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ORIGINAL ARTICLE

Does Removal of Tracheostomy Affect Dysphagia? A Kinematic Analysis

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Abstract Tracheostomy tubes are thought to increase the incidence of aspiration and several mechanisms that might cause this have been suggested. Some studies reported alterations in laryngeal elevation during swallowing, which they attributed to an anchoring effect of the tracheostomy tube resulting in dysphagia. The purpose of the present study was to kinematically investigate the effect of tracheostomy on the swallowing process in dysphagic patients. Thirteen patients (7 males, 6 females; mean age = 61.4 years) were prospectively enrolled between August 2008 and December 2009. The inclusion criteria for a patient who had undergone tracheostomy were an ability to tolerate tube plugging for 48 h and the capacity to expectorate without assistance. All patients underwent two videofluoroscopic swallow studies (VFSS), before and after decannulation. We measured 21 time interval variables during swallowing in the pharyngeal phase and the extent of laryngeal elevation. No patient exhibited any change in swallowing function status [Penetration − Aspiration Scale (PAS) (median value = 1)] in the interval between the two VFSS tests. Upon kinematic analysis, no significant difference in any variable pertaining to laryngeal elevation or pharyngeal constriction was found when pre- and post-decannulation VFSS test data were compared ($p > 0.05$). The present study thus showed that removal of a tracheostomy tube does not affect the kinematics of swallowing. Our results support previous findings that indicated no relationship between tracheostomy tube placement and dysphagia.

Keywords Tracheostomy · Deglutition disorder · Kinematic analysis · Videofluoroscopic swallowing study · Deglutition

Aspiration after tracheostomy has been reported in 50–87% of patients [1–6]. Tracheostomy has been thought to be associated with an increased incidence of such aspiration and several possible mechanisms causing it have been suggested [7–10]. These are, first, a decrease in laryngeal elevation caused by tethering of the larynx by the tracheostomy tube [6, 8, 11]; second, obstruction of the pharyngeal pathway by the tube cuff [9]; third, desensitization of the larynx and loss of the protective reflex because of chronic air diversion [11–13]; and fourth, uncoordinated laryngeal closure attributable to chronic upper airway bypass [14, 15]. However, all the earlier work was performed immediately after tracheostomy, with or without mechanical ventilation [6, 8], and only a few patients were studied; statistical reliability was thus absent [11]. Several recent reports have obtained contrary results [16–22]. Some studies found no increase in the incidence of aspiration after tracheostomy in patients with various diseases, having used fiberscopes to endoscopically evaluate swallowing [16, 18, 21, 22]. Although most studies reported no change in aspiration or penetration, those studies did not evaluate the mechanisms of aspiration. One report

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employed spatial analysis and found no difference in the extent of hyoid bone or laryngeal movement in the presence or absence of a tracheostomy tube [17].

In light of this, we utilized a videofluoroscopic swallowing study (VFSS) to prospectively investigate kinematic changes upon swallowing in patients with and without tracheostomy tubes. Whether placement of such a tube affects aspiration risk or the biomechanics of swallowing remains poorly understood because of a paucity of objective data. Our hypothesis was that coordination of the pharyngeal swallowing phase, including laryngeal elevation, pharyngeal constriction, and upper esophageal opening, might be affected by tracheostomy and that a kinematic analysis would be useful to explore this possibility.

Materials and Methods

Subjects

Patients were prospectively enrolled between August 2008 and December 2009. The inclusion criteria for patients who had undergone tracheostomy were an ability to tolerate tube plugging for 48 h and the capacity to expectorate without assistance. Thus, it was safe to remove tracheostomy tubes from all patients. The exclusion criteria were poor cognition, a prior history of laryngeal surgery, and/or poor general condition that rendered a patient unable to tolerate VFSS.

Procedures

Thick fluid (viscosity range $>$ 1750 cP), dysphagia I (viscosity range = 351–1750 cP, pureed diet), dysphagia II (same viscosity, mechanically altered), dysphagia III (same viscosity, regular texture), nectar-like (51–350 cP), and thin fluid (1–50 cP) were sequentially swallowed [23]. Subjects were presented with a 3-cc bolus (first, once) followed by a 5-cc bolus (second, twice). Thick fluid, nectar-like, and thin fluid were presented with syringes (10 cc) and dysphagia I, II, and III were presented by spoon. They were asked to hold it briefly in the oral cavity before a command to swallow. Swallowing was halted for safety reasons for patients who developed aspiration [24]. The first VFSS test was performed after tracheostomy tube placement, with the tube open and the cuff deflated; the second VFSS was performed within 2 weeks after decannulation and application of an occlusive dressing. To ensure data consistency, kinematic analyses were performed during the routine swallowing of 5 ml of thick fluid [24].

All VFSS images were recorded using a camcorder (Samsung SMX-C 14®) running at 30 frames per second. All images were saved on a personal computer and analyzed by one of the authors using a multimedia player (Gomplayer; Gretech®) [24].

Since all analyses were performed by one rater, interrater reliability tests were not performed. Intrarater reliability was established for ten patients and was found to be greater than 90% for each measure reported, except for LPPC (0.768) and DUEO (0.829).

We measured the values of 21 distinct timing variables during the pharyngeal phase of swallowing and the extent of laryngeal elevation. To measure the latter parameter, a 23-mm-diameter marker was taped to the lower mandible. This served as a reference point to correct for fluoroscopic magnification discrepancies among digitized images. The variables measured included the latency of bolus contact with the epiglottis, the extent of laryngeal elevation, the degree of pharyngeal contraction, the extent of opening of the upper esophageal sphincter, and interrelationships among these variables (please see the definitions of all variables in [21]); the variables measured in the current study are listed in Table 1.

The Rosenbek penetration–aspiration scale (PAS, scores range from 1 to 8) was obtained from the poorest swallow [25]. Our study protocol was approved by the Institutional Review Board of Bundang CHA Hospital.

Statistics

The Wilcoxon signed-rank test was used to compare values in patients who had undergone tracheostomy (before and after decannulation), using SPSS for Windows ver. 17.0 (SPSS Inc., Chicago, IL, USA). A p value $\leq$0.05 was considered to be statistically significant.

Results

Thirteen patients were enrolled, including 11 with disease of cerebral origin, 1 with a cervical cord injury, and 1 with a medical condition. The mean duration of tracheostomy was 103.2 (±72.4) days. The mean interval between decannulation and the second VFSS test was 6.6 (±4.6) days and the mean interval between the two VFSS tests was 23.6 (±16.9) days (Table 2).

Upon VFSS evaluation, no patient showed any change in PAS scores, whether or not tracheostomy had been performed (Table 3).

Descriptive data with kinematic analyses of all variables are given in Table 4. No statistically significant change in any variable between the first and second VFSS test was evident; the parameters evaluated included the latency and duration of laryngeal elevation and pharyngeal contraction and the extent of laryngeal elevation. Among-variable differences in intervals were also not significant.

Table 1 Abbreviations and definitions

Variables	Definition
LEC	Interval between the initiation of pharyngeal phase and epiglottis contract (arrival at vallecula)
LLE	Interval between the initiation of pharyngeal phase and the initiation of laryngeal elevation
LPC	Interval between the initiation of pharyngeal phase and the initiation of pharyngeal contraction
LUEO	Interval between the initiation of pharyngeal phase and the initiation of UES opening
LPLE	Interval between the initiation of pharyngeal phase and the peak laryngeal elevation
LPPC	Interval between the initiation of pharyngeal phase and the peak pharyngeal contraction
LLE–LEC	Interval between latency of epiglottis contact and latency of laryngeal elevation
LPC–LLE	Interval between latency of laryngeal elevation and latency of pharyngeal contraction
LPPC–LLE	Interval between latency of laryngeal elevation and latency of peak pharyngeal contraction
LUEO–LLE	Interval between latency of laryngeal elevation and latency of UES opening
LUEO–LPLE	Interval between latency of peak laryngeal elevation and latency of UES opening
LPLE–LPC	Interval between latency of pharyngeal contraction and latency of peak laryngeal elevation
LPPC–LPLE	Interval between latency of peak laryngeal elevation and latency of peak pharyngeal contraction
LPLE–LLE	Rise time of laryngeal elevation
DLE	Interval between the initiation and the end of laryngeal elevation
LPPC–LPC	Rise time of pharyngeal contraction
DUEO	Interval between the opening and closing of UES opening
LUEO–LPC	Interval between latency of pharyngeal contraction and latency of UES opening
LUEO–LPPC	Interval between latency of peak pharyngeal contraction and latency of UES opening
LPLE–LUEO	Interval between latency of UES opening and latency of peak pharyngeal elevation
DisLE	Extent of laryngeal elevation

Values are mean ± standard deviation. The unit of values is seconds

Table 2 Demographic data in all subjects ($N = 13$) and stroke subjects ($n = 11$)

	All subjects ($N = 13$)	Stroke subjects ($n = 11$)
Duration of tracheostomy (days)	103.2 (±72.4)	104.5 (±79.2)
Mean interval between decannulation and conduct of second VFSS	6.6 (±4.6)	6.1 (±4.1)
Mean interval between the two VFSS	23.6 (±16.9)	21.8 (±16.0)

Our patients included 11 stroke patients. When we analyzed the data from these patients, no significant difference in any of the 21 kinematic values (compared to initial data) was apparent (Table 4).

Discussion

The purpose of the present study was to explore whether a tracheostomy tube affects the coordination of laryngeal elevation, pharyngeal constriction, and the opening of the upper esophageal sphincter. This study showed that parameters related to laryngeal elevation, pharyngeal constriction, and esophageal opening were not changed significantly between patients with tracheostomy and without tracheostomy.

Recently, several studies have found that tracheostomy was not associated with an increased risk of dysphagia [16–22, 26], and two reports have sought to refute earlier theories that tracheostomy caused dysphagia [17, 26]. The use of cuff inflation did not affect PAS values collected with VFSS [22, 26], and neither the extent of hyoid bone displacement nor the larynx-to-hyoid bone approximation value differed significantly with reference to tracheostomy tube placement, tube cuff status, or tube capping [17]. Mean penetration–aspiration level on fiber-optic endoscopic examination of swallowing was not affected by occlusion conditions such as open, finger, capped, and removed tubes [22].

It was earlier suggested that a swallowing dysfunction that develops after tracheostomy was caused by "anchoring" of the larynx to the anterior neck [8, 11, 27]. The cited works had several limitations, one of which was observation of laryngeal elevation in only three patients using cinepharyngography [8]. Another report did not present

Table 3 Participant characteristics

Patient No.	Sex	Age (years)	Diagnosis	Duration of tracheostomy	PAS	
					With tracheostomy	Without tracheostomy
1	F	82	Subarachnoid hemorrhage	56	1	1
2	M	47	Putaminal hemorrhage	53	1	1
3	F	64	MCA infarction	75	1	1
4	F	46	Subarachnoid hemorrhage	44	1	1
5	F	62	Subarachnoid hemorrhage	99	1	1
6	M	73	Extradural and subdural hemorrhage	37	1	1
7	F	43	Subarachnoid hemorrhage	48	1	1
8	M	67	MCA infarction	112	7	7
9	M	70	Subarachnoid hemorrhage	197	8	8
10	F	78	Intracerebral hemorrhage	294	7	7
11	M	55	Subarachnoid hemorrhage	135	7	7
12	M	36	Spinal cord injury	99	1	1
13	M	75	Panperitonitis	92	6	6

PAS Rosenbek penetration–aspiration scale

Table 4 Descriptive analyses of variables in all subjects ($N = 13$) and stroke subjects ($n = 11$)

	All subjects ($N = 13$)			Stroke subjects ($n = 11$)		
	With tracheostomy	Without tracheostomy	p	With tracheostomy	Without tracheostomy	p
LEC	0.69 ± 0.64	0.65 ± 0.02	0.34	0.75 ± 0.68	0.73 ± 0.93	0.63
LLE	1.37 ± 1.70	1.09 ± 0.04	0.53	1.55 ± 1.79	1.26 ± 1.68	0.46
LPC	1.43 ± 1.71	1.13 ± 0.04	0.50	1.61 ± 1.81	1.31 ± 1.73	0.40
LUEO	1.25 ± 1.41	1.29 ± 0.04	0.59	1.10 ± 1.49	1.34 ± 1.73	0.21
LPLE	1.83 ± 1.84	1.55 ± 0.05	0.89	2.01 ± 1.94	1.75 ± 1.78	0.80
LPPC	2.01 ± 1.82	1.65 ± 0.06	0.31	2.19 ± 1.93	1.85 ± 1.80	0.26
LLE–LEC	0.68 ± 1.22	0.44 ± 0.01	0.45	0.81 ± 1.29	0.53 ± 0.94	0.25
LPC–LLE	0.06 ± 0.10	0.04 ± 0.00	0.88	0.06 ± 0.11	0.05 ± 0.09	0.81
LPPC–LLE	0.64 ± 0.25	0.56 ± 0.02	0.37	0.64 ± 0.14	0.59 ± 0.11	0.29
LUEO–LLE	−0.31 ± 1.64	0.11 ± 0.00	0.38	0.02 ± 0.90	0.08 ± 1.07	0.65
LUEO–LPLE	−0.77 ± 1.75	−0.36 ± 0.01	0.20	−0.40 ± 1.00	−0.41 ± 1.04	0.25
LPLE–LPC	0.40 ± 0.15	0.42 ± 0.01	0.48	0.40 ± 0.16	0.44 ± 0.10	0.33
LPPC–LPLE	0.18 ± 0.18	0.10 ± 0.00	0.15	0.18 ± 0.20	0.11 ± 0.07	0.93
LPLE–LLE	0.46 ± 0.18	0.46 ± 0.02	0.93	0.46 ± 0.14	0.48 ± 0.09	0.94
DLE	1.42 ± 0.47	1.22 ± 0.04	0.12	0.83 ± 0.20	1.24 ± 0.26	0.91
LPPC–LPC	0.58 ± 0.20	0.52 ± 0.02	0.39	0.58 ± 0.21	0.55 ± 0.12	0.43
DUEO	1.69 ± 1.38	1.75 ± 0.06	0.53	1.49 ± 1.53	1.78 ± 1.77	0.33
LUEO–LPC	−0.37 ± 1.67	0.06 ± 0.00	0.38	−0.05 ± 0.99	0.03 ± 1.05	0.46
LUEO–LPPC	−0.95 ± 1.83	−0.46 ± 0.02	0.72	−0.59 ± 1.14	−0.52 ± 1.01	0.83
LPLE–LUEO	0.77 ± 1.75	0.36 ± 0.01	0.20	0.40 ± 1.00	0.41 ± 1.04	0.25
DisLE (mm)	19.45 ± 5.39	19.39 ± 5.04	0.75	19.82 ± 5.01	19.60 ± 4.52	0.92

Values are mean ± standard deviation. The unit of values is seconds except for DisLE. For definition of abbreviations see Table 1

objective evidence of any decrease in the extent of laryngeal elevation [11].

We found no statistically significant change in any swallowing process of either the pharyngeal or the early esophageal phase when patients with and without tracheostomy were compared. The swallowing features examined included the latency and peak latency of laryngeal elevation, the details of pharyngeal constriction, and parameters associated with opening of the upper esophageal sphincter. We also evaluated the extent of laryngeal

elevation. These parameters reflect the kinematics of swallowing and the extent of swallowing coordination in the pharyngeal phase. Our results are useful in that we employed an objective method to explore changes in coordination of the pharyngeal phase after displacement of a tracheostomy tube. Our data are consistent with those of a previous study that found that hyoid bone displacement and the extent of larynx-to-hyoid bone approximation did not differ significantly between tracheostomy patients and controls [17].

When evaluated at a mean interval of 6.6 days after tube removal, no patient exhibited differences in PAS values before and after decannulation. Our data are in agreement with those of recent reports that found no relationship between the extent of dysphagia and tracheostomy status in patients with various conditions including trauma, those in acute care settings, and patients with head and neck cancer [16, 18, 19].

Our patients included 11 stroke patients. When we analyzed the data from these patients, no significant difference in any of the 21 kinematic values (compared to initial data) was apparent at a mean interval of 6.1 days after tube removal. Similarly, the PAS values assessing functional dysphagia were unchanged (Table 3). This report provides the additional evidence of a specific population, although the number of subjects is small.

We thus found that swallowing function, evaluated by PAS, was unaffected by tracheostomy. As has been emphasized, it was found recently that there is no casual relationship between tracheostomy tube placement and aspiration status [20, 21]. Our kinematic data make it clear that tracheostomy tube placement does not affect swallowing function in patients undergoing long-term tracheostomy.

Our present study had some limitations. All enrolled patients had experienced tracheostomy tube placement of long duration, a mean of 103.2 days. It is possible that several different forms of adaptation took place over such a long period, rendering the kinematics of swallowing unchanged after decannulation. However, it is difficult to investigate any possible "anchoring effect" in a medically problematic patient on whom tracheostomy has been newly performed. Second, we have no information on the tracheostomy procedures used or the types and diameters of tubes employed. One study that identified the development of swallowing dysfunctions after tracheostomy used vertical skin incisions for tracheostomy tube placement [8], whereas another report that explored the pathophysiology of aspiration after tracheostomy featured horizontal incision and fixation of the larynx upon tracheostomy; a "Bjork flap" was created [10, 27]. In the present study, patients underwent tracheostomy in either the Otorhinolaryngology or the Neurosurgery Department in various hospitals. Differences in tracheostomy procedures may influence the level of dysphagia to a greater or lesser extent.

Conclusion

The removal of a tracheostomy tube did not affect the kinematics of the swallowing mechanism in either the pharyngeal or the early esophageal phase. In addition, we found no association between tracheostomy tube placement and the extent of aspiration or penetration in chronically dysphagic patients. Future work should examine the effects of various types of tracheostomy on dysphagia and employ a larger number of study participants.

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