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Manometric Findings of the Upper Esophageal Sphincter in Esophageal Achalasia

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Abstract. Pharyngeal and upper esophageal sphincter (UES) manometry was performed in 15 patients with esophageal achalasia and compared with that in 10 healthy controls. Neither the pharyngeal contraction pressure nor the UES resting pressure were significantly different between the two groups, although the UES residual pressure in patients with achalasia was significantly increased compared with that in controls. Pneumatic dilatation of the lower esophageal sphincter (LES) was performed in these patients. After successful LES dilatation, the increased UES residual pressure in patients with esophageal achalasia decreased significantly. Our results suggest that UES relaxation in patients with esophageal achalasia is incomplete compared with that in healthy adults. This UES abnormality is not a primary defect but a secondary phenomenon.

The manometric characteristics of the lower esophageal sphincter (LES) and esophageal body in patients with esophageal achalasia have been well described [1–3], but the role of upper esophageal sphincter (UES) function in achalasia is not clear. A few studies have demonstrated abnormal UES function in esophageal achalasia [4–6], and case reports of esophageal achalasia with acute airway obstruction suggest an association between this unusual complication and dysfunction of the UES [7, 8].

In this study, manometric findings of the UES and pharynx in patients with esophageal achalasia were examined and compared with those in normal controls. Changes in the manometric findings of patients with esophageal achalasia prior to and following pneumatic dilatation of the LES were also evaluated. These findings were compared with those in previous reports of UES function in esophageal achalasia.

Materials and Methods

Subjects

Fifteen patients with a diagnosis of esophageal achalasia (9 men, 6 women; age range 20–74 years, mean 44 years) and 10 healthy adults (6 men, 4 women; age range 22–76 years, mean 52 years) underwent manometric evaluation of the LES, esophageal body, UES, and pharynx in our institution from July 1990 to December 1994. None of the patients with achalasia had previously been treated. The chief complaints of these patients included dysphagia, vomiting, and weight loss that had been present for 2 months to 7 years. Two patients (a 36-year-old man and a 39-year-old woman) had a history of aspiration pneumonia, but the other patients had no history of respiratory complications. Prior to manometric evaluation, each patient underwent a barium swallow. Barium esophagograms demonstrated that the maximum diameter of the esophagus was less than 3.5 cm in one patient, 3.5 to 6.0 cm in nine patients, and more than 6.0 cm in five patients.

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Equipment

Esophageal pressure was measured with a water-infusion system. The infusion catheter consisted of eight small capillary tubes joined together around a larger central tube. Each of the eight capillary tubes had side holes at predetermined points along its length spaced 5 cm apart and positioned along the external face of the tubes at 90-degree angles; four of the proximal side holes were used for the manometric study. The capillary tubes were continuously perfused with distilled water at a constant rate by a low-compliance, pneumohydraulic, capillary-infusion pump powered by compressed nitrogen (Arndorfer Medical Specialties, Greenfield, WI, USA). The individual capillary tubes were interfaced by an external transducer to a physiograph.

Manometric Studies

Subjects were studied fasting and supine. The catheter was placed with nasoesophageal intubation, and manometric studies were conducted with the station pull-through technique. The resting pressure of the LES, esophageal body, and UES were measured. The UES residual and peak pharyngeal contraction pressures, which were induced by dry swallowing, were also measured. The UES residual pressure was defined as the nadir of the UES relaxation during swallowing, which was measured when the orifice was positioned at the middle of the high pressure zone of the UES. Esophageal baseline pressure was used as zero reference for UES resting pressure and UES residual pressure. Relaxation of the UES was considered complete if the residual pressure had a negative value (Fig. 1). The pressure recording represented the mean value of the measurements obtained from the four

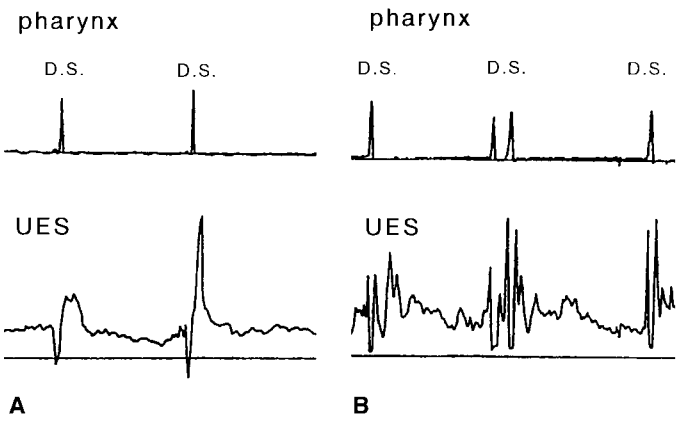

Fig. 1. Upper esophageal sphincter (UES) residual pressures in a healthy adult (**A**) and a patient with achalasia (**B**). The top tracings represent the output of the transducer located in the pharynx. The bottom tracings represent the output of the UES transducer. The esophageal baseline pressure was used as zero reference for the UES tracing. D.S.: dry swallowing. A. In the healthy adult the nadir of UES relaxation is below the esophageal baseline. B. In a patient with achalasia, the nadir of UES relaxation is above the esophageal baseline.

proximal orifices oriented at 90-degree angles because of the asymmetry of the UES. The manometric findings of patients with esophageal achalasia were compared with those of normal controls. Data are presented as the mean ± SD. A nonpaired *t*-test was used for statistical analysis.

Pneumatic Dilatation of the LES

All 15 patients with achalasia underwent pneumatic dilatation of the LES with a Rigiflex Achalasia Dilator (30 or 35 mm in diameter; Microvasive, Milford, MA, USA). All patients gave their informed consent to undergo this therapy. A session of LES dilatation consisted of two episodes of balloon inflation for 1 minute at a pressure of 9 psi. No significant complications were observed. Manometric studies were repeated again 4 to 14 weeks after dilatation of the LES and were compared with the manometric data before dilatation. A paired *t*-test was used for analysis.

Results

The manometric data from patients with achalasia and healthy controls are shown in Table 1. The LES resting pressure in patients with achalasia was significantly increased compared with that in controls (44.1 ± 15.2 mmHg vs. 25.1 ± 7.2 mmHg, respectively; $p < 0.01$). The resting pressure of the esophageal body in patients with achalasia was also significantly increased compared with that in controls (8.4 ± 6.0 mmHg vs. −3.5 ± 1.8 mmHg, respectively; $p < 0.001$). These differences are well known manometric characteristics of esophageal achalasia. Neither the UES resting pressure nor the pharyngeal contraction pressure were significantly different between patients with achalasia and healthy controls. The UES residual pressure in patients with achalasia was 1.7 ± 1.5 mmHg and was significantly ($p < 0.01$) higher than that in controls (−4.1 ± 4.2 mmHg). Nine of ten healthy controls had a negative UES residual pressure, whereas only one patient with achalasia had a negative UES residual pressure (Fig. 2). This finding indicates that UES relaxation in patients with achalasia is incomplete. Correlation between the UES residual pressure and the resting pressure of the LES or esophageal body was not statistically significant.

Table 1. Changes of manometric parameters following LES dilatation in patients with achalasia.

Parameter	Achalasia patients (n = 15) Before dilatation	After dilatation	Controls (n = 10)
LES resting pressure	44.1 ± 15.2*	16.6 ± 4.4***	25.1 ± 7.2
Resting pressure of esophageal body	8.4 ± 6.0**	−3.4 ± 2.8***	−3.5 ± 1.8
UES resting pressure	45.0 ± 26.5	34.0 ± 17.4	50.4 ± 23.4
UES residual pressure	1.7 ± 1.5*	0.0 ± 2.1****	−4.1 ± 4.2
Pharyngeal contraction pressure	58.3 ± 13.8	54.1 ± 20.4	72.4 ± 18.7

Results are means ± SD, in millimeters of mercury (mmHg).
*$p < 0.01$, **$p < 0.001$ (vs. control); ***$p < 0.001$, ****$p < 0.01$ (vs. before dilatation).

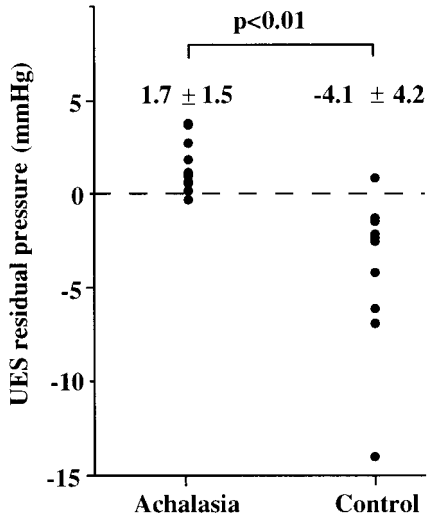

Fig. 2. Distribution of UES residual pressure in patients with achalasia and controls. UES residual pressure in patients with achalasia was significantly ($p < 0.01$) increased compared with that in controls.

After dilatation of the LES, all patients reported alleviation of their symptoms. The manometric findings after dilatation of the LES were compared with those before dilatation. The LES resting pressure was significantly decreased (16.6 ± 4.4 mmHg, $p < 0.001$), as was the resting pressure of the esophageal body (−3.4 ± 2.8 mmHg, $p < 0.001$), which was identical to that in healthy controls. These changes indicated successful pneumatic dilatation of the LES. Neither the UES resting pressure nor the pharyngeal contraction pressure were affected by the LES dilatation. The UES residual pressure was significantly decreased after dilatation of the LES (1.7 ± 1.5 mmHg vs. 0.0 ± 2.1 mmHg; $p < 0.01$) (Fig. 3), and eight patients had a negative UES residual pressure. However, the UES residual pressure after dilatation of the LES in patients with achalasia remained significantly higher than that in controls.

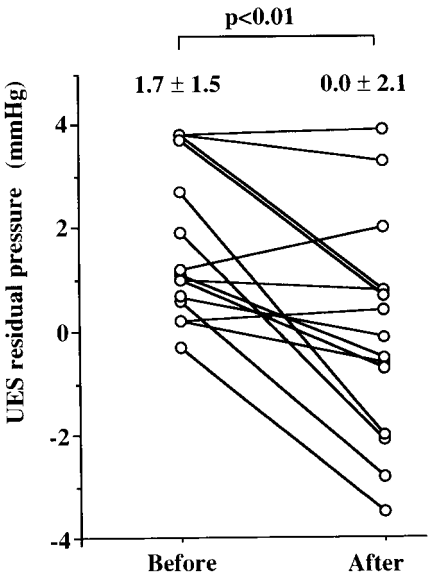

Fig. 3. UES residual pressure in achalasia patients before and after pneumatic dilatation of LES. The UES residual pressure was significantly ($p < 0.01$) decreased after dilatation of the LES.

Discussion

Esophageal achalasia is considered a primary motility disorder involving the smooth muscle of the esophageal body and LES. The characteristic findings of manometry in esophageal achalasia have been well known: incomplete relaxation of the LES, often increased LES resting pressure, and an absence of peristalsis in the esophageal body [1–3]. Increased resting pressure of the esophageal body has also been observed. However, the role of UES function in esophageal achalasia has not been clear. Little information is available in the literature about UES function in these patients. Furthermore, case reports of esophageal achalasia presenting an acute airway obstruction suggest an association between this unusual complication and dysfunction of the UES.

Bello et al. reported the first case in 1950; it involved an elderly woman with no prior esophageal disease who presented with megaesophagus and tracheal narrowing due to posterior compression [7]. In 1989 Becker and Castell reported a patient with esophageal achalasia who presented with acute airway obstruction and then summarized the previously reported 11 cases [8]. This unusual but emergent complication suggests the presence of UES dysfunction in esophageal achalasia.

Some authors have described dysfunction of the UES in esophageal achalasia. Jones et al. performed dynamic imaging of the pharynx in esophageal achalasia [4]. In 11 of 21 consecutive patients, abnormalities such as cricopharyngeal prominence, asymmetry of pharyngeal contraction, or epiglottic tilt and lateral pharyngeal pouch were demonstrated. They proposed that marked esophageal dilatation could result in compensatory changes, including a protective reflex to prevent esophagopharyngeal regurgitation. Dudnick et al. described abnormal UES function in esophageal achalasia [5]. In their study 19 patients with achalasia were compared with 14 healthy controls. Major abnormalities in UES function occurred with esophageal achalasia, which is characterized by an increased UES residual pressure during swallow-induced relaxation and shortened duration of the UES relaxation. Zhang and Diamant found repetitive contractions of the UES and upper esophageal body in 67% of patients with achalasia [6]. They observed a close relation of these repetitive contractions of the UES to increased intraesophageal pressure and elevated LES pressure.

In our study UES residual pressure in patients with achalasia was significantly increased compared with that in controls. Only one patient had a negative UES residual pressure. UES relaxation in patients with achalasia was incomplete compared with that in healthy adults. Neither the UES resting pressure nor the pharyngeal contraction pressure was significantly different for patients with achalasia and the controls. After successful pneumatic dilatation of the LES, elevated UES residual pressure was significantly decreased. The improvement in UES residual pressure was accompanied by a decrease in the pressure of the LES and the esophageal body. These data suggest that increased UES residual pressure in patients with achalasia is not a primary defect but, rather, a secondary phenomenon due to increased resting pressure of the esophageal body.

It is possible that increased UES residual pressure represents a protective effect against regurgitation. A neural feedback mechanism may exist between UES relaxation and tension in the esophageal wall, such that increased resting pressure in the esophageal body results in inhibition of UES relaxation. Some authors also reported increased UES resting pressures after intraesophageal distension in dogs and healthy men [9–11]. These investigators proposed that the UES serves as a dynamic barrier to esophagopharyngeal reflux and subsequent tracheobronchial aspiration.

Further studies are necessary to elucidate the mechanisms underlying increased UES residual pressures in patients with achalasia. In these patients, manometric examination is indispensable for precise diagnosis, determination of the therapeutic approach, and evaluation of the therapeutic results. It is important to perform UES and pharyngeal manometry in addition to that of the LES and esophageal body, so potential dysfunction of UES and pharynx is not overlooked.

Résumé

On a comparé les résultats de la manométrie du pharynx et du sphincter supérieur de l'oesophage (SSO) chez 15 patients achalasiques à ceux de 10 sujets sains (contrôles). Aucune différence significative de la pression de contraction du pharynx ou de la pression au repos du SSO n'a été retrouvée par rapport aux résultats des 10 sujets contrôles. La pression du SSO résiduelle était plus élevée chez le sujet achalasique par rapport aux sujets sains. Une dilatation pneumatique a été réalisée chez les patients achalasiques. En cas de succès, la pression du SSO résiduelle a diminué de façon significative par rapport aux contrôles. Nos résultats suggèrent que la pression de relaxation chez le patient achalasique est incomplète par rapport à celle de l'adulte sain. Il s'agit d'une anomalie secondaire, et non pas primitive.

Resumen

Se practicó manometría de la faringe y del esfínter esofágico superior (EES) en 15 pacientes con acalasia del esófago, a fin de comparar los valores con los de 10 personas saludables que sirvieron como grupo control. No se encontraron diferencias

significativas en cuanto a la presión de contracción ni a la presión en reposo del EES entre los dos grupos. Sin embargo, la presión residual del EES apareció significativamente más alta en los pacientes con acalasia. Se practicó dilatación neumática del esfínter esofágico inferior (EEI) en los pacientes, y luego de una dilatación exitosa se observó disminución significativa de la elevada presión residual del EES. Nuestros resultados sugieren que la relajación del EES en la acalasia es incompleta, en comparación con los adultos de buena salud. Tal anormalidad del EES no es un defecto primario, sino un fenómeno secundario.

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Invited Commentary

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Discussing the assessment of swallowing, Jones et al. stated: "simultaneous disorders of the pharynx and esophagus are so frequent that the complete swallowing chain should be examined in all patients with dysphagia" [1]. Further support for this concept is provided by Yoneyama and colleagues in this issue, where they examined upper esophageal sphincter (UES) function in patients with achalasia revealing significantly increased residual pressure after dry swallows.

There are two aspects of UES function to consider: first, the question of UES dysfunction in achalasia patients in general; and second, the significance of these general changes in patients presenting with the rare complication of acute respiratory embarrassment. Yoneyama et al. observed changes in a series of 15 patients with achalasia that are similar to those reported by others [2, 3]. The example illustrated (their Fig. 1) shows repetitive contraction, a feature noted by Zhang and Diamant [4]. In addition, there appears to be an increase in pressure in the UES before relaxation, a feature not reported in other studies and not commented on by the authors. Following pneumatic dilatation of the lower esophageal sphincter (LES) there is usually an improvement in esophageal emptying and a reduction in esophageal diameter. These changes contribute to the reduction in the resting pressure in the esophageal body observed by Yoneyama et al., but do they explain the posttreatment changes in UES function? The UES residual pressure was less but still significantly higher than in controls due either to incomplete relaxation or to altered compliance.

Although none of their patients had respiratory embarrassment, the authors suggest that the changes may contribute to respiratory embarrassment seen in occasional patients. Massey et al. showed that 95% of their achalasia patients reported difficulty with belching [5]. Similarly, Becker and Castell suggested that distension of the esophagus by swallowed air against a nonrelaxing UES caused tracheal compression [6].

In a case reported from our unit [7] of respiratory embarrassment in an 87-year-old woman with achalasia our initial treatment was by Heller myotomy, but during the early postoperative period respiratory distress recurred. Physiologic studies after the Heller myotomy showed defective UES function. There was incomplete sphincter relaxation in response to dry and wet swallows. Furthermore, injection of air into the esophagus, a stimulus that usually causes relaxation of the UES and a belch, produced no relaxation in our case and paradoxical postinflation augmentation. Our case provides evidence to support failure of the belch reflex in patients presenting with respiratory embarrassment. The demonstration of postinflation augmentation indicates intact innervation.

Yoneyama et al. suggest that the UES abnormality is a secondary phenomenon, because pneumatic dilatation of LES, to some extent, led to reversal of some of the observed differences. In our case, only cricopharyngeal myotomy corrected respiratory embarrassment. We know that patients with Parkinson's disease often show abnormalities in UES function. Loss of some of the more complex functions of UES probably requires a lesion in the medullary swallow center adjacent to the vagal nuclei [8]. It is not possible to state categorically that it is secondary to LES dysfunction. After all, esophageal achalasia can be induced in the cat by a selective electrolytic lesion in the dorsal motor nucleus of the vagus [9].

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