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Vocal fold paralysis following carotid endarterectomy

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Abstract

Injury to the vagus nerve or one of its branches during carotid endarterectomy (CEA) can result in vocal fold paralysis (VFP). This study assessed prospectively 73 patients undergoing CEA. A total of 76 procedures were performed in these patients over a one-year period. All patients underwent pre-operative and post-operative assessment of vocal fold mobility by indirect laryngoscopy and/or flexible nasendoscopy. All patients had normal vocal fold mobility pre-operatively. Eight patients (10 per cent) complained of hoarseness after surgery and in three patients (four per cent) examination confirmed an ipsilateral VFP. This persists in all three patients at six-month follow-up. Vocal fold assessment is important in patients undergoing CEA, particularly when performing second side surgery. We recommend that patients should be informed of the risk of VFP following CEA when obtaining consent.

Key words: Vocal cord paralysis; Carotid artery disease; Endarterectomy

Introduction

Carotid endarterectomy (CEA) is the most common procedure performed for the treatment of extra-cranial cerebrovascular atherosclerosis (Maniglia and Han, 1991). Although this procedure has evolved in technique over the years, accidental injury to cranial nerves as a consequence of this operation continues to be a significant complication. Vocal fold paralysis (VFP) resulting from injury to the vagus or recurrent laryngeal nerve can be distressing for the affected patient and even life-threatening if bilateral (Verta *et al.*, 1977). The aims of this study were to establish the incidence of VFP following CEA in our hospital and to detect the affected patients in order to offer them appropriate and early treatment when necessary.

Materials and methods

Seventy-three patients admitted to the vascular surgical ward at Gartnavel General Hospital for elective CEA over a 12-month period were studied prospectively. Seventy-six procedures were performed in these patients by four senior consultants in vascular surgery. A standardized surgical technique was used and there were no intraoperative incidents reported. All patients were submitted to a pre- and post-operative assessment of vocal fold function. Visualization of the vocal folds was performed by indirect laryngoscopy or fibre-optic laryngoscopy. This was performed on the day prior to the operation and on the second post-operative day where possible. The patients own perception of their voice was recorded. All pre- and post-operative assessments were performed by the same person. Patients who had impaired vocal fold mobility at their post-operative assessment were submitted to further examination of the larynx with a rigid laryngoscope and a video recording of the findings was also performed in these patients. They were subsequently referred to speech therapy and followed up in the voice clinic where video laryngoscopy was used to monitor their progress.

Results

A total of 76 CEA were performed in 73 patients over a one-year period. Three patients underwent staged bilateral procedures. There were 47 (65 per cent) men and 26 (35 per cent) women in the study group. Their ages ranged from 53 to 86 years (mean: 68 years). Two patients were excluded from this study; in one case the patient died on the first post-operative day and in the other follow-up was not possible due to a stroke. All 73 patients had normal vocal fold mobility on their pre-operative assessment. Eight patients (10 per cent) complained of hoarseness after surgery. Post-operative assessment of these patients confirmed an ipsilateral VFP in three of them (four per cent). The remaining group

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of five hoarse patients had normal mobile folds on examination and their voice had spontaneously recovered before discharge. None of the asymptomatic patients had impaired vocal fold mobility on examination. Follow-up of the affected patients at six months showed persistent VFP with some improvement of voice quality in all three due to compensation from the contralateral vocal fold.

Discussion

Significant advances in surgical technique and post-operative care have resulted in reduced perioperative mortality from CEA. Less emphasis has been placed on peripheral cranial nerve injuries, which if unrecognized, can cause significant post-operative morbidity and can be life-threatening in cases of bilateral VFP. The reported incidence of VFP following CEA varies according to different authors and in our series is four per cent. Hertzer *et al.* (1980) reviewed 240 patients prospectively and reported a 5.8 per cent incidence of VFP. Sannella *et al.* (1990) reported a seven per cent incidence in a series of 411 patients studied retrospectively, while a retrospective study by Matsumoto *et al.* (1977) showed a two per cent incidence. In our study the three affected patients presented with hoarseness but there are other series where VFP, as a result of recurrent laryngeal nerve damage during CEA, has been reported in patients without hoarseness. Curran *et al.* (1997) in their prospective study of 50 patients found six patients (12 per cent) with VFP following CEA and half of them were asymptomatic (six per cent). Hertzer *et al.* (1980) reported 14 patients (5.8 per cent) with post-operative recurrent laryngeal nerve dysfunction and four of them (1.6 per cent) had no symptoms whatsoever. The injury in those cases would have escaped detection if routine otolaryngological examination had not been performed. Some patients with bilateral carotid atherosclerosis disease will undergo second side surgery and if they have an undiagnosed VFP they run a small risk of sustaining a second VFP which will compromise their airway.

The vagus nerve is usually identified within the carotid sheath posterior to the common carotid artery but it is occasionally found anterior and lateral to the artery and medial to the internal jugular vein. The recurrent laryngeal nerve usually branches from the vagus nerve within the mediastinum, loops around the subclavian artery on the right side and the aortic arc on the left side and lies in the tracheoesophageal groove. Injury to either the recurrent laryngeal nerve or the vagus nerve causes paralysis of the ipsilateral vocal fold. Injury to the superior laryngeal nerve principally results in mild relaxation of the ipsilateral vocal fold manifest by early fatigability of the voice and impairment in phonation at a high pitch. This can represent a disability to vocalists or public speakers (Hertzer *et al.*, 1980). The exact mechanisms responsible for VFP subsequent to CEA are unclear but several different insults can occur. Knight *et al.* (1987) mention in their study that most injuries are transient and result not from transection but from blunt trauma during dissection, retraction and clamping of the vessels. The mechanism of injury in Abu-Rahma and Lim's (1996) study was thought to be from damage by self-retaining retractors, diathermy burns in the vicinity of the nerve or inclusion of the nerve in the arterial clamp. It is possible that disruption of the nerve's microvascular supply may be responsible for the nerve damage (Curran *et al.*, 1997). Electrocautery is a possible source of nerve injury and bipolar cautery is reported to be less injurious than monopolar (Vällfors and Erlandson, 1980). Transient post-operative dysfunction of adjacent cranial nerves and their branches may occur despite gentle dissection and retraction during CEA (Hertzer *et al.*, 1980).

At six-month follow-up none of the three affected patients from our study have yet recovered function of their paralysed vocal fold. All of them have improved their voice quality by contralateral vocal fold compensation after speech therapy. Spontaneous recovery of VFP after CEA has been reported in many previous studies. In the series reported by Sannella *et al.* (1990) 15 (50 per cent) of the affected patients recovered vocal fold function as early as six days and as late as eight months. Only one of the six affected patients followed up by Curran *et al.* (1997) had a persistent VFP after one year while the other five affected patients recovered vocal fold function within six weeks. Eight (5.7 per cent) of the 14 patients who had dysfunction of the recurrent laryngeal nerve recovered normal function between one and 12 months after the operation (mean five months) according to the study performed by Hertzer *et al.* (1980). It would appear that when spontaneous recovery of vocal fold function occurs, this happens within six months of the operation in the majority of patients. In those patients with persistent VFP after six months who continue to have a weak and breathy voice despite speech therapy, surgery to improve voice quality may well be considered particularly if a reversible surgical procedure is performed or if electrical studies confirm a low likelihood of recovery.

Conclusions

VFP is a significant and recognized complication of CEA. As mortality from this procedure decreases we are more likely to see patients with hoarseness following CEA. The exact mechanism of the nerve injury is unclear. Although spontaneous recovery does occur in the majority of patients, early diagnosis of this complication is useful. Speech therapy can be implemented at an early stage and surgery can be considered when appropriate. The timing and type of surgery is a matter of further debate. Vocal fold assessment is important in patients undergoing CEA. It is essential that all patients undergoing bilateral procedures have pre-operative assessment of their vocal fold function as asymptomatic VFP have been found following CEA. Given today's litigious society we recommend that patients should be informed of the risk of VFP following CEA when obtaining consent.

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