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Glottal Stop in Cleft Palate Speech

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Introduction

There is a great deal of literature that deals with the glottal stop, one of the abnormal articulations found in cleft palate speech. Except for some earlier research by Kawano,[1] very few descriptions of the articulatory movements involved in these glottal stops is available in the literature. The present study expands upon that earlier research and examines two cases in order to illustrate the process by which glottal stop production is corrected.

Method

The subjects were 26 cleft palate patients who were seen at our clinic during the 5 years from 1984 to 1988. Their productions of Japanese voiceless stops were auditorily judged to be glottal stops which were confirmed by fiberscopic assessment of their laryngeal behavior. Age at the time of fiberscopic evaluation ranged from 5 to 53 years, with the mean age being 23.6. Eighteen of the subjects were judged to exhibit significant velopharyngeal insufficiency while 8 demonstrated slight velopharyngeal insufficiency. Individuals with mental retardation or bilateral hearing loss were excluded from the study (see Table 1).

Fiberscopic observation of the larynx was done during production of Japanese voiceless stop (CV) syllables using an Olympus fiberscope (3.5 mm. diameter) and a Sony video recorder (30 frames or 60 fields per second). Simultaneous recording of speech sounds was superimposed on the videotapes as a sound wave using a videograph. Acoustic analysis was accomplished with a sound spectrograph (MacSpeech Lab II) having a 10 kHz frequency range and 300 Hz bandpass filter. In addition to the fiberscopic assessment, some patients were examined using videofluoroscopy. In these cases tongue movements during /k/ production was observed.

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Table 1. Subject characters.

(Cleft type)	CL/P	CP	SMCP	CVPI
total subject (n=26)	18	6	1	1

(V-P function)	incompetence			slight incompetence	
total subject (n=26)	18			8	

(age)	–5	6–10	11–20	21–30	31–40	41–50	51– years
mean=23.6	3	6	2	9	4	1	1

CL/P: cleft lip and palate
 CP: cleft palate
SMCP: submucous cleft palate
CVPI: congenital velopharyngeal incompetence

RESULTS

Types of Glottal Stops

Fiberscopic observation of the larynx revealed two types of laryngeal movement during glottal stops.

Type I.   Prior to voice onset the vocal folds and, in most of the cases, the ventricular folds, were firmly adducted.   At voice onset only the ventricular folds were released, resulting in a noise followed by vowel production.   During phonation the vocal fold closure was maintained although a spindle-shaped glottal opening was noted (see Fig. 1, 2).   Among the 26 patients producing glottal stops, 11 were categorized as having Type I production.
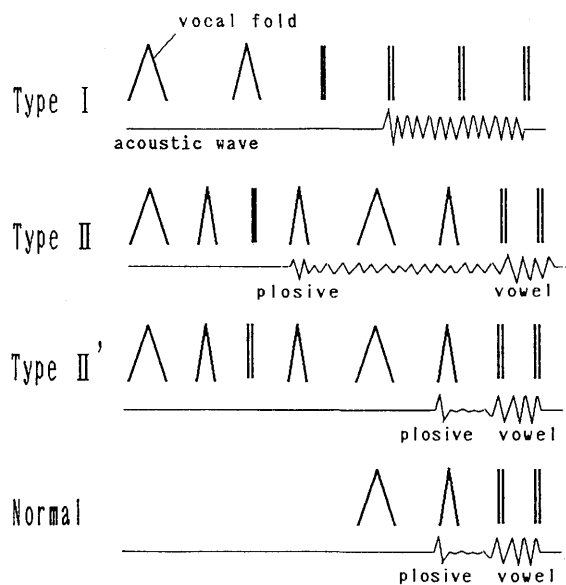

Fig. 1.   The different types of vocal fold movement and associated acoustic waves (read left to right).

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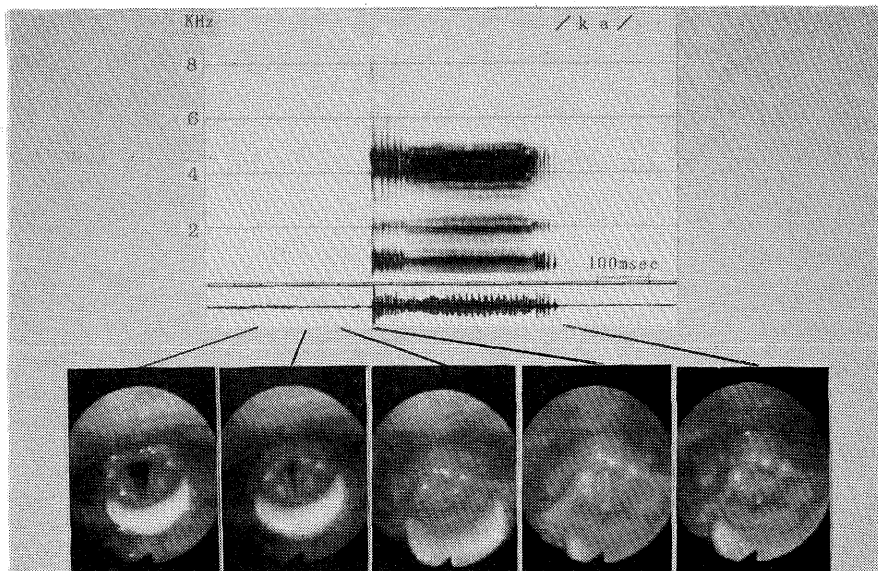Iwao HONJO and Hisayoshi KOJIMA

Fig. 2.   Spectrogram and laryngeal movement associated with production of a Type I glottal stop.
The VOT can't be identified in the spectrogram, as vocal folds adduction prior to voice onset
are sustained too close during the ensuing vowel production.

Adduction of the ventricular folds before phonation was observed in 10 of the
11 patients (Table 2).

Abnormal laryngeal constriction was observed in 4 patients.   That is, before
voice onset the arytenoids moved upward and approached the base of the epiglottis.
Constriction of the airway, which was apparent in the entire laryngeal region, was
associated with adduction of the arytenoids and with medial movements of the ary-
epiglottic folds and backward displacement of the basal portion of the epiglottis
(Fig. 3).

Acoustic analysis by sound spectrography revealed that the noise component

Table 2.   Findings for Type I patients.

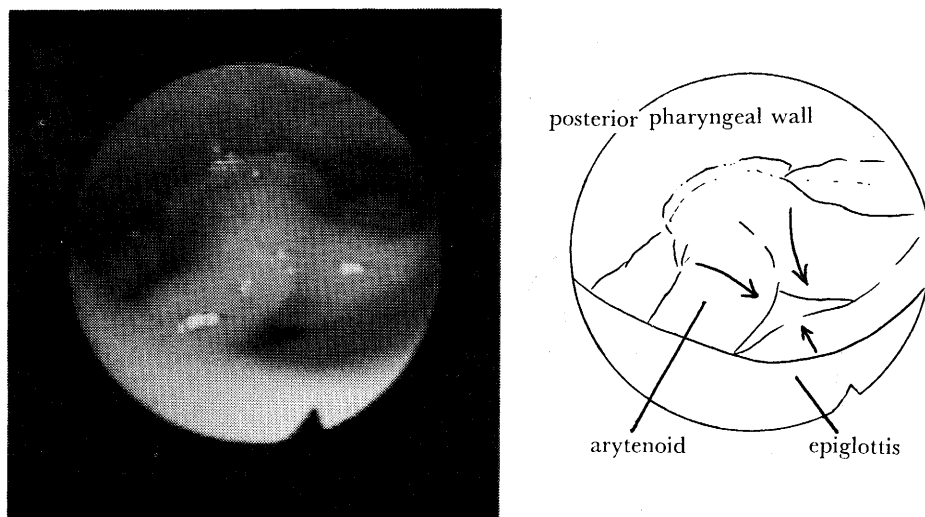| case	cleft type	age	V-P funct.	ventricular fold adduction	laryngeal constriction
1	CL/P	5	slight VPI	+	—
2	CL/P	19	VPI	+	—
3	CL/P	20	VPI	+	—
4	CL/P	31	VPI	+	+
5	CL/P	48	VPI	+	+
6	CP	7	VPI	+	—
7	CP	29	VPI	+	—
8	CP	33	VPI	—	+
9	CP	38	slight VPI	+	—
10	CP	53	VPI	+	+
11	CVPI	5	VPI	+	—

Fig. 3.   Abnormal laryngeal constriction.

and vowel sound began at the same time.   No voice onset time could be measured
(Fig. 2).   The auditory impression created by a Type I glottal stop was that of
vowel production with the hard vocal attack which was judged to be typical of glottal
stops.

Type II.   In contrast to Type I, adduction of both the vocal and ventricular
folds in most of these cases occured before voice onset.   Soon after ventricular fold
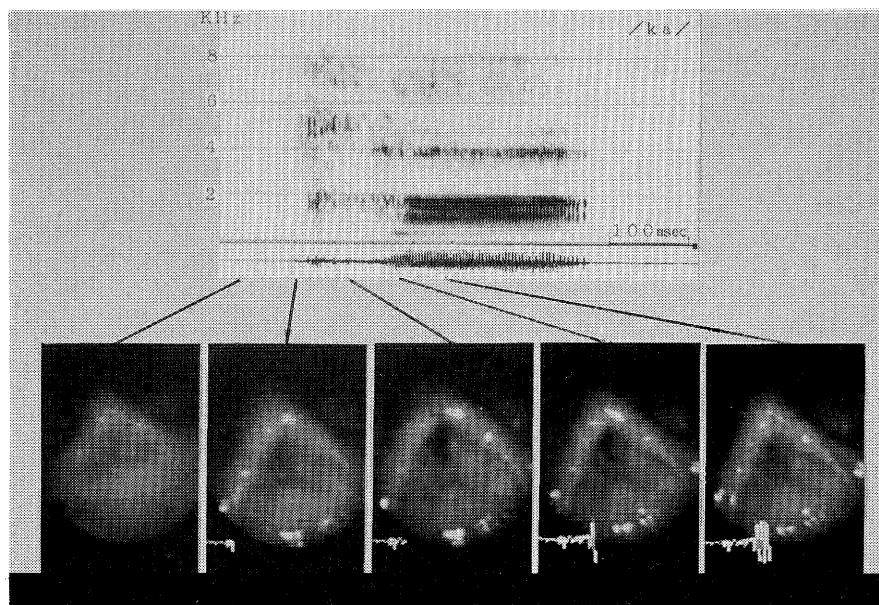

Fig. 4.   Spectrogram and laryngeal movement associated with production of a Type II glottal stop.
The vocal folds are shown to adduct prior to voice onset, as apparent from the soundspectrogram.
The vocal folds then abduct quickly to produce a plosive noise and adduct again to produce
the vowel.

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Table 3. Findings for Type II patients.

case	cleft type	age	V-P funct.	ventricular fold adduction	laryngeal constriction
1	CL/P	5	VPI	—	—
2	CL/P	8	VPI	+	+
3	CL/P	8	VPI		+
4	CL/P	9	slight VPI	+	+
5	CL/P	10	VPI	—	—
6	CL/P	21	slight VPI	+	+
7	CL/P	24	slight VPI	+	+
8	CL/P	24	VPI	+	+
9	CL/P	24	slight VPI		+
10	CL/P	25	slight VPI	+	+
11	CL/P	26	VPI	—	—
12	CL/P	27	slight VPI	—	—
13	CL/P	37	VPI		+
14	CP	29	VPI	+	+
15	SMCP	7	VPI		+

abduction the vocal folds abducted and a plosive sound was produced. Then the vocal folds again adducted to produce a sustained vowel sound (Fig. 1, 4). This adduct-abduct-adduct pattern was observed in 15 out of 26 patients. In Type II glottal stops the participation of the ventricular folds was also relevant. Their adduction was observed in 7 of the 11 patients (Table 3).

Eleven patients showed abnormal laryngeal constriction. Involving the arytenoids, base of the epiglottis and the aryepiglottic folds, the constriction was much tighter than that of Type I glottal stops. In some cases such constriction prevented us from observing the glottis with the fiberscope. Furthermore, every patient who showed ventricular fold adduction presented such abnormal laryngeal constriction (Table 3).

Acoustically, the plosive or fricative segment preceded the vowel segment (Fig. 4). In this case voice onset time was consistently found to be from 60 to 120 msec. Since breathy noise was perceived auditorily, it was easy to distinguish Type II and Type I productions. The auditory impression varied from that of a typical glottal stop to comparatively normal stop production.

Glottal Stops in Relation to Velopharyngeal Function

Among the patients producing Type I glottal stops, 9 of 11 were judged to have significant velopharyngeal insufficiency, while 2 had slight insufficiency. In the case of those producing Type II glottal stops, 9 of 15 patients were significantly insufficient and the remaining 6 were slightly insufficient. The proportion of velopharyngeal insufficiency was thus higher among the Type I patients (Table 2, 3).

Videofluoroscopic Findings

In some of the cases we observed articulatory movement during production of Japanese CV syllable (/ka/) productions using videofluoroscopy. For Type I glottal stops the tongue did not make contact with the palate during single syllable productions. In most of the cases the place of articulation was not found in the oral cavity. On the other hand, in most of the Type II patients articulatory movement was observed in the oral cavity not only during single syllable productions but also during production of words (Table 4).

Improvement of Glottal Stop Production

The course of improvement of patients who produced glottal stops was analyzed in two representative cases.

Patient 1 modified her productions from Type I to Type II as a result of speech therapy. This patient was a 20-year-old woman with cleft lip and palate whose velopharyngeal function was incompetent at the time of her first visit. Auditory assessment revealed hypernasality and glottal stop substitution for all voiceless plosives. Fiberscopic observation of the larynx revealed no abduction of the vocal folds during plosive production which was of the Type I glottal stop. Speech therapy with this patient continued for a month, concentrating on /p, k, and t/. Although velopharyngeal function was still incompetent the auditory impression of word production was changed. Laryngeal movement during voiceless stop production changed from Type I to Type II.

Patient 2 modified her plosive production from a Type II glottal stop to normal stop production. She was a 29-year-old woman with cleft lip and palate. A pharyngeal flap operation had been performed at another hospital, but slight velopharyngeal incompetence remained because of the narrow attachment of the flap to the soft palate. Speech therapy was undertaken at our clinic. Laryngeal observation after eight months of therapy showed that her glottal stops were of the Type II pattern. Her plosives were auditorily judged to be glottal stops during the production of sentences, while they became normal during words. During the sentence-level

Table 4. The results of fluorovideoscopy.

Type I				Type II			
case No.	/ka/	/kakji/	/ʃika/	case No.	/ka/	/kakji/	/ʃika/
3	±	—	—	5	+	+	+
6	—	—	—	8	+	+	
7	—	—	—	10	+		+
8	—	—	—	11	+		+
				12	+	+	+
				2	×	×	—
				3	×	×	—
				4	×		—
				6	×		—
				13	×		

+: tongue made contact with the palate

—: tongue did not make contact with the palate

×: tongue made contact with the posterior pharyngeal wall

plosives, which we judged to be glottal stops, the ventricular folds adducted and
the vocal fold abduction and noise production occured simultaneously. The noise
was perceived as a glottal stop. When producing stops in words, which were audi-
torilly judged to be normal, the ventricular folds did not adduct. Vocal fold ab-
duction preceded plosive sound production. In other words, the plosive was not
produced during the vocal fold abduction. Instead, plosives were generated by
normal articulatory movement and were auditorilly judged to be normal (Type II' in
Fig. 1). Since it proved difficult to improve her faulty articulation by speech therapy
alone, another pharyngeal flap operation was performed. Laryngeal observation one
year after this surgery revealed that laryngeal movement had become normal and
there was improvement of the perceived quality of the stop sounds in sentences.

DISCUSSION

On the whole, prior reports concerning glottal stops in cleft palate speech have
only rarely described laryngeal movements. Although assertions to the effect that
glottal stops are "caused by the abrupt opening of the vocal cords which have been
drawn together by hypertension in the laryngeal muscles"[4] have been advanced,
they have not been supported by objective evidence.

Fiberscopic and sound spectrographic analysis shows that there are two distinct
types of glottal stop production. Type I appears most likely to correspond to the
glottal stop production described in the literature. However, no record has been
found of Type II laryngeal movement.

Although both Type I and Type II glottal stops were auditorilly perceived as
so-called glottal stops, it seems necessary to distinguish these two types of articula-
tory movement. In most of the cases Type I is associated with a greater degree of
velopharyngeal dysfunction, as a result of which it is difficult to build oral pressure
to produce plosive sounds. In order to produce plosive noises, self regulation of the
larynx takes place. Meanwhile, in cases of Type II production, it is considered
that air pressure has been built beneath the glottis prior to voice onset, and an abrupt
release of the impounded air helps to produce plosive sounds in the oral cavity.
This conceptualization seems to be supported by the videofluoroscopic findings.

Observation of the process of articulatory improvement revealed that in Patient
1 glottal stops changed from Type I to Type II through speech therapy. In Pati-
ent 2 speech therapy and surgical improvement of velopharyngeal function resulted
in replacement of glottal stops by normal plosive productions. Adding to these re-
sults we also found that the plosive sound of Type II movements was not a product
of the vocal folds and that such sounds were auditorilly perceived as normal. In the
process of changing from Type II glottal stops to normal sounds, noise production
at the vocal folds disappeared and the subsequent disappearance of the mimic clo-
sure occured.

On the basis of this study it is suggested that Type I glottal stops change into normal productions via Type II glottal stops. Further longitudinal studies will be needed to clarify these dynamic alterations of laryngeal movement. More detailed analysis of laryngeal constrictions during glottal stop production may provide some useful ideas by which to improve the methods of speech therapy.

CONCLUSIONS

1. Combined sound spectrographic, fiberscopic, and videofluoroscopic analysis of glottal stops revealed two distinct types of stop consonant production.
2. It is suggested that Type I glottal stops modify to normal production via Type II glottal stops.

ACKNOWLEDGEMENT

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