The Production of Stops in VCV Sequences in Children with a Cleft Palate
An Acoustic Study


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ABSTRACT

Objectives: The present investigation was conducted to examine characteristic features of speech production in children with a repaired cleft palate. The cause of cleft lip and cleft palate formation can be genetic in nature. They are morphological accidents which appear at the second embryonic month. Stops may be difficult to produce for speakers with a cleft lip and/or a cleft palate because of problems in obtaining occlusions that are sufficiently tight. It is usually reported in the literature (as in the present study) that the burst of the plosive is not clear, and that plosives may have the aspect of constrictives. Such a disability in controlling the closure of the plosive thus affects the nature of the burst-release and, consequently, Voice Onset Time or VOT.

Speakers with a cleft palate will use compensatory articulations [1]. As shown by Powers [2], Lawrence and Phillips [3] or Trost [4], several strategies are deployed by speakers to compensate for their articulation problems. Consonantal durations are usually higher than those of the productions of pilot children. A study undertaken by Ha, Sim, Minje and Kuehn [5] testify these findings. Method: Acoustic recordings were obtained from three 10 year old kids characterized by a unilateral posterior cleft palate. Their data were compared with that of a healthy speaker of the same age. All subjects produced six French plosives, embedded in carrier sentences, several times. Recordings of the pathological subjects were carried out in a quiet room at the Hautepierre University Hospital in Strasbourg.

For each occurrence, the duration of the flanking vowels was measured as the interval between onset and offset of a clear vowel formant structure. Within this consonant hold, two other measures were taken: Voice Termination Time or VTT, which is the delay, after offset of the formant structure of V1, for voicing decay during closure of the voiceless stop. VOT was also measured as the interval between burst-release and onset of a clear formant structure for V2.

Results reveal significantly longer VOT values for the pathological subjects, especially when place of articulation is located in the pathological zone. It has been noticed, for example, that absolute and relative VOT values are similar in the [ati] context for the pilot speaker and the cleft palate subjects. On the contrary, relative VOT values are higher for cleft palate subjects in the [aki] sequence, thus confirming results obtained in the previous [ita] and [ika] contexts; indeed the combined effect of producing the alveolar voiceless stop [t] and the soft-palate stop [k] in a back vowel context, [a], attracts the tongue dorsum towards this posterior zone
Furthermore, at the qualitative level, it is observed that voiceless stops usually present irregular voicing pulses during the supposedly silent phase, and significant presence of frication is also remarkable during the consonantal hold of the voiced stops.

**Conclusion:**
Our investigations are related to productions of children who have received speech therapy. If they do have a perfectly comprehensible language, traces in the acoustic signal indicate remains of perturbed productions of certain sounds. The pilot speaker is clearly distinguishable from pathological speakers on the timing level, particularly with regards to control of the VOT of the stops studied. Indeed VOT values of the children with cleft palate are always significantly longer than that of the healthy speaker. Thus it seems coherent that children who have had a posterior cleft palate, although operated and rehabilitated, conserve articulatory difficulties, as highlighted by timing and qualitative anomalies in the acoustic signals. Such anomalies are not depicted in productions by children with a normal language development.

**References**