under enflurane and nitrous oxide. These agents may all produce respiratory depression, but the length of time following the induction, his alertness, and subjective dyspnea and tachypnea prior to nasotracheal intubation would suggest minimum contribution of drug-induced central respiratory depression. Also, his ability to sit unassisted and recruit accessory muscles of respiration without subjective weakness argue against significant residual paralysis from muscle relaxants. Chest wall motor blockade with loss of the contribution of intercostal and rectus muscles to inspiration and exhalation appears to be the major cause of respiratory failure in this patient.

The loss of abdominal and lower intercostal muscle power can compromise ventilation, even in normal subjects. Eisele *et al.* found that four patients without preexisting pulmonary disease who were given spinal or epidural anesthesia to the T-1 motor level, had a 42–80 per cent reduction in inspiratory capacity.[4] Freund *et al.* have similarly found that spinal anesthesia to the T-5 level also reduces expiratory reserve volume by 48 per cent.[5] The work of Jakobson and Ivarsson suggested that functional residual capacity is reduced following intercostal block.[6,7] Sharp *et al.* have further shown that patients with chronic obstructive disease may depend more on the rib-cage muscles than the diaphragm to produce tidal ventilation compared to healthy patients.[8] Fourteen of their 20 patients produced maximal forced inspiration mostly by the use of rib-cage muscular motion. In 5 to 20 patients, they saw an unusual decrease in abdominal girth in normal tidal breathing on inspiration, and concluded that patients with COPD are liable to have abnormal diaphragmatic function and dependence on rib-cage musculature.

Thus, in patients with chronic pulmonary disease, the intercostal muscles may play a crucial role for ventilation. Intercostal nerve block following surgery may enhance the development of respiratory failure in this group. Patients with preexisting pulmonary disease would profit from a critical clinical appraisal of the contribution of their intercostal musculature to their "normal" ventilatory pattern before considering an intercostal nerve block. If an intercostal block is performed, an assessment of arterial blood gases and ventilatory flow measurements should be performed to assure the adequacy of ventilation.

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Anesthesiology
54:419–421, 1981

The Case of the Errant Epidural Catheter

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Continuous lumbar epidural analgesia via catheter for analgesia during labor is a popular and effective technique. Occasional difficulty during removal of the catheter is usually resolved by flexing the patient's back and applying steady traction to the catheter. We present an instance of an epidural catheter which knotted itself around a strand of ligamentum flavum

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Received from the Departments of Anesthesiology and Surgery, Wright State University School of Medicine, Dayton, Ohio 45435. Accepted for publication November 3, 1980.

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Key words: Anesthetic techniques: epidural, lumbar. Anesthesia: obstetric. Equipment: catheters, peridural. Complications.

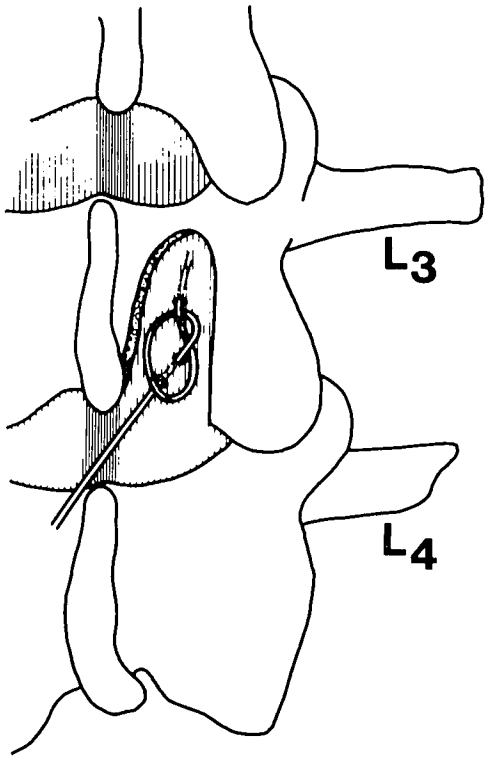

FIG. 1. Illustration showing position of epidural catheter as revealed by partial laminectomy of 3rd lumbar vertebra.

which prevented removal thus necessitating lumbar laminectomy.

REPORT OF A CASE

A 24-year-old, gravida 1, para 1 woman entered the hospital at term, in early labor. The patient's height was 165 cm and her weight 113 kg, having gained 28 kg during her pregnancy. Five hours later, an epidural catheter§ was inserted in the L3–4 interspace using loss of pressure technique with the patient in the sitting position. No unusual resistance to passage of the catheter was felt. Following a test dose of 2 per cent 2-chloroprocaine 3 ml, 0.25 per cent bupivacaine was used for analgesia. The patient remained comfortable throughout the labor with minimal loss of motor function. Labor progressed uneventfully, and the cervix was completely dilated six hours later. Attempted vaginal delivery by forceps was unsuccessful because of the operator's inability to obtain satisfactory application of the blades of the forceps. The operator believed that continued attempts at vaginal delivery would be traumatic to the neonate's head, and therefore a cesarean section was performed under epidural anesthesia. The infant had Apgar scores of 9 and 10.

The epidural catheter remained *in situ* until the patient was ready to be discharged from the obstetric recovery room. At this time, approximately 8 hours postpartum, there was difficulty in removing the epidural catheter. With the patient's back flexed, gentle traction stretched the catheter but did not withdraw it. The fifth black mark indicating a 10-cm length was visible just outside the skin. A possible knot in the catheter was postulated. In a portable roentgenogram the catheter could not be visualized in the ligaments or epidural space, perhaps due to the patient's obesity. The patient was informed that the catheter might have a knot in it. The alternatives presented were surgical removal or leaving it *in situ*, cut just beneath the skin. The patient at that time stated that she would rather have it removed.

After consultation with a number of colleagues we decided that the patency of the catheter should be checked and if patent, a water soluble radiopaque dye should be injected through the catheter and a roentgenogram taken to visualize its path. The catheter was found to be patent and the patient was scheduled for roentgenogram the following morning.

That evening, the member of the staff who had administered the epidural anesthetic returned to duty and attempted to remove the catheter. Unfortunately, during the procedure the catheter snapped and slid back beneath the skin. The projected roentgenogram was thus abruptly aborted and neurosurgical consultation was obtained. Since the cut end of the catheter was potentially contaminated, exploration of the back was scheduled under general anesthesia. Partial laminectomy was performed and the catheter found tied into a knot around a strand of the ligamentum flavum around the epidural space and was removed (fig. 1). Postoperatively, the patient did well with no motor or sensory deprivation, and was discharged six days later.

DISCUSSION

An account of a knot in a lumbar epidural catheter which was successfully removed by steady gentle traction was reported by Bromage[1] who stated that it was the only case encountered in over 30,000 catheter insertions. Three further cases of knotted catheters (all in the caudal canal) have been described.[2,3] In the first case, the end of the catheter was sheared with the needle, leaving a frayed end protruding from the patient's back. The catheter was removed by surgical exploration of the sacral area. In the second case, following a successful caudal anesthetic for delivery, attempts to remove the catheter were unsuccessful. The catheter was grasped with forceps and tension applied forcing the catheter to break. Again surgical exploration was performed to remove the frayed portion of the catheter, which had looped itself into a complete double knot deep in the sacro-coccygeal ligament. In the third case, a catheter supposedly was located in the caudal canal; however, satisfactory anesthesia was not accomplished. The patient was then anesthetized with nitrous oxide and pudenal block. Attempts to remove the caudal catheter were met by resistance and stretching of the catheter. A roentgenogram revealed a knot in the distal end of the catheter lying anterior to the sacrum. Prolonged gentle traction resulted in its successful removal.

The possibility of knotting probably is related to the insertion of too much catheter into the epidural space. In our case, the catheter had pierced the ligamentum flavum three times and knotted itself outside the ligament between the second and third ligamentum punctures. It is difficult to conceive of a fairly soft catheter

§ Abbott Laboratories.

performing this maneuver while appearing to pass easily. We believe the first two punctures were caused by the needle. The catheter then advanced superficial to the ligament, coiled around in a knot, and then fortuitously pierced the ligament once to enter the epidural space. Withdrawal of the needle then left the catheter passing through the ligament three times.

Shearing of the end of the catheter within the epidural space by erroneously retracting the catheter with the needle still in the patient is probably not an uncommon occurrence. Surgical removal is almost certainly unnecessary.[4] However, when the catheter cannot be removed and the end of the catheter is outside the skin, a potential tract for infection into the epidural space is present. In our case, the end of the catheter had been outside and in contact with the skin and subsequently retracted into the tissues. As such, these catheters should be removed.

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Anesthesiology
54:421–423, 1981

Stellate Ganglion Block for Sudden Profound Hearing Loss

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The onset of a sudden sensorineural hearing loss in a practicing anesthesiologist stimulated a review of the pathophysiology of labyrinthine structures and the recommended treatment for sudden deafness which is often referred to as sudden profound hearing loss (SPHL). Although a stellate ganglion block has been recommended for therapy of SPHL,[1–4] clinical experience has not been reported. The following is a case report of the use of stellate ganglion block in a patient with SPHL.

Report of a Case

A healthy, 34-year-old man was swimming and noticed tinnitus in his right ear after diving into 12 feet of water. There was no associated vertigo or ear pain. During the next three days, the tinnitus persisted and increased. On the evening of the third day, the patient noted inability to hear the telephone ring while listening with his right ear and sought consultation the following day. An audiogram was performed on the fourth day which revealed a pure tone average in the right ear of 65 dB (fig. 1). The patient was not vertiginous, had no perforation of the tympanic membrane, hemotympanum or fluid in the middle ear space. He had a negative fistula test and a normally mobile tympanic membrane by tympanometry. Since there was no clear-cut evidence for a round or oval window rupture, surgery was not performed and a series of stellate ganglion blocks were planned.

A series of stellate ganglion blocks were performed using the anterior paratracheal approach. Because of the success of Haug,[4] the blocks were repeated every 12 hours utilizing 0.5 per cent bupivacaine, 10 ml. There was rapid improvement in his hearing documented by serial audiograms (fig. 1). A visible Horner's syndrome (miosis, ptosis, anhidrosis) with unilateral nasal congestion gave evidence of an effective block. These unpleasant side effects were successfully reversed following instillation of 1–2 drops of 10 per cent phenylepherine HCl in the affected eye.[5] A block of the recurrent laryngeal nerve was elicited twice and a partial brachial plexus block once during the series. Upon completing eight blocks, full recovery was noted by audiogram (fig. 1). Three months later, this was still present without relapse.

Discussion

Although the cause of SPHL has not been elucidated, conditions that are known to produce abrupt, usually unilateral sensorineural losses are mumps, measles, meningitis, encephalitis, acoustic tumors, ear surgery, skull fracture, ototoxic drugs, and perilymphatic fistula from exertion or barotrauma. There are several reports of idiopathic sensorineural hearing loss.[2–4] The basic mechanisms are broken down to either a viral or vascular etiology responsible for 25 and 75 per cent of cases, respectively. They all have a similar clinical picture. The typical patient is one with serviceable hearing bilaterally who suddenly becomes deaf in one ear. The abruptness of onset may

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Received from the Departments of Anesthesiology and Otolaryngology, Naval Regional Medical Center, Camp Lejeune, North Carolina, and the Department of Anesthesiology, Naval Regional Center, Newport, Rhode Island. Accepted for publication November 6, 1980.

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0003-3022/81/0500/0421 $00.65