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CASE REPORT

Neuromuscular monitoring in myasthenic syndrome

H. Itoh,[1] K. Shibata[2] and S. Nitta[3]

1 Instructor, Department of Anaesthesiology and Intensive Care Medicine, and 2 Associate Professor, Department of Emergency and Critical Care Medicine, Kanazawa University School of Medicine, 13-1 Takara-machi, Kanazawa 920-8641, Japan
3 Chief, Division of Anaesthesia, Ishikawa Prefectural Central Hospital, Kanazawa, Japan

Summary

We describe the anaesthetic management of a 72-year-old man with myasthenic syndrome. Pre-operatively, he was treated with 3,4-diaminopyridine and showed a strong hand grip. During general anaesthesia with nitrous oxide and sevoflurane in oxygen, a mechanomyograph and two accelerographs were set up for the hands and left foot to monitor neuromuscular function. Insufficient force and acceleration of contraction with 1 Hz stimulation was observed in the hands. In the foot, the twitches produced by 1 Hz and train-of-four stimulation could barely be detected using the accelerograph, and the train-of-four ratio fluctuated between 70 and 100%. No neuromuscular blocking drugs were used during surgery. After discontinuation of sevoflurane, responses to train-of-four stimulation remained small, but a strong response to tetanic stimulation was observed, with post-tetanic facilitation. Extubation was successful, and recovery from anaesthesia was uneventful. Tetanic stimulation and post-tetanic facilitation are important in monitoring neuromuscular function in patients with myasthenic syndrome whose train-of-four responses are insufficient.

Keywords *Monitoring*: neuromuscular function. *Lambert–Eaton myasthenic syndrome*: drug therapy.

Correspondence to: Dr H. Itoh
E-mail: hironori@med.kanazawa-u.ac.jp
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Myasthenic syndrome (Eaton–Lambert or Lambert–Eaton syndrome), characterised by weakness of proximal limb muscles, may show abnormal sensitivity to both depolarising and non-depolarising neuromuscular blocking drugs [1]. As a result, administration of these agents can cause prolonged neuromuscular blockade [2–5]. Although monitoring of neuromuscular function during anaesthesia is considered necessary for patients with myasthenic syndrome, little information is available concerning the details of such monitoring. We describe a case of myasthenic syndrome in which monitoring of neuromuscular function during general anaesthesia was particularly difficult.

Case history

A 72-year-old man with myasthenic syndrome was scheduled for thoracotomy for excisional biopsy of a suspected small-cell cancer of the lung. A coin lesion had been discovered in the left lung on a chest radiograph 2 months after the onset of neuromuscular symptoms. Electromyography (EMG) showed marked diminution of amplitude of the initial compound muscle action potential evoked by sural nerve stimulation. A decremental response was seen with low-frequency repetitive stimulation (2 Hz), but up to 300% facilitation occurred with high-frequency repetitive stimulation (50 Hz). The patient was diagnosed with myasthenic syndrome based on these characteristic findings, absence of detectable antibodies to the acetylcholine receptor and a negative edrophonium test. At pre-operative assessment, the patient was 175 cm tall and weighed 63 kg. Weakness of the lower limbs precluded walking without assistance. Although he complained of weakness of the upper limbs, dynamometry showed a strong hand grip (> 30 kg). Other pre-operative investigations, including

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blood haematology and biochemistry screening, respiratory function tests and blood gas analysis, were unremarkable.

The patient had taken 3,4-diaminopyridine 60 mg daily beginning 1 week pre-operatively, but did not take the drug on the morning of surgery. Without premedication, anaesthesia was induced with thiopental 225 mg, followed by inhalation of 50% nitrous oxide and 2.0% sevoflurane in oxygen. To assess neuromuscular function during anaesthesia, two neuromuscular monitoring devices, the Myograph 2000 (Biometer International, Odense, Denmark) for the left hand and TOF-Guard (Biometer) for the right hand, were then set up in the usual manner. Even when a maximal current (60 mA) was applied at 1 Hz to each ulnar nerve, responses of both thumbs were extremely weak and no peak was recorded on either monitor even at maximal sensitivity of recording. Accordingly, another TOF-Guard was attached to the left foot to monitor plantar flexion of the first toe by the flexor hallucis brevis muscle when the posterior tibial nerve was stimulated [6]. Responses of the first toe to 1 Hz stimulation at 60 mA were also weak and twitches were barely discernible on the monitor. Function in this foot was monitored continuously during anaesthesia. When train-of-four (TOF) stimulation (0.2 ms duration, 2 Hz every 15 s) was begun, twitch amplitude for the first response to TOF stimulation (T1) was very small, and the TOF ratio fluctuated between 70 and 100%. A tentative plan to administer non-depolarising neuromuscular blocking drugs was abandoned because of the unreliability of neuromuscular monitoring.

Using only inhaled anaesthetics, ventilation with a bag and face-mask was continued to induce sufficient relaxation for tracheal intubation, which was performed uneventfully 15 min after induction. During surgery, anaesthesia was maintained with 60% nitrous oxide and 0.5–2.0% sevoflurane in oxygen, and T1 decreased gradually. At the end of surgery after 130 min, T1 was 0–20% of control with decrement upon TOF stimulation. Twenty minutes after discontinuation of sevoflurane, T1 showed no increase; ventilation was judged to be adequate based on sufficient tidal volume. The stimulation sequence for post-tetanic count was then applied, consisting of 1 Hz single stimuli for 30 s followed by 50 Hz tetanic stimulation for 5 s, a pause of 3 s and then 1 Hz single stimuli again for 15 s [7]. Both a strong response to tetanic stimulation and post-tetanic facilitation were observed (twitch height in the first response after tetanic stimulation was 16 times greater than the 1 Hz single twitch responses just before tetanic stimulation; by the 15th stimulus, twitch height was still 10 times greater). Five minutes after the post-tetanic count stimulation, the patient was able to lift his head for 5 s and the trachea was extubated. The post-operative course was uneventful.

Discussion

Myasthenic syndrome typically presents with proximal muscle weakness, depressed tendon reflexes and complaints of dry mouth [8]. The characteristic EMG finding is an abnormally low amplitude of the compound muscle action potential evoked by low-frequency nerve stimulation, with a progressive increase during high-frequency stimulation [9, 10]. Our patient's clinical presentation and the results of examination were consistent with a diagnosis of myasthenic syndrome. Surgery is performed only in some cases of myasthenic syndrome because of its relationship to small-cell lung cancers often treatable by other modalities [9, 10]. In surgical cases, myasthenic syndrome shows abnormal sensitivity to both depolarising and non-depolarising neuromuscular blocking drugs [1].

Neuromuscular monitoring during anaesthesia is considered essential in patients with myasthenic syndrome, but methods for monitoring neuromuscular function in these cases have not been standardised. While TOF stimulation is a widespread monitoring method in general anaesthesia, we believe that it has only limited value in patients with myasthenic syndrome, because our patient showed insufficient responses. This failure was not surprising because the amplitude of the initial compound muscle action potential was abnormally small on the pre-operative EMG. The aetiology of myasthenic syndrome is thought to involve development of antibodies to voltage-gated calcium channels at motor-nerve terminals, inhibiting calcium flux and thereby diminishing the release of acetylcholine; these cross-reacting antibodies are incited by neural crest tumours [11]. Marked post-tetanic facilitation is seen in the syndrome [1], possibly from accumulation of acetylcholine released during the course of such frequent stimulation. Low rates of nerve stimulation, such as single stimuli or TOF, are rarely used in neurophysiological testing; nerves are almost always stimulated at ≈ 50 Hz [12]. Such frequent nerve stimulation leads to great muscle force corresponding to an incremental compound muscle action potential response. Post-tetanic count, however, was introduced as a method for assessing intense neuromuscular blockade. A close correlation was reported between post-tetanic count and recovery from intense blockade caused by clinically used non-depolarising neuromuscular blocking drugs [7]. Post-tetanic count is based on the concept of post-tetanic facilitation, resulting from mechanisms including accelerated flux of calcium ions into nerve terminals followed by mobilisation and release of acetylcholine [13]. This mechanism, reflecting

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the pathophysiology of myasthenic syndrome, may be the reason that pre-operative muscle force was not markedly limited in our patient.

In this case, neuromuscular function was evaluated using two accelerograph monitors in addition to a standard mechanomyograph. Introduced recently into clinical practice, the accelerograph is simpler and more easily used than a standard mechanomyograph, while showing good correlation with mechanomyographic data [14, 15]. In stimulation of a nerve by post-tetanic count, the device can record the acceleration during post-tetanic facilitation and up to 15 post-tetanic twitches. Little or no post-tetanic facilitation occurs in the absence of neuromuscular blocking drugs in healthy individuals [7]. Our case showed an initial post-tetanic twitch height 16 times greater than that at a 1-Hz stimulation rate just before tetanic stimulation, whereas the final post-tetanic twitch height remained 10 times greater. This phenomenon is compatible with the mechanisms of compound muscle action potential responses. Few reports have described neuromuscular function monitoring using the post-tetanic count method in myasthenic syndrome patients. The informative response that we obtained by post-tetanic count suggests that this technique may be used to monitor such patients to determine the degree of neuromuscular blockade when neuromuscular blocking drugs are administered.

In patients with myasthenic syndrome, prolongation of neuromuscular blockade has been reported [2–5], requiring decreased doses of neuromuscular blocking drugs. Indeed, neuromuscular blocking drugs should be avoided if possible. If their use is necessary, neuromuscular transmission should be monitored closely. In some cases, prolongation of neuromuscular blockade has been avoided by adding regional or local anaesthesia, such as epidural [16] or superior laryngeal nerve blocks [17], to light general anaesthesia instead of using neuromuscular blocking drugs. We initially planned to use minimal doses of non-depolarising neuromuscular blocking drugs while monitoring neuromuscular transmission, but abandoned this plan because monitoring using the TOF method was unreliable in our patient. During surgery, sufficient paralysis was obtained with inhaled anaesthetics alone. However, when residual neuromuscular effects of neuromuscular blocking drugs must be assessed, specific methods for neuromuscular monitoring must be chosen carefully.

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CASE REPORT

Epidural anaesthesia for Caesarean section in a patient with Devic's Syndrome

B. Gunaydin,[1] D. Akcali[2] and M. Alkan[2]

1 Assistant Professor, and 2 Resident, Department of Anaesthesiology, Faculty of Medicine, Gazi University, Ankara, Turkey

Summary

The anaesthetic management of a 29-year-old paraplegic woman suffering from Devic's Syndrome scheduled to undergo Caesarean section under epidural anaesthesia is presented. The case is discussed with particular reference to the risk of autonomic hyperreflexia.

Keywords *Anaesthetic techniques*: epidural. *Complications*: tranverse myelitis; autonomic hyperreflexia.

Correspondence to: Dr B. Gunaydin
E-mail: gunaydin@med.gazi.edu.tr
Accepted: 6 February 2001

Devic's Syndrome (neuromyelitis optica) may necessitate special anaesthetic considerations in Caesarean section because of the possibility of autonomic hyperreflexia, a complication arising from transverse myelitis, one of the components of the syndrome [1–3]. We report a case detailing the anaesthetic approach in managing a paraplegic parturient with this condition.

Case history

A 29-year-old woman was admitted to the hospital at 20 weeks' gestation suffering from weakness, involuntary contractions in her lower limbs and back pain. She had a history of herpes zoster and bilateral optic neuritis. Physical examination revealed that she was able to sit only with support, that she had hypoaesthesia below the T_2 dermatome and that her lower extremities were flaccid. She had urinary and faecal incontinence. Neurological examination revealed progressive paraparesis and paraplegia associated with pathological reflexes (Babinski and clonus), and a hyperintense lesion between C_5 and T_4 was observed in the magnetic resonance imaging (MRI) of the vertebral column and confirmed by contrast uptake.

Laboratory results were within normal limits except for a haemoglobin level of 9.3 $g.dl^{-1}$. Levels of angiotensin converting enzyme (ACE) and oligoclonal band were measured in cerebrospinal fluid to exclude the differential diagnosis of sarcoidosis and multiple sclerosis, respectively. Low ACE levels (3 $U.l^{-1}$, normal: 8–52 $U.l^{-1}$) and negative oligoclonal band results ruled out both of these conditions.

One month later, there was decreased hyperintensity without contrast uptake in the second MRI examination; the clinical signs and symptoms were unchanged except that the hypoaesthesia was now below T_6. She was treated with intravenous immunoglobulin (0.5 $g.kg^{-1}.day^{-1}$) and methyl prednisolone sodium succinate (1000 $mg.month^{-1}$) until the end of the pregnancy. She complained of painful spasticity especially during fetal movements, and baclofen was given to relieve this. Her chronic urinary tract infection was treated by antibiotics. After her request for termination of pregnancy was refused at 29 weeks' gestation, psychological support was offered and antidepressants were prescribed. Ritodrine was started to treat premature uterine contractions and continued until 37 weeks' gestation.

Caesarean section was planned by the obstetricians because of developing fetal distress. After performing epidural puncture with a 20 G Tuohy needle in the sitting position at the L_{3-4} interspace, an epidural catheter was inserted and advanced 5 cm into the epidural space and secured. Following administration of 3 ml bupivacaine 0.5% as a test dose through the catheter, a further bolus of 5 ml bupivacaine 0.5% and 50 μg fentanyl was

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administered. Caesarean section was started after an anaesthesia level at T_4 was obtained. A healthy female neonate was delivered with Apgar scores of 9 and 10 at 1 and 5 min, respectively. Fifteen units of oxytocin were given by intravenous infusion after clamping the umbilical cord, and the procedure was completed uneventfully. The patient was very comfortable throughout the procedure and felt no pain; there were no untoward haemodynamic disturbances at any time. Bupivacaine 0.125% with 2 mg morphine in a total volume of 10 ml was administered through the catheter to relieve postoperative pain 2 h after the initial injection, followed by removal of the epidural catheter.

Her neurological symptoms improved in the postpartum period and physical rehabilitation is continuing.

Discussion

Several cases of pregnancy with pre-existing transverse myelitis have been reported, although there have to date been no reports of patients with Devic's Syndrome undergoing Caesarean section under epidural anaesthesia, since this syndrome is rarely seen in pregnancy [2, 4].

Devic's Syndrome is characterised by acute bilateral optic neuritis and transverse myelitis which may occur either simultaneously or separately [1]. Transverse myelitis is an inflammatory disease that causes functional transection of the spinal cord and may be triggered by either infections or autoimmune disorders [3]. The most common symptoms of the syndrome are back pain and progressive paraparesis as were observed in the present case. The diagnosis of transverse myelitis was suspected because of the possibility of the occurrence of the disease secondary to herpes zoster infection. The clinical diagnosis was supported by neurological examination and confirmed by MRI scan. Finally, the presence of a history of optic neuritis in addition to transverse myelitis led the neurologists to the diagnosis of Devic's Syndrome.

Pregnant women with transverse myelitis are at increased risk for recurrent urinary tract infections, anaemia, premature labour and delivery and autonomic hyperreflexia, depending upon the spinal level of the pathological process [2]. Our patient was receiving antibiotic treatment because of chronic urinary tract infection, and was also suffering from anaemia. Although anaemia mostly results from iron deficiency during pregnancy, it has been reported that it also occurs in 12% of pregnant women with spinal cord injuries [4]. The risk of preterm delivery because of the inability to detect early signs of labour is dependent upon the functional level of the spinal cord lesion [2]. A patient with a functionally complete transection above the T_6 level is at risk of preterm labour, and this occurred in our case. This was managed by treating premature uterine contractions until the decision was made to proceed to Caesarean section because of fetal bradycardia.

Autonomic hyperreflexia is a life-threatening complication characterised by severe hypertension and bradycardia associated with facial flushing, sweating and pilomotor erection and may cause convulsions, cerebral haemorrhage and even death [5, 6]. Epidural and spinal anaesthesia are effective in producing prompt cessation of autonomic hyperreflexia by interrupting the reflex arc extending from the contracting uterus to the cardiovascular system via the spinal cord [7]. We preferred epidural to spinal anaesthesia because we felt that it would be easier to control the onset of sympathetic block and thus minimise cardiovascular instability.

There are limited case reports of pre-existing transverse myelitis during pregnancy and most had uneventful spontaneous vaginal delivery. However, conduction anaesthesia is still required for pain relief as well as control of autonomic hyperreflexia in patients with complete or partial cord transection during labour and delivery [3, 4, 8, 9]. General or regional anaesthesia are the options for Caesarean section, although there is some controversy in the literature about which technique should be preferred. In one case report, transverse myelitis developed after general anaesthesia with propofol and atracurium followed by enflurane in nitrous oxide and oxygen [10]. Therefore, it appears that it is not always true to assume a close temporal relationship between regional anaesthesia and transverse myelits since the aetiology remains unknown in the majority of the cases.

Once we had chosen to use epidural anaesthesia, we were faced with deciding on the drug, the concentration and the volume to administer epidurally. We preferred bupivacaine like Pauzner *et al.* [11], who described a successful delivery using epidural bupivacaine. We avoided adding any adjunctive drugs such as epinephrine in the epidural test dose because of the possibility of triggering autonomic hyperreflexia by precipitating a hypertensive response. However, after inserting the epidural catheter, we performed regular aspirations to check for intravascular placement of the catheter and then administered 3 ml of bupivacaine 0.5% in order to rule out subarachnoid injection. Since the required volume of local anaesthetic for an adequate block in this patient might be difficult to predict, we administered a small initial dose of 5 ml of bupivacaine 0.5%, aware also of the difficulty in assessing block level because of the existing paraparesis. The epidural use of opioid drugs such as pethidine and morphine has succesfully blocked autonomic hyperreflexia during labour and delivery [7, 11]. Although epidural bupivacaine alone could control autonomic hyperreflexia [12], we decided to add 50 μg

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of fentanyl to the epidural bupivacaine in order to improve the quality of block and contribute to the protective effect against autonomic hyperreflexia. Fentanyl alone has failed to control visceral nociceptive impulses caused by uterine contractions in a quadriplegic parturient [9]. We were surprised to find that the level of anaesthesia had reached as high as T_4 after 10 min with such a low dose of bupivacaine, but felt that this block was satisfactory for Caesarean section, as indeed it proved to be.

Prior to administration of the epidural drugs, the mean arterial pressure and heart rate were 91 mmHg and 62 beat.min^{-1}, respectively. The mean arterial pressure neither decreased below 85 mmHg nor increased above baseline values during the operation. We did not observe any haemodynamic alterations suggestive of autonomic hyperreflexia as described in the literature during parturition in quadriplegic and paraplegic patients [12, 13].

With respect to general anaesthesia, it is recommended to avoid using succinylcholine in these cases because of the potential risk of inducing hyperkalaemia and muscle contracture in patients with denervating neurological deficits and muscle atrophy. Hyperkalaemia occurs because of the release of potassium ions from the affected denervated muscle, can occur for several months after denervation and may be sufficiently severe to precipitate cardiac arrest [14]. In contrast, the denervated muscle frequently responds normally to non-depolarising neuromuscular blocking agents [14].

In summary, the pregnant paraplegic patient presents a challenge to the anaesthetist, especially in rare conditions such as Devic's Syndrome. Although some critics point out that regional anaesthesia is contraindicated because of the possible difficulty in evaluating neurological complications that might develop in pre-existing paraplegia, many practitioners feel that regional anaesthesia is the treatment of choice, particularly if there is a risk of associated autonomic hyperreflexia. We performed epidural anaesthesia with low-dose bupivacaine combined with fentanyl in this case, and had an uncomplicated and an uneventful course of anaesthesia not only for mother but also for the baby. It is important to be aware of all the potential problems that may arise peroperatively in Devic's Syndrome and to consider the use of epidural analgesia to prevent the dangerous complication of autonomic hyperreflexia.

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