

Research report

Genesis of the decrement of intraluminal pressure in the UES during swallowing in rabbits

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Abstract

The intraluminal pressure in the upper esophageal sphincter (UES) briefly decreases during swallowing. This decrement in pressure plays an important role in smooth transport of the ingested bolus from the pharynx to the esophagus. It is known that the decrement is caused by cessation of tonic activity of the cricopharyngeus (CP) muscle and also by elevation of the larynx. On the other hand, it is suspected that the recurrent laryngeal nerve (RLN) also contributes to the decrement, since our preliminary study showed for the first time that the decrement in UES pressure was much reduced after the RLN was sectioned. In the present study, we examined the genesis of the decrement of the UES pressure in anesthetized rabbits. When swallowing was elicited by repetitive electrical stimulation of the superior laryngeal nerve, the UES pressure briefly decreased and then abruptly increased. After bilateral sectioning of the RLN, the decrement of the pressure was significantly reduced, whereas the increment was little altered. Sectioning of the pharyngeal branch of the vagus nerve (X-ph) and the RLN mostly eliminated both the decrement and increment of the pressure, and abolished tonic and burst activities of the CP muscle. Electrical stimulation of peripheral end of the RLN decreased the pressure. These results indicate that the RLN and X-ph are involved in the decrement of the UES pressure during swallowing. The RLN generates the decrement by adducting the arytenoid cartilage and closing the glottis. The X-ph contributes to the decrement both by suppressing the tonic activity of the CP muscle and by regulating the laryngeal elevation.

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1. Introduction

The upper esophageal sphincter (UES) serves as a gateway between the pharynx and the esophagus, and plays an important role in smooth transport of an ingested bolus from the pharynx to the esophagus during swallowing. The UES remains closed at rest to prevent reflux of esophageal contents. Once swallowing is evoked, the UES opens briefly

to permit passage of an ingested bolus into the esophagus, and then closes to propel the bolus along the esophagus [1,2]. Opening of the UES is particularly important in swallowing, because incomplete opening of the UES occasionally causes dysphagia (e.g., cricopharyngeal achalasia) [6,9].

To evaluate the opening and closing of the UES, the intraluminal pressure in the UES has been often used. Several previous studies have found that the decrement in UES pressure during swallowing is caused by cessation of tonic activity of the cricopharyngeus (CP) muscle and also by elevation of the larynx and hyoid [1,3]. On the other

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hand, we found for the first time that the decrement in UES pressure during swallowing was much reduced after bilateral sectioning of the recurrent laryngeal nerve (RLN), which innervates the intrinsic laryngeal muscles [7]. Therefore, it is suspected that the RLN also contributes to the decrement in UES pressure during swallowing.

The main purpose of the present study was to determine the genesis of the decrement in UES pressure during swallowing in rabbits. We examined the effects of sectioning of the RLN and the pharyngeal branch of the vagus nerve (X-ph, motor nerve for the CP muscle) on UES pressure during swallowing. In addition, we confirmed the effects of sectioned nerves on UES pressure by means of electrical stimulation.

2. Materials and methods

Experiments were performed in 8 male rabbits weighing 2.5–4.0 kg. The animals were anesthetized with urethan (0.75 g/kg, ip) and then placed in the supine position. A midline incision was made in the ventral surface of the neck. The trachea was isolated, transected 2–3 cm caudal to the cricoid cartilage, and cannulated for free breathing. To provide adequate access to the pharynx and larynx, the sternohyoid muscle was transected at its insertion on the hyoid bone [11]. Bipolar Enamel–Nichrome wire electrodes were placed in the right side of the CP muscle to record the electromyographic (EMG) activity.

The superior laryngeal nerve (SLN) was dissected free from the surrounding tissue and then sectioned bilaterally. The central cut end of the right side of the SLN was electrically stimulated using bipolar silver wire electrodes. Application of repetitive electrical stimulation with rectangular pulses (intensity: 50–100 μ A, frequency: 20–30 Hz, duration: 1 ms) to the SLN induced sequential swallowing [12]. Swallowing was identified by EMG activity of the CP muscle and by visual observation of laryngeal elevation. The RLN and X-ph were also dissected free from the surrounding tissue in advance of their sectioning. The RLN was exposed in the groove between the trachea and the esophagus, and the X-ph was exposed under the thyrohyoid muscle after transection of the stylohyoid muscle [8].

Intraluminal pressure in the UES was recorded using a silicon catheter (outer diameter = 4.7 mm) and a pressure transducer (DX-360, Nihon Kohden). The catheter was sealed up in the tip and had a lateral recording orifice (diameter = 3.0 mm) 1.5 cm proximal to the tip of the catheter. It was perfused continuously with saline and was connected to the pressure transducer. The catheter was inserted into the UES from the rostral cut end of the esophagus, which was transected 4–5 cm caudal to the cricoid cartilage, and fixed to orient the recording orifice ventrally.

The UES pressure and EMG activity of the CP muscle during swallowing were recorded in the following states: (1) before sectioning of the nerves (in control), (2) after bilateral

sectioning of the RLN, and (3) after bilateral sectioning of the RLN and X-ph. Magnitude of the decrement in UES pressure during swallowing was defined as the difference between the value of the resting pressure and the minimal value of the decrement of pressure during swallowing. Similarly, magnitude of the increment in UES pressure was defined as the difference between the value of the resting pressure and the maximal value of the increment during swallowing. The mean values of the decrement and increment in 8 animals in those three states are shown as mean \pm SE. One-way repeated-measures ANOVA and Fisher's PLSD were used to identify differences between the respective states. The significant level was set at $P < 0.05$.

The UES pressure during electrical stimulation of each peripheral end of the RLN and X-ph (intensity: 50–200 μ A, frequency: 20–100 Hz, duration: 1 ms, 20 pulses) was recorded in 6 animals. Magnitude of the decrement and increment in UES pressure during electrical stimulation of the RLN and X-ph was measured in a similar manner as that used during swallowing. The mean values of the decrement and increment in 6 animals are shown as mean \pm SE.

3. Results

3.1. Profile of UES pressure and EMG activity of CP muscle during swallowing

Fig. 1A shows typical recordings of intraluminal pressure in the UES and EMG activity of the CP muscle during swallowing. The resting pressure in the UES was always positive, 4.5 ± 1.8 mm Hg (mean \pm SE, $n = 8$), relative to atmosphere. When swallowing was elicited by repetitive electrical stimulation of the SLN (50 μ A, 20 Hz and 1 ms), the UES pressure decreased briefly at first (brief decrement), then increased abruptly (abrupt increment), and finally returned to the resting pressure. The CP muscle exhibited tonic activity before swallowing. When swallowing occurred, the tonic activity of the CP muscle ceased for 150–200 ms; the period was almost identical to the duration of the decrement in UES pressure. Following the cessation, discharges with small amplitude of the CP muscle appeared for 50–100 ms and the UES pressure began to rise during this period. Finally, large burst activity of the CP muscle occurred with marked increment in UES pressure.

3.2. Effects of sectioning of RLN and X-ph on UES pressure during swallowing

Fig. 1B shows changes in UES pressure after sectioning of the RLN. After bilateral sectioning of the RLN, the magnitude of brief decrement in UES pressure was greatly reduced, whereas the magnitude of abrupt increment was little reduced. Sectioning of the RLN altered neither tonic nor burst activities of the CP muscle. Fig. 1C shows an example of recordings of the UES pressure and CP-EMG

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