Repetitive synchronized cyclical oscillations of multisystem parameters subsequent to high-dose thiopental therapy for status epilepticus secondary to herpes encephalitis


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We report a case of status epilepticus secondary to herpes encephalitis, treated with thiopental infusion and mechanical ventilation. The computerized storage and analysis of physiological data led to the detection of repetitive synchronized cyclical oscillations of arterial pressure, heart rate, EEG parameters, peripheral temperature and core temperature. Arterial pressure oscillations have been described in patients who are severely systemicill; cardiovascular and brain electrical activity may also oscillate in the presence of raised intracranial pressure. In contrast, this patient had no features of severe systemic illness or of raised intracranial pressure. Our hypothesis is that high-dose thiopental may have been a cause of our findings by producing autonomic dysfunction.

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Case report

An 18-yr-old male with no history of epilepsy was treated in the emergency unit for status epilepticus. There were no focal neurological signs or evidence of raised intracranial pressure. Seizure activity persisted despite cumulative total doses of phenytoin 1500 mg and diazepam 60 mg. A loading dose of thiopental 1000 mg was therefore given followed by tracheal intubation and mechanical ventilation. Positive herpes zoster antibody serology and cerebrospinal fluid polymerase chain reaction studies later confirmed the diagnosis of viral encephalitis.

On admission to the intensive care unit (ICU), intermittent positive-pressure ventilation was continued, together with a thiopental infusion titrated up to 500 mg h⁻¹ under the guidance of a continuously running paper electroencephalogram (EEG), which confirmed the presence of burst suppression and the absence of seizure activity. Standard monitoring included continuous electrocardiography (ECG), invasive arterial pressure, capnography, respiratory flow/volume measurements, hourly urine output measurements and repeated arterial blood gas analysis. The cardiorespiratory, haematological, hepatic, renal, biochemical and blood gas profiles were within normal limits. In particular, no signs of poor tissue oxygenation or pH imbalance were observed. Cranial CT and MRI scans showed no abnormalities. Additional therapy included acyclovir 750 mg 8-hourly and a crystalloid infusion at 100–250 ml h⁻¹. No sympathomimetic therapy was required. No subsequent seizures were detected clinically or electroencephalographically, and the thiopental infusion was stopped on day 2, by which time the patient had received 7975 mg over 20 h. Regular intravenous phenytoin therapy (250 mg 12-hourly) was continued.

On day 3, with the relatives’ assent and research ethics committee approval, specialized monitoring was set up as part of the EU Biomed 1 IMPROVE intensive care project in order to establish an annotated data library. This involved acquiring and storing continuous physiological variables (including arterial pressure, ECG and capnographs at sampling rates between 25 and 100 Hz) and intermittent annotations (exact timing of drug doses, insertion of lines and any other interventions). This period of specialized
monitoring lasted 24 h, during which time continuous digitized EEG was also recorded.2 No notable clinical events occurred in this 24 h period, and biochemical and blood gas profiles remained within normal limits. As on admission, there were no signs of raised intracranial pressure.

Subsequently, the patient made an uncomplicated recovery; his level of consciousness improved and the tracheal tube was removed on day 6. At this point his Glasgow Coma Scale score was 15 and gross intellectual functions were normal. He left the ICU on day 7 and went home on day 14.

Retrospective trend analysis revealed marked cyclical oscillations of the multisystem physiological variables over 24 h (Fig. 1). Assessments were made of raw data (peripheral and core temperature, mean arterial pressure, heart rate) and frequently used processed EEG parameters. The latter consisted of root mean squared amplitude (each EEG amplitude digital value within a sampling period is squared, a mean is subsequently taken and the square root of this is taken) and median power frequency (frequency at which half the spectral power is above and half is below). Peaks of relative hypertension occurred synchronously with peaks in EEG amplitude. Oscillations in heart rate and EEG frequency both showed a close inverse relationship to arterial pressure and EEG amplitude. Peripheral temperature and urine output also showed oscillations, though these did not show exact synchrony with the other parameters.

Discussion
The variability of physiological parameters is an important indicator used in assessment and prediction in intensive care patients. Several types of variability have been observed: the simplest example is the ECG R–R interval, which is a good measure of (i) reactivity to stress during critical illness

Fig 1 Twenty-four-hour display of polygraphic physiological measurements. The variables are shown on separate graphs for clarity. (a) Root mean square EEG amplitude, a frequently used processing technique in which digitized values for EEG amplitude are individually squared within a sampled period; subsequently the mean is extracted and the square root of the mean is taken. (b) EEG median power frequency, the frequency at which half the spectral power is above and half is below within a sampled period. (c) Mean arterial pressure. (d) Heart rate. (e) Core temperature. (f) Urine output.
and (ii) autonomic neuropathy. Similarly, EEG reactivity to stimuli carries the same implications; the presence of reactivity is reassuring, and the lack of reactivity (in the absence of drug-induced or hypothermic coma) suggests a poor prognosis.5

More orderly variations exist in both cardiac and neurological signals. First-, second-, third- and, more recently, fourth-order blood-pressure waves have been described and summarized elegantly by Seiver and colleagues.4 First- and second-order waves are descriptions of blood-pressure variations with each systolic beat and respiratory cycle, respectively. Third-order waves, with a cycle of 10–160 s, are a consequence of disordered autonomic feedback. Fourth-order waves (cycles of minutes to hours, as seen in this case) have been thought to be due to poor oxygen delivery to the tissues and are associated with a poor prognosis.4 5 Electroencephalographers have described cyclical changes in association with sleep patterns;6 the presence or absence of these features has, in turn, relevance to prognostication in coma.7 8 Repetitive waves with cycling of minutes to hours are also seen in the EEG monitoring of patients with brain insult as a consequence of raised intracranial pressure.9

In this case, we considered three possible causes of the multiparameter oscillations. Classical fourth-order changes seemed unlikely, as severe systemic dysfunction was not evident. Covertly raised intracranial pressure may have been an alternative cause, though there was never any clinical or radiological evidence of it, or of persisting brain injury. Lastly, we could not exclude high-dose thiopental infusion as an important cause. Plasma levels were not measured, as these findings became apparent only on trend analysis after the cessation of monitoring. Our hypothesis is that high-dose thiopental may cause autonomic dysfunction, leading to loss of ‘randomicity’; this would be similar to the hypothesis of fourth-order oscillations caused by poor tissue oxygen delivery in ill patients.

The interesting phenomenon of synchronized cyclical oscillations of physiological parameters should not automatically instil concern, and may prove to be a recognized finding when high-dose thiopental infusion is used or there is covert brain dysfunction. This may, in time, be clarified with the increasing sophistication of polygraphic trend analysis in intensive care, without which our findings would not have been apparent. To validate these findings, prospective studies of high-dose thiopental infusions (including the simultaneous measurement of plasma levels) and intracranial pressure monitoring should be encouraged.

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Loss of consciousness following spinal anaesthesia for Caesarean section

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A healthy parturient under spinal anaesthesia for Caesarean section lost consciousness for an hour, 20 min after the intrathecal injection of 2 ml of 0.5% heavy bupivacaine. The patient was haemodynamically stable before losing consciousness. The differential diagnosis is discussed.

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Spinal anaesthesia is increasingly used for Caesarean section as it is remarkably safe and effective. Its safety lies in the fact that parturients are conscious throughout anaesthesia and are not exposed to the hazards of intubation. Patients can lose consciousness in the presence of a total spinal block, usually characterized by severe hypotension. However, loss of consciousness with this technique in a haemodynamically stable patient is unusual. We report a parturient who lost consciousness 20 min after a successful spinal anaesthetic block.

Case report

The parturient, a 25 yr old, 160 cm, 55 kg normotensive primigravida with mild polyhydramnios, required an elective Caesarean section for delivery of a baby with bilateral pleural effusions of unknown cause. She had no other medical condition of note; in particular, she had no psychiatric history. Plasma electrolytes were normal before surgery. Sodium citrate and oral ranitidine were given in the ward before delivery; no other drugs were administered and the patient was not on any medication. Immediately before spinal blockade, Unasyn 1.5 g (subactam 500 mg and ampicillin 1000 mg) diluted in sterile water was administered as an intravenous infusion. For spinal block, a 26 G Atraucan (B Braun) spinal needle was used at L3/4 in the midline with the patient in the sitting position. Cerebrospinal fluid flowed from the needle after a single attempt and 2 ml of 0.5% heavy bupivacaine was injected after initial aspiration. The patient was then placed horizontally with a left lateral tilt and a single pillow to support the head. A T4 level of block was obtained by 5 min and the Bromage score before surgery was zero. Before delivery of the baby, the parturient’s arterial pressure was measured every 5 min with a Dinamap. This averaged around 110/60 mm Hg with one transient episode of hypotension (90/50 mm Hg) about 5 min after the spinal injection, which improved with the administration of ephedrine 5 mg. The heart rate averaged 80–100 beats min⁻¹. Three litres of oxygen were administered by nasal prongs; oxygen saturation was 99–100%. Hartmann’s solution (500 ml) was given as a preload and a second 500 ml was commenced before the baby was delivered. During this period the patient was alert and communicating with the anaesthetist. No other drugs were administered for 15 min before delivery of the baby.

At the time of delivery, 20 min after administration of the intrathecal drug, the patient suddenly stopped communicating, as if going off to sleep. She was not responding to verbal commands or to deep pain. There was no frothing, and no uprolling of the eyeballs. The incident was not preceded by nausea and vomiting. There were no complaints of chest pain, inability to breathe or weakness of the upper limbs immediately before loss of consciousness. There was a transient period of apnoea with a dramatic drop in oxygen saturation to around 75%. The patient’s lungs were ventilated with a face mask for 1 min on the circle system after which spontaneous ventilation resumed. No bronchospasm or urticaria was noted. The anaesthetist tried to intubate the patient at this stage but was unsuccessful as the patient had a very strong gag reflex. A decision was made not to give additional drugs to sedate or paralyse the patient as it was noted that she was able to protect her airway. Both of her pupils were about 3 mm wide and reactive to light. Throughout this period of loss of consciousness, arterial pressure averaged around 100/55 mm Hg and the pulse rate was about 80 min⁻¹. Ten minutes into the incident the patient was responding to deep pain.
with non-purposeful movements of the upper limbs. Gradually the movements on deep pain became more purposeful but there was still no verbal response. One hour after the onset of the incident, the patient opened her eyes to commands and had slurred speech when she attempted to talk. Muscle power in the upper limbs at this stage was grade 4 and her sensory level was at T2 bilaterally. The blood sugar concentration was 3.9 mmol litre\(^{-1}\). One and a half hours after the onset of the loss of consciousness, the patient was fully alert and her upper limb muscle power was grade 5 with a sensory level at T5. When questioned about her loss of consciousness, she said that she had felt extremely sleepy, had gone to sleep and was unable to recall the event. Four hours after loss of consciousness, the spinal block had totally worn off, with grade 5 muscle power in all four limbs. The patient was discharged back to the ward where the anaesthetist visited her for the next few days. The baby died because of severe heart failure, with a single atrium, single ventricle and pulmonary atresia, 2 h after delivery.

Twenty-four hours after spinal injection, the patient complained of giddiness and a postural frontal headache which gradually worsened. CT scan results were normal and an EEG showed no abnormal foci. An epidural blood patch relieved the headache immediately. The patient was discharged 1 week after the incident with no further complaints.

**Discussion**

A non-sedated parturient given an intrathecal local anaesthetic injection should never lose consciousness. In this particular parturient, there was no history of epilepsy or diabetes to cause loss of consciousness. A blood sugar concentration of 3.9 mmol litre\(^{-1}\) is at the low end of the normal range but was not low enough to account for the loss of consciousness. The normal EEG indicated that the event could not be explained by an epileptic seizure. A CT scan done when the patient developed a headache 24 h after the incident was normal. This indicated that there was no space-occupying lesion intracranially to account for the loss of consciousness during the Caesarean section. As magnetic resonance imaging (MRI) is superior to CT scanning for evaluating most suspected central nervous system pathology,\(^6\) we should, in retrospect, have subjected the patient to MRI instead. Only 2 ml of 0.5% bupivacaine was given intrathecally and the patient was subsequently placed horizontally, so the signs could not be attributed to a total spinal block. The patient did not develop significant hypotension, respiratory depression or pupillary dilation to warrant a diagnosis of total spinal block.\(^4\) The anaesthetist was certain that the loss of consciousness could not be attributed to any intravenous sedative drugs being given inadvertently, as no drugs were administered for 15 min before delivery of the baby. It was unlikely that the patient had an amniotic fluid embolism,\(^7\) although the event occurred at the time of delivery of the baby. She was stable cardiovascularily and at no time was there any coagulopathy. The event was also unlikely to be the result of a pulmonary embolism,\(^8\) as the patient had a normal cardiac rhythm. There was no cardiovascular compromise at the time of the event and no residual neurological deficit after regaining consciousness. However, echocardiography was not attempted. Air embolism occurring at the time of delivery can also be responsible for the loss of consciousness. For loss of consciousness to have occurred, air must have reached the left side of the heart through a patent foramen ovale. This was not ruled out by echocardiography, but air embolism is unlikely as there were no complaints of dyspnoea or chest pain,\(^9\) nor was there residual neurological deficit beyond the period of loss of consciousness. The patient described by Davis, Glover and Maycock,\(^10\) with cerebral arterial air embolism, not only had a prolonged recovery from general anaesthesia but had a neurological deficit for several days despite hyperbaric oxygen therapy.

Skowronska and Rigg\(^11\) and Philip and Walter\(^12\) described loss of consciousness in patients following the administration of epidural anaesthesia for labour. In both these parturients, the loss of consciousness had occurred, as in our patient, in the presence of remarkable haemodynamic stability. These were described as total spinal anaesthesia. These complications were probably the result of subdural spread. The signs that alerted the anaesthetists to the cranial extension in these case reports were difficulty in breathing,\(^11\) arm weakness\(^12\) and dysarthria.\(^12\) These signs developed slowly with time and the anaesthetists involved were able to detect them. In our case, the loss of consciousness after 20 min of relatively uneventful spinal blockade was the first sign of a possible cranial extension. The loss of consciousness precluded detailed sensory and motor testing in the head, but the gradual and progressive return of function indicated that it could have been compatible with anaesthetic blockade that receded with time. Although we do not have radiological evidence to prove that this incident was caused by subdural blockade, we believe that the clinical events that occurred in our patient are in keeping with this suggestion.\(^5\) These include relatively stable arterial pressure, slow onset of symptoms after 20 min and complete recovery after almost 2 h. The absence of sympathetic blockade\(^14\)\(^15\) is consistent with subdural blockade.

The parturient could have lost consciousness as a psychogenic response to distress felt at the time of the Caesarean section, knowing that her newborn was critically ill and not expected to survive the neonatal period. Hysteria cannot be excluded from the differential diagnosis since measurements taken during the incident were normal except for a transient episode of hypoxia and the 1 h loss of consciousness. Normal antenatal and postnatal mental health makes this diagnosis improbable.

In summary, we have described loss of consciousness in a parturient who had a successful spinal block. The loss of consciousness occurred in the presence of remarkable haemodynamic stability. Despite investigations, we were
unable to determine the cause. We can only speculate that a subdural block complicating the spinal block may have accounted for this most unusual event.

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Traumatic bilateral internal carotid artery dissection following airbag deployment in a patient with fibromuscular dysplasia

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This case describes a 39-yr-old male, presenting with left hemiplegia after a road traffic accident involving frontal deceleration and airbag deployment. Brain computerized tomography (CT) scan revealed a right parietal lobe infarct. Contrast angiography demonstrated bilateral internal carotid artery dissection and fibromuscular dysplasia. The patient was treated with systemic heparinization. Neurological improvement, evidenced by full return of touch sensation, proprioception and nociception began 10 days after the injury. To our knowledge, this is the first case report of carotid artery dissection associated with airbag deployment. Forced neck extension in such settings may result in carotid artery dissection because of shear force injury at the junction of the extracranial and intrapetrous segments of the vessel. Clinicians should consider carotid artery injury when deterioration in neurological status occurs after airbag deployment. We propose that the risk of carotid artery dissection was increased by the presence of fibromuscular dysplasia.

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Airbags were developed to decrease the incidence of death and severe injury following road traffic accidents. Airbags are reported to cause injuries in 43% of deployments. The majority of these are minor (abrasions and contusions), but severe injury such as upper limb fracture occurs in 4% of cases. This case report describes bilateral internal carotid artery dissection, associated with airbag deployment, in a patient with fibromuscular dysplasia.

Case report
A 39-yr-old male driver, height 165 cm, was involved in an acute deceleration, frontal-impact collision. He was restrained by both a seatbelt and an airbag. He was transferred by road ambulance to the Accident & Emergency unit at our institution. On initial examination the patient was haemodynamically stable. His Glasgow Coma Score (GCS) was 10/15 at the time of initial assessment but improved to 13/15 thereafter. Neurological examination revealed a left homonymous hemianopia, left hemiplegia and an upper motor neurone left 7th nerve palsy. Other injuries sustained included metatarsal fractures and anterior neck abrasions. Standard haematological and biochemical investigations and chest and cervical spine radiographs were normal. A brain computerized tomography (CT) scan showed a right parietal lobe infarct.

The patient was transferred to the neurosurgical intensive care unit for invasive arterial pressure monitoring and neurological observations. Supplemental oxygen was administered by facemask. Aspirin 300 mg once daily by mouth was prescribed for its antiplatelet effect. A second brain CT on hospital day 2 demonstrated haemorrhagic transformation of the right parietal lobe infarct. Duplex scan and magnetic resonance angiogram of the carotid arteries was inconclusive. Contrast angiography revealed bilateral internal carotid artery dissection, fibromuscular dysplasia of the internal carotid arteries and thrombus in the right internal carotid artery (Fig. 1). Heparin was commenced with a target activated partial thromboplastin time of 60 s. Warfarin was commenced with a target prothrombin time of 26 s.

Recovery of neurological function began with the return of normal GCS on hospital day 3. By day 10, touch sensation, proprioception and nociception in the left upper and lower limbs were evident. Left lower limb power had improved to grade 3/5 on day 24. However, the left facio-brachial palsy persisted. The patient was transferred to a rehabilitation hospital on day 26.

Discussion
Blunt carotid injury presents typically with neck abrasions and neurological sequelae, including hemiplegia, low GCS and a co-existing Horner’s syndrome when the sympathetic chain is stretched across the transverse process of the first cervical vertebrae. Road traffic accidents are the most frequent cause of blunt carotid injury. The mechanism of injury to the internal carotid artery is thought to be a shear force applied to the vessel during forced neck extension, causing intimal dissection at the junction of the immobile intracranial and relatively mobile extracranial portions of the internal carotid artery.

Fibromuscular dysplasia is an arterial developmental abnormality of unknown aetiology affecting medium-sized arteries. The incidence of the disorder based on a series of 20 244 post-mortems is two per 1000 population. There are four known subtypes, each with its own unique histological appearance (intimal fibroplasia, medial fibroplasia, medial hyperplasia and perimedial dysplasia). Affected vessels are elongated, kinked and punctuated with stenoses and microaneurysms. These abnormalities produce unique angiographic features which radiologists call the ‘string of beads’ appearance (Fig. 1). The presence of fibromuscular dysplasia may compromise the structural integrity of the affected vessels, including the carotid artery, and therefore predispose to vessel dissection after traumatic injury.

Patients with fibromuscular dysplasia are asymptomatic until they sustain a vessel dissection. They then present with
ischaemia of the organs supplied by the affected vessel, for example cerebrovascular accident. Fibromuscular dysplasia is found in 15% of cases of spontaneous carotid dissection. A report of bilateral internal carotid and vertebral artery dissection in a patient with fibromuscular dysplasia after a road traffic accident has been described. Arteriography is the gold standard diagnostic test for both fibromuscular dysplasia and blunt carotid injury.

The management of carotid artery dissection is controversial. Heparinization was associated with an improved outcome in the largest series of blunt carotid injuries. However, the safety and efficacy of anticoagulation in the management of stroke following carotid injury has not been demonstrated. In our patient, the apparent benefit of heparinization had to be balanced against the risks of a worsening stroke.

Airbag usage is increasing worldwide and is now mandatory in new cars in some countries. Airbags function by preventing traumatic impact between the vehicle occupant and the vehicle interior. During a road traffic accident, sensors in the vehicle body detect deceleration and trigger airbag deployment. To protect against injury, airbags must be fully expanded before the vehicle occupant is propelled forward by the deceleration of the accident. An airbag expansion speed of 200 mph is necessary. This rapid forceful expansion causes airbag-associated injury in 43% of deployments. In addition, fatal rupture of the ventral ligaments of the cervical spine and brain stem has been described secondary to forced neck extension.

An increase in the incidence of airbag-associated injury is predicted. Drivers of short stature, as in this case, are at increased risk of these injuries. Their short stature makes it necessary to move the vehicle seat forward in order to reach the foot pedals, thereby bringing the upper body into the path of the expanding airbag. In the USA it is now recommended that airbags should be decommissioned if drivers cannot keep a wheel–chest distance of 10 inches.

The evidence for airbag-mediated blunt carotid injury by means of forced neck extension is compelling in this case. A less likely explanation is that the patient initially sustained spontaneous carotid artery dissection because of fibromuscular dysplasia and then crashed. However, spontaneous dissections of the carotid artery typically present initially with minor clinical manifestations (e.g. headache), and only develop neurological signs after several days.

In summary, this is the first case report of bilateral internal carotid artery dissection, in a patient with fibromuscular dysplasia, associated with airbag deployment. The report suggests that clinicians should consider the possibility of carotid injury when neurological deterioration occurs after a road traffic accident with airbag deployment. Associated conditions, such as fibromuscular dysplasia, may increase the risk of carotid artery injury in this setting. Early carotid angiography is warranted to make the diagnosis.

References

Anaesthetic management of a pregnant patient in a persistent vegetative state

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Pregnancy in a patient in a persistent vegetative state presents challenging therapeutic questions about the level of supportive management required, the assessment of fetal well-being, the timing and mode of delivery and the anaesthetic management of labour and delivery. We report the case of a 29-yr-old woman who had a favourable fetal outcome despite suffering hypoxic brain damage after a suicide attempt by a drug overdose. She was managed until the onset of labour on an intensive care unit and had a spontaneous vaginal delivery assisted by epidural anaesthesia.

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The term persistent vegetative state (PVS) describes a state of continuing ‘wakefulness without awareness’ that is usually due to a severe brain insult from a variety of possible causes.¹ PVS involves damage to the central nervous system, other systems being essentially normal. A pregnancy may be viable, creating an unusual scenario requiring difficult clinical and ethical decisions. In this report, we discuss the anaesthetic and intensive care management of a 29-yr-old patient who suffered hypoxic brain injury after a failed suicide attempt at 12 weeks gestation.

Case report

A 29-yr-old lady was 12 weeks pregnant with her second pregnancy. She had a history of depressive illness. There was also a history of marital discord. While abroad with her family, she attempted suicide and overdosed on a cocktail of antihypertensives (which belonged to her father-in-law), anxiolytics and antidepressants. The patient was found unconscious, and after admission to an intensive care unit (ICU) a computer tomographic (CT) scan of her brain showed gross ischaemic changes in the cerebral cortex consistent with severe hypoxic brain damage.

After air transfer to our ICU, 4 weeks after the suicide attempt, she had a Glasgow Coma Score (GCS) of 7 (spontaneous eye opening, no verbal response and an extensor response to pain). She responded to distressing situations by becoming increasingly agitated, sweaty and tachypnoeic. A tracheostomy had been performed at the referring hospital. The tracheostomy tube was changed to a fenestrated type to facilitate spontaneous ventilation, an enterostomy feeding tube was sited and a central venous catheter (CVP) was inserted to aid fluid management. She was treated for multiple respiratory, urinary and gastrointestinal infections with appropriate antibiotics, but continued to demonstrate intermittent pyrexia and persistent tachycardia.

A fetal ultrasound scan showed that the fetus was intact and small for date at 16 weeks, but did not appear to have any anatomical abnormality. A plan to manage the pregnancy expectantly was made. This involved weekly clinical assessment supplemented by regular ultrasound assessment of fetal growth, liquor volume and umbilical artery waveforms. In the absence of fetal or maternal indications, a requirement for early delivery or Caesarean section was not anticipated. These findings were discussed extensively with the family, who were particularly concerned about fetal survival. Arrangements were made initially to transfer the patient to a rehabilitation unit once her condition had stabilized. However, this decision was reversed after careful consideration of the risks to the patient and the fetus. She therefore remained in the ICU until delivery. Whilst in the ICU she required substantial nursing care, although she was not artificially ventilated.

The ICU nursing staff were instructed in the palpation of uterine contractions and encouraged to request midwifery support if in doubt. At 35 weeks gestation, the patient developed regular uterine contractions, which seemed to be
causing considerable distress. A lumbar epidural catheter was sited in the lateral position and an infusion of 0.25% bupivacaine commenced at a rate of 6–10 ml h⁻¹, which was adjusted according to the patient’s agitation and the cardiovascular parameters. She was transferred to the delivery suite after spontaneous rupture of the membranes occurred. Continuous fetal heart rate monitoring identified a baseline rate of 140 beats min⁻¹ with normal variability.

Taking into account the patient’s previous normal delivery and the small size of the fetus, it was decided to allow a trial of labour and to anticipate vaginal delivery. However, it was felt that there should be a low threshold for operative delivery. As the patient was not in established labour, the fetal heart rate decelerations, the need for maternal analgesia and the suspected rupture of membranes prompted a decision to induce labour by artificial membrane rupture with oxytocin augmentation. The patient appeared comfortable with the epidural solution being infused at a rate of 8 ml h⁻¹. Four hours after admission to the delivery suite, she delivered a healthy 2.2 kg boy by spontaneous vaginal delivery, who had an Apgar score of 10 at 5 min. She was transferred to a rehabilitation unit 2 days later.

Discussion

This is the first report, to our knowledge, of a pregnancy in which the fetus survived maternal PVS during the entire second and third trimesters, thus presenting a therapeutic dilemma during the pregnancy and at delivery of the fetus. PVS is a state of eyes-open unconsciousness with sleep–wake cycles in which patients are incapable of awareness of themselves or their environment.⁵ The use of the term PVS usually implies that the vegetative state has continued for more than 1 month.⁶ A patient in this state is not expected to recover, although this is the subject of controversy. There have been rare documented cases of late recovery of patients who had been diagnosed as being in a PVS.⁷ Hence the diagnosis should be considered carefully before it is made because of the enormous financial, emotional and social implications of the management of these patients. Apart from establishing lack of awareness, the cause and irreversibility of the syndrome must be obvious.⁸ The differential diagnosis includes prolonged coma (a state of unconsciousness in which the eyes are closed and sleep–wake cycles are absent) and the locked-in syndrome (resulting from brainstem lesions which disrupt the voluntary control of movement without abolishing either arousal or the content of awareness), both of which can also result from severe brain injury.⁹

Patients with PVS usually breathe spontaneously, although a tracheostomy may be necessary to ensure airway protection. Patients may exhibit a bizarre variety of neurological signs, including arousal to painful stimuli, grimacing, limb movement, spontaneous head movements, chewing, and grinding the teeth.⁴ Most of these features were present in our patient, but with the additional feature that she was pregnant, exposing her to the potential risk of obstetric complications such as aortocaval compression, pregnancy-induced hypertension and premature labour. Termination of the pregnancy was not considered appropriate as the fetus had survived the hypoxic insult of a suicide attempt and had a good chance of survival as long as the mother was alive.

The choice of an appropriate place to manage the patient was a contentious issue. Managing her in a side room in the delivery suite would have been considered as a cheaper alternative to high-dependency or intensive care. However, this would not have provided optimal patient care in view of the presence of a tracheostomy tube and the lack of appropriate expertise in airway management among the labour ward staff. The potential requirement for artificial ventilation, the degree of nursing and physician input required and the obstetric and fetal risks indicated that the intensive care unit, rather than the high-dependency unit, was the most appropriate place to optimize her management. Once the fetus had been delivered without any maternal complications, the patient was deemed to present a risk profile similar to a non-pregnant patient in a PVS, who would usually be managed safely in a rehabilitation unit.

Assessment of pain and the provision of safe and effective analgesia during labour and delivery in a patient in a PVS is challenging because the patient cannot communicate. Physiological parameters such as tachycardia, hypertension and tachypnoea are commonly used as indicators of inadequate analgesia, but all lack specificity. The safety and efficacy of epidural analgesia for the management of labour pain is well documented.⁵ Despite the absence of reports describing the use of epidural analgesia for a similar case, we could not find any justifiable reason for withholding epidural analgesia in our patient. We decided to use a plain bupivacaine infusion to avoid the further neurological depression that may have been induced by opioids. A continuous infusion, rather than bolus doses of epidural local anaesthetic, was chosen to minimize cardiovascular compromise, especially since dermal sensory levels of the block could not be assessed. The medical condition of the patient, the fetal heart rate decelerations in the intrapartum period and the anaesthetic considerations indicated a low threshold for emergency Caesarean section. Although regional anaesthesia is now established as the preferred technique for Caesarean section,⁹ this requires a dense sensory block extending from the fourth thoracic dermatome to the fifth sacral dermatome.¹⁰ However, we could not establish the extent of the block in this patient. Therefore, our anaesthetic plan in the event of emergency Caesarean section was to administer a general anaesthetic. Provision was made for a cuffed tracheostomy tube and antacid prophylaxis, as for any obstetric patient undergoing general anaesthesia. The epidural could also have been used for postoperative analgesia.

In conclusion, this unusual case illustrates that pregnant patients in a PVS may receive low-dose epidural analgesia

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in labour, but general anaesthesia would be indicated for Caesarean section. Nursing support, medical management, fetal assessment and deciding the optimum time for delivery are challenging decisions requiring a multidisciplinary approach to this clinical situation.

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