was used as the method of insertion. Blood flow was not assessed until one hour after decannulation in order to minimize the possibility of reduced flow, resulting from vessel spasm, ascribed to thrombus formation. Blood flow was also assessed five days after decannulation. This period of time was chosen because thrombus formation occurring later than five days after decannulation is uncommon and the possibility of recanalization occurring within four days is also uncommon.[4]

The incidence of thrombus formation occurring after cannulation with 20-gauge, non-tapered Teflon® catheters is low. Five days after decannulation the incidence in our study, regardless of the method of cannulation, was 5 per cent. This compares with an 8 per cent incidence of occlusion previously reported by Bedford.[5]

Although we were unable to demonstrate a difference in thrombus formation comparing the two techniques, the low overall incidence of thrombosis means we cannot exclude such a possibility. However, if such a difference does exist, it must be small. We conclude that the commonly practiced technique of transfixing does not carry an appreciable increased risk to the patient.

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Postoperative Paralysis of Phrenic and Recurrent Laryngeal Nerves

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Postoperative paralysis of the recurrent laryngeal nerve or the phrenic nerve usually follows a direct trauma during neck or thoracic surgery.[1–4] The present report describes an unusual case of postoperative unilateral paralysis of both phrenic and recurrent laryngeal nerves in a patient undergoing surgery at a site far from the anatomic course of these two nerves.

Report of a Case

A 72-year-old man with right hydronephrosis was scheduled for right nephrectomy. Preoperative radiologic examination was normal except for degenerative changes of the lumbar spine with spur formation. He was premedicated with atropine, 0.6 mg, intramuscularly. Anesthesia was induced with the intravenous administration of thiopental, 250 mg, and succinylcholine, 75 mg. Endotracheal intubation was performed using 9-mm low-residual volume cuffed tube. The cuff was inflated with air just to the point of preventing an air leak. Anesthesia was then maintained with 75 per cent nitrous oxide and supplemented with alloferine. The patient was placed in the kidney position with the right side up. The patient was sharply angulated at the T9 to L1 level by means of a kidney bridge placed under his lower most flank. This angulation was accentuated further by "breaking" the mid-portion of the surgical table. A pillow, 15 cm in height, was placed under the patient's head. He was stable throughout surgery which lasted two hours after which the trachea was extubated. Following extubation and recovery of consciousness, the patient was hoarse which was attributed to intubation of the trachea. However, the hoarseness persisted on the second postoperative day and the patient became febrile. By indirect laryngoscopy, left recurrent laryngeal paralysis was evident. A chest roentgenogram showed left lower lobe consolidation and elevation of the left leaflet of the diaphragm. Thoracic fluoroscopy demonstrated the left diaphragmatic leaflet to be

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paralyzed. No other neurologic deficits were detected. Antibiotic therapy was initiated. One week later, he became afebrile and his chest roentgenogram showed clearance of the lung infiltrate. He was discharged three days later with hoarseness and elevation of the leaflet of the diaphragm. Resolution of the vocal cord paralysis was documented 6 months later, but a repeat roentgenogram did not show any change in the position of the left diaphragmatic leaflet.

Discussion

Postoperative peripheral nerve paralysis usually results from faulty positioning of the anesthetized patient and is attributed to pressure or stretching of a nerve at a vulnerable point along its course. This complication most commonly affects branches of the brachial plexus, and less frequently, nerves supplying the lower extremities.[5–7] In contrast, the most common cause of postoperative paralysis of the phrenic and recurrent laryngeal nerves is a direct trauma during neck or thoracic surgery.[1–4] However, recurrent laryngeal nerve paralysis can occur following surgical intervention at sites far from the anatomic course of the nerve. A coincidental viral infection and exposure to cold, acting in a manner similar to that suggested by Bell's palsy, have been proposed as causes.[8] Also, hyperextension of the neck,[9] excessive retraction of the chest wall associated with median sternotomy,[10] and traction on structures such as the lower end of the esophagus may lead to stretching of the recurrent laryngeal nerve with subsequent vocal cord paralysis.[11] Recently, factors related to the endotracheal tube have been incriminated in recurrent laryngeal nerve injury.[9,12,13] In these cases, paralysis would result from the toxic effects of substances present in the tube,[12] or by direct compression of the nerve endings against the thyroid cartilage.[13] The latter accident is more likely to occur if the cuff of the tube is overexpanded, irregularly inflated, or placed just below the vocal cords. Our patient was anesthetized with nitrous oxide, 75 per cent. Nitrous oxide is thirty-four times more soluble than nitrogen, and can diffuse into air-containing cavities more readily than nitrogen can diffuse out.[14] Nitrous oxide may have diffused through the rubber cuff and increased the intracuff pressure of the low-residual volume cuffed endotracheal tube.[15]

Similar to the recurrent laryngeal nerve, the most common cause of postoperative paralysis of the phrenic nerve is a direct surgical trauma.[3,4] However, postoperative paralysis of the phrenic nerve in our patient followed surgery at a site far from the anatomical course of the nerve. The injury may be attributed to factors coincidental or associated with surgery such as neuritis, whether idiopathic, toxic, or inflammatory in nature.[3] The adjacent pulmonary infection might have been a predisposing factor. It may have also resulted from indirect trauma secondary to stretching of the phrenic nerve which has been reported following crush injury of the chest.[16]

Separate etiologies may have been involved in the pathogenesis of the postoperative complications observed in our patient. For instance, paralysis of the vocal cord may have resulted as a sequelae of endotracheal intubation, while the diaphragmatic paralysis was secondary to the adjacent infection. However, the occurrence of unilateral paralysis of both the phrenic and the recurrent laryngeal nerves in the same patient, at the same side and essentially at the same time supports a single etiology for the two lesions. Nerve stretch secondary to malpositioning may be considered as an etiologic factor responsible for both the phrenic nerve and the recurrent laryngeal nerve paralysis. Anesthesia associated with the extreme muscular relaxation allows the adoption of nonphysiologic positions. In the lateral decubitus, proper positioning of the head and neck in relation to the trunk must be ensured in order to avoid overstretch of the neck. The neck may be overstretched on the uppermost side if a small pillow or doughnut is placed under the patient's head. However, in our patient, the nerve paralysis occurred on the opposite site; a relatively high pillow was placed beneath his head which may have overstretched the neck on the opposite side of surgery. The stretch might have been also exaggerated by the immobility of the lower shoulder and by the sharp lateral angulation of the trunk during the kidney position.

Individual variations of anatomy, such as the presence of a cervical rib, the size of the cervico-axillary canal, the shape of the first rib and the slope of the shoulder may all play a part in rendering the brachial plexus and the nerves having a cervico-thoracic anatomic course such as the phrenic and recurrent laryngeal nerves vulnerable to stretch injury. Also, pathological factors such as degenerative changes of the cervical spines with spur formation, diabetes, periarteritis nodosa, and alcoholism are known predisposing factors.[17] None of these conditions were present in the reported case.

Stretch nerve injuries often produce a readily reversible neurapraxia, but may also produce axonotmesis[18] or even neurotmesis.[16] Fortunately, most of these stretch nerve injuries clear up spontaneously from two weeks to one year.[7] Recovery time depends on the degree of injury and the time required for nerve fiber regeneration between the site of injury and the denervated muscle. In our patient, resolution of the vocal cord paralysis occurred spontaneously after six months, when the left leaflet of the diaphragm was

still paralyzed. The phrenic nerve has a longer anatomic course than the recurrent laryngeal nerve and may subsequently show a slower rate of recovery. Following stretch trauma of the phrenic nerve, the normal diaphragmatic function gradually returns over the succeeding six to twelve months.[16]

In conclusion, the present case report describes an interesting complication of postoperative unilateral paralysis of both the phrenic nerve and the recurrent laryngeal nerve, following surgery at a site far from their anatomical pathways. The possible etiologic factors predisposing to this complication are discussed. The occurrence of unilateral paralysis of both nerves in the same patient, on the same side and at the same time suggests a single etiology. However, the possibility still remains that separate etiologies were involved.

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Anesthesia for a Child with Leigh's Syndrome

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Leigh's syndrome[1] or subacute necrotizing encephalomyelopathy (SNE) is a chronic neurologic disease, usually discovered during infancy. Feeding problems, weakness, external ophthalmoplegia, swallowing difficulties, ataxia, and convulsions are frequent clinical features; with respiratory difficulties occurring late in the course of the disease. The neuropathologic findings, both histologically and topologically, are distinctive,[2] with several similarities to Wernicke's encephalopathy. Increased blood lactate and pyruvate concentrations are also frequently present. The most likely enzyme abnormality is a defect in activation of pyruvate dehydrogenase.[3]

These patients have a chronic relapsing course, and acute exacerbation has been reported following surgery.[4] The anesthetic course in a fourteen-month-old child who required an open liver biopsy for diagnosis is described below.

Report of a Case

The patient was a 14.5-month, 7-kg girl who was born prematurely at 28-weeks gestation with Apgar scores of 6 and 9. She was treated with continuous positive airway pressure and oxygen, and was discharged at age two months. The child was mildly developmentally retarded, but was pulling to stand and imitating sounds by

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