

needle. The dural fistula allows continued leakage of cerebral spinal fluid (CSF) with caudal displacement of the brain and tearing of intracranial subdural veins with formation of a subdural hematoma.¹ Dural fistulae following spinal anesthesia have been shown to persist for as long as 18 weeks,² and the volume of CSF lost may be over 200 ml per day which could exceed normal production.³ Under these circumstances, the rupture of subdural veins is certainly conceivable. Ordinarily, one would not expect a dural puncture using a 25-G needle to produce a tear sufficiently large to cause such a serious problem. It is possible, however, that multiple dural punctures could result in sufficient CSF leakage to tear fragile veins in the cranium.

The symptoms of subdural hematoma are somewhat different from the usual postspinal headache in that the headache is more severe and persistent, even in the recumbent position. Associated symptoms of subdural hematoma include vomiting, blurring of vision, drowsiness, and disorientation. It must be emphasized, however, that many or all of these symptoms may not be apparent. The patient that we report demonstrated none of the above associated symptoms. Also, there were no localizing neurologic signs or papilledema. It is possible that the subdural hematoma occurred coincidentally and was entirely unrelated to the lumbar puncture. Trivial head injury may lead to a subdural hematoma in elderly patients and spontaneous hematoma may occur in patient treated with anticoagulants.⁴ This patient, though somewhat elderly, gave

no history of even minor trauma and was not taking anticoagulants. Whether the subdural hematoma was coincidental or not, the fact that the patient had a spinal anesthetic must not cloud the possibility that the symptoms are a result of subdural hematoma.

The failure to make an early diagnosis of subdural hematoma may result in additional complications. Four of 14 cases reviewed by Newrick and Read resulted in death, and one other patient suffered persistent visual loss.⁵ Consequently, when a patient complains of severe, persistent headache following spinal anesthesia unrelieved by conservative measures or epidural blood patch, one should consider the possibility of subdural hematoma. Current diagnostic procedures such as computed axial tomography should be employed so that appropriate treatment can be initiated before death or irreversible neurologic damage occurs.

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Epidural Opioids for the Management of Pain in a Patient with the Guillain-Barré Syndrome

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The Guillain-Barré Syndrome (GBS) is an acute post-infectious polyneuropathy characterized by demyelination of the peripheral nervous system and rapidly progressive

paralysis. The primary neurological deficit is motor loss, often accompanied by paresthesias. Pain, often severe, is a common manifestation of the disease that poses a difficult therapeutic challenge. We report a case of GBS in which severe pain was a prominent symptom that was effectively treated with epidural opioids.

CASE REPORT

A 34-yr-old female presented to the emergency room with complaints of pain in her lower back, later accompanied by weakness and paresthesias in her lower extremities. She was admitted to the hospital and analysis of CSF was consistent with a diagnosis of GBS with an elevated protein of 51 mg/dl. During the first 24 h in the hospital she was transferred to the intensive care unit where her trachea was later intubated when her vital capacity decreased. She then rapidly developed flaccid quadriplegia, diplopia, and facial muscle weakness. An EMG

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was consistent with a demyelinating peripheral neuropathy. A tracheostomy was performed and she was treated with eight courses of plasmapheresis. Steroids were not administered. After 9 days she began to regain function of her facial muscles; however, she required mechanical ventilation of her lungs for 35 days. She was transferred from the intensive care unit on hospital day 45 and continued to show slow but steady improvement.

From the time of admission, pain was severe and difficult to manage. While her lungs were being ventilated she could only relate that the pain was severe in her back and lower extremities and less severe in her upper extremities. Later in her recovery she was able to describe two types of pain. The most severe was deep and sharp in her extremities and lower back. Another distinct complaint was a constant burning sensation in her feet.

Intravenous opioids were ineffective in controlling her pain without the patient being unresponsive. Based upon the effectiveness of anti-convulsant medications in the treatment of neuralgic pain,¹ phenytoin and later carbamazepine were administered with therapeutic serum concentrations. Neither medication provided effective analgesia. Phenothiazines demonstrate opiate receptor affinity and several case reports document their effectiveness in the treatment of chronic pain.¹ Fluphenazine was administered to our patient with a significant decrease in her pain, but was discontinued due to the development of dyskinesias and visual hallucinations. A transcutaneous nerve stimulator was also ineffective.

Because of our inability to control her pain and given the report by Rosenfield, Burel, and Hanley describing the use of epidural morphine for the treatment of pain in patients with GBS,² an epidural catheter was inserted *via* the L3-4 interspace on day 30. Fentanyl was used initially (75 µg in 10 ml of NS) with complete resolution of the pain in her back, upper, and lower extremities. We then substituted intermittent epidural injections of morphine with continued good results. On day 37 a permanent Infusaport (Infusaid Co., Norwood, MA) epidural catheter was placed. In order to provide continuous analgesia a continuous infusion of epidural morphine was begun and this continued to provide her with excellent analgesia as her recovery continued. On hospital day 67 epidural opioids were discontinued, and all opioids were discontinued after 3 days of oral morphine. Five days later the epidural catheter was removed under epidural anesthesia. The burning sensation in her feet persisted but was tolerated without difficulty and further trials of phenothiazines, tricyclic antidepressants, or use of local anesthetics in the epidural space were not pursued.

DISCUSSION

The incidence of pain as a manifestation of GBS ranges from 50–76%.³ Ropper and Shahani described the characteristics of pain in 29 patients with GBS.³ Sixteen of the 29 patients had pain early in the illness and 6 of 29 developed pain later in their course for an overall incidence of 76%. This correlates with Haymaker and Kernchan's series of 50 patients of whom 28 experienced pain early in their course.⁴

In an attempt to define clinical correlates of pain in GBS, Ropper and Shahani demonstrated the "absence of correlation between the incidence of pain and inflammation of dorsal root ganglion." Serum creatine kinase concentrations were increased in 10 of 13 patients with pain and only 1 of 8 patients without pain. Based on these findings, and the distribution of pain in the regions of weakest muscles, Ropper and Shahani postulated that al-

terations in neural input and sensation from muscle are the causes of this type of pain. It is this pain that responds well to epidural opioids.

Pain associated with GBS appears to be of two distinct types. One is a deep pain most commonly in the back and lower extremities and less commonly in the upper extremities and correlating with the distribution of motor loss. This is associated with tenderness and pain with passive movement of the affected muscle groups.⁵ Distinguished from this are symptoms that resemble causalgia with hyperesthesia and a constant burning sensation in the extremities. Trophic changes in the extremities have not been described. It was the "muscular" type of pain that most bothered our patient and responded well to epidural opioids. Although the burning sensation was tolerable, it did not respond to epidural opioids and supports a distinction between the two types of pain, suggesting a different mechanism for each.

Although the use of epidural opioids for the treatment of pain in GBS has been reported previously,¹ we report a distinction between the symptoms of sharp, deep pain in the distribution of paralyzed muscle groups that responded well to epidural opioids, and the symptoms of dysesthesia that did not respond to epidural opioids. Although the mechanism of pain in GBS is not well understood, this distinction would suggest at least two different mechanisms. Possible mechanisms include radicular pain related to inflammation and entrapment of nerve roots, peripheral neuralgia related to alteration in function as a result of demyelination, and an imbalance in neural input to the dorsal horn of the spinal cord. The perception of pain is transmitted to the dorsal horn of the spinal cord by small unmyelinated C fibers and larger myelinated A delta fibers. Melzack and Wall proposed the "gate control" theory of pain to explain the modulation of sensory input in the dorsal horn of the spinal cord and the subsequent perception of pain.⁶ According to this theory the larger myelinated fibers exert an inhibitory influence on cells in the substantia gelatinosa (SG), while smaller unmyelinated fibers exert an excitatory influence. One could postulate that demyelination of peripheral nerves alters the normal balance of sensory input from myelinated and unmyelinated fibers to the dorsal horn of the spinal column accompanied by the perception of pain. One could further speculate that stimulation of opiate receptors in the dorsal horn of the spinal cord provides analgesia by modulating this sensory imbalance in a similar way that spinal opiates are thought to provide analgesia in a variety of other situations.

Although parenteral opioids were helpful initially, tolerance developed quickly and we were unable to achieve analgesia without excessive sedation. Other therapeutic interventions were unsuccessful as described. Epidural opioids were effective in treating what may be pain of

muscular origin and should be considered early in the course of GBS when other modalities are not effective.

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Beneficial Effect of Delivery in a Patient with Adult Respiratory Distress Syndrome

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The management of adult respiratory distress syndrome (ARDS) often requires support of respiratory and cardiovascular function and is particularly difficult in pregnant patients. ARDS has been reported to occur in pregnancy secondary to many different etiologies.¹⁻¹⁵ However, there is limited information in these studies concerning the effect of delivery on gas exchange in ARDS. We report a patient in her third trimester who developed ARDS requiring mechanical ventilation and PEEP. Following delivery her pulmonary status improved markedly.

CASE REPORT

A 49-kg, 160-cm, 19-yr-old woman G1P0 was hospitalized with a complaint of a 2-day history of abdominal pain. Her 31-week pregnancy had been uneventful except for hyperemesis in the first trimester. She denied any previous medical problems. Her vital signs were temperature 38.5° C, blood pressure 93/41 mmHg, pulse 102 beats/min, and respiratory rate 16 breaths/min. Physical examination revealed an enlarged uterus consistent with 31-week gestation and right lower quadrant tenderness. Uterine contractions were occurring every 2-5 min. Laboratory studies included 19,900 leukocyte/mm³ with 64 polymorphonuclear cells, 32 bands, and 4 lymphocytes. Urinalysis was normal. A magnesium sulfate infusion was begun to treat preterm labor at 3:40 A.M. of her first hospital day. No sympathomimetic drugs were administered.

At 5 A.M. on the first hospital day, the patient was taken to the operating room with a diagnosis of acute appendicitis. A spinal anes-

thetic was performed at the L3-4 interspace with the patient in the left lateral decubitus position; 1.2 ml of bupivacaine 0.75% in 8.5% dextrose with 0.2 ml of epinephrine 1:1,000 was administered. Exploratory laparotomy revealed an erythematous appendix and no other abnormalities. Intraoperatively, the only additional drug the patient received was 1.25 mg droperidol for nausea. She vomited during the laparotomy but did not develop clinical signs of aspiration. She was discharged from the peri-anesthetic care unit (PACU) to her room at 8:30 A.M. with a respiratory rate of 26 breaths/min. At 9:40 P.M. on the second hospital day, 39 h after operation, she developed tachypnea of 40-50 breaths/min. Arterial blood gases (ABG) while breathing room air were pH 7.47, PaCO₂ 33 mmHg, and PaO₂ 42 mmHg (table 1). Magnesium sulfate was discontinued and she was transferred to the surgical intensive care unit (SICU). Upon admission to the SICU, ABG while breathing 100% O₂ via a nonbreathing mask were pH 7.44, PaCO₂ 34 mmHg, and PaO₂ 83 mmHg. Continuous positive airway pressure (CPAP) therapy via a mask failed to improve her ventilatory status sufficiently and her trachea was intubated at 4 A.M. on the third hospital day, 46 h after operation. Total fluid intake from hospital admission to intubation (50 h) was 6,010 ml and total output was 4,835 ml. Ventilator settings included tidal volume of 600 ml with an intermittent mandatory ventilation (IMV) rate of 10. Radial and pulmonary artery catheters were inserted. Initial hemodynamic measurement included pulmonary capillary wedge pressure (PCWP) of 15 mmHg and cardiac index of 4.59 l · min⁻¹ · m⁻² (table 2). A chest x-ray revealed extensive bilateral pulmonary edema. Fetal monitoring demonstrated a heart rate of 140 beats/min, good variability, and occasional contractions. PEEP was increased to 10 cmH₂O to decrease FI_{O₂} to 0.4. These ventilator settings resulted in PaO₂ 60 mmHg, SaO₂ 92.4%, cardiac index 4.63 l · min⁻¹ · m⁻², and Q_o/Q_t 28%. Five hours prior to delivery, she received 50 mg of meperidine iv for analgesia. Spontaneous labor progressed to unassisted vaginal delivery at 8:36 P.M. on the third hospital day, 62 h after operation. Peripartum arterial blood gas (table 1) and hemodynamic data (table 2) demonstrated the impact of delivery on the patient's ARDS. The patient's gas exchange was stable for several hours prior to delivery. At the time of delivery arterial desaturation was noted by pulse oximetry and FI_{O₂} was increased to 1.0. Blood gases and hemodynamic data collected during delivery revealed a 47% increase in oxygen consumption (\dot{V}_{O_2}) but only a 17% increase in cardiac index. Consequently, Sv_{O₂} decreased to 54.1% and oxygen extraction (SaO₂ - Sv_{O₂}/SaO₂) increased to 0.37. Following delivery gas exchange returned to predelivery levels within 2 h. The patient's ventilatory status improved sufficiently to allow tracheal extubation 18 h postpartum. Maternal fluid intake from delivery to extubation was

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