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Swallowing Performance in Patients with Vocal Fold Motion Impairment

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Abstract. Twenty-seven patients with vocal fold motion impairment underwent detailed pharyngoesophagel manometry with a strain gauge assembly linked to a computer recorder. Nine were known to have lesions of the central vagal trunk or nucleus, 9 had recurrent laryngeal nerve (RLN) palsy, and the remainder were idiopathic. The site of the lesion was a more important determinant of subjective swallowing performance than the position of the involved cord at laryngoscopy. Patients with cental lesions had lower tonic and contraction upper esophageal sphincter (UES) pressures than 25 age-matched controls, suggesting that high cervical branches of the lower cranial nerves are important in UES excitatory innervation. RLN palsy patients showed significantly increased pharyngeal contraction amplitude and reduced pharyngoesophageal wave durations. The results suggest that the dysphagia associated with vocal fold motion impairment is not simply due to the disruption of laryngeal deglutitive kinetics, but to independent effects on pharyngeal function.

Key words: Pharyngoesophageal manometry — Laryngeal nerve paralysis — Upper esophageal sphincter — Vocal fold motion impairment — Deglutition — Deglutition disorders.

Early cineradiographic studies of swallowing in patients with unilateral or bilateral recurrent laryngeal nerve palsy failed to demonstrate any abnormalities of cricopharyngeal function, and it was therefore believed that the motor nerve supply to the human upper esophageal sphincter (UES) was the pharyngeal branch of the vagus [1]. A recent anatomical investigation, however, indicated that the extrinsic innervation of the pharyngoesophagus comprised branches of the extrinsic pharyngeal plexus and recurrent laryngeal nerve and their pharyngeal, laryngeal, and esophageal branches [2]. Dysphagia in patients with vocal fold motion impairment may be attributable to a concomitant loss of pharyngeal innervation or to aspiration into the lower respiratory tract due to glottic incompetence [3,4] and diminished cough impulse [5]. Although the effects of vagal and recurrent laryngeal denervation on swallowing have been assessed in experimental animals [6–8], to our knowledge there has been only one previous report [9] of swallowing performance in patients with recurrent laryngeal nerve palsy, published before the advent of modern manometric techniques.

Methods

Over a 2-year period, 27 patients attending the dysphonia clinic of the otolaryngology department, Royal Infirmary, Edinburgh, with vocal fold motion impairment underwent pharyngoesophageal (PE) manometry. The patients gave informed consent to undergo manometry but were otherwise unselected. There were 14 males and 13 females aged 25–84 years (median = 64) The impairment was left-sided in 19 and right-sided in 8. Nine patients had clear evidence of a lesion causing recurrent laryngeal nerve (RLN) palsy. One palsy arose following parathyroid surgery while 8 had lesions of the main trunk of the RLN due to thoracotomy (n = 4) or intrathoracic lesions—aortic aneurysm (n = 1) or bronchial carcinoma (n = 3). Nine patients had more central lesions, of which 5 were known to be intracranial—acute demyelination (n = 1) or brainstem ischemia (n = 4), in 1 patient with associated facial and trigeminal weakness. The other 4 had weakness of the glossopharyn-

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geal and hypoglossal nerves due to glomus tumor (n = 1), metastatic squamous carcinoma (n = 1), or postirradiation collagenosis (n = 2). The vocal cord motion impairment in the remaining 9 patients was deemed idiopathic' on the basis of (1) absence of a clear-cut etiological factor in the history: 1 had hepatic carcinoid tumor, 1 had widespread vascular disease, and 1 had myocardial ischemia, but the relevance of these disorders to the pathogenesis was not certain; (2) normal otolaryngological examination; (3) normal hematological indices and ESR; (4) normal chest radiography and where indicated, by the presence of dysphagia; (5) normal upper gastrointestinal barium series.

The mean duration of vocal fold motion impairment was 7 months in the RLN palsy group (range 2–13 months), 19 months in the 'central' group (range 4–60 months) and 17 months in those where the etiology remained in doubt (range 2–60 months).

Following videolaryngoscopic assessment of the vocal cord position in the dysphonia clinic, PE manometry was performed using a six-sensor strain gauge assembly (Gaeltec Ltd, Dunvegan, Skye, Scotland) linked to a computer recorder (GR800, Albyn Medical, Dingwall, Scotland) with a pressure sample rate of 32 Hz. Lower esophageal sphincter (LES) pressure was averaged from the six sensors at 60° radial orientation, during a rapid pull-through (RPT) at 1 cm/sec and a station pull-through (SPT) at 1 cm intervals. Distal esophageal peristaltic amplitude duration and velocity were measured during 10 water swallows (5 ml bolus). Tonic UES pressure was derived from five of the sensors during an RPT. An SPT at 0.5 cm intervals with 20 sec at each station was used to derive two further parameters of UES tonic pressure—the average maximum tonic pressure in the five sensors and the greatest tonic pressure in any one sensor. Finally, PE dynamic motility was assessed during four water swallows (5 ml) and four bread swallows, as previously described [10]. The manometric findings were compared with those in 25 asymptomatic healthy volunteers, 13 males and 12 females aged 25–77 years (median = 65), using Mann-Whitney analysis. The study had the approval of the local hospital ethical committee.

Results

Fifteen of the 27 patients complained of symptomatic dysphagia. This was very mild in 7 patients who complained only of slight difficulty with dry foods or pills, or of occasional episodes of choking on liquids. The problem was more severe in 8 patients for whom eating presented a significant problem. One, with postradiation collagenosis of the skull base, was fed via an indwelling nasogastric tube. All but 1 of these 8 patients belonged to the 'central' group and included the 5 with multiple cranial nerve palsies. At laryngoscopy, 9 patients had a median/paramedian cord. Three of these had an RLN palsy, 2 a central lesion, and 4 were idiopathic. Five of the 9 patients with a median/paramedian cord and 10 of the remaining 18 patients with abducted cords were dysphagic. The shorter duration of vocal fold motion impairment in the RLN group reflects the surgical etiology in 5 patients, and the typically prompt referral of patients with bronchial carcinoma.

Initial statistical comparison was made between the total group of vocal cord palsy (VCP) patients and the 25 control subjects, and the results are given in Table 1 and Figures 1 and 2. There was no difference between the two groups in LES pressures nor in any of the peristaltic parameters (median duration = 2.8 sec in patients, 3.8 sec in controls; median peristaltic velocity = 3.4 cm/sec in patients, 3.5 cm/sec in controls). UES tonic pressures and, with the exception of UES water swallow relaxation pressure, PE dynamic pressures were also similar in both groups (Table 1). During both water and bread swallows, however, VCP patients showed reduced contraction duration in the pharynx, UES, and upper esophagus (Fig. 1).

Table 1. Manometric pressures in vocal cord palsy patients and controls

	VCP (n = 27) Median (range)		Controls (n = 25) Median (range)	
Pressures (mmHg)				
LES RPT	24	(13–65)	17	(10–51)
LES SPT	20[a]	(5–41)	15	(3–53)
Peristalsis	47	(15–145)	41	(2–226)
UES RPT	33	(8–82)	33	(19–76)
UES SPT-x tonic	30	(6–94)	34	(11–67)
-max tonic	48	(11–136)	54	(17–132)
Water swallow				
UES relaxation	−0.6[b]	(−(8.2–18)	4.7	(−3.3–18.6)
UES after-contraction	76	(29–199)	95	(47–270)
Pharyngeal contraction	90	(7–452)	68	(14–146)
Bread swallow				
UES relaxation	−2.2	(−11.7–6.3)	0.9	(−16.8–21)
UES after-contraction	85	(36–177)	102	(57–187)
Pharyngeal contraction	99	(7–260)	76	(30–177)

[a] $p < 0.05$.
[b] $p < 0.01$.

The results in patients with different underlying etiologies were then separately compared with those of the control group (Table 2, Figs 1 and 2). The patients with central lesions showed significantly lower UES tonic pressures during SPT, although the difference was not significant during RPT (Table 2). This group also had significantly lower UES after-contraction pressures during both water and bread swallows but no significant differences in any temporal parameter from the control group (Fig. 2). This was in contrast to the RLN palsy cohort who showed increased pharyngeal contraction amplitude (Table 2) compared with controls, together with a reduced duration of the pharyngoesophageal contraction waves (Fig. 2). Those whose vocal fold motion impairment was of idiopathic etiology shared the greater degree of UES relaxation shown by the RLN group (Table 2). The idiopathic group also showed reduced wave durations (Fig. 2).

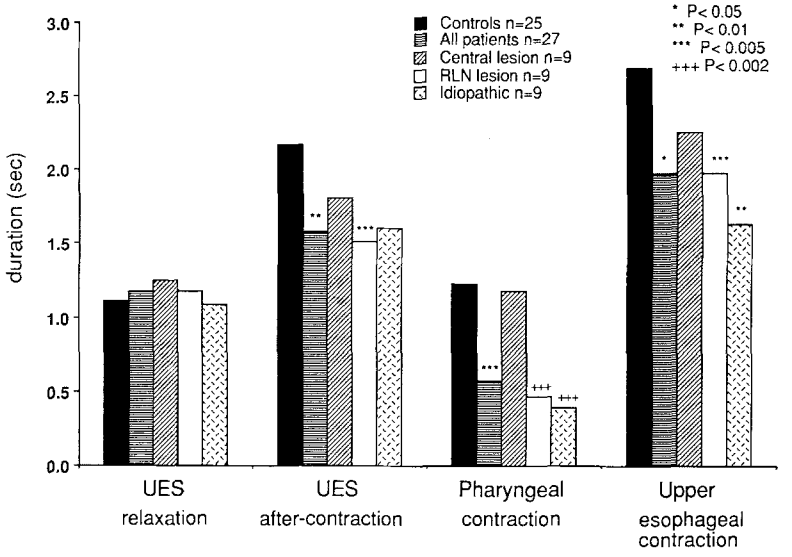

Fig. 1. Median durations of water swallow parameters in patients with vocal fold motion impairment and controls.

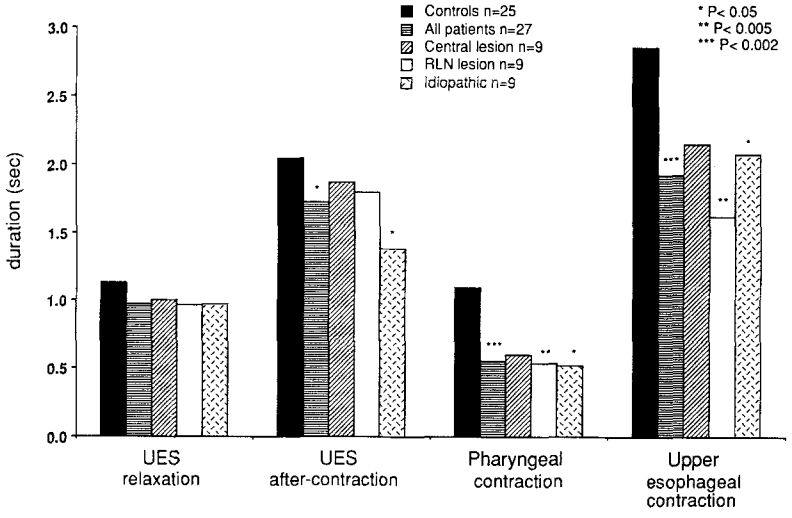

Fig. 2. Median durations of bread swallow parameters in patients with vocal fold motion impairment and controls.

Discussion

Symptomatic dysphagia was present in over half of this series of unselected patients with vocal cord paralysis presenting to a dysphonia clinic. It has been suggested that most patients with idiopathic palsy vocal fold motion impairment have a central lesion [11] and most patients with either a central lesion or an idiopathic problem complained of swallowing difficulties, compared with only one in the RLN group. In 4 patients, the associated glossopharyngeal and hypoglossal nerve paralysis probably contributed to the dysphagia. The central group showed consistent weakness of both UES in tonic pressure and of water and bread swallow UES aftercontraction pressures. The relationship of dysphagia and the site of lesion appeared independent of cord position, as equal proportions of adducted and abducted cords were present in each group. This reflects the clinical unreliability [11,12] of Semon's law (i.e., that RLN paralysis affects principally the abductors of the vocal cord) as the cord position reflects not only the completeness of paralysis but is probably dynamic rather than static, and is influenced by the extent of reinnervation, contraction, and atrophy [11].

Laryngeal kinetics are central to the performance of a normal swallow. In the traditionally accepted sequence of deglutitive muscular activity, the leading complex comprises genioglossus and the elevators of the hyoid bone, while thyroarytenoid activity and therefore, glottic closure is registered at a point between the appearance of activity in the superior and the middle constrictors [13]. An early report [9] of pharyngoesophageal dysphagia in 11 of 15 patients with RLN palsy included manometric assessment of 8 patients. No consistent observations were made, perhaps because of the perfused pressure measurement system used. A recent study of

Table 2. Manometric pressures in patients with vocal fold motion impairment of different etiology

	Central/Vagal palsy (n = 9)	RLN Palsy (n = 9)	Idiopathic Palsy (n = 9)	Controls[c] (n = 25)
Age (y)	64 (42–76)	63 (25–81)	72 (28–84)	65
Pressures (mmHg)				
UES RPT	25 (12–54)	39 (8–82)	33 (17–64)	33
UES SPT-x tonic	18[a] (6–50)	50 (10–94)	34 (9–77)	34
-max tonic	18[b] (11–89)	78 (18–136)	57 (15–125)	54
Water swallow				
UES relaxation	0.6 (−6.4–18)	−0.3[b] (−5.8–14.2)	−3.1[b] (−8.2–14.2)	4.7
UES after-contraction	55[a] (29–95)	88 (48–136)	103 (42–199)	95
Pharyngeal contraction	18 (7–452)	121[a] (38–399)	94 (28–111)	68
Bread swallow				
UES relaxation	1.0 (−1.7–4.3)	−9.3[b] (−11.7–0)	−5.5 (−8.2–6.3)	0.9
UES after-contraction	62[b] (36–119)	86 (61–177)	93 (40–157)	102
Pharyngeal contraction	37 (7–168)	119[b] (59–164)	95 (13–260)	76

[a] $p < 0.01$.
[b] $p < 0.05$, compared with control values.
[c] Ranges given in Table 1.

normal swallowing using concurrent transnasal videolaryngoscopy and pharyngeal manometry has revealed that onset of vocal cord adduction is the first recordable event in the normal swallow sequence, preceding even genioglossus electromyogram activity [14]. Similarly, the sensory pathways of laryngeal and pharyngeal function are closely related. Whether the response to experimental stimulation of the internal branch of the superior laryngeal nerve is a swallow reflex or a simpler event like glottic depends on the frequency of stimulation [15].

The fundamental question addressed by this study was whether the dysphagia associated with vocal fold motion impairment is due to the disruption of normal laryngeal function on swallowing or to an associated motor or sensory pharyngeal denervation. One of the problems in attempting to answer this question is the relative lack of knowledge on the innervation of the pharyngoesophageal segment. The technical problems encountered in the study of this topic include, e.g., the persistence of UES electrical activity after positioning a manometric catheter in the sphincter [16] and the species differences in autonomic innervation [1,6]. The human cervical esophagus is said to have a poor vagal supply, particularly in its middle third [17]. Horseradish peroxidase studies of canine pharyngoesophageal innervation have shown sensory cell bodies in the glossopharyngeal and vagal nuclei and in C2–T6 spinal ganglia [18], the two peak fields for the cervical esophagus being C2–C6 and T2–T4 [19]. There is a viscerotopic pattern also in vagal sensation: the esophageal cell bodies are more abundant in the proximal jugular vagal nucleus than those of the stomach or duodenum. The efferent supply to the esophageal musculature appears to arise from the caudal part of the dorsal vagal nucleus and the rostral part of the nucleus ambiguus [18]. The myenteric tension receptors and more superficial receptors project via the nodose ganglion to an area of nucleus tractus solitarius ('central subnucleus') whence they project to the esophageal motoneurones of the nucleus ambiguus [20]. Compared with the neurones of the thoracic and subdiaphragmmatic esophagus, the neurones of the laryngopharyngeal and cervical esophageal musculature have extensive extranuclear dendritic arborization into the adjacent reticular formation, creating a wide target area for multiple central afferents [21]. The central deglutition pattern generator appears to reside in the premotoneurones of the nucleus tractus solitarius [22].

In the cat, a fall in UES pressure follows bilateral vagosympathetic trunk blockade [6]. In this species, there is also clear evidence of a dual afferent cervical esophageal innervation so that the UES pressure response to intraesophageal balloon distension is preserved even during prolonged unilateral vagal blockade [23]. There also appears to be a dual motor innervation to the distal cervical esophagus in the dog, as bilateral pharyngoesophageal nerve section in this species results in a severe but only temporary dysphagia [9]. The distal cervical esophagus recovers more completely and stimulation of the RLN causes a cervical esophageal contraction. Thus, both the pharyngoesophageal nerve and the RLN seem to innervate the distal cervical canine esophagus. Study of the *in vitro* behavior of rabbit pharyngoesophageal muscle strips in response to electrical and chemical stimuli indicated that the cricopharyngeus and cervical esophagus had a somatic cholinergic supply, whereas the responses of thyropharyngeus and middle constrictor strips indicated the presence of nonmuscarinic, nonnicotinic, nonadrenergic receptors [24]. A small human autopsy

series found 'large' branches from both right and left RLNs entering cricopharyngeus in 5 subjects [9] but others' findings have been much less conclusive.

The numbers in each subgroup where etiology of the paralysis was known are small but there are interesting differences between patients with a central lesion and those with a RLN lesion. The RLN patients show a greater degree of UES relaxation and greater pharyngeal contraction amplitudes for both water and bread swallows than controls (who were age-matched to allow for the age-related changes which we have previously shown) [25]. We have also shown, however, that there are many confounding variables in the accurate registration of hypopharyngeal pressures [26] and the ranges observed in both patient groups during water swallows were skewed by the measurement of very high pressures (around 400 mmHg) in a few individuals. Ekberg et al. [27] reported radiological evidence of weakness of pharyngeal constrictor activity in 6 of a series of 22 patients thought to have RLN palsy, but 10 of these were of idiopathic/neoplastic etiology. Our own patients with idiopathic etiology have manometric pressures which tend to be intermediate between those of the other two diagnostic groups, suggesting that the 'idiopathic group' may be heterogeneous, including both central and peripheral (RLN) lesions.

The mechanisms for the maintenance of laryngeal competence were recently reviewed by Shin et al. [7,28] in a series of human and feline experiments. The protective increase in subglottic pressure during laryngeal descent was preserved even after section of one RLN [28], although peak glottic closing pressure was reduced by around one-third [7]. The latter study also showed a deglutitive synergism between the inferior constrictor and the laryngeal adductors in closing the laryngeal inlet which may, in part, explain the association of symptomatic dysphagia and the reduced UES pressures of the 'central' palsy patients in the present series (Table 2). Subsequent studies using synchronized videofluoroscopy and pharyngeal manometry in healthy volunteers showed that the duration of laryngeal exposure increased with increasing volume of swallowed bolus [29], a salutory reminder of the complexity of deglutition during eating.

The association of RLN paralysis with reduced pharyngoesophageal wave durations (Fig. 2) may be due to loss of RLN afferent discharge which increases during swallowing [28]. The human importance of the RLN in the innervation of the human cervical esophagus [30] is one possible explanation for the reduction in upper esophageal wave duration in bread swallows (Fig. 2). Alternatively, as there was a significant increase in pharyngeal contraction amplitudes following RLN paralysis (and a nonsignificant increase in tonic UES pressure) it is possible that this nerve has a physiological inhibitory function in the pharyngoesophageal segment. As the pharyngeal contraction wave follows the tail of the bolus, helping it to clear the pharyngeal inlet [31], a further possibility is that the augmented pharyngeal contraction in RLN paralysis may simply represent a compensatory mechanism to protect an unusually exposed subglottis in a group of patients whose motor pharyngeal supply is intact. Thus, the patients with central paralysis may have additional disruption of pharngeal innervation and one can speculate that it is their failure to generate a similar protective pharyngeal response that contributes to the dysphagia.

In conclusion, the present study is the first detailed manometric investigation of patients with vocal fold motion impairment. The results show that the level of vagal involvement significantly influences both the subjective swallowing performance and the manometric findings. The extent of the pharyngoesophageal dysmotility which we have demonstrated indicates that it is this, rather than the disruption of laryngeal kinetics, that is responsible for the subjective dysphagia.

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