Cricopharyngeus Dysfunction and Acute Dysphagia

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THE cricopharyngeus is the smaller and caudal component of the inferior pharyngeal constrictor (Figs. 1 and 2). In most cases the muscle is quite distinct from the higher thyropharyngeus muscle. On each side the cricopharyngeus takes origin from the postero-inferior margins of the lateral surfaces of the cricoid cartilage. The upper oblique fibres sweep posteriorly and upwards to be inserted in the posterior raphe which is common to all the pharyngeal constrictors. In contrast, the lower horizontal fibres form a continuous muscular sling without insertion into the central raphe. In the human, at the junction of the hypopharynx and esophagus, the muscular architecture does show considerable variability: in some cases the horizontal component of the cricopharyngeus forms a thick muscular band sharply demarcated from the circular fibres of the esophagus; in others this separation is less distinct, with fibres passing caudally and posteriorly to decussate with the longitudinal fibres of the esophagus; in a few cases no distinct muscular transition zone is recognized.[1]

Because of the anatomical arrangement of muscle fibres at the hypopharyngo-esophageal junction, there are two areas of relative weakness through which it is possible for herniation of mucosa to occur. The first is named Laimer's triangle, situated posteriorly, and defined superiorly by the cricopharyngeus and laterally by the spreading longitudinal fibres of the esophagus. The second, known as Killian's dehiscence, is between the oblique and horizontal fibres posterolaterally. It is through this area of weakened musculature that Zenker's diverticulum is said to protrude.

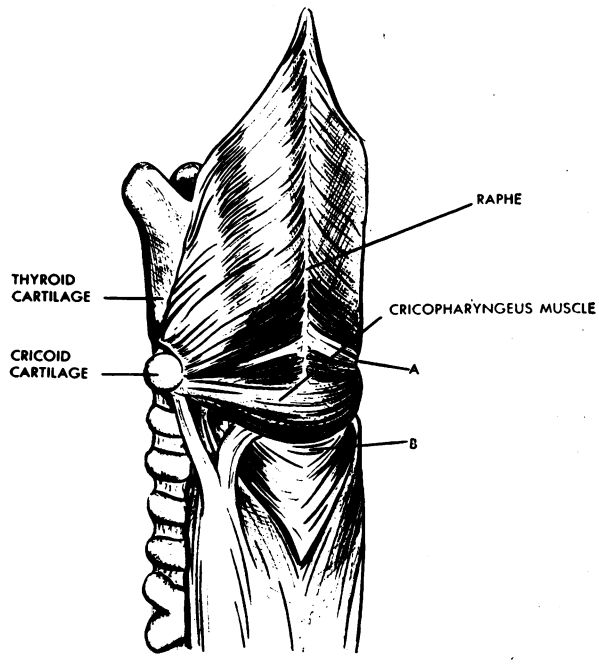

Fig. 1.—Inferior pharyngeal constrictor seen from behind: Killian's dehiscence at point A, Laimer's triangle at point B.

PHYSIOLOGY

Although the physiology of the sphincteric action at the pharyngo-esophageal junction is still in dispute, recent experimental work[1-3, 6] has challenged some of the time-honoured concepts.[4] The proximal anatomical boundary of the physiological sphincter at the pharyngo-esophageal junction is defined by the cricopharyngeus muscle. The sphincter itself, however, is represented at rest by a zone of elevated pressure 2 to 4 cm. in length which includes a segment of esophagus just distal to the cricopharyngeus. The sphincter is capable of withstanding pressures up to 11 cm. of water in the esophagus. The region of peak or maximal resting high pressure does, however, clearly correspond to the radiologically demonstrated cricopharyngeus.[5]

In the well-ordered and sequential train of events necessary for propulsion of a bolus from the mouth to the esophagus the normal response of relaxation of the cricopharyngeus plays a key

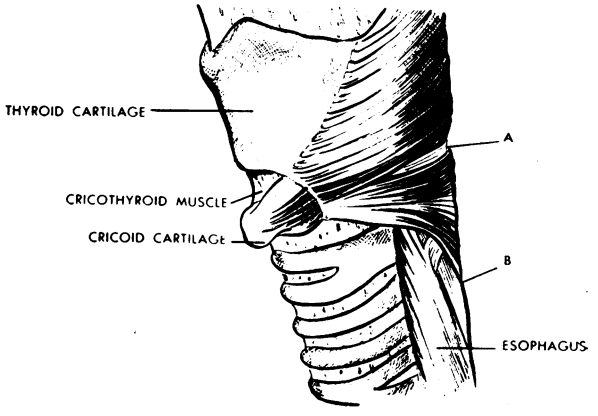

Fig. 2.—Inferior pharyngeal constrictor, lateral view.

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role and is dependent on uninterrupted transmission of vagal impulses. Experimentally the sympathetic nervous system does not contribute to the motor innervation of the sphincter.[6] In other words, there is no basis for a simple opening-and-closing mechanism controlled by opposing neural influences. It is more likely that vagal branches to the sphincter zone control both contraction and relaxation. The latter change is probably due to a reduction of nerve impulses which normally pass continuously to the sphincter, maintaining its resting tone.[7]

An additional fact established by experimentation is that the cricopharyngeus exhibits phasic rather than tonic or continuous activity,[2] and this activity is closely linked with the respiratory cycle and with deglutition.[3] Pressure levels at the sphincter zone are highest during the predeglutition and inspiratory phases. Increase in sphincteric pressure during inspiration minimizes the entrance of air into the esophagus.

The phasic activity of the cricopharyngeus is abruptly halted at the end of the oral or voluntary stage of swallowing, just before the bolus reaches the oropharynx and before the larynx and cricoid are elevated. The pharyngo-esophageal isthmus is then opened passively, as a result of elevation of the larynx in the presence of an inactive inferior constrictor. The bolus is then thrust from a high-pressure area to an area of relatively lower pressure by the forceful contractions of the base of the tongue combined with the primary stripping wave which originates in the superior pharyngeal constrictor. The essential role of the cricopharyngeus in the act of swallowing, therefore, is one of active inhibition.

RADIOLOGICAL FEATURES

The radiological demonstration of a cricopharyngeus bar is considered by most authorities to be an abnormal phenomenon[1, 8-11] even though the patient may not complain of difficulty in swallowing (Fig. 3). When dysphagia is the chief complaint, a fairly close correlation can be established between the prominence of the cricopharyngeus and the severity of dysphagia. Characteristically, the muscle band appears as a horizontally placed indentation on the posterior wall of the contrast-filled hypopharynx at the level of the bodies of C5 or C6, and will migrate upwards during swallowing, for it is tethered to the cricoid cartilage. Its prominence varies from a low mound to a sharply defined shelf which can virtually occlude the full width of the pharyngeal lumen. Its thickness seldom exceeds 1.2 cm.

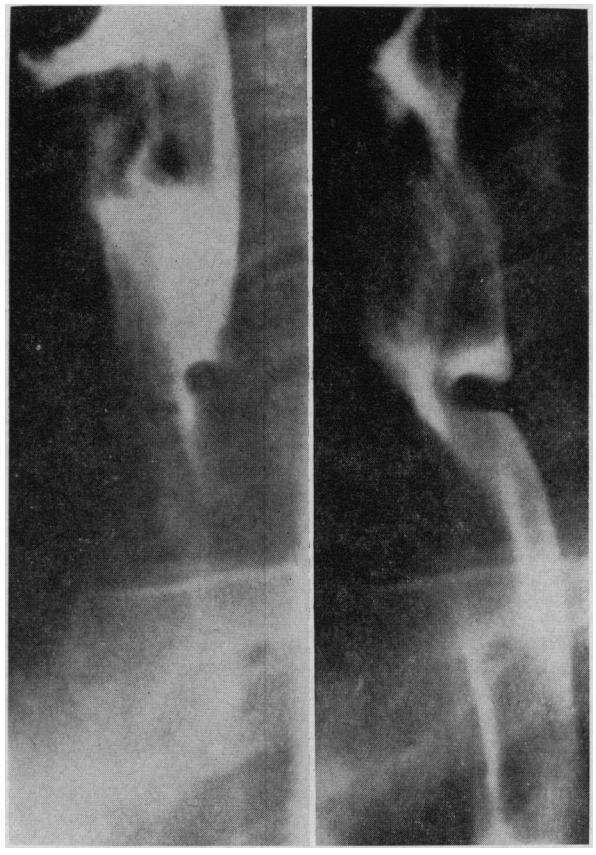

Fig. 3.—Two sequential projections of barium-filled hypopharynx with prominent posterior indentation produced by the cricopharyngeus muscle.

A knowledge of regional anatomy and physiology can be applied to explain some of the puzzling features relating to the abnormal radiological appearance of the cricopharyngeus. For example, the phasic activity of the sphincter accounts for the vagary of its demonstration; a distinct muscle shelf can be recognized with one swallow, yet this is inconspicuous or even absent with a second swallow. The anatomical variation in muscle thickness explains a difference in the prominence of the cricopharyngeal bar in two individuals, assuming that both were subjected to the same abnormal neural influences. If one accepts the premise that the presence of a cricopharyngeal bar is the result of a motor dysfunction alone, then one need not assume that muscular hyperplasia is necessarily present but that a normal muscle is in tonic contraction. I have not yet encountered any evidence that a cricopharyngeal bar, once demonstrated, increases in prominence as one would expect, were its presence the result of muscular hyperplasia alone.

The malfunctioning cricopharyngeus, out of sequence with the swallowing mechanism, acts

as an obturator to the passage of a bolus. As a cause of chronic high dysphagia the role of the cricopharyngeal bar has been well documented. It is not generally appreciated, however, that the same mechanism may be responsible for the production of acute dysphagia. Four such cases have been encountered during the past two years at the Toronto General Hospital and are unique in that an incapacitating degree of dysphagia occurred acutely in patients who, initially, gave no history of dysphagia (or at least minimized any such preceding dysphagia). The radiological feature common to these patients was the presence of a cricopharyngeal bar.

This reluctance voluntarily to admit to a history of dysphagia is a tribute to the adaptability of the human. The symptom is made little of because the patient has functionally compensated for the sphincter dysfunction by alteration of his diet to softer foods and by ingestion of a smaller bolus.

The following two cases illustrate the salient clinical and radiological features of cricopharyngeal dysfunction presenting acutely.

CASE HISTORIES

CASE 1.—M.W., an 83-year-old woman, sustained an undisplaced intertrochanteric fracture of the left hip when she fell while rising from a couch. She was admitted to the Toronto General Hospital, and on the same day the fracture was treated by internal fixation with Knowles' pins. The immediate postoperative period was complicated by acute dysphagia for which nasogastric feedings were required, supplemented by fluids given intravenously.

Three days after surgery a cine-recording of a barium swallow demonstrated a complete obstruction at the pharyngo-esophageal junction (Fig. 4a and b). On the twelfth postoperative day esophagoscopy disclosed no organic pathology in the hypopharynx or upper esophagus apart from slight resistance to passage of the instrument at the pharyngo-esophageal junction. The patient's ability to swallow did not improve. Increasing laryngeal stridor after esophagoscopy necessitated a tracheostomy and at the same time a cricopharyngeal myotomy was performed. After a stormy postoperative course complicated by what was probably a monilial esophagitis, the patient gradually recovered her ability to swallow over the succeeding three weeks (Fig. 5a and b).

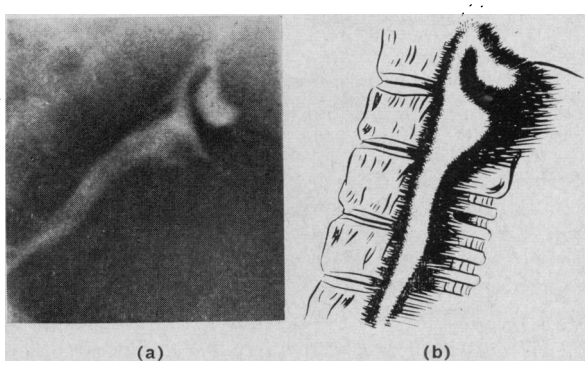

Fig. 5.—Case 1. Postoperative examination: (a) Following myotomy of the cricopharyngeus, restoration of unobstructed flow through the pharyngo-esophageal junction. (b) Line drawing of the same frame.

It is of interest to note that on close questioning the patient admitted to episodic high dysphagia during the previous three to four years, but she had not considered this of sufficient importance to deserve mention when she was admitted to hospital.

CASE 2.—R.R., a 79-year-old woman, had a one-year history of mild intermittent difficulty in swallowing, marked by a rapidly worsening and sustained dysphagia localized to the cervical area and associated occasionally with aspiration and spasmodic cough.

Radiological examination demonstrated a prominent bar indenting the posterior aspect of the barium column at the pharyngo-esophageal junction, and

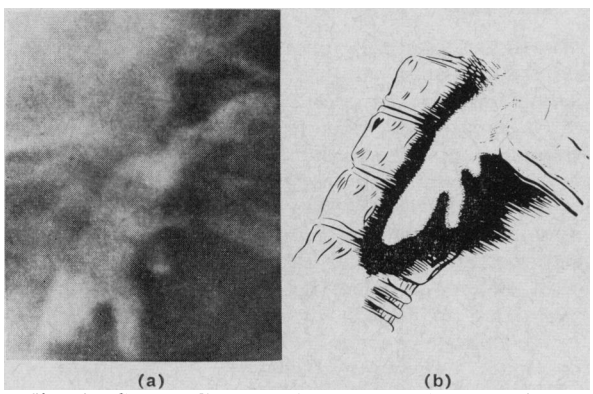

Fig. 4.—Case 1. Preoperative examination: (a) Single frame from a 16-mm. cine sequence showing, in lateral projection, complete obstruction at the pharyngo-esophageal junction. (b) Line drawing of the same frame.

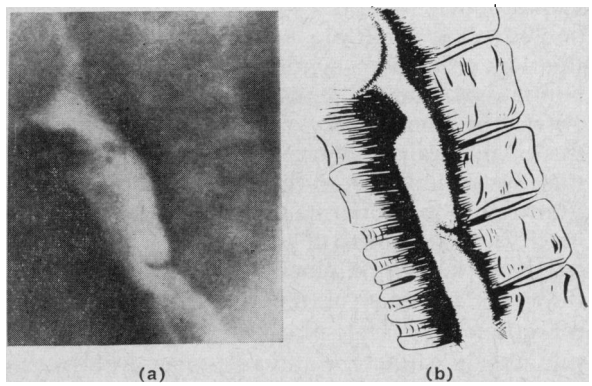

Fig. 6.—Case 2. Preoperative examination: (a) indentation on posterior wall of hypopharynx due to cricopharyngeal bar. The upper margin of the muscle is partially obscured by a small Zenker's diverticulum. (b) Line drawing of the same frame.

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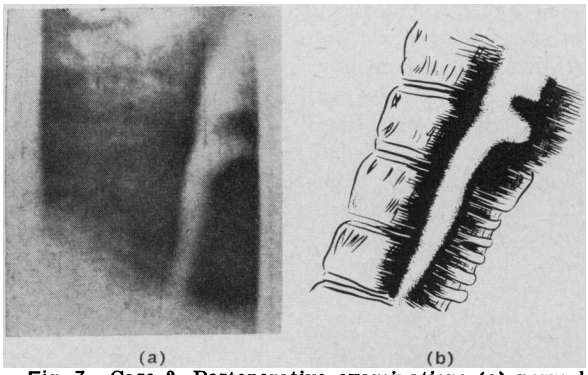

Fig. 7.—Case 2. Postoperative examination: (a) normal pharyngeal channel re-established following cricopharyngeal myotomy. (a) Line drawing of the same frame.

a small Zenker's diverticulum as well (Fig. 6a and b). At endoscopy, in spite of general anesthesia, the firm cricopharyngeal muscular bar remained hypertonic, which made introduction of the instrument beyond this point difficult. A local biopsy proved negative for malignancy.

The situation was remedied by a vertical cricopharyngeal myotomy three inches in length which divided the longitudinal and circular muscle. The small diverticulum was not excised.

The postoperative convalescence was uncomplicated, and 10 days after the operation the patient was enjoying a soft diet without difficulty and subjectively noticed a marked improvement in her ability to swallow (Fig. 7a and b).

DISCUSSION

It does not serve any useful purpose to categorize cricopharyngeal dysfunction under the broad heading of pharyngeal palsy. This term implies a diminished or absent contractile ability of pharyngeal musculature, while cricopharyngeal dysfunction denotes a failure of normal relaxation. Although both may sometimes occur in the same individual, they more often appear as separate entities.

Pharyngeal palsy has a diverse etiology: in amyotrophic lateral sclerosis, syringobulbia, poliomyelitis, multiple sclerosis and posterior-inferior cerebellar syndrome there is an abnormality of neural transmission; in myasthenia gravis the abnormality involves transmission at the myoneural junction,[12] and in dermatomyositis, systemic lupus erythematosus and muscular dystrophies the abnormality is in the musculature itself. In the majority of patients with pharyngeal palsy of known etiology, the cine recordings show no evidence of delayed or asynchronous relaxation of the sphincter.[6] The fact that sphincter dysfunction and pharyngeal palsy can occur concomitantly in a small minority of patients might imply that, in these patients, the pharyngeal constrictors and cricopharyngeus sphincter have a nerve supply derived from a common source.

The role of the malfunctioning cricopharyngeus in the production of dysphagia has a simple anatomical basis. It would be an understatement, however, to infer a simple physiological aberration. The fact that cricopharyngeal dysfunction is symptomatically slowly progressive and is more often encountered in the fifth decade and beyond has been offered as argument that it is a phenomenon of ageing. On a teleological basis, some authors suggest that the cricopharyngeal bar is a protective mechanism against the upward rush of gastro-esophageal reflux. Only one of the four cases in this report showed a small hiatus hernia with minimal gastro-esophageal reflux. Others suggest the possibility of limited bulbar damage selectively affecting the cricopharyngeus muscle. The abnormality may be at the muscle-receptor level with failure of normal pharyngeal reflex arc activity to be initiated during deglutition. Whatever the mechanism, the end result is a failure of normal relaxation.

Myotomy of the pharyngo-esophageal sphincter has been of proved value[13-15] in selected cases but should be undertaken with caution and only after a careful clinical, radiological and esophagoscopic evaluation. The efficiency of the entire swallowing mechanism must be assessed, and organic pathology excluded, both in the pharynx and in the esophagus. A thorough cineradiographic documentation should be made and the permanency of the dysphagia established. Some patients may make a remarkable and spontaneous recovery from an acute dysphagia, in which case myotomy is not justified. Subjective improvement following myotomy is generally quite striking and improved nutrition will result. Myotomy, however, as in the case of achalasia, is based on relief of an obstructing physiological abnormality.

Summary The proximal anatomical boundary of the physiological sphincter at the pharyngoesophageal junction is defined by the cricopharyngeus muscle. In the normal and sequential train of events necessary for propulsion of a bolus from the mouth to the esophagus, relaxation of the crichopharyngeal sphincter plays a key role and is dependent on intact vagal transmission. Selective interference with motor function may originate either centrally or peripherally and may be responsible for cricopharyngeal dysfunction with failure of relaxation. Such a mechanism is postulated to explain dysphagia occurring acutely in the absence of organic disease of the hypopharynx or upper esophagus. Under these conditions the radiological findings are frequently misinterpreted as indicating obstruction secondary to a tumour mass or to an impacted bolus.

Résumé La frontière anatomique proximale du sphincter physiologique au niveau de la jonction du pharynx et de l'œsophage est délimité par le muscle cricopharyngien. Dans la succession normale des phénomènes qui marquent la propulsion du bol alimentaire de la bouche vers l'œsophage, le relâchement du sphincter cricopharyngien joue un rôle capital et dépend de l'intégrité de la transmission de l'influx vagal. Une entrave sélective à la fonction motrice peut avoir un point de départ central ou périphérique et peut expliquer le dysfonctionnement cricopharyngien par absence de relâchement. Ce mécanisme peut faire comprendre la dysphagie aiguë qui survient en l'absence d'une lésion organique de l'hypopharynx ou de l'œsophage supérieur. Dans ces conditions, on risque souvent d'interpréter erronément les constatations radiologiques comme indiquant une occlusion secondaire à une masse tumorale ou à l'enclavement d'un bol alimentaire.

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Angiographic Assessment of the Ischemic Left Ventricle:
A Preliminary Report

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SINCE the popularization of selective coronary arteriography by Sones and Shirey,[15] many variations of the original technique have been described and lesions of the coronary arteries clearly demonstrated.[8, 12, 14, 17] In addition, some cardiovascular centres have gone so far as to favour angiographic assessment of left ventricular function in patients considered for revascularization procedures.[3, 4]

It is the purpose of this communication to outline a cinegraphic classification of abnormalities of left ventricular contraction for comparison with other hemodynamic parameters, in an effort to supply the clinician and cardiovascular surgeon with meaningful information with regard to the suitability of patients for revascularization procedures.

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MATERIALS AND METHODS

Over a 12-month period a series of 50 patients with chest pain have been studied by left ventriculography, right and left heart catheterization and selective coronary cineangiography. The group consisted of 43 males and 7 females, ranging in age from 28 to 67 years.

1. *Measurement of Left Ventricular Area*

Left ventricular cineangiograms recorded in the right anterior oblique position were projected on to a paper taped to a screen and advanced frame by frame so that optimum images could be selected. After tracing the end-systolic and end-diastolic ventricular and catheter images on to the paper, planimetric measurements of the ventricular areas were obtained (Fig. 2). Care was taken to select cycles in sinus rhythm. These areas could then be plotted as ratios or corrected for magnification, knowing the true catheter diameter, and expressed as absolute values.

2. *Classification of Left Ventricular Contraction*

Left ventricular contractions were divided by inspection of the angiogram and ventricular measurement into four groups: