145-cm, 0.0032-inch sterile J-wire (Cook) was threaded through the suction port of the FOB (with suction control ring removed) and its endotracheal position confirmed by visualization. The FOB and LMA were removed from the trachea, while the J-wire position was maintained carefully. The FOB then was reinserted into the trachea over the wire through its suction port in an antegrade fashion. Tracheal placement was again confirmed by fiberoptic visualization. A lubricated 7.0 endotracheal tube that previously had been loaded onto the FOB was passed over it into the trachea using a rotating motion and mild anterior traction of the tongue. The FOB and J-wire were removed and ventilation was achieved through the endotracheal tube.

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Application of Negative-pressure Ventilation When Changing Endotracheal Tubes

To the Editor:—Negative-pressure ventilation using a chest shell or cuirass without an artificial airway has been shown to be an effective form of ventilation in patients with chronic respiratory insufficiency.[1] Using this type of ventilatory support, we successfully performed the exchange of an endotracheal tube from oral to nasal route in a quadriplegic patient who could not breathe spontaneously.

Recently, we were asked to change an orotracheal tube to a nasotracheal tube in a 41-yr-old woman suffering from cervical spinal cord injury. Her head and neck were rigidly fixed with a halo and chest cast so that even her epiglottis could not be visualized with direct laryngoscopy. Cricothyrotomy or tracheostomy could not be performed because of an infected neck wound.

Initially, we tried to exchange the endotracheal tubes using two precautions: the passage of a hollow 6.0-mm JEM tube changer through the existing orotracheal tube and the passage of a 4.8-mm fiberoptic bronchoscope (p 10, Olympus) *via* the nose into the trachea alongside the tube changer. However, with the tube changer in place and even with the endotracheal tube withdrawn, the fiberoptic bronchoscope could not be advanced through the glottis. Thus, the orotracheal tube was reinserted over the tube changer.

We then prepared a small-sized tube changer (7-Fr Mettro, Cook) and a 3.5-mm fiberoptic bronchoscope (3C 20, Olympus). We also obtained a negative-pressure ventilator (NEV-100, Lifecare) to maintain ventilation during exchange of the endotracheal tubes because the prepared small-sized tube changer was not hollow and thus jet ventilation could not be applied.

The patient's lungs were ventilated with 100% O_2, and midazolam (10 mg), pentazocine (30 mg), and vecuronium (4 mg) were administered intravenously. The 3.5-mm fiberoptic bronchoscope, which was jacketed on its proximal end with a new endotracheal tube, was passed through the nostril, and the glottis was observed. The 7-Fr Mettro tube changer was inserted through the orotracheal tube, and the tube was withdrawn. At the same time, ventilation using the NEV-100 was begun. Thereafter, the fiberoptic bronchoscope was easily passed into the trachea, and the nasotracheal tube was passed over the fiberoptic bronchoscope into the trachea. During the 8 min that elapsed between removal of the orotracheal tube and insertion of the nasotracheal tube, ventilation was maintained by the negative-pressure ventilator with oxygen insufflation, resulting in the arterial oxygen saturation by pulse oximetry at 98–100%. Immediately after reintubation, end-tidal carbon dioxide was 48 mmHg.

In the literature, we found a report by Benumof[2] that elaborates on his successful change of the endotracheal tubes by use of a method similar to our first procedure. In his method, the tube changer allows for jet ventilation if the new endotracheal tube does not enter the trachea. However, we failed to exchange endotracheal tubes using this technique, because the sizes of the tube changer and fiberoptic bronchoscope were too large for the patient. Therefore, we prepared a small-sized tube changer. Since it was not hollow, jet ventilation could not be used. By using negative-pressure ventilation, however, we could safely perform the exchange of the endotracheal tubes in a patient who could not breathe. Negative-pressure ventilation should be considered as a useful additional safety measure when changing endotracheal tubes.

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Evaluation of Double Lesion Syndrome with Diagnostic Spinal Anesthesia

To the Editor:—Loubser and Clearman[1] described the use of diagnostic spinal anesthesia in evaluating mechanisms of central spinal cord injury (SCI) pain. The following case report highlights another application of diagnostic spinal anesthesia in double lesion syndrome[2] (a subtype of SCI).

A 62-yr-old woman with complete quadriplegia of 2 yr, presented with chronic dysesthetic pain in the buttocks and lower extremities, which commenced approximately 6 months after the SCI. Pain was distributed from the buttocks to the toes and described as continuous and burning or stinging in nature. Dysesthetic pain also occurred in the upper extremities from the shoulders to the fingers in an asymmetric patchy distribution, although not as severe as in the lower extremities. Physical examination did not reveal any obvious source of nociception. The patient was insensate below C7 with allodynia or hyperpathia in the areas of pain. Reflexes, clonus, and spasticity were absent in the lower extremities. All attempts at oral pharmacologic management, including antidepressant (amitriptyline), anticonvulsant (carbamazepine), and opioid and nonopioid analgesics provided limited analgesia. Physical therapeutic modalities, such as range of motion, transcutaneous or neuromuscular electrical stimulation, and local heat, were ineffective.

Radiologic studies of the lumbosacral vertebral column, pelvis, and lower extremities excluded the presence of fractures or other bony abnormality. An extensive urodynamic evaluation of bladder function, including pressure-flow studies with simultaneous videocystourethrography, revealed an areflexic bladder neck. Lumbosacral somatosensory evoked potentials (LSEPs) were recorded from electrodes placed over the lumbosacral vertebrae (S1, L2, T12, reference at T6) after tibial nerve stimulation. An analysis of the R and S components (amplitudes and latencies) compared to control values revealed grossly abnormal LSEPs. These findings suggested the presence of an occult lumbosacral spinal lesion compatible with double lesion syndrome. The patient's pain symptomatology was attributed to the lumbosacral spinal lesion, and diagnostic spinal anesthesia was planned for confirmation before a trial of dorsal column stimulation or intrathecal opioids.

A 23-G intrathecal catheter was placed *via* a 20-G Tuohy needle in the L3–L4 vertebral interspace and threaded cephalad for 3 cm. Double-blind pain assessments included a 10-cm visual analog scale and somatic diagram indicating distribution of pain. After aspiration of clear cerebrospinal fluid (CSF) from the catheter hub, 2 ml of placebo (normal saline) was administered per catheter. However, no change in status was recorded for 60 min, whereupon 50 mg lidocaine in 7.5% dextrose was administered. Pain assessments were continued every 15 min over the next 90 min, without any recorded change in pain intensity or distribution. Similarly, no change in pain symptomatology occurred in the upper extremities. To verify that the catheter had not migrated out of the intrathecal space during spinal anesthesia, aspiration of free-flowing CSF from the catheter hub was demonstrated before catheter removal.

Double lesion syndrome was described by Beric *et al.*[2] as a clinical and a neurophysiologic syndrome characterized by an areflexic bladder, abnormal LSEPs, and cauda equina-like pain in conjunction with a primary cervical or thoracic SCI. Several hypotheses have been advanced to explain the development of the lumbosacral spinal lesion, including nerve hypoxia, arachnoiditis, spinal stenosis, and disc herniation, although at present its pathogenesis is not fully understood.[2] Furthermore, with respect to the pain symptoms, origination of nociception within the lumbosacral region is doubtful in patients with complete SCI (*i.e.,* absence of sensory perception below the level of the lesion). However, Beric *et al.*[2] caution that nonrecognition of lumbosacral dysfunction as the cause of pain could result in pain being interpreted as central in origin, *i.e.,* originating at or above the upper SCI.

The patient response described above suggests that the source of nociception was not within the cauda equina or lumbosacral vertebral column. Spinal anesthesia failed to produce any reduction of pain in the buttocks or lower extremities and response to placebo was negative. In contrast, the clinical presentation of widely distributed dysesthesias in the buttocks and upper and lower extremities correlates more closely with central SCI pain.[3] Subsequently, the patient decided against further intervention, and aggressive medical treatment was pursued.

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