Influence of altered tongue contour and position on deglutitive pharyngeal and UES function

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Afferece input from oral and pharyngeal receptors may be important in regulating the temporal relationships among motor events during the oral-pharyngeal swallow. The sequence of motor events is not stereotyped, and the timing of hyoid motion and upper esophageal sphincter (UES) relaxation and opening can be modified by alterations in the characteristics of the swallowed bolus such as volume or viscosity (7, 8, 14). Blockade of all oral-pharyngeal mucosal receptors by topical anesthesia does not influence the temporal relationships among the deglutitive oral-pharyngeal motor events, suggesting that these volume- and viscosity-dependent temporal shifts are not mediated by tactile mucosal sensory receptors (2). It is possible that other receptors deep to the mucosa, such as mechanoreceptors, might mediate the observed temporal shifts. Alterations in tongue contour and position have been shown to serve important functions of bolus containment and loading before bolus propulsion (15). These processes involve deformation of the tongue at swallow initiation and are to an extent dependent on bolus volume (16). It is possible that a sensory cue that is modified by bolus variables might emanate from lingual mechanoreceptors at swallow initiation. We hypothesized that modification of bolus forces on the tongue, at or shortly after swallow initiation, influences bolus volume-dependent temporal events and that lingual mechanoreceptor activity is likely to be involved in this process. The aim of this study was to determine whether experimental modulation of tongue contour and position and thus lingual mechanoreceptor activity mediates volume-dependent changes in swallow coordination. We used this experimental procedure on the premise that it is likely to alter mechanoreceptor activity. Hence a positive finding would support the hypothesis that mechanoreceptor activity is involved in volume-dependent changes in swallow coordination.

METHODS

Subjects. We studied two groups of healthy volunteers recruited by advertisement from the community. Group 1 consisted of 6 healthy subjects (mean age 27.2 ± 6.3 yr) in whom we confirmed that the maxillary splint caused lingual deformity and group 2 consisted of 15 healthy volunteers (mean age 30 ± 11 yr) in whom the physiological effects of dental splints were examined. All were carefully screened, and none had swallowing difficulties, medical illnesses, or were taking any medications. Ethical approval was granted by Southern Sydney Area Health Service Ethics Committee, and all subjects gave written informed consent.

Videoradiography. Subjects were studied by simultaneous videoradiography and manometry as previously described (7). Briefly, subjects were studied seated, and images of bolus swallows were recorded in the lateral projections using a 9-in. Toshiba (Kawasaki, Japan) image intensifier. Fluoroscopic images were recorded on videotape at 25 frames/s by a VHS video recorder (Panasonic, AG6500, Osaka, Japan) for later analysis. The correction factor for magnification was determined before each study by placing two metallic markers set 3 cm apart in the field of the image intensifier, above the subject’s head but in the plane of the UES. Subjects swallowed duplicate 5- and 10-ml boluses of two different densities: 1) high-density liquid barium suspension (250% wt/vol), E-Z-HD, E-Z EM, Westbury, NY) and 2) a low-density water-soluble iodinated radiopaque contrast MD-Gastroview (Mallinkrodt Medical, St. Louis, MO). The rationale for using two densities was to examine the effect of bolus weight on the tongue. All boluses were delivered to the mouth by a syringe. Included in the field of view were the incisor teeth anteriorly, hard palate superiorly, cervical spine posteriorly, and proximal cervical esophagus inferiorly.

Manometry. Pharyngeal pressures were measured with a catheter combining three solid-state transducers (Gaeltec, Dunvegan, Isle of Skye, Scotland) measuring pharyngeal pressures and a 6-cm perfused sleeve assembly (Dentsleeve, Belair, South Australia) measuring UES pressure. The solid-state catheter (OD 2.3 mm) was inlaid into a six-lumen silicon rubber-polivinyl chloride perfused manometric catheter (ID each lumen 0.51 mm). The overall catheter diameter was 6 mm, and the sleeve assembly distal to the transducers had a 5 × 3 mm oval cross section to maintain its anteroposterior orientation within the UES (13). The manometric assembly...
was passed transnasally, and the transducers and the sleeve were orientated posteriorly with the sleeve straddling the UES. The posterior orientation of the transducers was readily verified radiographically. The solid-state transducers were spaced 3 cm apart with the middle transducer lying at the level of the valleculae in the midpharynx and the distal transducer lying at the upper margin of the sleeve in the hypopharynx, just proximal to the UES at the time of maximal sphincter ascent during swallowing. Four perfused side holes, spaced at 1.5-cm intervals in the pharynx with the distal side hole located at the midsleeve position, were used to aid in positioning the sleeve such that its midpoint was in the center of the UES high-pressure zone at rest. The sleeve assembly and side holes were perfused by a low-compliance pneumohydraulic perfusion system at 0.6 ml/min, and UES pressures were registered by external transducers (Spectramed Medical Products), and all signals were amplified and digitized at 200 Hz by preamplifiers (Neomedix Systems, Sydney, Australia) and recorded on a Macintosh computer (Apple, Cupertino, CA) using Gastromac software (Neomedix Systems). The height-adjustable perfusion pump was positioned so that the external transducers were level with the midsleeve position. All pressures were referenced to basal hypopharyngeal (atmospheric) pressure. A purpose-built, video-digital timer unit (Practel Sales, Holden Hill, South Australia) imprinted simultaneously the elapsed time on the video images in hundredths of seconds and a signal on the pressure tracing each whole second, to permit precise temporal correlation of video images with pressure.

Validation experiment. A validation experiment was conducted in the six group 1 subjects in whom we examined radiologically the tongue surface contour relative to the palate to confirm that a maxillary splint did exaggerate tongue deformity for a given bolus volume. In these six subjects we applied three tantalum markers (3-mm diam) to the ventral surface of the tongue in the midsagittal plane. The height-adjustable perfusion pump was positioned so that the external transducers were level with the midsleeve position. All pressures were referenced to basal hypopharyngeal (atmospheric) pressure. A purpose-built, video-digital timer unit (Practel Sales, Holden Hill, South Australia) imprinted simultaneously the elapsed time on the video images in hundredths of seconds and a signal on the pressure tracing each whole second, to permit precise temporal correlation of video images with pressure.

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with denture adhesive (Poly-grip, Stafford-Miller, New South Wales, Australia). The anterior tantalum marker was placed 0.5 cm from the tongue tip, the posterior marker was positioned adjacent to the last lower molar tooth, and the middle marker was placed midway between the other two. A reference marker was placed on the hard palate in the midline (Fig. 1A). We then recorded lateral videoradiographic images of duplicate 5- and 10-ml water swallows to determine splint-induced alteration in tongue contour at rest and during oral bolus delivery. Rays from the hard palate reference marker were traced to each tongue marker, and distances along these rays were measured at three time points: preswallow rest position (T1); at swallow onset (T2), defined as the initial movement of the tongue tip against the posterior surface of the maxillary incisors; and at the mid oral phase (T3), 200 ms after swallow onset.

Experimental protocol. Swallows in each of the 15 group 2 subjects were recorded under control conditions (no oral splint) and separately under two experimental conditions, after insertion of either a mandibular splint or a maxillary splint. Full arch mandibular and maxillary splints were made from Hydroplastic (Tak Systems, Boston, MA), a custom formulated plastic that softened in hot water to enable molding. A “U-shaped” mandibular splint of uniform thickness covered the occlusal, lingual, and labial surfaces of the lower teeth. The maxillary splint covered the mucosa of the hard palate in addition to covering the upper teeth. The maxillary splint served to reduce the supralingual, subpalatal space, thereby exaggerating bolus forces on the tongue for a given bolus volume and altering tongue contour. Because the maxillary splint had the potential to influence extralingual mechanoreceptors (e.g., periodontal and temporomandibular joint proprioceptors) and increase salivation, the lingual marker study confirmed that, with maxillary splint in situ, the distance between the reference and tongue markers was greater than would be predicted by the thickness of the splint. This increased palatal lingual separation was more pronounced posteriorly and at rest (P = 0.02). This effect was also significant at swallow onset T2 (P = 0.02) but diminished as the oral phase progressed (Fig. 1). The effects were comparable for 5- and 10-ml water boluses.

With the mandibular splint in situ the timing of swallow events did not differ from that under control flow were defined fluoroscopically (6). UES relaxation onset was defined as the time point when the basal UES pressure began to fall abruptly. Maximum UES relaxation was defined as the point in time when the UES relaxation profile ceased to fall rapidly and leveled off. Because the proximal sleeve margin projects into the hypopharynx, the sleeve registers prematurely the apparent termination of UES relaxation (14). Accordingly, termination of UES relaxation was measured from the tracing recorded by the side hole 1.5 cm distal to the proximal sleeve margin, which was seen fluoroscopically to lie within the UES at the time of sphincter closure. We measured the onset of superior and anterior motion of the hyoid and larynx as well as timing of peak anterosuperior motion of these structures. Total swallow duration was defined as the interval between swallow onset and UES relaxation. Peak pharyngeal pressure was measured at the two distal solid-state transducers positioned in the hypopharynx and the midpharynx. Hypopharyngeal intrabolus pressure measured at the side hole immediately proximal to the UES represents the pressure within the bolus as it traverses the pyriform sinuses (7).

Duplicate values for each subject were averaged before calculation of group mean data for each volume swallowed. Statistical inferences were made regarding the bolus volume effect, splint effect, and splint-volume interaction using a two-way mixed design analysis of variance for repeated measures (3). All values, both numeric and graphic, are represented as means ± SE unless stated otherwise.

RESULTS

The lingual marker study confirmed that, with maxillary splint in situ, the distance between the reference and tongue markers was greater than would be predicted by the thickness of the splint. This increased palatal lingual separation was more pronounced posteriorly and at rest (P = 0.02). This effect was also significant at swallow onset T2 (P = 0.02) but diminished as the oral phase progressed (Fig. 1). The effects were comparable for 5- and 10-ml water boluses.

With the mandibular splint in situ the timing of swallow events did not differ from that under control

Table 1. Timing of swallow events referenced to swallow onset

<table>
<thead>
<tr>
<th>Variable</th>
<th>Density</th>
<th>Volume, ml</th>
<th>Control</th>
<th>Mandibular</th>
<th>Maxillary</th>
<th>P Values</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Splint 0.05</td>
<td>Splint 0.04</td>
<td></td>
</tr>
<tr>
<td>Anterior hyoid onset</td>
<td>High</td>
<td>2</td>
<td>0.34 ± 0.05</td>
<td>0.33 ± 0.04</td>
<td>0.30 ± 0.04</td>
<td>0.003 NS NS NS NS</td>
</tr>
<tr>
<td></td>
<td>Low</td>
<td>10</td>
<td>0.15 ± 0.04</td>
<td>0.19 ± 0.05</td>
<td>0.22 ± 0.05</td>
<td>NS NS 0.001 0.001</td>
</tr>
<tr>
<td>UES relaxation onset</td>
<td>High</td>
<td>2</td>
<td>0.46 ± 0.05</td>
<td>0.43 ± 0.06</td>
<td>0.42 ± 0.04</td>
<td>0.003 NS 0.03 NS</td>
</tr>
<tr>
<td></td>
<td>Low</td>
<td>10</td>
<td>0.24 ± 0.04</td>
<td>0.28 ± 0.06</td>
<td>0.34 ± 0.05</td>
<td>NS NS 0.001 0.003</td>
</tr>
<tr>
<td>UES opening</td>
<td>High</td>
<td>2</td>
<td>0.21 ± 0.05</td>
<td>0.22 ± 0.04</td>
<td>0.28 ± 0.06</td>
<td>0.02 NS 0.002 0.03</td>
</tr>
<tr>
<td></td>
<td>Low</td>
<td>10</td>
<td>0.63 ± 0.05</td>
<td>0.62 ± 0.05</td>
<td>0.58 ± 0.04</td>
<td>NS NS 0.001 NS</td>
</tr>
<tr>
<td>UES closure</td>
<td>High</td>
<td>2</td>
<td>0.33 ± 0.06</td>
<td>0.34 ± 0.04</td>
<td>0.38 ± 0.06</td>
<td>0.01 NS 0.0003 0.0001</td>
</tr>
<tr>
<td></td>
<td>Low</td>
<td>10</td>
<td>0.89 ± 0.04</td>
<td>0.94 ± 0.06</td>
<td>0.96 ± 0.05</td>
<td>NS NS NS NS</td>
</tr>
<tr>
<td></td>
<td>Low</td>
<td>2</td>
<td>0.93 ± 0.05</td>
<td>0.93 ± 0.04</td>
<td>0.99 ± 0.05</td>
<td>NS NS NS NS</td>
</tr>
</tbody>
</table>

Values are means ± SE in seconds; n = 15 subjects. UES, upper esophageal sphincter; high, high-density bolus (barium); low, low-density bolus (Gastroview); NS, not significant.
conditions. Hence all subsequent comparisons were made between control state (i.e., no splint) and maxillary splint in situ. Under control conditions the timing of UES relaxation onset and UES opening relative to the swallow onset occurred earlier with increasing swallowed bolus volume (Table 1), and this effect was preserved with the maxillary splint in place (Fig. 2). However, for any given bolus volume the maxillary splint caused a delay in the onset of hyoid motion, the onset and termination of UES relaxation, and UES opening and closure (Fig. 3). This effect was statistically significant with low-density bolus swallows ($P < 0.006$) but was less marked and did not reach statistical significance with high-density bolus swallows. Hence the volume-dependent earlier timing of UES relaxation and opening was preserved for both bolus densities, whereas for low-density boluses, the maxillary splint causes a rightward temporal shift in these variables. The timing of UES relaxation onset, maximum relaxation, and opening did not differ significantly between high- and low-density swallowed boluses, indicating that density per se did not influence coordination (Fig. 4).

Oral and pharyngeal transit times, total swallow duration, and pharyngeal clearance times were not significantly influenced by the maxillary splint (Fig. 5). Because the splints occupied space within the oral cavity, we examined whether this effect might cause earlier entry of the bolus head into the pharynx. The timing of the arrival of the bolus head into the pharynx was not significantly influenced by either splint.

Bolus volume did not influence peak pharyngeal pressure. Midpharyngeal, but not hypopharyngeal, peak pressure was significantly reduced by both splints during high-density barium bolus swallows ($P < 0.01$; Table 2). Hypopharyngeal intrabolus pressure was also significantly reduced by both maxillary and mandibular splints during high-density barium boluses ($P < 0.05$; Table 2) but not during low-density boluses.

**DISCUSSION**

In this study we hypothesized that modification of bolus forces on the tongue, at or shortly after swallow initiation, influences bolus volume-dependent temporal events and that lingual mechanoreceptor activity is likely to be involved in this process. We demonstrated that the presence of a maxillary splint during the swallow alters tongue contour during the early stages of the swallow. The maxillary splint delays the onset of anterior hyoid motion, UES relaxation, and UES opening without influencing bolus transit times or total swallow duration. However, contrary to our initial hypothesis, the maxillary splint did not influence the normal bolus volume-dependent earlier onset of anterior hyoid motion, UES relaxation, or UES opening.
Influence of oral splints on pharyngeal pressures

Table 2. Influence of oral splints on pharyngeal pressures

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control</th>
<th>Mandibular Splint</th>
<th>Maxillary Splint</th>
<th>P Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Midpharyngeal peak pressure</td>
<td>2</td>
<td>100 ± 7</td>
<td>85 ± 7</td>
<td>82 ± 10</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>90 ± 9</td>
<td>86 ± 9</td>
<td>80 ± 7</td>
</tr>
<tr>
<td>Hypopharyngeal peak pressure</td>
<td>2</td>
<td>147 ± 11</td>
<td>150 ± 14</td>
<td>148 ± 14</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>149 ± 13</td>
<td>159 ± 14</td>
<td>174 ± 5</td>
</tr>
<tr>
<td>Intrabolus pressure</td>
<td>2</td>
<td>6.7 ± 1.6</td>
<td>4.36 ± 2</td>
<td>3.18 ± 2.3</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>8.95 ± 2</td>
<td>5.73 ± 2.1</td>
<td>5.23 ± 2.3</td>
</tr>
</tbody>
</table>

Values are means ± SE in mmHg; n = 15 subjects. Data only shown for high-density barium boluses.
Lingual Modulation of Deglutition

geal but not hypopharyngeal contraction amplitude during swallowing. Unlike UES relaxation and opening pharyngeal peristaltic velocity, contraction duration, and maximal contraction amplitude are stereotyped among swallow volumes (16). As distinct from the hypopharynx, the midpharyngeal chamber is bound anteriorly by the tongue base. Augmentation of supralingual, retrolingual, and hypopharyngeal peak pressures by bolus viscosity suggests that bolus propulsion is a function of both tongue and pharyngeal constrictor activity (10, 25). Alterations in mandibular position by a maxillary splint can influence tongue function (5), and electromyographic recordings confirm that changes in the mandibular position have a definite effect on tongue activity (19). These observations indicate that alterations in mandibular position, with or without altered tongue activity, may influence pharyngeal propulsive forces.

The finding of reduced hypopharyngeal intrabolus pressure is also likely to be due to modification of lingual forces by the splint. During pharyngeal bolus propulsion the initial phase of the intrabolus pressure domain is generated by tongue forces, whereas the later segment is due to a combination of tongue and pharyngeal constrictor forces (4, 20). The intrabolus pressure domain is similar in the retrolingual and hypopharyngeal regions (10). The reduction of the intrabolus pressure domain in our study was uniform and therefore not delineating the exact contribution of the tongue in the generation of this pressure domain.

Although both splints reduced hypopharyngeal intrabolus and midpharyngeal peak pressures, the pressures observed under experimental conditions still lay within the normal range. It is likely that the magnitude of change was insufficient to influence bolus transit time. Alternatively, the absence of any effect on transit time might reflect activation of nonlingual mechanoreceptors. The changes in propulsive forces demonstrated in our study may have implications for bolus transport in patients with oral prostheses and dentures, because, although unknown, it is highly likely that maxillary dentures may have similar effects. Therefore any attenuation of propulsive forces during deglutition with such devices might further impair bolus transport in the context of preexisting compromise of pharyngeal function.

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