Unilateral Vocal-cord Paralysis Following Endotracheal Intubation

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Unilateral vocal-cord paralysis following surgical procedures unrelated to the neck and recurrent laryngeal nerve has hitherto been reported only rarely in the anesthetic literature and infrequently in other specialties. In 1970, Hahn *et al.* described five such cases, in which the patients had had no laryngeal symptoms prior to operation, had been anesthetized for operations unrelated to the neck region using a cuffed endotracheal tube and, on subsequent examination, manifested no visible evidence of trauma secondary to the intubation.[1] More recently, Ellis and Pallister reported four similar cases[2] and suggested a way in which paralysis from trauma to a branch of the recurrent laryngeal nerve could account for the palsy. By cadaver dissection they supported a mechanism previously postulated by Hahn *et al.*[1]

Report of a Case

A 47-year-old woman of average size was admitted to hospital for a hysterectomy for a benign conditon. Except for her gynecologic complaints, she was healthy and gave no history of any laryngeal symptom. Induction of anesthesia consisted of thiopental, succinylcholine (1 mg/kg), and atraumatic intubation with a #8 cuffed endotracheal tube. The cuff was inflated to a point of no air leakage and anesthesia maintained with a mixture of nitrous oxide, oxygen and halothane. The surgical procedure lasted 90 minutes and was completely uneventful, as was the postoperative period. The patient was discharged eight days after operation, and shortly thereafter began to complain of increasing hoarseness. She was seen by an otolaryngologist, who found left vocal-cord paralysis. No local lesion was seen. Six months later movement of the cord began, and movement returned to normal after almost a year. There has been no recurrence of symptoms in almost four years.

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Comment

Reviews of laryngeal complications following endotracheal intubation testify to their frequent occurrence.[3–6] These complications include severe pain due to laryngitis and tracheitis, granulomas of the vocal cords and subglottic regions, avulsion of the vocal cords, dislocation of the arytenoid cartilage, tracheal perforations, vocal-cord palsies, etc. Vocal-cord paralysis, whose chief sympton is hoarseness, following tracheal intubation is usually associated with readily discernible damage to tracheal or laryngeal mucosa, or visible evidence of trauma to the arytenoid cartilage or joint. This last form of injury may be caused by pronounced extension of the neck, driving the tube back against the arytenoids with or without dislocation of the cricoarytenoid joint.[6] On the other hand, recurrent nerve palsies following surgical procedures without evidence of local trauma are also frequently seen by laryngologists. Such palsies are easy to explain when they follow surgical procedures of the head or neck, or anywhere along the course of the recurrent laryngeal nerves, but they may, on rare occasions, be seen following operations in other areas.

In 1975, Ellis and Pallister[2] dissected out the recurrent laryngeal nerves in cadavers. They were able to demonstrate that the recurrent laryngeal nerve, before reaching the superior rim of the cricoid cartilage, divides into anterior and posterior branches. The anterior branch passes medial to the lamina of the thyroid cartilage to supply the lateral cricoarytenoid and thyroarytenoid muscles. During their dissection they were able to show that when an endotracheal tube was passed and the cuff inflated within the larynx, it compressed the anterior branch between the cuff and the interior surface of the thyroid lamina. Just such a mechanism had been postulated by Hahn *et al.*[1] five years earlier.

Irregular inflation of a cuff, due to a defect in manufacture or following many sterilizations, may exert unduly high pressure localized to the intralaryngeal course of the anterior branch of the recurrent laryngeal nerve. Many anesthesiologists, to preclude inadvertent endobronchial intubation, make it a practice to place the endotracheal tube so that the top of the cuff is immediately below the vocal cords—thus, part of the cuff, at least, lies within the larynx.

Recent experiments have demonstrated that oxygen and anesthetic gases diffuse into endotracheal tube cuffs more rapidly than nitrogen diffuses out.[7] This results in a marked increase in the pressure within the cuff which, in turn, may be associated with trauma to the mucosa of the larynx and trachea and undue pressure on any nerves in the vicinity.[8]

Prevention of this apparently rare complication lies in a) eliminating the use of endotracheal tubes with cuffs that, on testing, inflate unevenly, b) desisting from the practice of deliberately placing the cuff within the larynx, and c) filling the cuff with a sample of the inspired mixture of gases, regular deflation of the cuff, or alternately, routine use of a simple pressure-relief valve. These last measures have been recently advocated by Stanley.[9]

A case of left vocal-cord palsy in a patient undergoing a hysterectomy has been presented. A cuffed endotracheal tube was used for maintenance of anesthesia. No local lesion was seen by the consultant laryngologist, and it took almost a year before resolution was complete. The course and ultimate outcome of the complication fit in with the postulated theory that a branch of the recurrent laryngeal nerve within the larynx may have been damaged by the inflated cuff.

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