Perception of Place of Articulation by Children With Cleft Palate and Posterior Placement

Tara L. Whitehill
Alexander L. Francis
University of Hong Kong

Christine K-Y. Ching
United Christian Hospital, Hong Kong

The aim of this study was to determine if children with repaired cleft palate who demonstrate posterior placement of alveolar targets (e.g., /t^h/ → [k^h]), known as Group P, differ from children with cleft palate without such an error pattern (Group NP) and from normally developing children without cleft palate (Group N) in the perception of /t^h/ and /k^h/. Ten age-matched children in each of these three groups identified 8 synthetic stimuli along an acoustic continuum ranging from /t^h/ to /k^h/. The children with posterior placement performed at random levels, appearing unable to distinguish /t^h/ from /k^h/. In contrast, both groups of children without posterior placement demonstrated a clear identification pattern. These results, which suggest that children with cleft palate and posterior placement have a perceptual deficit, contribute to discussion of the possible etiology of speech deficits in this population.

KEY WORDS: cleft palate, phonology, perception, identification

Cleft palate is a congenital anomaly affecting approximately 1 in every 750 live births (McWilliams, Morris, & Shelton, 1990). Birth prevalence in Chinese populations appears similar to rates for Caucasian populations (Cooper et al., 2000; Croen, Shaw, Wasserman, & Tolarova, 1998). Speech disorders associated with cleft palate have been well-documented (for recent a review, see Peterson-Falzone, Hardin-Jones, & Karnell, 2001). Although there is considerable variation within this heterogeneous population, consistently reported problems include hypernasality, nasal emission, and articulation errors affecting consonants requiring intraoral air pressure (Peterson-Falzone et al., 2001). A distinction is currently made between "passive" or "obligatory" errors (such as hypernasality, pervasive nasal emission, and weak obstruents), which appear to be a direct result of velopharyngeal incompetence or oronasal fistulae, and "active" or "compensatory" errors, which presumably arise in a learned attempt to compensate for physiological constraints but which might persist after the physical anomaly has been corrected (Peterson-Falzone et al., 2001). These compensatory errors, which are atypical in English-speaking noncleft populations, include glottal stop, pharyngeal fricative, velar fricative, pharyngeal stop, posterior nasal fricative, and middorsum palatal stop (Trost, 1981).

One common general error pattern observed in speakers with repaired cleft palate is posterior placement of oral targets (Lawrence & Philips, 1975; Moller, 1990). Different terms have been used to describe this error pattern, and there is some variation regarding what is included

under this pattern. Gibbon and Crampin (2001) referred to a "retracted or 'backed' tongue placement" (p. 96), which affects sibilants, alveolar stops, and velar stops. "Palatal misarticulation" is a pattern demonstrated via electropalatography, which involves contact between the tongue dorsum/middorsum and the hard palate (Michi, Suzuki, Yamashita, & Imai, 1986; Yamashita, Michi, Imai, Suzuki, & Yoshida, 1992). This pattern has been shown to occur frequently in Japanese speakers with cleft palate, and it affects dental and alveolar targets (Yamashita et al., 1992). "Backing" has been reported as a commonly occurring phonological process in children with cleft palate by several investigators (e.g., Chapman, 1993; Peterson-Falzone et al., 2001; Russell & Grunwell, 1993). The term "posterior placement," rather than "backing," is used in this study in order to avoid, at this stage, debate regarding the phonetic versus phonological nature of such a pattern.

Posterior placement may affect many targets. In this study, we focus in particular on children who have velar realization of alveolar stops (e.g., /t/ → [k]). However, the possible realization of alveolar targets as middorsum palatal stops is also considered. Middorsum palatal stops were described by Trost (1981) as a possible compensatory substitution for either alveolar or velar stops. When occurring as a substitution for alveolar targets, they may be considered part of a posterior placement pattern (Peterson-Falzone, 1986). Middorsum palatal stops are notoriously difficult to transcribe (Gibbon & Crampin, 2001; Gooch, Hardin-Jones, Chapman, Trost-Cardamone, & Sussman, 2001; Santelmann, Sussman, & Chapman, 1999) and may be inaccurately transcribed as either alveolar or velar stops (Santelmann et al., 1999; Trost, 1981).

Several explanations have been offered for the occurrence of a pattern of posterior placement in speakers with cleft palate. The most common explanations have been current or past velopharyngeal incompetence (Chapman, 1993; D'Antonio & Scherer, 1995; McWilliams et al., 1990; Peterson-Falzone, 1986) and current or past oronasal fistulae (Hoch, Golding-Kushner, Siegel-Sadowitz, & Shprintzen, 1986; LeBlanc, 1996). In the case of velopharyngeal incompetence, the speaker presumably subconsciously is attempting to achieve valving at a point inferior to the velopharyngeal valve, in an effort to produce plosion or frication before pressure is lost through the velopharyngeal port. In the case of oronasal fistulae, the speaker may similarly subconsciously be attempting to achieve a valve at a place posterior to the fistulae to prevent nasal escape of air. Other explanations that have been suggested are dental/occlusal anomalies (Golding-Kushner, 1995), where normal tongue posture may not be possible; reduced sensation in the alveolar region relating to scar tissue following surgery (Hardcastle, Morgan Barry, & Nunn, 1989; Stengelhofen, 1989), where the speaker may be subconsciously targeting a palatal region with more sensation; hearing impairment (Hardcastle et al., 1989; Trost-Cardamone, 1990); and an early-established posterior tongue posture, possibly related to early feeding patterns (Hardcastle et al., 1989; Moller, 1990; Russell & Grunwell, 1993) and, again, presumably a subconscious attempt to achieve velopharyngeal closure or to block escape of fluid through an unrepaired palate. Henningsson and Isberg (1990) have also noted a posterior placement pattern in speakers with "residual clefts" in the case of delayed hard palate closure. Unfortunately, there is little evidence to support or refute any of these hypotheses (D'Antonio & Scherer, 1995; Peterson-Falzone, 1986). The fact that this abnormal articulatory pattern is observed in speakers following palatal repair and the provision of a seemingly adequate physiological speech mechanism (i.e., normal occlusion, no fistula, adequate velopharyngeal closure) suggests that there may be a habitual, learned aspect to the pattern (Peterson-Falzone et al., 2001).

Most explanations of speech disorders in persons with cleft palate have focused on structural factors. However, several recent studies have advocated a phonological approach toward understanding speech disorders in this population. Phonological delay or disorder unrelated to the cleft could of course occur in a child with cleft palate. However, several authors have suggested that speech errors that initially occur as a result of structural limitations (phonetic disorder) may later result in a phonological disorder (Bzoch, 1997; Chapman, 1993; Grunwell & Russell, 1988; Russell & Grunwell, 1993).

Other authors have been more skeptical about appropriateness or usefulness of a phonological approach or explanation for this population (e.g., Foster, Riley, & Parker, 1985; Peterson-Falzone et al., 2001). The dispute regarding the value and appropriateness of a phonological approach may be at least partially explained by differing definitions of what constitutes a phonological disorder (see McWilliams et al., 1990, for a discussion). Although some authors appear to use the term "phonological process" to refer simply to an error pattern that affects more than one phoneme in a sound class (e.g., Middleton & Pannbacker, 1997; Moller, 1990), others appear to reserve the term "phonological disorder" for error patterns that are linguistically based, implying a deficit in the child's abstract knowledge or underlying representation of the sound system (e.g., Dinnsen, 1984; Milroy, 1985).

Empirical testing of the representations underlying children's sound system is generally regarded as either unlikely or impossible (Milroy, 1985). However, it is possible to test children's ability to discriminate or identify phonological contrasts. There have been relatively few investigations of such abilities in children with cleft palate (but see Finnegan, 1974). Children with phonological disorders (without structural abnormalities) have received

more attention, although the results have been conflicting. Although many studies have demonstrated that children with phonological disorders also demonstrate perceptual deficits, others have not (see Rvachew & Jamieson, 1989, for a review). Many of these previous studies of the perceptual abilities of children with phonological disorder may have been limited by problems with the perceptual tasks that were used, as outlined by Locke (1980), Raaymakers and Crul (1988), and Rvachew and Jamieson (1989). Recommendations for appropriate perceptual testing include making the perceptual task specific to the production disorder, multiple trials, an easy response task, and determination of the direction of misrepresentation (Locke, 1980). Raaymakers and Crul (1988) recommended the inclusion of a control group of children with different production errors to the contrast under investigation.

The use of synthetic stimuli for identification or discrimination tasks allows for systematic control of input in perceptual experiments, avoiding possible nonphonemic features such as differences in pitch or aspiration (Rvachew & Jamieson, 1989). The synthetic continuum task permits participants to focus on salient acoustic cues (Groenen, Maassen, & Crul, 1998; Rvachew & Jamieson, 1989). This approach has been applied to several populations. In one recent example, Maassen, Groenen, Curl, Assman-Hulsmans, and Gabreels (2001) demonstrated that children with developmental dyslexia had poorer discrimination of voicing and place of articulation than did two control groups (matched for age and reading level). This was interpreted as supporting evidence for a functional link between perception and dyslexia (Maassen et al., 2001). To our knowledge, this approach has not been applied to children with a structural abnormality (such as cleft palate) where there is no known cognitive, neurological, or hearing impairment.

In summary, speech errors in children with cleft palate have been well-documented, and one commonly observed error pattern is posterior placement of oral targets. Several explanations have been offered for this pattern, but there is little evidence to support any of the explanations. Most explanations have focused on physiological factors, such as velopharyngeal incompetence. More recently, there has been an interest in phonological explanations of speech disorder in this population, in learned versus physiologically determined patterns of errors, and in the possible interaction between production and perception. However, there have been few investigations of the perceptual abilities of children with cleft palate. Such an investigation could contribute to our understanding of the nature and cause of a posterior placement error pattern in this group of children.

The aim of the present study was to determine whether children with repaired cleft palate and a posterior-placement production error pattern (e.g., /t/ → [k]) also demonstrated difficulty in the perception of anterior versus posterior place of articulation. We tested the children's ability to identify /t^h/ versus /k^h/ in a word-identification task, using synthetic stimuli that varied systematically between /t^h/ and /k^h/. The performance of this target group of children was compared with two control groups: children with repaired cleft palate but no posterior placement and normally developing children without cleft palate.

Method

Participants

Three groups of children participated in this experiment. Two groups of children with repaired cleft palate differed in terms of their speech production errors, as determined during the screening procedures. Group P consisted of 10 children (mean age = 6;9 [years;months], range = 4;6–12;9) with repaired cleft palate who displayed posterior placement of alveolar targets (e.g., /t/ → [k]). Group NP consisted of 10 children with repaired cleft palate matched in age to the children in Group P (mean age = 6;9, range = 4;6–12;8). These children did not have posterior placement of alveolar targets but most (8 of 10) demonstrated other speech production errors. The children in these two groups were recruited from the Prince Philip Dental Hospital/University of Hong Kong Cleft Lip and Palate Centre and from the Hong Kong Cleft Lip and Palate Association. Preselection information was obtained from medical records and parent reports. None of the children in these two groups had been identified by a member of the multidisciplinary cleft palate team or by any other professional caring for them as having any syndrome associated with cleft palate. All had surgical closure of the palate between 12 and 18 months. None of the children had a fistula at the time of testing, although several were scheduled for bone grafting of a residual alveolar cleft, according to the treatment protocol of the Centre. One of the children in Group P (P5) had mild hypernasality, and 1 of the children in Group NP (NP6) had mild hypernasality and borderline velopharyngeal closure. The remaining participants had resonance and velopharyngeal status (where known) within normal limits, as determined by experienced members of a multidisciplinary cleft palate team. Participant details are shown in Table 1, which includes a summary of type of cleft, whether the children had received speech therapy previously, and whether there was any parent report of recurrent otitis media (ROM), according to the patient record. Children with three or more reported episodes of otitis media were classified as "yes" for ROM, and those with less than three ocurrences as "no".

The third group (Group N) consisted of 10 normally developing children matched in age to the participants

Table 1. Participant details.

Participant	Age	Sex	Type of cleft	Previous speech therapy	Parent report of ROM
P1	4;6	F	U(R)CLP	No	Yes
P2	4;7	M	U(L)CLP	No	Yes
P3	4;10	M	U(R)CLP	Yes	Yes
P4	5;6	F	U(L)CLP	No	No
P5	6;1	M	U(L)CLP	Yes	Yes
P6	6;9	M	BCLP	Yes	Yes
P7	7;1	F	U(L)CLP	Yes	No
P8	9;3	M	BCLP	Yes	Yes
P9	12;2	M	U(R)CLP	Yes	No
P10	12;9	M	BCLP	Yes	Yes
NP1	4;6	F	U(R)CLP	No	Yes
NP2	4;6	M	U(R)CLP	No	Yes
NP3	4;11	M	U(L)CLP	Yes	Yes
NP4	5;6	M	U(L)CLP	Yes	Yes
NP5	6;2	M	U(R)CLP	Yes	Yes
NP6	6;9	F	BCLP	Yes	No
NP7	7;0	M	U(L)CLP	Yes	Yes
NP8	9;6	M	BCLP	Yes	Yes
NP9	12;5	M	U(L)CLP	Yes	Yes
NP10	12;8	M	BCLP	Yes	Yes
N1	4;5	F	—	—	—
N2	4;8	F	—	—	—
N3	4;10	M	—	—	—
N4	5;4	M	—	—	—
N5	6;0	M	—	—	—
N6	6;7	F	—	—	—
N7	7;2	M	—	—	—
N8	9;6	M	—	—	—
N9	11;11	M	—	—	—
N10	12;10	M	—	—	—

Notes. ROM = recurrent otitis media; P = children with repaired cleft palate and posterior placement of alveolars; NP = children with repaired cleft palate and no posterior placement of alveolars; N = normally developing children; U(R)CLP = unilateral (right) cleft lip and palate; U(L)CLP = unilateral (left) cleft lip and palate; BCLP = bilateral cleft lip and palate; Dashes indicate not applicable.

in Group P and Group NP (mean age = 6;9, range = 4;5–12;10). These children were recruited from the local community. According to parent report, none of these children had a history of speech, language, hearing, or learning problems. The participants in all three groups were native Cantonese speakers.

Screening Procedures

All participants demonstrated hearing within normal limits bilaterally as determined by pure-tone screening at 20 dB hearing threshold level from 250 Hz to 4000 Hz (International Organization for Standardization, 1985), conducted on the same day as experimental testing. None of the children had middle ear infection or tympanostomy tubes at the time of testing, as determined by parent report and otoscopic examination. According to parent reports, all children were performing well in their regular classroom studies (kindergarten, primary, or secondary), suggesting normal levels of cognitive functioning.

Children meeting these criteria then received a speech evaluation with two word lists. Part I of the Cantonese Segmental Phonology Test (CSPT; So, 1993) is a 31-item picture-identification test that samples each Cantonese phoneme and tone at least once. A "Deep Test" was constructed for this study, consisting of 36 items in a picture-identification test. Each Cantonese syllable-initial alveolar ([t, t^h, s, ts, ts^h, l]) was sampled six times, in six different monosyllabic and bisyllabic words varying in vowel and final consonant context. All the children were able to name all the pictures spontaneously. The speech evaluation was conducted in a quiet clinic room. All productions were recorded using an Aiwa AM-F70 minidisc recorder and a Sony XII-57 microphone, which was attached to the collar of the participant's clothing.

Productions from both tests were transcribed live by the third author using IPA conventions. Broad transcription was used, with the addition of narrow transcription for the following: lateral /s/, palatalized /s/, and labialized /f/. Hypernasality and nasal emission are not reported in this study. All productions were retranscribed from audiorecordings by another final-year speech-language student trained in IPA transcription. Agreement scores for syllable-initial alveolar targets only were calculated by dividing the number of agreements by the total number of agreements plus disagreements. Interjudge agreement was 92% (1,350 of 1,470). Discrepancies were resolved by consensus agreement between these two transcribers to ensure the inclusion of all data.

The production errors of the children in Group P are summarized in Table 2. All participants had posterior placement for at least 8 of 17 alveolar plosive targets. Four children demonstrated posterior placement for /s/ (including Participant P5, who produced /s/ → [k]), and 2 demonstrated posterior placement (as well as stop substitution) for affricate targets. Several children in this group produced errors in addition to posterior placement (Participant P2: /s/ → [t^h]; Participant P4: /s/ → [ɬ], Participant P8: /s/ → [f]).

The children in Group NP produced a variety of errors, including de-aspiration (NP1), affrication (NP1 and

Table 2. Production errors of children in Group P (repaired cleft palate and posterior placement) on the Cantonese Segmental Phonology Test and the Deep Test.

	Target					
Participant	**/t/**	**/ tʰ/**	**/s/**	**/ts/**	**/tsʰ/**	**/l/**
P1	[k] 8/8	[kʰ] 9/9	[sʲ] 7/9	—	—	—
P2	[k] 6/8	[kʰ] 9/9	[tʰ] 9/9	—	—	—
P3	[k] 5/8	[kʰ] 7/9	—	—	—	—
P4	[k] 3/8	[kʰ] 5/9	[t] 7/9	—	—	—
P5	[k] 5/8	[kʰ] 5/9	[k] 4/9	—	—	—
P6	[k] 7/8	[kʰ] 9/9	—	[k] 5/9	[kʰ] 6/7	—
P7	[k] 5/8	[kʰ] 7/9	[sʲ] 7/9	—	—	—
P8	[k] 6/8	[kʰ] 7/9	[f] 6/9	[k] 8/9	[k] 7/7	
P9	[k] 4/8	[kʰ] 6/9	—	—	—	—
P10	[k] 4/8	[kʰ] 4/9	[sʲ] 8/9	—	—	—

Note. Dashes indicate no production errors noted.

NP8), lateralization (NP2 and NP6), and stopping (NP3). Two participants in this group had no articulatory errors. The children without cleft palate (Group N) produced no speech errors on the CSPT or the deep test.

Stimuli

Two words were selected that were within the vocabularies of young Cantonese children: /t^hau_4/ *head* and /k^hau_4/ *ball*. Cantonese has six contrastive tones that can be described by tone numbers (1–6) as well as by labels that indicate the approximate starting and ending pitch of the tone (high level, high rising, mid-level, low falling, low rising, and low level). The target words both have Tone 4 (low falling). The target words produced by a 23-year-old male native speaker of Cantonese were recorded using the external microphone of an Apple Power Macintosh G3 (a unidirectional electret microphone with a minimum 56dB signal-to-noise ratio) and SoundScope 16™, a sound and speech analysis software program (GW Instruments, Inc., 1993). Recorded syllables were analyzed acoustically using SoundScope 16 running on an Apple Power Macintosh G3. The "best" sounding /t^hau_4/ syllable (as identified by a native speaker of Cantonese) was normalized in amplitude and used as a basis for synthesizing the syllable continuum. Six measures were taken at every millisecond throughout the duration of the target syllable—F0, the center frequencies of the first four formants (F1, F2, F3, F4), and the amplitude. These raw measures were used as the initial settings for Sensimetrics Corporation's SenSyn™, a Klatt-style cascade/parallel formant synthesizer (Klatt, 1980) implemented on an Apple Macintosh G3. The amplitude envelope was used as a rough estimate of parameter amplitude of voicing, and F0, F1, F2, F3, and F4 were used as initial approximations of the parameters of the same name. Following synthesis, parameters were adjusted by hand until the synthetic stimulus was judged both intelligible and natural sounding by a panel of three native-Cantonese-speaking student clinicians. The onset frequency of the third formant of this synthetic /t^hau_4/ syllable was then manipulated (alone) until the resulting syllable sounded sufficiently /k^hau_4/-like to satisfy a native speaker. Both synthetic syllables (/t^hau_4/ and /k^hau_4/) were then adjusted in tandem (any adjustment made to one was made to the other simultaneously) until three native Cantonese listeners judged both to be acceptable representatives of the respective consonants. These two synthetic /t^hau_4/ and /k^hau_4/ syllables were used as the endpoints of an eight-interval continuum varying in perceptually equal steps only in the onset frequency of F3. Specific stimulus parameters are given in Table 3.

Procedures

Perceptual testing immediately followed the screening procedures described above. Each participant was tested in a quiet room. Stimuli were played to listeners via a Hypercard stack running on a Macintosh PowerBook 1400. Presentation was via Sennheiser HD 545 headphones. Playback level was set at a listening level judged to be comfortable by the experimenter and the participants. This level was maintained across listeners.

Each participant was tested with 11 blocks of trials to assess their ability to identify the test stimuli. Each block consisted of eight trials, in random order, one trial for each of the eight test stimuli. Each trial consisted of a single presentation of the test stimulus in a two-alternative, forced-choice paradigm. Each child

Table 3. Stimulus parameters for the synthetic /t^hau_4/ and /k^hau_4/ stimuli.

Parameter	Onset frequency level	Specifications
F0	100 Hz	0–95 ms, level at 100 Hz; 96–125 ms, linear fall to 98 Hz; 126–425 ms, linear fall to 80 Hz; 426–500 ms, linear fall to 75 Hz.
F1	487 Hz	0–65 ms, linear rise to 165 Hz; 66–165 ms, level at 165 Hz; 166–429 ms, linear fall to 429 Hz; 430–500 ms, level at 429 Hz.
F2	1790 Hz	0–96 ms, linear fall to 1434 Hz; 97–113 ms, level at 1434 Hz; 114–243 ms, linear fall to 777 Hz; 243–500 ms, level at 777 Hz.
F3	Token 1 – 3109.00 Hz Token 2 – 2961.59 Hz Token 3 – 2819.89 Hz Token 4 – 2683.67 Hz Token 5 – 2552.73 Hz Token 6 – 2426.85 Hz Token 7 – 2305.84 Hz Token 8 – 2189.51 Hz	0-97 ms, linear fall or rise to 2374 Hz; 98–340 ms, linear rise to 2700 Hz; 341–500 ms, level at 2700 Hz.
F4	4303 Hz	0–342 ms, linear fall to 3338 Hz; 343–500 ms, level at 3338 Hz.
F5	4561 Hz	0–500 ms, level at 4561 Hz.
AH	80 dB	0–13 ms, linear fall to 77 dB; 14–95 ms, linear rise to 78 dB; 96–98 ms, fall to 0 dB
AV	0 dB	0–70 ms, level at 0 dB; 70–90 ms, linear rise to 60 dB; 61–63 ms, linear rise to 63 dB; 63–64 ms, linear rise to 100 dB; 65–130 ms, level at 100 dB; 101–155 ms, fall to 62 dB; 156–214 ms, fall to 59 dB; 215–272 ms, level at 59 dB; 273–250 ms, fall to 56 dB; 251–412 ms, fall to 51 dB; 413–500 ms, fall to 34 dB.

Note. F1, F2, F4, AH, and AV were the same for all stimuli; F3 onset varied along the continuum. AH = amplitude of aspiration; AV = amplitude of voicing.

was told that he or she was going to listen to words that could be identified by clicking on one of two pictures: a picture of a head, representing the stimulus /t^hau_4/, and a picture of some soccer balls, representing the stimulus /k^hau_4/. The first block was treated as a practice block and was not scored, although listeners were not aware of this at the time of testing.

Results

Group Identification Functions

The number of /k^hau_4/ responses for each of the eight stimuli in the continuum was calculated for each participant. Group identification values were plotted by averaging across the participants within each group. The identification functions for the three groups of participants are displayed in Figure 1 as linear interpolations between each groups' data points.

One way to locate a phonemic boundary along a stimulus continuum is to determine the point along the continuum at which the identification function is 50% (Repp & Liberman, 1987, p. 90). Using this approach, the group phonemic boundaries for the normally developing children (Group N) and the children with repaired cleft palate but without posterior placement (Group NP) were both found to lie between Stimulus 4 and Stimulus 5. Evidence to support this phonemic boundary location can be found by examining the group response proportions on either side of the boundary. Children in Group N and Group NP identified an average of 90% of the stimuli to the right of this boundary as /k^h/ and identified about 95% of the stimuli to the left of this boundary as /t^h/. A pattern of identifying two categories was clearly shown. This suggests that these two groups of children appropriately perceived differences in F3 onset frequencies as signaling either /t^h/ or /k^h/. Stimuli 1–3, with F3 onset frequencies above 2600 Hz, were generally perceived as /t^h/, whereas Stimuli 5–8, with F3 onset frequencies below 2600 Hz, were most often identified as /k^h/.

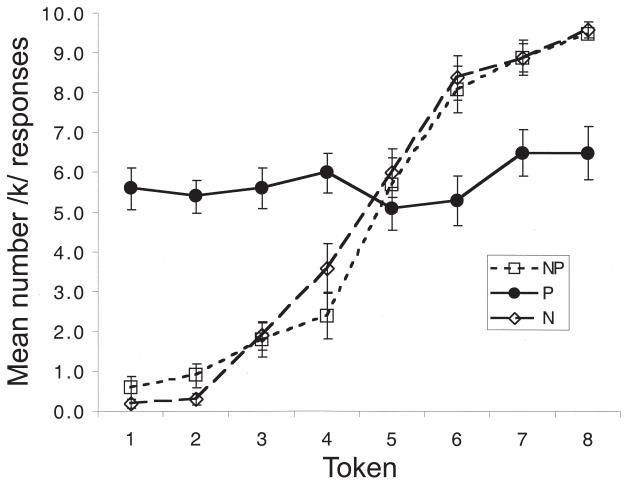
In contrast, the children with repaired cleft palate and posterior placement (Group P) generally identified

51% to 65% of trials as /k^h/ for all stimuli (1–8) along the continuum. The flatter identification function shown in Figure 1 suggests that this group was virtually at chance level in their identification, and did not perceive two distinct categories along the /t^h–k^h/ continuum. The apparent lack of any category boundary along the continuum for this group is supported statistically. Proportions were transformed using the arcsin transform to more closely approximate a normal distribution (Kirk, 1995). One-way repeated-measures analysis of variance of the arcsine transform of these proportions showed no effect of token, $F(7, 63) = 1.53$, $p = .18$. Post hoc (Tukey's honestly significant difference) analysis further showed no significant difference in response percentages, even between Tokens 5 and 8 ($p = .32$).

Individual Identification Functions

Examination of identification functions for individual speakers showed that all children in Group NP and Group N were able to classify the stimuli into two distinct groups, whereas none of the children in Group P were able to identify the stimuli appropriately. Intragroup variability was low, as indicated by the standard error bars in Figure 1. Qualitative analysis of individual identification functions within each group indicated that intragroup variability could not be easily explained by any of the participant variables included (age, type of cleft, previous speech therapy, or parent report of ROM). The results for the 2 children with mild hypernasality (P5 and NP6) did not appear to differ from those of the other children in their respective groups in any meaningful way.

Figure 1. Mean number of /k^h/ responses to eight synthetic stimuli on a /t^h–k^h/ continuum by children with repaired cleft palate and posterior placement (P), children with repaired cleft palate but without posterior placement (NP), and normally developing children (N). Error bars show standard error.



Intraparticipant Variability

Table 4 displays the identification patterns for each child in Group NP and Group N. For example, the first participant in Group NP identified Stimuli 1–3 as phoneme /t^h/ at least 7 out of 10 times. The fourth stimulus was identified at only chance levels (which was conservatively defined as less than 7 out of 10 responses), thus forming a phonemic boundary region. Stimuli 5–8 were identified as /k^h/ at above chance level, forming the /k^h/ phonemic space. The phonemic boundaries of the children in these two groups varied from Stimulus 3 to Stimulus 7, with the majority falling between Stimulus 4 and Stimulus 5. All the children in these two groups demonstrated identification patterns that involved a stable /t^h/ region starting with Stimulus 1 and 2 and a stable /k^h/ region that ended with Stimulus 8.

Table 5 shows the individual identification patterns for the children in Group P. The children in this group performed at chance levels for many of the stimuli (ranging from two to seven of the eight stimuli). When a phoneme was identified (i.e., at least 7 out of 10 times), it was more likely to be identified as /k/ (see, in particular, Participants P1, P6, P7, and P10, who never identified a stimulus as /t/). None of the children appeared to have clear phonemic spaces for the two phonemes or an identifiable phonemic boundary.

Discussion

Posterior placement of oral targets is a well-documented error pattern in speakers with repaired cleft palate, although the etiology of this pattern has not been well-established. A relatively recent focus on phonological disorders in children with cleft palate has complemented physiological explanations of this and other error patterns. However, most studies offering phonological explanations have examined speech output data only. There have been very few investigations of the ability of children with cleft palate to identify or discriminate phonological contrasts.

The aim of the current study was to evaluate the ability of children with repaired cleft palate and posterior placement of alveolar targets (e.g., /t^h/ → [k^h]) to identify /t^h/ versus /k^h/ targets from a range of synthetic stimuli. Participants from two control groups, children with repaired cleft palate but without posterior placement (Group NP) and normally developing children without cleft palate (Group N), performed similarly on the categorical perception task; the children in both groups were able to identify the stimuli appropriately, and they showed a clear phonemic boundary between /t^h/ and /k^h/. In contrast, the children in the posterior placement group (Group P) demonstrated almost chance-level performance and showed

Table 4. Identification of each stimulus by each child in Group NP (children with cleft palate but without posterior placement) and Group N (normally developing children).

	Stimulus number									Stimulus number							
Participant	1	2	3	4	5	6	7	8	Participant	1	2	3	4	5	6	7	8
NP 1	/t/	/t/	/t/	–	/k/	/k/	/k/	/k/	N 1	/t/	/t/	/t/	/t/	–	–	–	/k/
NP 2	/t/	/t/	/t/	/t/	/k/	/k/	/k/	/k/	N2	/t/	/t/	/t/	/k/	/k/	/k/	/k/	/k/
NP 3	/t/	/t/	/t/	/t/	–	/k/	/k/	/k/	N3	/t/	/t/	/t/	/t/	–	/k/	/k/	/k/
NP 4	/t/	/t/	/t/	–	–	/k/	/k/	/k/	N 4	/t/	/t/	–	/t/	–	–	/k/	/k/
NP 5	/t/	/t/	–	–	/k/	/k/	/k/	/k/	N 5	/t/	/t/	/t/	–	/k/	/k/	/k/	/k/
NP 6	/t/	/t/	/t/	/t/	–	/k/	/k/	/k/	N 6	/t/	/t/	/t/	/t/	/k/	/k/	/k/	/k/
NP 7	/t/	/t/	/t/	/t/	/t/	–	–	/k/	N 7	/t/	/t/	/t/	/t/	/t/	/k/	/k/	/k/
NP 8	/t/	/t/	/t/	/t/	/t/	–	/k/	/k/	N 8	/t/	/t/	/t/	–	/k/	/k/	/k/	/k/
NP 9	/t/	/t/	/t/	/t/	/k/	/k/	/k/	/k/	N 9	/t/	/t/	/t/	–	/k/	/k/	/k/	/k/
NP10	/t/	/t/	/t/	/t/	–	/k/	/k/	/k/	N10	/t/	/t/	/t/	–	/k/	/k/	/k/	/k/

Note. The criterion for identification was 7 of 10 responses. A dash indicates that the stimulus was identified at chance level (below 7 of 10 responses). Shaded areas represent the apparent phonemic boundary (between /t/ and /k/) for each child.

no clearly defined division of the acoustic space across the range of synthetic stimuli into /t/ and /k/ categories.

Finnegan (1974) showed that 7- and 8-year-old boys with cleft palate had poorer phoneme discrimination than age-matched controls. We selected an identification task, rather than a discrimination task, for this study. Identification tasks involve phonemic judgement, which is considered more relevant to investigations of phonological disorder than discrimination tasks (Groenen et al., 1998; Monnin & Huntington, 1974; Raaymakers & Crul, 1988; Rvachew & Jamieson, 1989). However, given the poor performance of the participants in Group P (those with posterior placement of alveolar targets), it may be of interest to conduct further discrimination tasks in order to identify the locus of breakdown in processing more precisely.

Several studies of children with phonological disorder have demonstrated that perceptual deficits, when they exist, are specific to the production error (see Groenen et al., 1998, for a recent review). In the current study, only one phonemic contrast was investigated in the perceptual identification task. It is not known whether the children with posterior placement demonstrate a more general perceptual deficit, affecting other contrasts. However, it is of interest that the children in Group NP, most of whom had production errors (but not posterior placement of alveolar targets), were indistinguishable from the normally developing, noncleft group (Group N) on the perceptual task. In addition, the children with posterior placement (Group P) tended to make a higher percentage of /k/ than /t/ responses (see Figure 1 and Table 5). Although it is possible that these children had a response bias (e.g., preferring one picture over another; Rvachew & Jamieson, 1989), a more likely explanation is that the children had a perceptual bias related to their own production error (Raaymakers & Crul, 1988). This suggests a production-specific perception disorder.

A range of linguistic deficits have been documented in children with cleft palate (see Kuehn & Moller, 2000, for a review). Although reduced length of utterance has been the most consistent finding across studies, children with cleft palate have been shown to perform significantly

Table 5. Identification of each stimulus syllable by each child in Group P (children with cleft palate and posterior placement).

Participant	Stimulus number							
	1	2	3	4	5	6	7	8
P 1	/k/	/k/	/k/	—	—	/k/	/k/	/k/
P 2	—	—	/k/	—	—	/t/	—	—
P 3	—	/k/	—	—	/t/	—	/k/	/k/
P 4	/t/	—	—	/k/	—	/k/	/k/	/k/
P 5	—	—	/t/	—	/t/	—	—	/t/
P 6	—	—	—	—	—	—	/k/	/k/
P 7	—	—	—	/k/	—	—	—	—
P 8	—	—	/k/	—	—	/t/	—	—
P 9	/k/	/t/	—	—	—	/t/	—	—
P10	—	/k/	—	/k/	—	/k/	/k/	/k/

Note. The criterion for identification was 7 of 10 responses. A dash indicates that the stimulus was identified at chance level (below 7 of 10 responses).

poorer than their noncleft peers on a number of expressive language tasks (e.g., grammatical complexity, amount of verbal output, number of different words, and age of appearance of first words and first phrases) as well as on receptive language tasks such as vocabulary comprehension (Kuehn & Moller, 2000).

Although some authors report that these deficits represent a delay and that children "catch up" by midchildhood, several other studies report persisting language problems through adolescence and adulthood (Kuehn & Moller, 2000). It is possible that the perceptual deficit shown by the children in Group P (children with repaired cleft palate and posterior placement of alveolar targets) represents a delay rather than deviance. This distinction is a difficult one to demonstrate empirically (Groenen et al., 1998). However, the consistently poor performance on the perceptual task of all the children in Group P across a wide age range (4;6 to 12;9 years) suggests that this is not a developmental issue. A longitudinal or systematic cross-sectional study would address this issue directly (Groenen et al., 1998; Rvachew & Jamieson, 1989).

Phonemic categorization requires both intact processing of primary acoustic cues and stable representation of contrasts in phonological representation (Cutting & Pisoni, 1978). All 30 participants in the present study had normal hearing (as defined by passing a puretone audiological screening examination) at the time of the perceptual experiment, suggesting that the same acoustic cues must have been equally available to all children in each group. However, the incidence of otitis media and conductive hearing loss in children born with cleft palate is known to be very high (McWilliams et al., 1990) and the possibility of fluctuating or persistent conductive hearing loss during development cannot be ruled out. Finnegan (1974) attributed the poorer performance of boys with cleft palate on a perceptual discrimination task, as compared with age-matched controls without cleft palate, to a history of ROM. However, his study did not include a second, control group of children with cleft palate but without the specific output disorder under investigation. The two groups of children with cleft palate in our study had very similar profiles of parent report of ROM (7 of 10 in Group P and 8 of 10 in Group NP). However, we cannot rule out the possibility that the children in Group P may have had a higher incidence of ROM during development.

The children in Group P demonstrated both production and perception deficits related to place of articulation. Our experimental design did not allow exploration of the direction of the relationship between production and perception; the results of this study could be seen as supporting the view that production errors may lead to perceptual deficits, the position that perceptual deficits may lead to production errors, or that some underlying deficit accounts for both the perceptual and the production problems.

Evidence that production shapes perception might be taken to support the motor theory of speech perception developed by Liberman, Cooper, Shankweiler, and Studdert-Kennedy (1967). Several previous studies have claimed support for this position (e.g., Hoffman, Daniloff, Bengoa, & Schuckers, 1985; Monnin & Huntington, 1974). There are, however, other ways to account for the influence of production on perception.

Hewlett (1985) discussed the possibility that phonetic factors might have a possible effect on a child's developing phonology (see also Monnin & Huntington, 1974). Interestingly, Hewlett (1990) suggested that children with cleft palate might represent a case of a phonetic disorder with an intact phonological system. However, the results of our study may be interpreted as supporting the view that speech errors that are originally phonetic in nature, as a result of structural abnormality, may lead to phonological errors (see, e.g., Bzoch, 1997; Chapman, 1993; Grunwell & Russell, 1988; Russell & Grunwell, 1993). This explanation is related to the notion of "the biasing of perception by expectation", described by Menn (1983, p. 11) to explain a situation whereby a child maintains an incorrect production when the child is capable of improving it. Menn suggested that the child has stopped monitoring the adult model, assuming that the child's own production is correct: "It seems that he has replaced his original input representation with a new one which is based on his own output" (Menn, 1983, p. 11). This is a plausible explanation for the maintenance of error productions. However, no direct evidence has yet been provided to support the claim that representation in the input lexicon is incorrect in such cases. Furthermore, the relationship between phonological models of input versus output lexicons and models of perception/perceptual processing remains unclear.

Another possible explanation for the results of our study is that an underlying perceptual deficit (in some children with cleft palate) contributes to both perceptual and production errors. A recent series of studies conducted in Helsinki (Ceponiene et al., 1999, 2000; Cheour et al., 1998, 1999) used the auditory event-related potential component known as mismatch negativity to examine auditory sensory memory in children with cleft palate. These studies demonstrated deficits in pre-attentive auditory discrimination in neonates (Ceponiene et al., 2000; Cheour et al., 1999), infants (Ceponiene et al., 2000), and older children with cleft palate (Ceponiene et al., 1999; Cheour et al., 1998), with some differences according to type of cleft (Ceponiene et al., 1999). These authors highlighted the role of auditory discrimination in language development and use and linked the deficits they found

to language and learning disabilities documented by other investigators in children with oral clefts.

Further investigations of the perceptual abilities of children with cleft palate are clearly needed. Such studies will contribute not only to our understanding of speech production errors such as posterior placement of alveolar targets, but potentially to our understanding of the deficits in other areas of language evidenced in some children with cleft palate.

Acknowledgments

Alexander L. Francis is now at Department of Audiology and Speech Sciences, Purdue University. This work was based on an undergraduate honors dissertation conducted by the third author, under the supervision of the first two authors. Portions of this study were presented at the Annual Convention of the American Speech-Language-Hearing Association, New Orleans, LA, November 15–18, 2001. We are grateful to the Hong Kong Cleft Lip and Palate Association and to the children and families who agreed to participate.

References

Bzoch, K. R. (1997). *Communicative disorders related to cleft lip and palate* (4th ed.). Boston: Little Brown.

Ceponiene, R., Hukki, J., Cheour, M., Haapanen, M. L., Koskinen, M., Alho, K., et al. (2000). Dysfunction of the auditory cortex persists in infants with certain cleft types. *Developmental Medicine and Child Neurology, 42*, 258–265.

Ceponiene, R., Hukki, J., Cheour, M., Haapanen, M. L., Ranta, R., & Näätänen, R. (1999). Cortical auditory dysfunction in children with oral clefts. *Clinical Neurophysiology, 110*, 1921–1926.

Chapman, K. L. (1993). Phonological processes in children with cleft palate. *Cleft Palate-Craniofacial Journal, 30*, 64–72.

Cheour, M., Ceponiene, R., Hukki, J., Haapanen, M. L., Näätänen, R., & Alho, K. (1999). Brain dysfunction in neonates with cleft palate revealed by the mismatch negativity. *Clinical Neurophysiology, 110*, 324–328.

Cheour, M., Haapanen, M. L., Ceponiene, R., Hukki, J., Ranta, R., & Näätänen, R. (1998). MMN as an index of auditory sensory memory deficit in cleft palate and CATCH children. *NeuroReport, 9*, 2709–2712.

Cooper, M. E., Stone, R. A., Liu, Y-E., Hu, D-N., Melnick, M., & Marazita, M. L. (2000). Descriptive epidemiology of nonsyndromic cleft lip with or without cleft palate in Shanghai, China, from 1980 to 1989. *Cleft Palate-Craniofacial Journal, 37*, 274–280.

Croen, L. A., Shaw, G. M., Wasserman, C. R., & Tolarova, M. M. (1998). Racial and ethnic variations in the prevalence of orofacial clefts in California, 1983–1992. *American Journal of Medical Genetics, 79*, 42–47.

Cutting, J., & Pisoni, D. (1978). An information-processing approach to speech perception. In J. Kavanaugh & W. Strange (Eds.), *Speech and language in the laboratory, school, and clinic* (pp. 38–72). Cambridge, MA: MIT Press.

D'Antonio, L. L., & Scherer, N. J. (1995). The evaluation of speech disorders associated with clefting. In R. J. Shprintzen & J. Bardach (Eds.), *Cleft palate speech management: A multidisciplinary approach* (pp. 176–220). St. Louis, MO: Mosby.

Dinnsen, D. A. (1984). Methods and empirical issues in analysing functional misarticulations. In M. Elbert, D. Dinnsen, & G. Weismer (Eds.), *Phonological theory and the misarticulating child* (ASHA Monographs No. 22). Rockville, MD: American Speech-Language-Hearing Association.

Finnegan, D. E. (1974). Speech sound discrimination skills of seven- and eight-year-old cleft palate males. *Cleft Palate Journal, 11*, 111–122.

Foster, D., Riley, K., & Parker, E. (1985). Some problems in the clinical application of phonological theory. *Journal of Speech and Hearing Disorders, 50*, 294–297.

Gibbon, F. E., & Crampin, L. (2001). An electropalatographic investigation of middorsum palatal stops in an adult with repaired cleft palate. *Cleft Palate-Craniofacial Journal, 38*, 96–105.

Golding-Kushner, K. J. (1995). Treatment of articulation and resonance disorders associated with cleft palate and VPI. In R. J. Shprintzen & J. Bardach (Eds.), *Cleft palate speech management: A multidisciplinary approach* (pp. 327–351). St. Louis, MO: Mosby.

Gooch, J. L., Hardin-Jones, M., Chapman, K. L., Trost-Cardamone, J. E., & Sussman, J. (2001). Reliability of listener transcriptions of compensatory articulations. *Cleft Palate-Craniofacial Journal, 38*, 59–67.

Groenen, P., Maassen, B., & Crul, T. (1998). Formant transition duration and place perception in misarticulating children and adolescents. *Clinical Linguistics & Phonetics*, *12,* 439–457.

Grunwell, P., & Russell, V. J. (1988). Phonological development in children with cleft lip and palate. *Clinical Linguistics and Phonetics, 2*, 75–95.

GW Instruments, Inc. (1993). SoundScope 16™ [Computer software]. Somerville, MA: Author. Available at http://www.gwinst.com

Hardcastle, W., Morgan Barry, R., & Nunn, M. (1989). Instrumental articulatory phonetics in assessment and remediation: Case study with the electropalatograph. In J. Stengelhofen (Ed.), *Cleft palate: The nature and remediation of communication problems* (pp. 136–164). London: Churchill Livingstone.

Henningsson, G., & Isberg, A. (1990). Oronasal fistulas and speech production. In J. Bardach & H. L. Morris (Eds.), *Multidisciplinary management of cleft lip and palate* (pp. 787–792). Philadelphia: W. B. Saunders.

Hewlett, N. (1985). Phonological versus phonetic disorders: Some suggested modifications to the current use of the distinction. *British Journal of Disorders of Communication, 20*, 155–164.

Hewlett, N. (1990). Processes of development and production. In P. Grunwell (Ed.), *Developmental speech disorders* (pp. 17-36). London: Churchill Livingstone.

Hoch, L., Golding-Kushner, K., Siegel-Sadowitz, V., & Shprintzen, R. J. (1986). Speech therapy. In B. J. McWilliams (Ed.), Current methods of assessing and

treating children with cleft palates. *Seminars in Speech and Language, 7*, 313–325.

Hoffman, P. R., Daniloff, R. G., Bengoa, D., & Schuckers, G. H. (1985). Misarticulating and normally articulating children's identification and discrimination of synthetic [r] and [w]. *Journal of Speech and Hearing Disorders, 50,* 46–53.

International Organization for Standardization. (1985). *Recommendation R 389: Standard reference zero for the calibration of pure-tone audiometers* (2nd ed.). Basel, Switzerland: Author.

Kirk, R. E. (1995). *Experimental design: Procedures for the behavioral sciences* (3rd ed.). Pacific Grove, CA: Brooks/Cole.

Klatt, D. H. (1980). Software for a cascade/parallel formant synthesizer. *Journal of the Acoustical Society of America, 67*, 971–995.

Kuehn, D. P., & Moller, K. T. (2000). Speech and language issues in the cleft palate population: The state of the art. *Cleft Palate-Craniofacial Journal, 37*, 348.

Lawrence, C., & Philips, B. J. (1975). A telefluoroscopic study of lingual contacts made by persons with palatal defects. *Cleft Palate Journal, 12*, 85–94.

LeBlanc, E. M. (1996). Fundamental principles in the speech management of cleft lip and palate. In S. Berkowitz (Ed.), *Cleft lip and palate: Perspectives in management* (Vol. 2, pp. 75–84). San Diego, CA: Singular.

Liberman, A. M., Cooper, F. S., Shankweiler, D. P., & Studdert-Kennedy, M. (1967). Perception of the speech code. *Psychological Review, 74*, 431–461.

Locke, J. L. (1980). The inference of speech perception in the phonologically disordered child. Part I: A rationale, some criteria, the conventional tests. *Journal of Speech and Hearing Disorders, 45*, 431–444.

Maassen, B., Groenen, P., Crul, T., Assman-Hulsmans, C., & Gabreels, F. (2001). Identification and discrimination of voicing and place-of-articulation in developmental dyslexia. *Clinical Linguistics and Phonetics, 15*, 319–339.

McWilliams, B. J., Morris, H. L., & Sheldon, R. L. (1990). *Cleft palate speech* (2nd ed.). Toronto, Ontario, Canada: B.C. Decker.

Menn, L. (1983). Development of articulatory, phonetic, and phonological capabilities. In B. Butterworth (Ed.), *Language production* (Vol. 2, pp. 3–50). London: Academic Press.

Michi, K., Suzuki, N., Yamashita, Y., & Imai, S. (1986). Visual training and correction of articulation disorders by use of dynamic palatography: Serial observation in a case of cleft palate. *Journal of Speech and Hearing Disorders, 51*, 226–238.

Middleton, G. F., & Pannbacker, M. (1997). *Cleft palate and related disorders*. Bisbee, AZ: Imaginart International.

Milroy, L. (1985). Phonological analysis and speech disorders: A comment. *British Journal of Disorders of Communication, 20*, 121–180.

Moller, K. T. (1990). Early speech development: The interaction of learning and structure. In J. Bardach & H. L. Morris (Eds.), *Multidisciplinary management of cleft lip and palate* (pp. 726–731). Philadelphia: W. B. Saunders.

Monnin, L. M., & Huntington, D. A. (1974). Relationship of articulatory defects to speech-sound identification. *Journal of Speech and Hearing Research*, *17*, 352–366.

Peterson-Falzone, S. J. (1986). Speech characteristics: Updating clinical decisions. In B. J. McWilliams (Ed.), Assessment and treatment of children with cleft palate. *Seminars in Speech and Language, 7*, 269–295.

Peterson-Falzone, S. J., Hardin-Jones, M. A., & Karnell, M. P. (2001). *Cleft palate speech* (3rd ed.). St. Louis, MO: Mosby.

Raaymakers, E. M. J. A., & Crul, T. A. M. (1988). Perception and production of the final /s-ts/ contrast in Dutch by misarticulating children. *Journal of speech and Hearing Disorders*, *53*, 262–270.

Repp, B. H., & Liberman, A. M. (1987). Phonetic category boundaries are flexible. In S. Harnad (Ed.), *Categorical perception* (pp. 89–112). Cambridge, U.K.: Cambridge University Press.

Russell, J., & Grunwell, P. (1993). Speech development in children with cleft lip and palate. In P. Grunwell (Ed.), *Analysing cleft palate speech* (pp. 19–47). London: Whurr.

Rvachew, S., & Jamieson, D. G. (1989). Perception of voiceless fricatives by children with a functional articulation disorder. *Journal of Speech and Hearing Disorders*, *54*, 193–208.

Santelmann, L., Sussman, J., & Chapman, K. (1999). Perception of middorsum palatal stops from the speech of three children with repaired cleft palate. *Cleft Palate-Craniofacial Journal, 36*, 233–242.

So, L. K. H. (1993). *Cantonese Segmental Phonology Test.* Hong Kong: Bradford.

Stengelhofen, J. (1989). The nature and causes of communication problems in cleft palate. In J. Stengelhofen (Ed.), *Cleft palate: The nature and remediation of communication problems* (pp. 1–30). London: Churchill Livingstone.

Trost, J. E. (1981). Articulatory additions to the classical descriptions of the speech of persons with cleft palate. *Cleft Palate Journal, 18*, 193–203.

Trost-Cardamone, J. E. (1990). The development of speech: Assessing cleft palate misarticulations. In D. A. Kernahan & S. W. Rosenstein (Eds.), *Cleft lip and palate: A system of management* (pp. 227–235). Baltimore: Williams & Wilkins.

Yamashita, Y., Michi, K., Imai, S., Suzuki, N., & Yoshida, H. (1992). Electropalatographic investigation of abnormal lingual-palatal contact patterns in cleft palate patients. *Clinical Linguistics and Phonetics, 6*, 201–217.

Received March 22, 2002

Accepted October 24, 2002

DOI: 10.1044/1092-4388(2003/037)

Contact author: Tara L. Whitehill, PhD, Department of Speech and Hearing Sciences, University of Hong Kong, 34 Hospital Road (5th Floor), Hong Kong.
E-mail: tara@hku.hk