facts:

- (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 post-dural puncture headache (PDPH), rather than to reduce

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

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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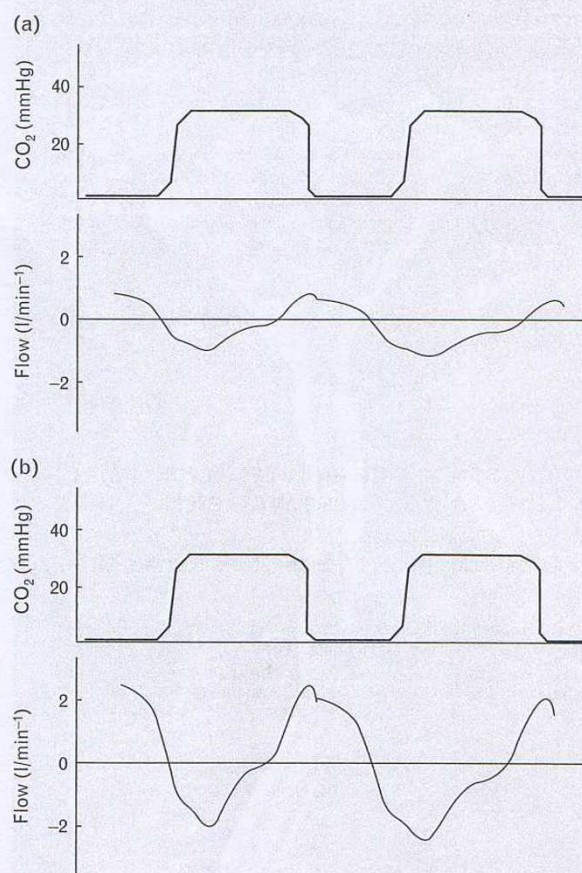
Editor,

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).