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Editorial Review

Voice disorders following road traffic accidents

LESLEY MATHIESON, DIP.C.S.T., F.R.C.S.L.T.

The number of patients presenting with voice disorders following road traffic accidents (RTAs) appears to be relatively small but dysphonia or aphonia resulting from the injuries sustained can have a significant impact on the individual's recovery and future. For many RTA survivors a voice disorder might be regarded as a relatively minor problem. Not only have they survived but in many cases there will be a range of physical problems of reduced mobility, disfigurement and neurological damage which demand considerable attention. Central nervous system trauma frequently results in a combination of cognitive deficits, aphasia and dysarthria which can also be regarded as more serious communication disorders than vocal abnormality. In reality, successful rehabilitation is adversely affected by phonatory difficulties whether they are part of a complex picture of impairment or exist alone. During the early stages of recovery, physically and psychologically traumatized aphonic or dysphonic patients often feel ineffective and helpless. Aphasic individuals who are aphonic or dysphonic have the additional difficulty of being less able to monitor their disordered language output. As rehabilitation progresses, the residual clinical voice disorder has important implications socially and for employment prospects.

The comparative rarity of post-RTA voice problems is reflected in the paucity of specific literature. This is probably due, in part, to the fact that laryngotracheal injuries of any type are relatively rare and that their mortality rate is fairly high (Hosny *et al.*, 1995). Understandably, immediate management of the cervical airway trauma in order to ensure the patient's survival is the focus of the majority of papers in the field. Single cases of vocal tract injury are reported (Myers and Iko, 1987; Guertler, 1988; Mittleman, 1988; Wenig *et al.*, 1990; De Lorenzo *et al.*, 1991; Asai *et al.*, 1996) as are groups of patients who have sustained particular types of injury (Reece and Shatney, 1988; Hoy and Cole, 1993; Bent and Porubsky, 1994; Yen *et al.*, 1994; Danic *et al.*, 1996). Authorities such as Tucker (1987) and Casiano and Goodwin (1991) have contributed to the surgical literature on managing laryngeal trauma. Much of the above literature considers laryngeal trauma sustained in RTAs as part of wider studies which address laryngeal trauma in general. Voice problems related to RTAs, however, are frequently the result of more extensive damage or of aetiology which is multi-factorial. Consequently, best-practice techniques of diagnosis, evaluation and treatment of this patient group require particular consideration.

RTA injuries affecting phonation

Although the aetiologies of voice problems related to RTAs are wide-ranging, they can be classified into three groups:

(1) problems affecting laryngeal structure and function;
(2) supra- and sub-glottic vocal tract involvement;
(3) concomitant problems speech-breathing difficulties for example.

Some voice disorders are aquired at the time of the accident but others are the result of subsequent management during intensive care. In addition, the patient's reaction to the accident and to any litigation can also affect the severity and type of voice disorder. The injuries which give rise to voice problems occur at four main sites, with some patients sustaining mixed injuries:

(1) front neck and laryngeal trauma;
(2) damage to the peripheral nervous system (recurrent laryngeal nerve (RLN));
(3) head injury/central nervous system (CNS) damage;
(4) spinal injury.

Laryngeal trauma is frequently sustained as the body is propelled forward on vehicle impact, whether or not a seatbelt is worn. The larynx is fixed by neck extension during this process so that its normal mobility laterally and antero-posteriorly is reduced (Casiano and Goodwin, 1991). As a result it is

Chief Speech and Language Therapist (ENT), Northwick Park and St Mark's Hospitals, London, Visiting Lecturer in Voice Pathology, Department of Linguistic Science, University of Reading

subjected to maximum force if contact is made with any object or person. A high antero-posterior blow to the larynx can crush the thyroid and cricoid cartilages against the cervical vertebrae causing a mid-line vertical fracture. During this process, the thyroid alae will splay out like the covers of a partially opened book when its spine is pressed. As a result, the internal laryngeal structures can be disturbed and do not necessarily return to their original position. Injuries can include mucosal lacerations, haematomas, tears, vocal fold injury, cricoarytenoid separation and oedema (Yen *et al.*, 1994). A low blow can lead to cricoid fractures and to laryngotracheal separation (Roh and Fazzalaro, 1993). The laryngeal skeleton is fractured so extensively in some cases that there is no possibility of realigning the structures with the result that the vocal folds, even if undamaged and mobile, are no longer on the same plane. The most severe laryngeal fractures can result in such marked irreversible stenosis of the airway that laryngectomy is the only alternative.

Laryngeal trauma can also present as thermal damage to the upper airway if a fire has been caused by the vehicle impact. Even when gross vocal fold movements are relatively normal, smoke inhalation can result in stiff, red vocal fold mucosa with markedly reduced mucosal waves.

Unilateral or bilateral *recurrent laryngeal nerve (RLN) injury* sustained on impact might not be revealed until extubation. The patient will have increasing breathing difficulties on removal of the endotracheal tube, despite a reasonably normal voice, in the case of bilateral vocal fold abductor paralysis. Unilateral RLN paralysis will present with typical breathy voice quality and inefficient cough. Aspiration will occur in some cases.

Head injury acquired in high impact RTAs can result in central nervous system damage which will affect vocal fold adduction. This is seen usually as a feature of dysarthria with the resulting neurological dysphonia giving rise to either hypo- or hyper-adduction of the vocal folds, according to the site of the damage. Phonation in these cases is compromised further in many instances by speech-breathing difficulties, with the resulting inability to establish and maintain adequate sub-glottal air pressure. The phonatory effects of this are predominantly reduced vocal loudness and reduced phrase length. Similar speech breathing problems are seen in cases of spinal injury.

Vocal tract changes acquired during post-RTA management

Following an RTA, vocal tract changes are frequently acquired during immediate and long term management. These changes can give rise to voice problems in patients who would otherwise have had no vocal problems or they can exacerbate voice problems which are attributable directly to the accident. Establishing and maintaining the airway is the principal management priority, but endotracheal intubation can give rise to a range of vocal tract changes which become apparent on extubation. Gallivan *et al.* (1989), in their videolaryngoscopic study of patients following endotracheal intubation note that these changes can include ulceration, oedema, granuloma, loss of vocal fold substance in the posterior vocal folds and arytenoid dislocation followed by scar formation. These changes can eventually result in vocal fold contraction, arytenoid approximation and ankylosis of the cricoarytenoid joints. Sub-glottic stenosis as the result of friction of the tube on the tracheal mucosa or excessive cuff pressure (Messahel, 1994) can be so severe that a tracheostomy is necessary.

Effects of patient reaction

The patient's reaction to the accident is also relevant to diagnosis and management whether or not there is an organic basis for the voice disorder. In cases of organic dysphonia, many individuals will use inappropriate compensatory strategies in an attempt to overcome the problem unless they understand how to deal with their difficulties. Their misguided, but understandable, attempts to phonate effectively usually involve increased effort which tends to be counter-productive. Non-organic voice problems include conversion symptom aphonia which can occur as a result of the psychological trauma of the RTA in vulnerable individuals. (This was particularly pertinent in the case of a female patient who was travelling with her brother and boyfriend from the scene of a crime which they had committed when their car was involved in a collision with a police car. Although she was not injured, she became aphonic immediately.) In cases of head injury, however, a diagnosis of conversion symptom aphonia is less straightforward even if laryngoscopic examination appears to be normal. Frontal lobe damage resulting in poor affect and motivation can impair the ability to initiate phonation (Sapir and Aronson, 1985, 1987). Oromotor dyspraxia can cause similar difficulties in differential diagnosis when involuntary voicing appears to be normal but the patient is otherwise aphonic.

Less dramatic reactions following the accident include severe anxiety, possibly accompanied by other intense emotions such as anger, so that the individual experiences intermittent voice loss or constant dysphonia. In these cases, globus symptoms frequently accompany the dysphonia. The possible effects of litigation on the maintenance of abnormal phonation should not be overlooked.

Evaluation

In view of the complexity and frequently multi factorial nature of voice disorders following RTAs, these patients should be seen by a laryngologist and specialist speech and language therapist working as a co-ordinated team. Clinical experience suggests that patients with voice disorders following RTAs are best served when the problem is recognized and investigated at an early stage. Usually, the patient's other injuries will determine when a comprehensive evaluation can be undertaken but for those who have been intubated, a husky voice persisting after

24 hours indicates the need for thorough laryngoscopic examination in order to prevent future problems. It is essential to obtain the best image possible of laryngeal structure and function before decisions are made regarding phonosurgery or voice therapy. Where possible, phonatory function should be assessed initially by the laryngologist and specialist speech and language therapist in a joint voice clinic using videostroboscopy so that vocal fold mucosal waves can be seen in addition to the general architecture of the larynx and gross movements of the vocal folds. The minimum standard is nasendoscopic laryngoscopy; laryngeal mirror examination is inadequate. A comprehensive picture of laryngeal phonatory behaviour is essential for soundly based surgical and therapeutic management hypotheses to be established.

Evaluation of the disordered voice begins with perceptual and instrumental acquisition of an acoustic profile. This provides not only a baseline from which progress can be monitored but should also highlight aspects of phonation which do not appear to correlate with the laryngoscopic findings. For example, in cases of unilateral vocal fold paralysis, incomplete adduction of the vocal folds is observed and the paralysed, flaccid fold has a chaotic vibratory pattern on stroboscopy with the vibrations of the paralysed fold occurring at a lower frequency than those of the healthy fold. The expected acoustic result, therefore, is a vocal note with a low signal-to-noise ratio, lowered speaking fundamental frequency, and reduced range of pitch and intensity. If the patient is using a particularly high speaking fundamental frequency, however, this suggests that inappropriate compensatory strategies are being used. These have to be eliminated before treatment can be directed at improving vocal fold closure if progress is to be made. Conversely, an acoustic profile which concurs with the laryngoscopic findings can reduce the time unnecessarily spent on exploratory treatment to improve aspects of vocal function which are unlikely to improve. In the case of a fractured larynx, for example, the final laryngeal state might result in a false vocal fold approximating to the contra-lateral true vocal fold. As the mass of a false fold is greater than the mass of a true fold, it is to be expected that the patient's speaking fundamental frequency will be considerably lowered. Consequently, treatment directed at raising the vocal pitch is unlikely to be successful. Clinical time might be spent more productively in working on improving sub-glottic air-pressure with the aim of improving vocal note quality and volume.

In addition to evaluation of vocal function directly related to the vocal tract, co-existing factors such as impaired speech-breathing must also be taken into account during treatment.

Intervention

The surgical aims are to provide the best airway possible and to repair vocal tract damage in order to establish the most efficient phonatory structure and formation. Whether or not phonosurgery is involved, the aim of vocal therapy is to achieve maximum functional efficiency of the damaged mechanism in the light of its biochemical limitations.

Therapeutic techniques will include the elimination of secondary phonatory behaviours which develop as inappropriate compensatory strategies. Patients who are investigated as soon as possible will have less time to habituate these behaviours. Increased capacity and control of speech breathing will be encouraged. Hypofunctional vocal fold adduction can be addressed with laryngeal valving techniques while RLN paralysis can be helped with medial compression of the thyroid cartilage. In cases of bilateral abductor paralysis where a tracheostomy has been necessary, speech therapy can facilitate increasing tolerance of stoma occlusion in cases where closure of the stoma is a realistic possibility.

Patients exhibiting conversion symptom aphonia should be referred to the speech and language therapist for treatment immediately the condition is diagnosed. Normal voice should be restored in the first or second treatment session in most cases (Greene and Mathieson, 1989).

There is evidence that patients with an in-dwelling tracheostomy tube can benefit considerably from an expiratory speaking valve being supplied as soon as possible during their recovery. Not only does this enable them to phonate but the increased sub-glottic air pressure which it creates can decrease aspiration and improve swallowing function (Eibling and Diez Gross, 1996).

The patient's motivation, emotional status and compliance with therapy are further factors which will inevitably affect the final outcome.

Management principles

Certain management tenets emerge from the perspective of a joint voice clinic team which sees patients with post-RTA voice problems, including those from the regional rehabilitation unit.

(i) All voice problems following RTAs should be investigated thoroughly as soon as possible.
(ii) The laryngologist and specialist speech and language therapist should work closely to:
  (a) assess laryngeal structure and function initially.
  (b) monitor and redefine the original management programme and treatment stategies by regular review procedure.
(iii) Ideally, patients should be assessed by a voice clinic team using videostroboscopy. In cases of persisting voice disorders where this instrumentation is not available to the laryngologist, referral to a voice clinic should be made in order to clarify the reasons for the failure to improve.
(iv) It has to be acknowledged that restoration of a 'normal' voice is an unrealistic goal for many patients with vocal tract damage following an RTA. Appropriate intervention, however, can help the patient to achieve optimal phonation or prevent further vocal fold misuse.

(v) It is essential to ensure, therefore, that vocal rehabilitation is undertaken by a specialist speech and language therapist whenever possible so that maximum improvement can take place given the biomechanical limitations.

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Address for correspondence:
Lesley Mathieson, Dip.C.S.T., P.R.C.S.L.T.,
Alloway,
Maplefield Lane,
Chalfont St Giles,
Bucks HP8 4TY.