SOME ACOUSTIC, AERODYNAMIC AND LARYNGEAL CORRELATES OF STUTTERING: PRE-POST THERAPY COMPARISON.

Registration No. 8605
Mohan Murthy G

A Dissertation Submitted in Part fulfilment for Second year M. Sc., (Speech and hearing) to the University of Mysore

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May-1988

DEDICATED TO:

Amma, nānna, babai, pinni

& All the brave Researchers who work(ed) hard to tell us why some of us stutter

Human Researcher is a sat of living cells. These cells are unique because they consume only glucose and oxygen to know about other man's cells. I wish, they will tell us the story of stuttering cells and molecules very soon.

CERTIFICATE

This is to certify that the Dissertation entitled "SOME ACOUSTIC, AERODYNAMIC AND LARYNGEAL CORRELATES OF STUTTERING" - PRE-POST THERAPY COMPARISION is the bonafide work in part fulfilment for the degree of M.Sc, (SDeech and Hearing) of the student with Register No.

Director
All India Institute of
Soeech and Hearing,
Mysore-6

Date: May 1988

Mysore.

CERTIFICATE

This is to certify that this DISSERTATI0N entitled "SOME ACOUSTIC, AERODYNAMIC AND LARYNGEAL CORRELATES OF STUTTERING" - PRE-POST THERAPY COMPARISION has been prepared under my suoervision and guidance.

Savithri. S.R

(DR.S R SAVITHRI) May 1988
GUIDE

DECLARATION

This Dissertation entitled "SOME ACOUSTIC, AERODYNAMIC AND LARYNGEAL CORRELATES OF STUTTERING" - PRE-POST THERAPY COMPARISION is the result of my own study undertaken under the guidance of Dr. SR Savithri, Lecturer in Speech Sciences, All India Institute of SDeech and Hearing, Mysore and has not been submitted earlier at any University for any other Diploma or Degree.

Mysore. Register No.

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INTRODUCTION

INTRODUCTION

To speak, a person generates precise amount of muscular forces at precise time instants, at any particular locus in his speech production system. Force at a particular locus decreases or increases with appropriate velocity (speed and direction) as the speaker transits from one sound to another.

Dysfluencies as in stuttering, perhaps occur when either absolute or relative force(s) at a particular locus or a few loci are not generated as required. This could occur due to:

(1) Inability of the neural speech and language systems to generate normal and required impulses for the muscles,

(2) interference of the neural speech & language systems by other areas of the CNS, when they are generating 'normal and required' impulses for the speech muscles,

(3) anamoly in the conducting pathways that will finally bring impulses to the muscles and

(4) abnormal response of the muscle tissue itself, to the 'otherwise normal' neural impulses.

An attempt to construct a general theory of speech production that explains the mechanisms underlying the production of error-free fluent speech and dysfluencies seen in stuttering was made by Mackay and Mac Donald (1984). According to them, there are three levels of control in speech production: the sentential, the phonological and the muscle movement system. Each system consists of content nodes, which has one or wore neurous. They argue that stuttering originates in the muscle movement system and add that high-level factors such as anxiety and syntactic ambiguity can affect motor control at every level.

Research on stuttering has not pointed out clearly the locus of anamoly in the neuromuscular system (that includes all neural and muscular tissue subserving speech and language). In the oast, scientists have implicated genetic mechanisms (Kidd, 1983), Right hemisphere (Moore, 1984; Boberg et al, 1983), soeech motor system (Cullinan & Springer, 1980), central auditory system (Hall and Jergar, 1978) and auditory-motor connections (Sussman & Mac Ncilage, 1975) as the possible loci of the anamoly. Some resorted to the concept of learning (a behavioural concept that obviously involves some cell-circuits and biochemical events) to explain stuttering (Sheehan, 1953), Bruttan & Shoemaker (1967). However, it has not been possible so far, to localize the aberrant cell-circuit or bio-chemical event at the neural or muscular level which leads to stuttering.

Research has also demonstrated several deviant events in the peripheral speech mechanism of stutterers. For example: Reduced articulatory speed and displacement and longer segment durations (Zimmermann, 1980), and greater glottal adduction per glottic cycle (Conture, 1984). These are directly related to 'deviant muscular forces'. Interpretation of these 'deviant force patterns' has not been strong enough to hint at cell-circuits or biochemical events. What is interesting to note is that quite a few stutterers seem to benefit from speech therapy. Some have maintained fluent speech for a long time. Post therapy fluency in stutterers is all the more intriguing when one realises that the therapy is mainly an endeavour of instructing the patient to alter certain covert cognitive patterns and overt speech patterns voluntarily through self-monitoring and practice. The patient is doing something unknown, when he is overcoming chronic dyafluencies, and acquiring and maintaining fluent speech patterns. Aberrant cell-circuits and biochemical events might have got altered in this process.

The relevant 'facts from the patient's history', 'deviant forces operating in the patient's speech mechanism', 'the activities a patient indulges in during speech therapy' and 'cell-circuits and biochemical events' should all be correlated for a better understanding of stuttering.

The present study is an attempt to analyze and to compare the pre and post therapy speech patterns of a stutterer. The relevant facts from the case history, the deviant forces operating in his speech mechanism and the 'activities he was required to indulge in during speech therapy' are accessed in this study.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

The review is organized under the following sections:

1. Research on laryngeal involvement in stuttering.
2. Acoustic studiea of post therapy fluency in stutterers.
3. Acoustic studies of fluency of stutterers.
4. Research on speech initiation time.
5. Two interesting hypotheses.

Research on laryngeal involvement in stuttering:

I Laryngeal behaviour in stutterers has mainly been observed through fibroscopy, Electromyography and Electroglottography. Available information can be summarized as follows:

(1) During perceived stuttering there were higher levels of muscle activity in the agonist-antagonist laryngeal muscles and a disruption of reciprocal contraction (Freeman, 1984).

(2) Fibroscopic observations show inappropriate adductory/abductory behaviour during perceived stuttering. For sound prolongations, there was much larger percentage of adductory behaviour. For sound/syllable repetitions the percentage of laryngeal behaviours were more evenly distributed among the adductory, intermediate and abductory categories. It was also reported that a greater amount of supraglottal activity may be associated with sound/syllable repetitions (Confture et al, 1985).

These authors observed that there was a more restricted laryngeal target for voiced than for voiceless sounds. This might make the production of voiced sounds an act that requires a finer degree of neuromuscular coordination than that for voiceless sounds. They postulate a hypothesis that the "coordinations necessary for laryngeal adjustments between a word-initial voiced-sound /eg/d / and the subsequent vowel /eg/i/ tray actually require finer gradations of laryngeal muscle adjustments than those between a voiceless word-initial sound and a subsequent vowel where the laryngeal adjustments are actually more quantal in nature".

They also speculate that "just as we are beginning to understand that it is not nature or nurture but nature interacting with nurture that produces a variety of human problems, we are likely to find that it is the larynx inappropriately interacting with supra-laryngeal and respiratory events that produces the physiological backdrop for the problem of stuttering."

(3) Conture et al (1986) opine that it is unclear if laryngeal disruptions as described above are merely 'reactions to some other aspect of the stuttering'. For example, they note that if the supraglottal articulatory configuration is fixated in an open position during an instance of stuttering the vocal folds may adduct to conserve lung air. For this reason, they also studied fluent utterances of stutterers. Electromyographic studies have also been conducted on fluent utterances of sutterers.

i) Shapiro (1980) and Freeman (1984) reported that abnormal electromyographic activity was observed during fluent utterances of stutterers, particularly during periods of acoustic silence preceeding an utterance. This disruptions in the muscular activity patterns were observed during perceptually fluent utterances also.

ii) Electroglottographic studies have bean conducted by Conture (1984) and Conture et al (1986). The former study reported longer closed phases in stutterer's glottal cycles. The later study was a detailed one. In the 1986 study, Conture et al used an Instrument that gave a video display of a trignometric transform of the open quotient, on the basis of the suggestions given by Rothenberg & Mahshie (in press). They found that during perceptually fluent speech, young stutterer's laryngeal behaviour Mas most likely to be subtly inappropriate during transitions betMeen sounds (cv or vc) but essentially normal within sounds. Typical trace of abduction measure, slopes from larger (more open or towards 1 values) values to smaller (more closed or toMards 0 values) for a CV transition. The trace slopes in opposite direction for a VC transition.

II <u>Normal values of speed quotient for young adults</u>

Normal speakers were studied to find out norms for S.Q. for vowels /a/ /i/ /u/ by Shridhara (1986). They are as follows:

	Male				Female			
	/a/	/i/	/u/	Mean for 3 vowels	/a/	/i/	/u/	Mean for 3 vowels
Mean S.Q.	1.91	1.8	1.8	1.84	2.20	2.16	2.13	2.17
S.D.	0.5	0.37	0.36	0.34	0.37	0.46	0.45	0.38

Table 1 : S.Q. values for normal males and females.

Subjects were 30 in number. 10 males and 15 females in the age range of 17 to 30 years were studied. Mean age of subjects was 21.2 years.

Acoustic studies of cost therapy fluency in stutterers

A few researchers have investigated the acoustic differences between the fluency of untreated stuttarers and the fluency of the same subjects after successful speech therapy.

(1) Metz et al (1979): Van Riperian treatment was given. Significant increase was found in

(i) Vowel duration and

(ii) the amount of voicing (the number of times voicing appeared spectrograohically in target words where one would normally predict an acoustic gap).

(2) Metz, Samar, Sacco (1983): 5 weeks of daily Van Riparian therapy was given. Significant increase was found in

(i) vowel duration,

(ii) frication duration associated with intervocalic intervals of voiceless stop consonants and

(iii) the duration of voicing associated with intervocalic intervals for voiced stop consonants.

It was not mentioned whether this increase was independent of speech rate .

(3) Prosek and Runyan (1983): They reported increase in vowel and consonant duration after therapy.

(4) Ramig (1984): They adapted Van Riperian treatment. They reported increase in word duration and vowel duration during oral reading tasks. But these findings were associated with reduced speaking rates.

Mallard & Westbrook (1985) reported same findings but with a use of prolonged speech program.

(5) Robb et al (1985): Prolonged speech type program as given by Shames and Florence (1980) was used. They reported significant increase in the percentage of vocalization time (on sentence length samples).

All the above studies employed only adult subjects.

Acoustic studies of fluency of stutterers

I. Several Studies have investigated the fluency of nontreated stutterers. It seems to differ from the fluency of nonstutterers.

(1) Disimoni (1974): Adult subjects ware studied. Vowel duration was found to be longer in stutterers. On the average, it was 137 ms longer than nonstutterers.

(2) Starkweather & Meyers (1979); Adult subjects were studied. Speed of transition was slower in the transitional subsegmants within an intervocalic interval but had normal speed in the steady state subsegments. They interpreted that stutterers were not able to move their laryngeal and suoralaryngeal structures as quickly as nonstutterers.

(3) Zimmermann (1980): He also studied adult subjects and used high speed cineradiography to describe the temporal organization of perceptually fluent speech in stutterers and nonstutterers. Movements of lower lip and jaw were analyzed in three CVC syllables. It uas reported that the stutterers consistently showed-

(i) longer transition tlmes for downward movements of the articulators,

(ii) longer times between movement onset and peak velocity in the CV gesture and

(iii) longer steady state positions of the lip and jaw during the vowel portion of the syllable.

These findings indicated that stutterers were slower than normals in coarticulatory movements.

(4) Healey (1981): The results of his study indicate that the adult stutterers were slower in completing the transitions from frication onset to peak amplitude during the production of the /s/ phoneme.

(5) Klich & May (1982): Found that durations of /i/ /y/ and /u/ did not vary in different conditions and that the rate of forwent transitions of /i/ /y/ & /u/ were also relatively invariant. They interoreted that as the articulatory details in different conditions did not vary much, their vowel productions are more spatially and temporally restricted.

Prosek R.A. et al (1987) found contrary observations and argued that stutterer's vowel productions were not spatially restricted.

(6) Suchitra (1985) reported abnormal coarticulation in the fluent VCV utterances of stutterers.

(7) Zebrouski at al (1985): They studied children in the age range of 3.1 years to 6.8 years. They did not find any difference in the stop gap duration, frication duration, vouel duration and the rate of CV & VC transitions. However, they speculated that "unlike normals, stutterers do not shout any systematic relationship betueen the peakglottal opening and the articulatory release". This was based on the stop gap duration data. They interpreted that "stutterers show less control and stabilization of laryngeal and supralaryngeal temporal coordination."

(8) Healey & Ramig (1986): They noticed that

(i) greater differences existed between stutterer's and non-stutterer's fluent durational treasures extracted from a reading sample than from a short, isolated nonsense phrase, and

(ii) durational treasures for the stutterers retrained relatively stable during multiple repetitions of both the short phrase and the reading passage.

(9) Pindzoia (1987) reported that the stutterers spend longer time in static articulatory positions (duration of the steady state formant was reportedly longer). Vowel duration was reported to be same in both normals and stutterers. Stutterers had faster V_1C transitions but equivalent CV_2 transitions. Total word durations also remained same in both the groups.

Invariance of total duration of word and difficulty with initiating movements (as seen in lengthened steady states) must be reconciled. It was speculated that if temporal compensation was the effect which ooerates to modify the durations of internal segments of the articulatory units so that the overall duration of the unit remains relatively constant, then the brains articulatory programmes in stutterers was forced by these temporal constraints to move faster throughout the transitional subsegment. However, they opine that it should be confirmed whether stutterers limited their movements to conform with temporal constraints or whether they 'sped up' their movements to accomplish the same extent.

II. Implication for speech therapy

If pretreatad stutterers are inherently slower than normals in coarticulatory movements then the sense of sloping them down further in treatment seems questionable. On the contrary if what Webster (1974) says (that stutterers use articulatory patterns which are too forceful and coarticulatory movements that are too rapid) and what Pindzoia (1987) interprets (that stutterers are using too rapid transitional movements) holds good then rate control methods such as prolonged speech seem appropriate (Pindzoia 1987).

Speech Initiation time

Ever since researchers have viewed the beginning stuttering behaviour such as repetitions or prolongations as implying a difficulty of initiation, they have been interested in the vocal reaction time as an indicator of a stutterer's ability to start and terminate speech. Reaction time is defined as the time one would like to respond to a stimulus. When linguistic stimuli are used as cues for initiation or when the response required is of linguistic nature, the reaction time data are believed to give an insight into both the motor and the linguistic processing systems. Experiments have been conducted to answer the following specific questions about the motor system in stutterers.

(1) Is the speech motor system slow in executing movements?

(2) Is it the auditory-motor process which is aberrant or is it just the motor process in isolation?

(3) In general does their motor system show some overall deficiency resulting in both poor speech and nonspeech manual task responses?

(4) If they are slow in speech movements may it be initiation or termination, will some training and practice improve their performance?

However, research is scanty, where the goal is to see whether speech therapy improves these reaction times or to identify the ingredients in a given speech therapy regimen that is responsible for post therapy speech gains and apparently seems to tackle these minimal motoric differences of stutterer's speech mechanism.

Slow vocal reactions are reported in adult stutterers in several studies. (Adams & Hayden, 1976; Starkweatner et al 1976; Starkweatner et al 1984). That the auditory motor process and not just the motor process which is abberrant was noticed by Mcfarlane & Prins 1978; Sunman and Mac Neilage, 1975; McFarlane & Shiprey, 1981) slower voice and finger reaction times were observed by Luper & Cross, 1978. However, Reich at al (19B1) found only a slower speech reaction time and not the manual reaction time. They believe that longer speech reaction times in stutterers may not mean anything more than the 'learned anticipatory fears of phonatory initiation and maladaptive prephonatory sets. Thus the answer for the third question appears more unresolved.

Recently Borden (1983) has reported that some stutterer's need more time to coordinate serially ordered events, regardless of whether they involve:- speech or hand coordination.

Regarding the fourth and the most pragmatic question, Gregory (1986) believes that practice in rapid initiation and termination of speech segments, vowels and consonant-vowel combinations should be a part of therapy in both adult and child stutterers. He provides the following sources of evidence to support his belief:

(1) Motor responses improve with practice. Articulatory dynamics seem to improve in an adaptation procedure (Zimmermann & Hanley, 1983).

(2) Stutterers responded to training to perform specific articulatory or phonatory maneuvers equally well as nonstutterers. (Peters and Boves, 1985).

(3) A personal observation that children's ability to sequence sounds more rapidly improves with practice.

(4) Fluency enhancing procedures such as vivid modeling of slowing of speech production showing increased length of segments and smoother blending seem to tackle tha minimal motoric differences of speech mechanism may be due to some general motor nervous system deficit or simply maladaptive learning.

All these studies were conducted to investigate the reaction time of pretreated stutterers. However, there are no specific atudies showing formal post-speech therapy effects on reaction times.

Two interesting hypothesis:

There have been two interesting hypothesis during 1980's. Both require detailed experimentation. They are as follows:

I. Perkins (1986) hypothesized that "what is frightening to stutterers is the temporary feeling of 'loss of control of the flow of speech' and their response to thia feeling largely determines the topography of their stuttering. Essence of stuttering could be the 'experience of momentary loss of speech control'."

II. Parry (1985) proposed the valsalva hypothesis. This is an interesting possibility suggested by a nonprofessional who has experienced stuttering & underwent speech therapy at various centres. His own study of scientific literature and his close association with professionals has enabled him to propose this hypothesis. Certain supporting evidences were collected by Atkins, on his (Parry's) speech mechanism, through fibroscopy. The hypothesis were

(1) "Certain types of stuttering behaviour may be proceeded by excessive 'tuning' of the neural pathways to the muscles of the vaisalva mechanism".

(2) "The tuning of the vaisalva mechanism would increase the excitability of those muscles to stimuli that would trigger a valsalva maneuver (for instance, the abrupt rise in subglottal pressure that occurs when speech is initiated)."

(3) "The triggering impulses might be sent to the muscle groups comprising the valsalva mechanism (as well as to the oral structures that caused the closure of the upper airway) to stimulate their simultaneous constraction so as to continue the increase of subglottal pressure".

(4) "As subglottal pressure rises, the oral or laryngeal structures blocking the upper airways would be stimulated to close with proportionately greater force so as to resist the increased pressure."

From the review, it is apparent that a great deal of research has been conducted on the acoustic and the laryngeal events during stuttering. However, these have been in isolation. In the present study an attempt has been made to study the acoustic and the laryngeal events during stuttering simultaneously. Also the acoustic and the aerodynamic events have been studied simultaneously.

METHODOLOGY

METHODOLOGY

This study was conducted on a 17 year old male subject. He had chronic stuttering, with onset in early childhood. Detailed history-taking was done to collect all the relevant information. Screening tests were done for hearing, language, vital capacity and phonation volume.

Prior to therapy, the speech patterns (Microphone output, Electrolaryngograph output and electroaerometer output) were recorded on magnetic tapes. After this, the subject underwent speech therapy. Following therapy again the speech patterns (Microphone, Electrolaryngograph, Electroaerometer outputs) were recorded on magnetic tapes. Speech initiation time was also measured before and after therapy. Details are described under the following subheadings:

I Subject

II Material

III Procedure

IV Identification and Measurements

SUBJECT

A seventeen year old mala Kannada speaking stutterer was selected for this study. The following information were obtained from an interview with the subject:

1. Mother tongue: Lambadi
   Proficiant in speaking Kannada
2. Occupation: Agriculturist
3. Natal and post natal history: No significant history reported.
4. Scholastic achievement: Studied upto 3rd Standard but cannot read or write in any language.

5. Sibling history: One elder brother and 3 siaters who are reported to be normal
6. Family history: Maternal uncle and his two male children are reported to have stuttering.
7. Onset of the problem: Started stuttering at the age of four years. At this age he started imitating his maternal uncle's speech and his secondary characteristics.
8. Environment: Some of the 'age peers' of the subject, who have imitated his uncle's speech are reported to have stuttering,

His dysfluencies were-

1) intraword audible pause (Inhalatory noise)
2) intraword silent pause,
3) whole word repetition,
4) syllable repetition,
5) intrasyllabic repetition,
6) phoneme prolongation,
7) phoneme repetition and
8) phoneme interjection.

He exhibited tension of laryngeal muscles and eye twitching as secondaries. On screening tests his hearing, language, vital capacity and phonation duration were found to be within normal limits.

MATERIAL

I. Dysfluent utterances were elicited in four types of speech tasks.

1) Spontaneous speech.

The experimenter engaged in a general conversation with the subject and enquired about his food habits, family, occupation, festivals, village, etc.

2) Spontaneous picture discription.

Picture charts depicting short stories were used to elicit speech.

3) word repetition.

The words in the Kannada articulation test were used.

4) Sentence repetition.

Recorded sentences including declarative, interrogative and exclamatory types were employed for this task.

II. A list of 'difficult-to-utter' words was prepared on the basis of the subject's report. This list was used in speech initiation time (SIT) measurements.

PROCEDURE

I. There were three sessions:

1. Speech and Electrolaryngographic recordings

In the first session the subject was seated on a chair and a microphone (Unidynamic Ahuja AMD 535 MS) was placed 10 cms away from the subject's mouth. Two spherical electrodes of the Electrolaryngograph (Kay elemetrics 80138) were positipned on the thyroid cartilage of the subject. The Microphone was connected to the internal tape recorder of the sound spectrograph (VII 700). The output of the Electrolaryngograph was connected to a cassette deck recorder. Spontaneous speech was elicited by conversaticn and the subject was instructed to describe tha story, repeat the words and sentences as uttered by a Kannada speaker. Thus simultaneous speech and Electrclaryngographic signals were recorded on high fidelity magnetic tapes.

2. Speech and mouth_expiration recording

In the second session the subject was seated on a chair and held the mask of the Electroaerometer (F.J. Electronics Type EA 510/4) tightly to his face. The microphone output of the Electroaerometer was fed to the internal taperecorder of the Sound Spectrograph. Tha output of the mouth expiration was fed to a cassette deck recorder. Speech samples were elicited in the same way as in sesaion one. Thus simultaneous recordings of speech and expiratory mouth airflow were obtained.

3. Speech recording for SIT

The experimenter uttered the words one each at a time. The subject was instructed to close his eyes and listen carefully to the word and to utter the same as soon as he heard a tap sound. The tap sound and the subject's utterances were recorded on a cassette tape.

All these recordings were obtained before and after therapy using the same material.

Wideband bar type pf spectrograms along with Average amplitude functions were obtained only for the pretherapy nonfluent words. Similar type of spectrograms were taken for the same post therapy fluent words. Laryngograms and airflow wave analysis of only those words, which depicted abnormal air-flow and vocal fold vibrations on the spectrcgrams, were obtained. The high resolution signal analyser (B&K type 2033) was used as a video display unit and the cassette deck recorder was connected to it. The laryngograms and the airflow waves were analysed after displaying it on the HRsA. The spectrograms, laryngeal waves and the airflows of the nonfluent words (pretherapy) and the fluent words (post therapy) were analysed and compared. To measure speech initiation times the recorded samples were displayed on the screen of high resolution signal analyser. The SIT's before and after therapy were recorded and compared.

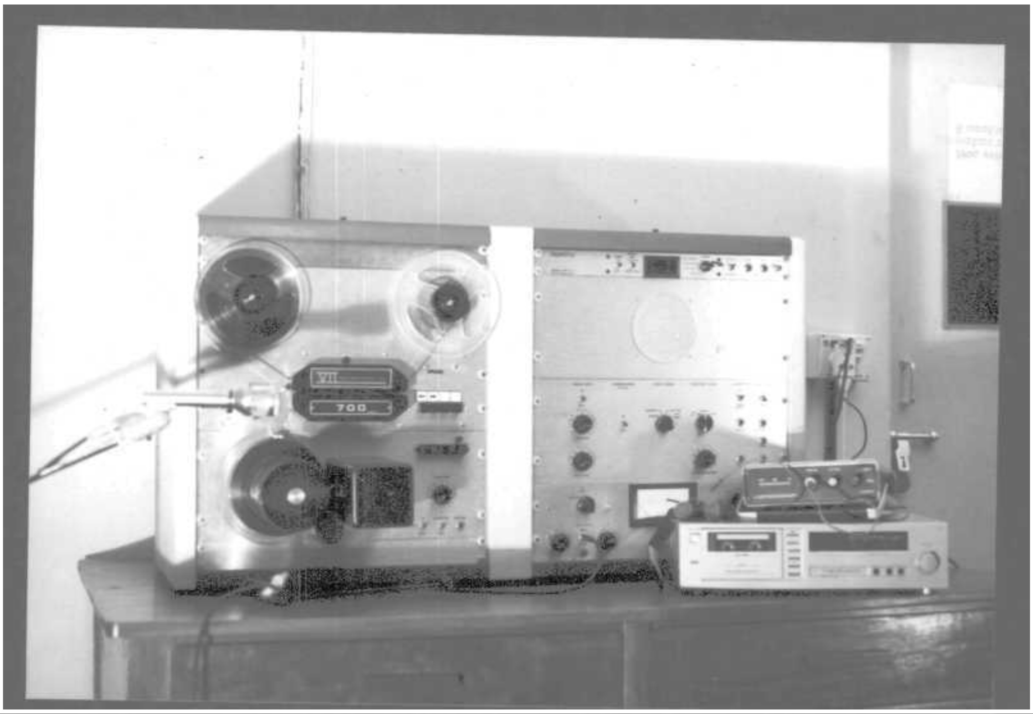


fig (1) Microphone, spectrograph, electrolaryngograph and cassette deck recorde

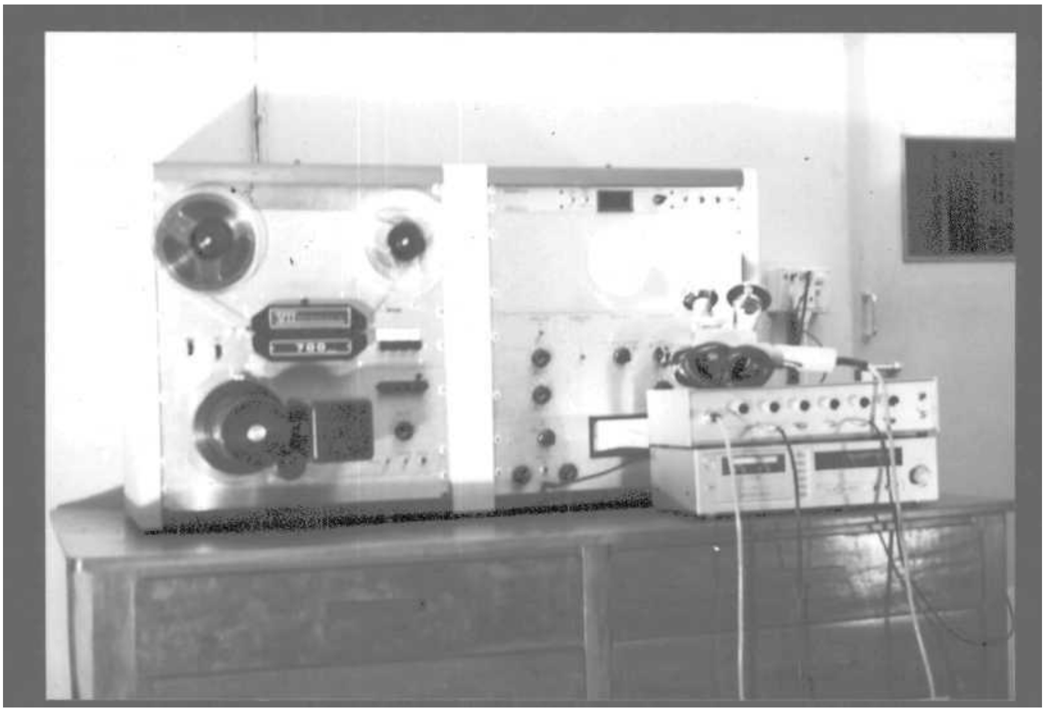


fig (2) Spectrograph, Electroaerometer (with Mask) and cassette deck recorder

fig (3) Cassetta deck recorder and high resolution signal analyser

II. Description of speech therapy

The subject attended a total of 23 sessions. Each session lasted for 45 minutes. The experimenter instructed the subject to alter certain covert cognitive patterns and overt speech patterns. Secondary behaviours were reduced by increasing his self-awareness about them. Desired speech patterns were modelled by the experimenter. The experimenter also gave an unconditional positive regard and interacted with the subject outside the clinic.

1. Cognitive patterns

The subject was instructed (i) to accept the speech problem and to acknowledge about it, when people enquired, without developing any negative covert feelings,

(ii) to look at the listner and speak and

(iii) to suggest himself that he was speaking well and that everything was fine.

He was also instructed

i) not to think about himself as a parson with a speech problem,

ii) not to think of dysfluencies that might occur and how to speak certain feared words, while speaking,

iii) not to develop fears, anxieties or inhibitions to speak and not to remember unrelated thoughts, events or previous unpleasant circumstances,

iv) not to think about what the listner(s) might feel about him when he is dysfluent and

v) not to consider tha dysfluancies as the signs of his inability and not to develop negative feelings, when they occur.

2. Speech patterns

1) Initially the subject was instructed to name the objects, people, animals in single words. He was instructed to prolong the initial part of

each word. Also while speaking he was to keep his hand on his neck for monitoring unnecessary muscle tensions. He was explained the difference of making hard and soft articulatory contacts. Ha was encouraged to detect hard articulatory contacts through self-monitoring. When he detected abnormal muscle tensions in the neck or any instance of hard articulatory contact he was advised to stop speaking and out a tick mark on a white paper, to 'hight light' this instance. When he did so he was stopped and was instructed to inhale air into lungs, slowly exhale it out in an audible /h/ sound and utter the dysfluent word slowly.

2) Following this he was instructed to speak a two or three word sentence about each picture shown to him. No specific attempts to make him prolong were made at this instant. However, a card with a horizontal arrow printed on it was always fixed on a table kept in front of the subject to remind him to speak slowly. His family members were also instructed to speak with slower rates and to remind the subject to speak slowly when he spoke fast. When dysfluencies occurred, he was to stop, inhale air, exhale it in a /h/ uttering the dysfluent word slowly with soft articulatory contact. This was done at all the stages of therapy - whenever dysfluency occured. He was also encouraged to utter sentences with prosodic variations. Prosodic variations and soft contacts were also symbolized on the 'card' to remind him. He was encouraged to carry this card with him all the time. When the subject was fluent in the clinic he was taken outside and was instructed to speak simple sentences about various things in the environment.

3) At this stage he was instructed to narrate stories, converse with one person at a time and speak in a dialogue (in a mono act) and to converse in a group of people.

4) When the subject was fluent in the above activities 'tranafer' phase was carried out. The experimenter accompanied the subject to real life situations uhare the subject made enquiries with strangers or purchased items in shops;using hand to self-monitor and practicing dysfluent words with airflow procedure were encouraged in real life situations.

5) The subject uent to his village and tried to transfer his fluency. He came back after 15 days and reported that at his village he was as fluent as he was in the clinic.

However, though his nonfluencies were reduced, speech did not appear perfectly normal perceptually.

IDENTIFICATION AND MEASUREMENTS

On the wideband spectrograms speech sounds were identified on the basis of the descriptions given by Pattor, Kopp and Green (1947) and various measurements were done as follows:

1. Speech sound duration: Tha procedures given by House & Fairbanks (1953) and Peterson & Lehiste (1960) were followed in the measurement of the durations of speech sounds.
2. Formant frequency: The dark bands on the wideband spectrograms were identified as the formants and a horizontal line across the formant was drawn. The frequency of the midpoint of these were considered as formant. frequencies. The first darkband from the base was considered as the first formant F1 and its frequency as F_1 and the second darkband was considered as the second formant F2 and its frequency as F_2 and so on.

3. Speed of transition: The frequency in the beginning and the end of the transitions was measured. The duration of this was also treasured. Tha difference between these frequencies were divided by the duration to calculate the speed of transition.

4. Soeech initiation time: The time duration between the tap sound and the onset of speech was considered as SIT.

5. Speed quotient: It was calculated as the ratio of the opening phase duration to the closing phase duration in the glottogram.

All these measurements were done and the data thus obtained was tabulated.

RESULTS & DISCUSSIONS

RESULTS AND DISCUSSION

Totally 29 dysfluent words were analysed. These dysfluent words were studied and were compared with the post therapy utterances (same words elicited after therapy).

The dysfluencies were of 5 types.

I Intraword audible pause (Inhalatory noise)
(12 words)

II Intraword silent pause
(2 words)

III Whole word repetition
(1 word)

IV Intraword repetition
(8 words)

V Phoneme prolongation and repetition
(6 words)

For all the words pre and post therapy spectrograms are shown. For 5 words abnormal Electrolaryngographic recordings and for 2 words abnormal electroaerometric recordings are shown. For each word the major pre and post therapy differences are summarized in a table. For convenience of description the pre-therapy utterance was divided into areas labelled (1),(2),(3) etc.

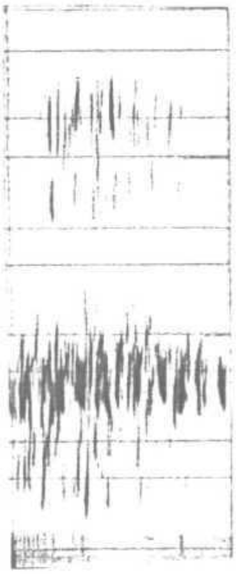
1. Intraword audible pause (Inhalatory friction)

In this type of dysfluency it was observed that the initial one or two phonemes of the words were produced. Following this was a brief inhalation of air resulting in an aperiodic friction noise. This aperiodic friction noise was accompanied by a few vocal cord vibrations in some instances. whenever present the vibrations were not distributed over the entire friction duration but were intermittent. This (sound syllable + friction noise) was repeated until the word was produced finally. To check whether this friction noise was generated during inspiration or expiration, two words - one with intraword audible inhalation and the other with intraword audible exhalation, were recorded and wideband spectrograms were obtained. It was observed that the spactrgrams of the friction noise and the intraword audible inhalation were similar. Further evidence for the friction noise during inhalation came from the analysis of expiratory airflow recordings. while the spectrograms depicted the friction noise, the waveforms of the mouth expiration was absent indicating that the event was taking place during inhalation. Intraword inhalatory friction indicates a sudden reflexive contraction of respiratory muscles that act on lungs in such a way as to cause a rapid inhalation. The sudden gush of air through the constricted glottis causes friction noise.

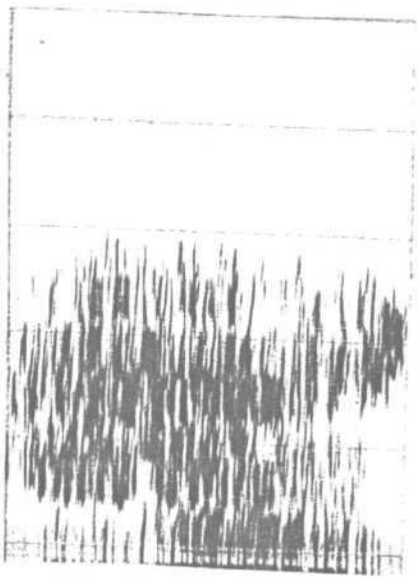
Dysfluent words under this category were as follows:

1. Words starting uith a vowel
   1) /aṣṭe/
   2) /aivattu/
2. Words starting with a plosive
   1) /bartāide/
   2) /ḍisṭicṭalla/
   3) /dipāvaḷige/
3. Words starting with an afflicate
   1) /jana/
   2) /jagaḷa/
4. Words starting with a nasal continuant
   1) /māḍide/
   2) /nōḍilla/
   3) /nōḍidivi/

Intraword Audible Inhalation



Intraword Audible Exhalation



5. Words starting With a trill

1) /rājamma/

6. Words starting with a fricative

1)/Sarṭu/

In all these inhalatory friction is denoted by the symbole 'If'.

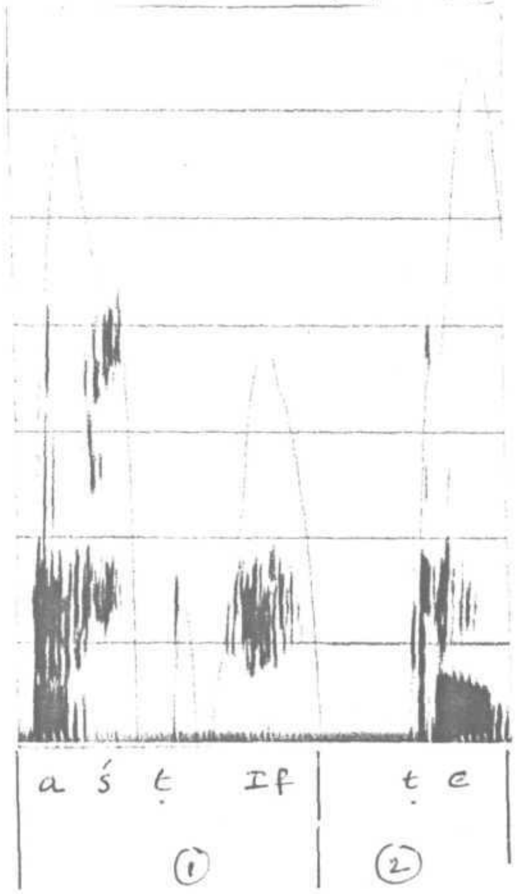
1) /aṣṭe/ (aṣṭ If ṭe)

Speech Sound	Duration in Pre-therapy utterance	Duration in Post-therapy utterance
/a/	50	135
/s/	40	50
t (i)	50 no aspi-ration	80 20 ms of aspiration
'If' .	65	-
/e/	65	70

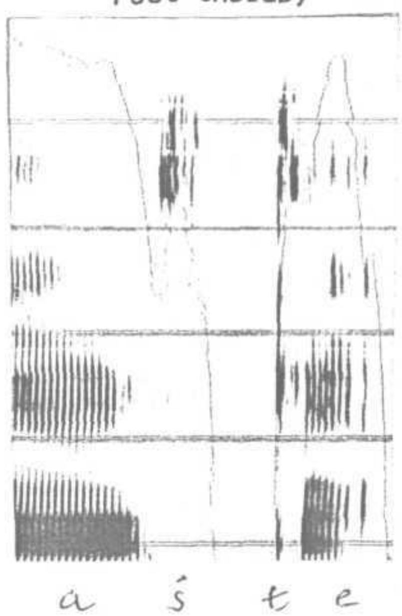
Table 2: Pre and Post Therapy durations of the segments in the word /aṣṭe/ (in m.secs).

The data showed that the duration of segments was short in pre-therapy utterance.

Pretherapy /aṣṭe/



Post therapy



2) /aivattu/ (ai If aivattu)

Speech Sound	Speed of transition in Pre-therapy utterance	Post-therapy utterance
/ai/ (1)	F_2 rised at a rate of 6.4 hz/ms	F_2 rised at a rate of 8 hz/ms and fell at a rate of 11.43 hz/ms
'If'	180 ms	-
/ai/ (2)	F_2 rised at a rate of 5 hz/ms and fell at a rate of 9.3 hz/ms	

Table 3: Speed of F_2 transitions in the word /aivattu/ (in hz/m.sec)

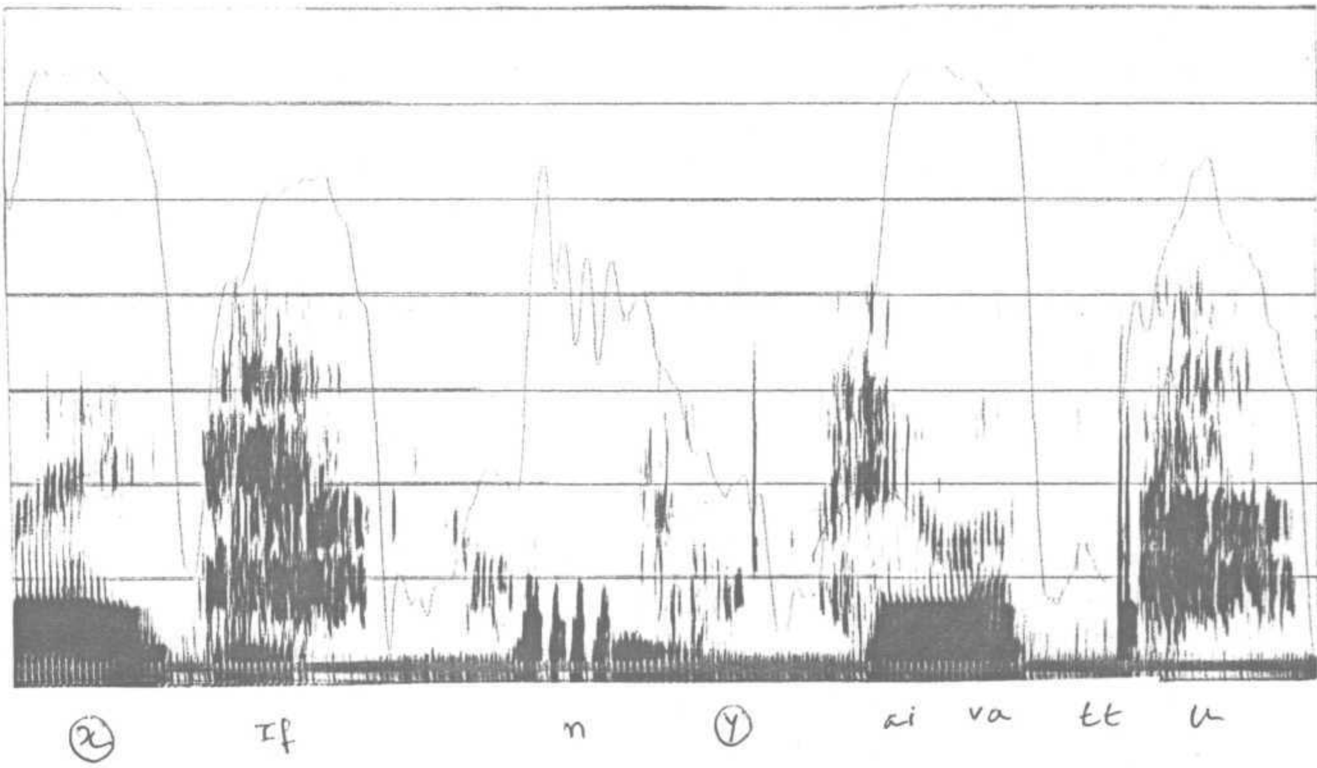
i) In the pre-therapy utterance there were two attempts (Marked x & y) to produce /ai/. In both, falling F_2 transitions were absent. Also the F_2 transition speeds were different in the fluent pretherapy utterance and fluent post therapy utterance.

Absnece of an appropriate F_2 transition (indicating inability of the tongue to move to the next segment) seemed to trigger an inhalatory /friction.

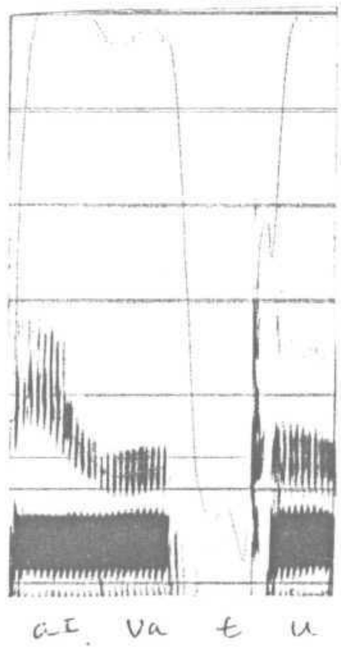
ii) Region marked 'n' showed intermittent vocal cord vibrations but no energy of higher frequencies, possibly indicating an abnormal positioning of supraglottal articulators.

From this, it may be speculated that when the required movement of the articulator is absent and is fed back to the CNS, the CNS may issue a random surge of impulses to various speech muscles which might result in an inspiratory friction, intermittent vocal cord activity

Pretherapy /aivattu/



Post therapy



and abnormal supraglottal positioning.

2. /bartaide/ (ba If bartaide)

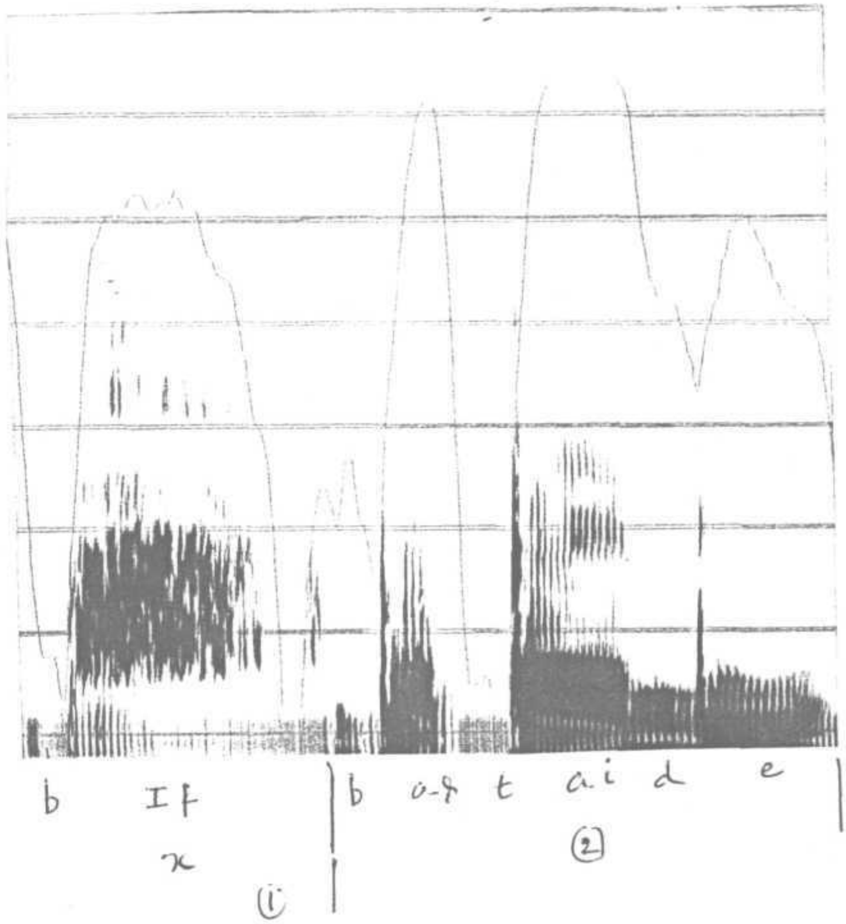
Speech sound	Duration in Pre-therapy utterance	Duration in Post-therapy utterance
/b/	60 (1) 55 (2)	30
If	140. This was accompanied by vocal cord vibrations	–
/a/	55	50
/r/	20	20

Table 4: Durations of the segments in the word /bartaide/ (in m.secs)

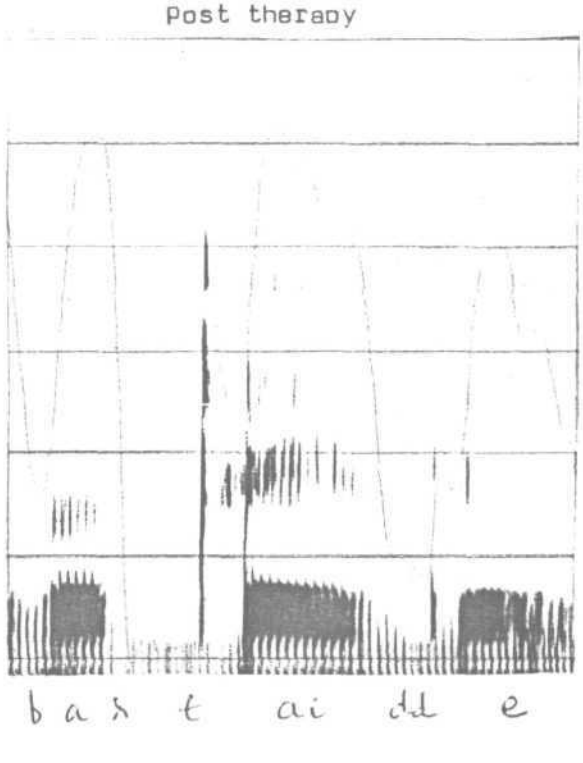
S.Q. of cycles during If in region x	S.Q. of cycles in /a/ in the syllable /bar/ in fluent pre-therapy utterance	S.Q. of cycles in /a/ in the syllable /bar/ in fluent post therapy utterance
1.0	0.8	0.8 k = 3.3.
1.1	0.8	0.9 k = 3.5
1.2	0.9	0.9 k = 3.3
1.5		0.9 k = 3.7
1.8		0.9 k = 3.3
1.2		0.9 k = 3.5
1.5		0.8 k = 3.7
1.2		

Table 5: Speed Quotients of different segments

Pretherapy /baɣtaide/



Post therapy



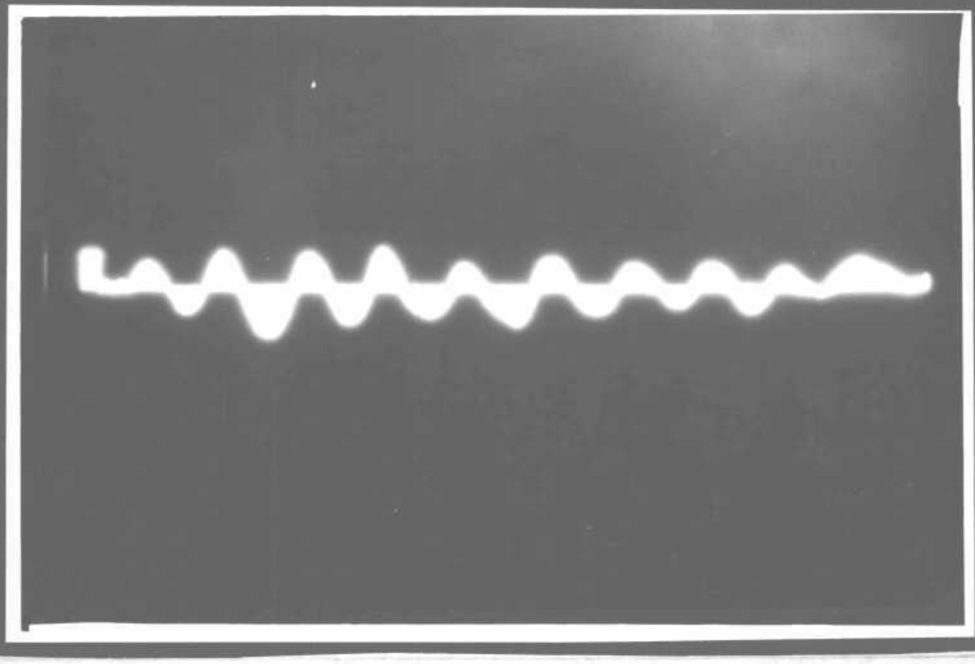


fig (4) Bartaide:Vocal fold (Lx) cyclas during Insoiratory frication ('If') at region)*

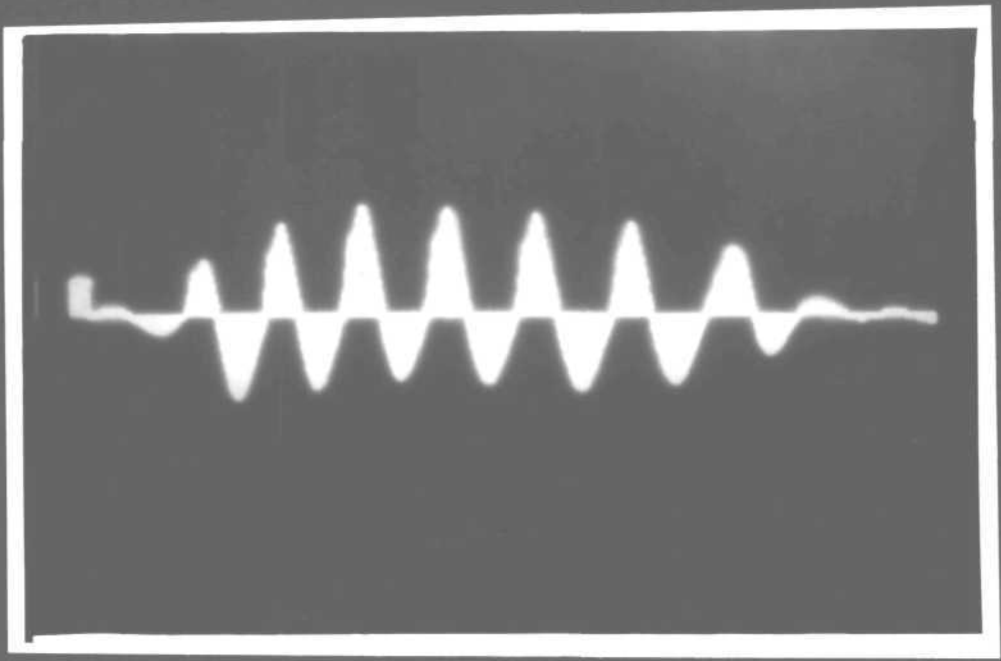


fig (S) BartaidmL* cycles of /a/ in syllabla /bar/ in Pretheraoy utterance
ore

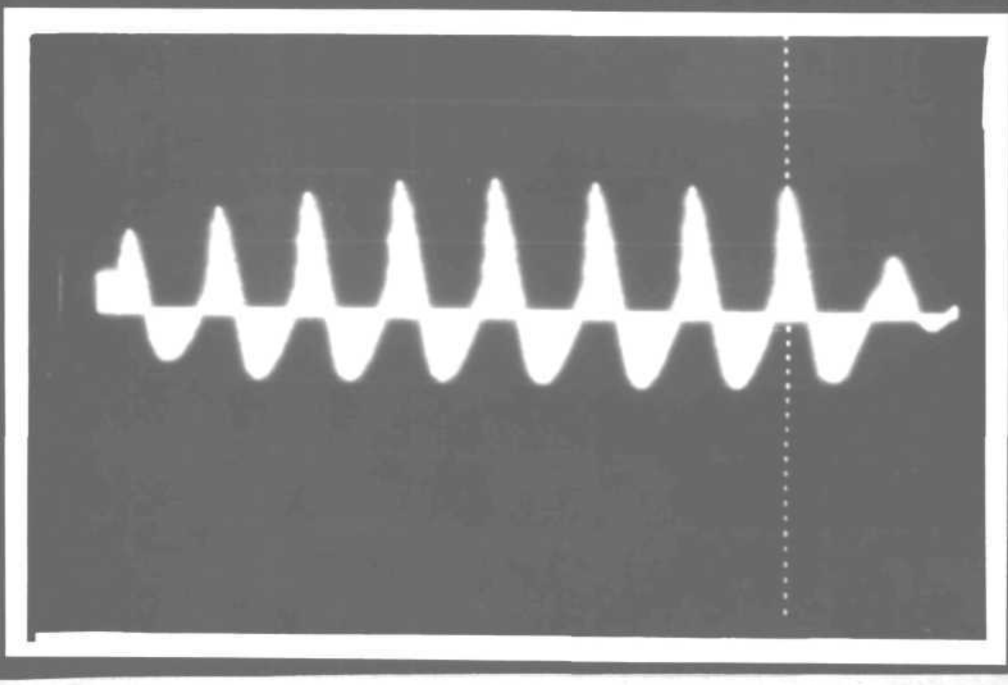


fig (5) Post Bartaide:Lx cycles of /a/ in syllable /bar in Post therapy utterance.

(Note: K refers to the time (Re: beginning of closing phase) at which a kneepoint occurs in closing phase).

In the pretherapy utterance the duration of /b/ was longer but the durations of /a/ and /r/ were almost same aa in the post therapy utterance.

Tha S.Q. of the vocal fold vibrations during Insoiratory friction varied randomly. 2 cycles showed a peculiar stairstap pattarn. (see fig.4)

S.Q. was invariant and closing phase was longer than opening phase in /a/ of syllable /bar/in both fluent pretherapy and post therapy utterances.

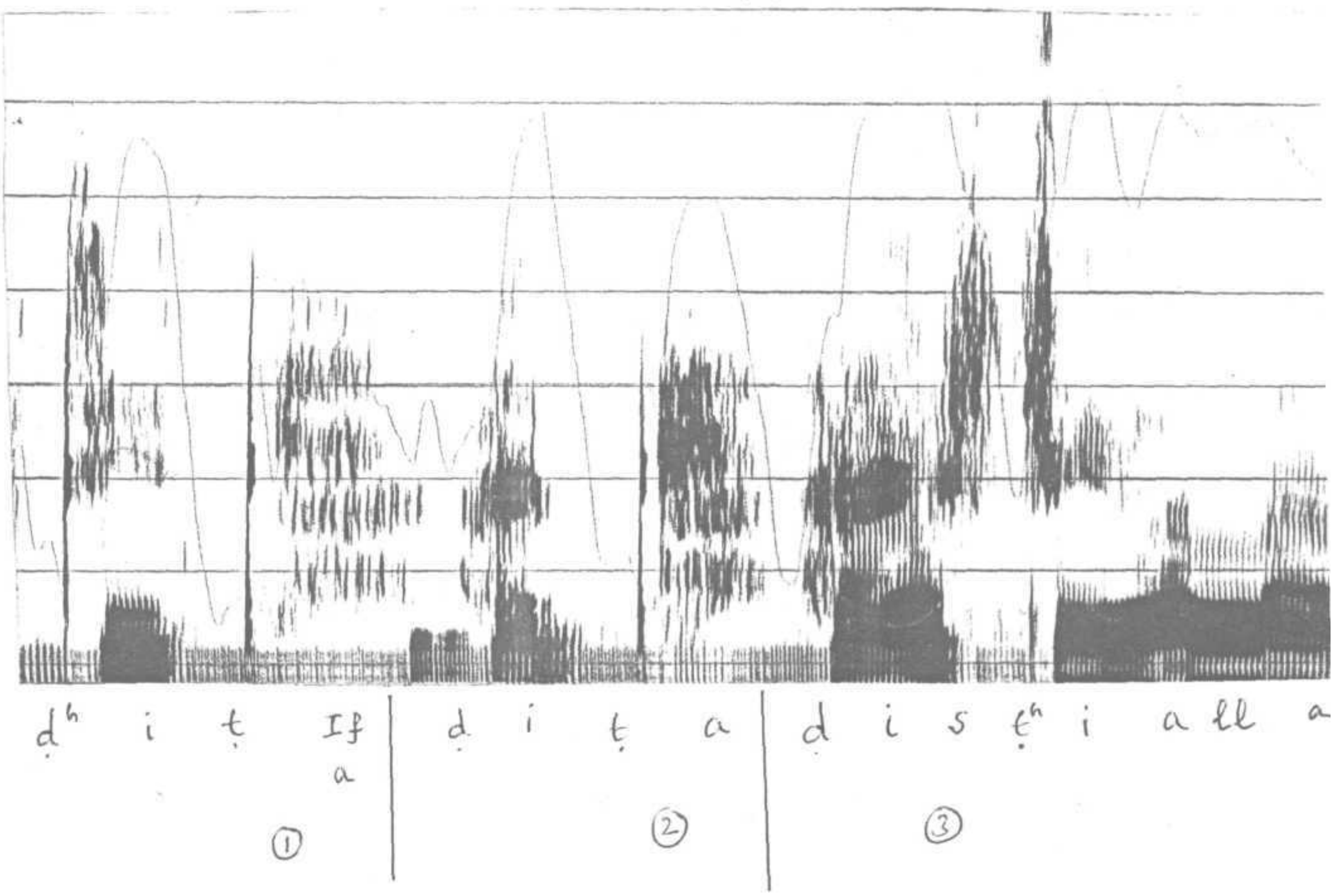
However, only post therapy cycles shouted clear kneepoints in the closing phase. On the average kneepoint occurred 3.5 ms after the beginning of closing phase (see fig.5)

2. /distiktalla/ (di If distiktalla)

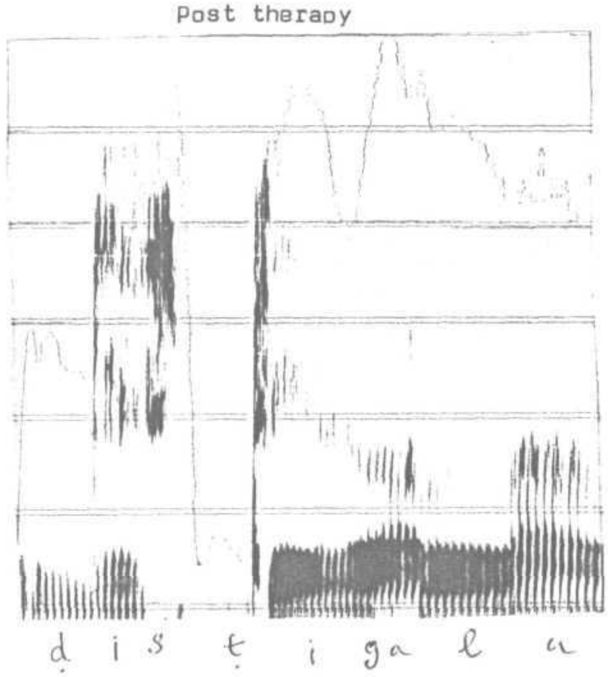
	Speed of transition in	
	Pre-therapy Utterance	Post-therapy Utterance
F_2 transition between /d/ & /i/	rise at a rate of 8.9 hz/ms (1) absent (2) rises at a rate of 4.3hz/ms (3)	falls at a rate of 33.3 hz/ms
F_2 transition from /t/ to/i/	absent (1) & (2) rises at a rate of 20 hz/ms (3)	rises at rate of 40 hz/ms

Table 6: Speed of transitions at different segment junctions in the word /distiktalla/ (in Hz/m.sec.)

Pretherapy /ḍistikṭalla/



Post therapy



In the pretherapy utterance F_2 transitions were absent or slower. Sometimes they were in opposite direction. Also /s/ was omitted in the pretherapy utterance.

Steady formantic during the inhalatory friction indicated an articulatory fixation at that moment.

3) /dipavalige/ (di If If di dipavalige)

	Pre-therapy	Post-therapy
duration of /d/	15 (1) 100 (3)	50
duration of /i/	210 (1) 100 (2)	90
F_2 transition from /i/ to /p/	absent (1) falls at a rate of 2 hz/ ms(3)	rises at a rate of 5.7 hz/ms
If	130 (x) 260 (y)	--

Table 7: Duration and soeed of transition in the word /dipavalige/ (in m.sec and Hz/m.secs respectively).

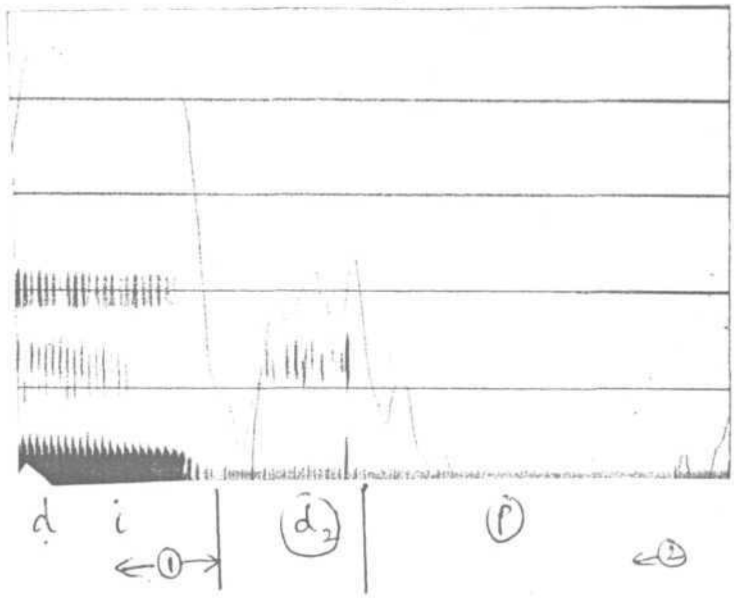
In the ore-therapy utterance the durations of segments were shorter /d/ or longer /i/ compared to those of the post-therapy utterance.

F_2 transition from /i/ to /p/ was absent or slower and opposite in direction in the pre-therapy utterance.

A steady F_2 of /i/ indicating prolonged articulatory gesture for /i/ seemed to trigger a set of unnecessary events including two inhalatory frications, strong and weak vocal fold activity coupled with abnormal positioning of the articulators. The possibility of a supraglottal constriction was evident by the absence of any wave in the air flou recordings corresponding to the area 'b'.

Airflow recording corresponding to region 'c' also showed absence of any kind of airflow waveform. This mas interesting because region 'c' unlike region 'b' showed some formants. It appears that the filter characteristics of the supraglottal cavity may be different at different instants. It is also possible that different types of constrictions may be occuring at different instants. Further, some irregular expiratory air flow waveforms were observed in regions d_1 and d_2 indicating the articulator may be released intermittently. Airflow recordings during (x, y), (c), (d_1 and d_2) are shown in figures.

Pretherapy /dīpavaḷige/



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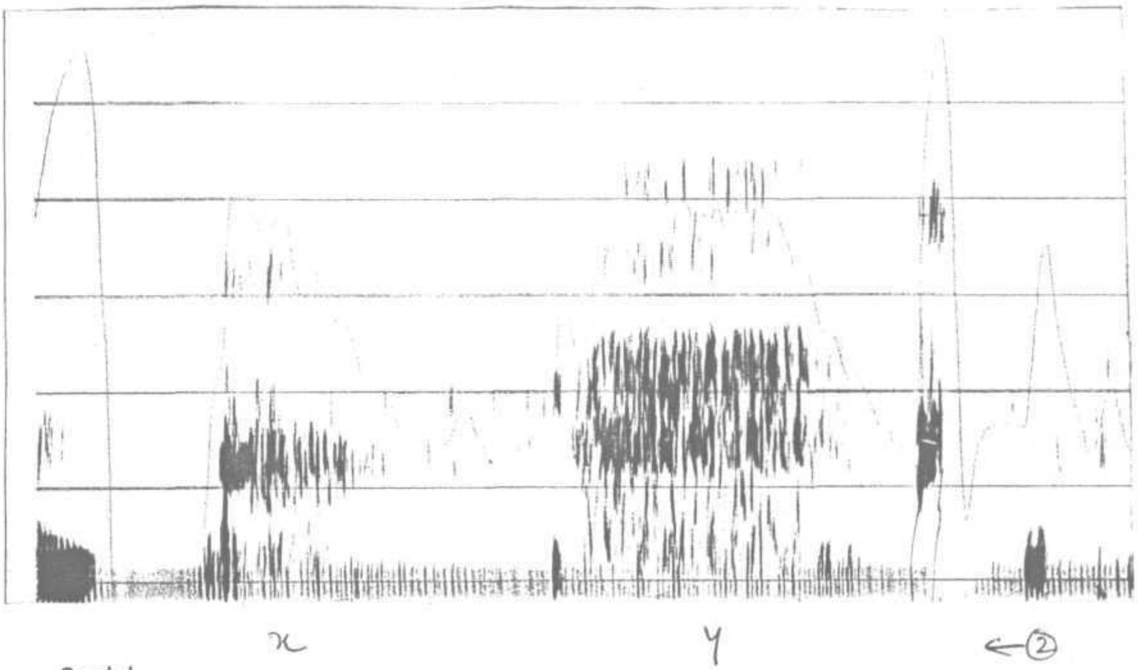

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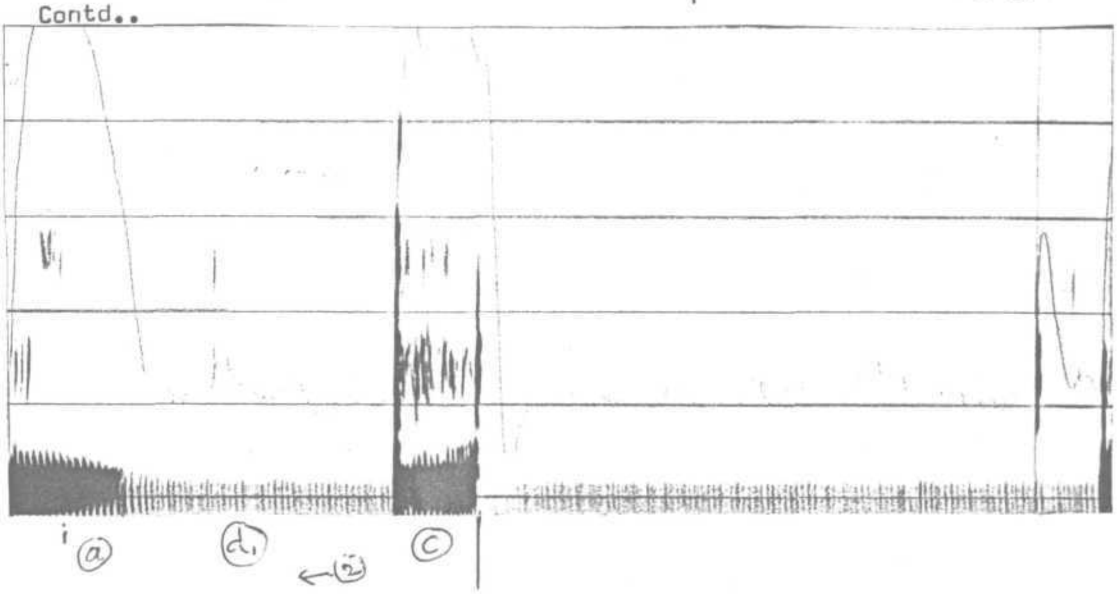


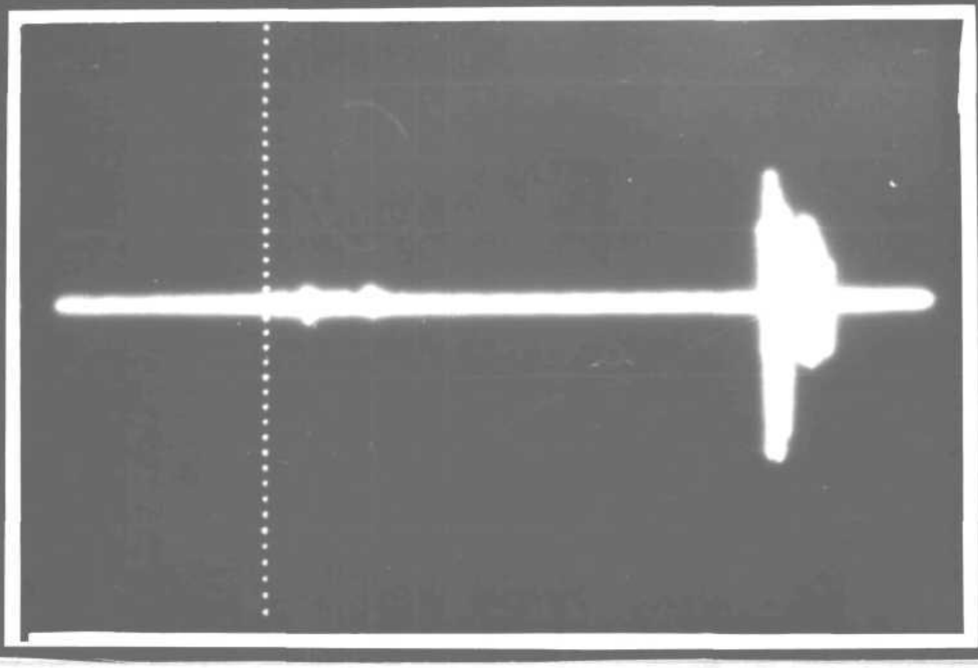


fig (6) dipavalige: Mouth expiratory airflow waveform (MEAW) at region x & y.

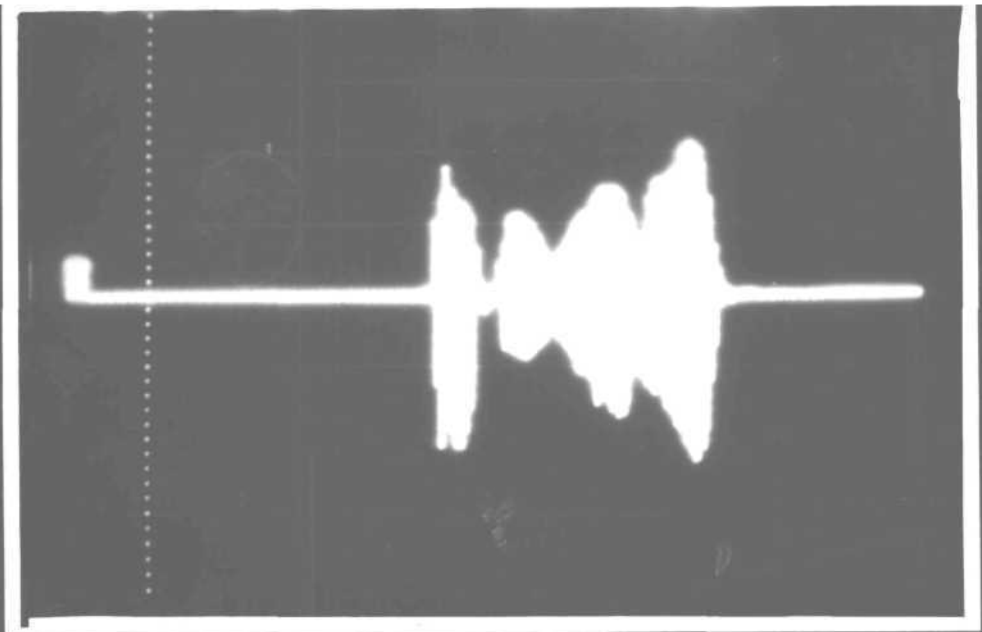


fig (7) dipavalige: MEAW at region b

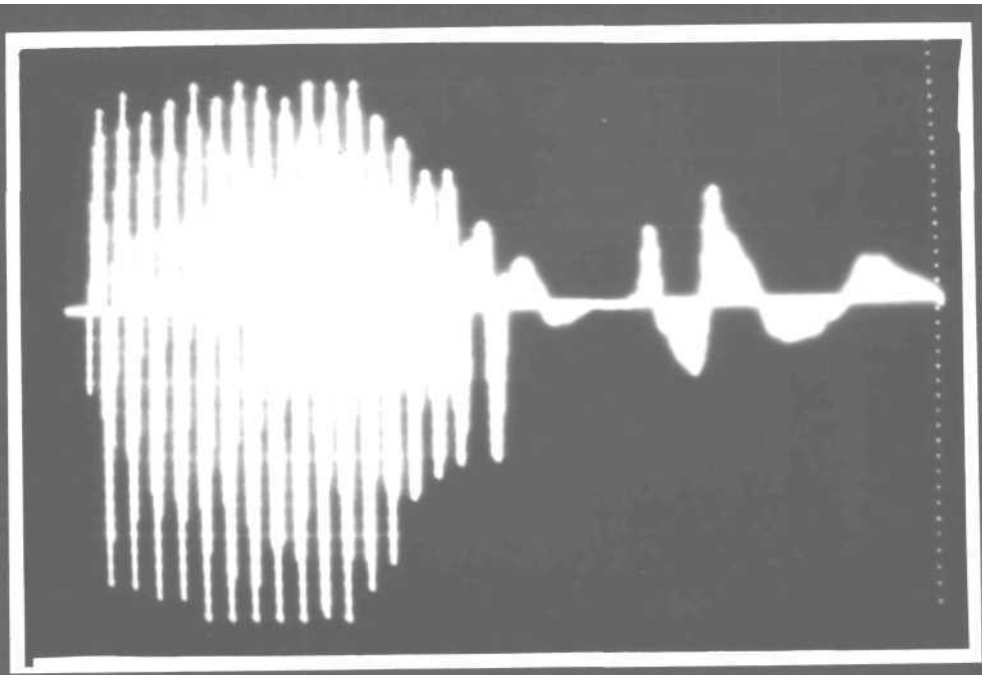
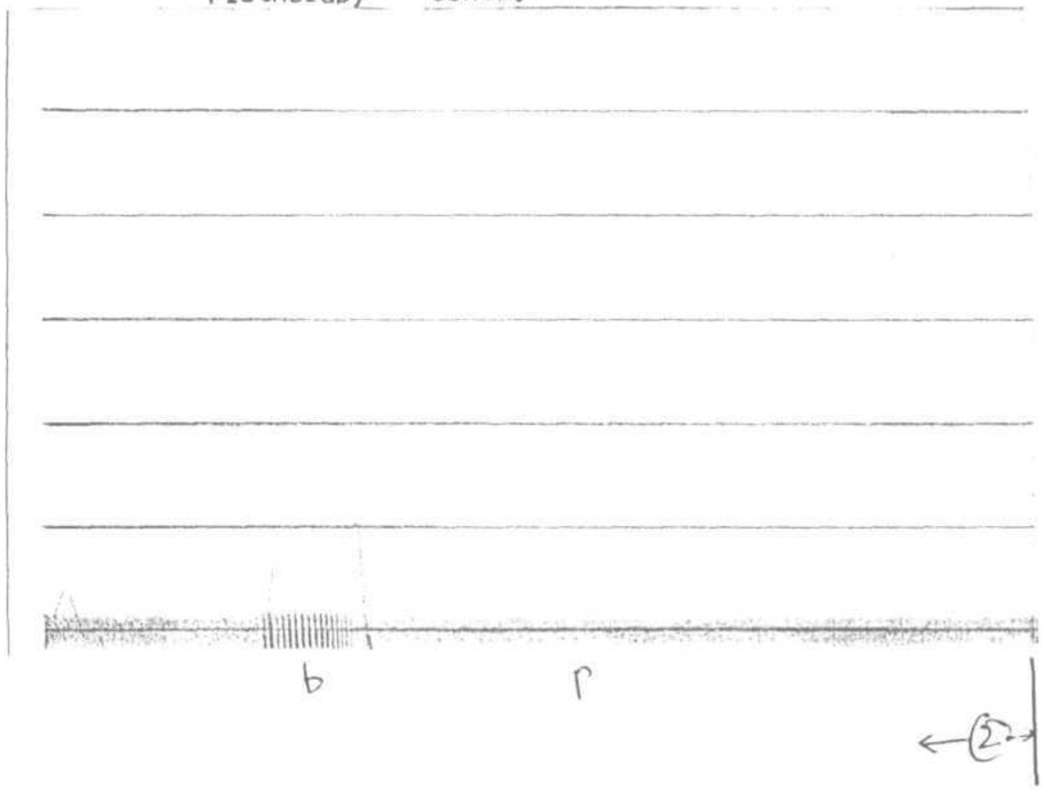


fig (10) dioavalige: MEAM at region d_2

Pretherapy contd.



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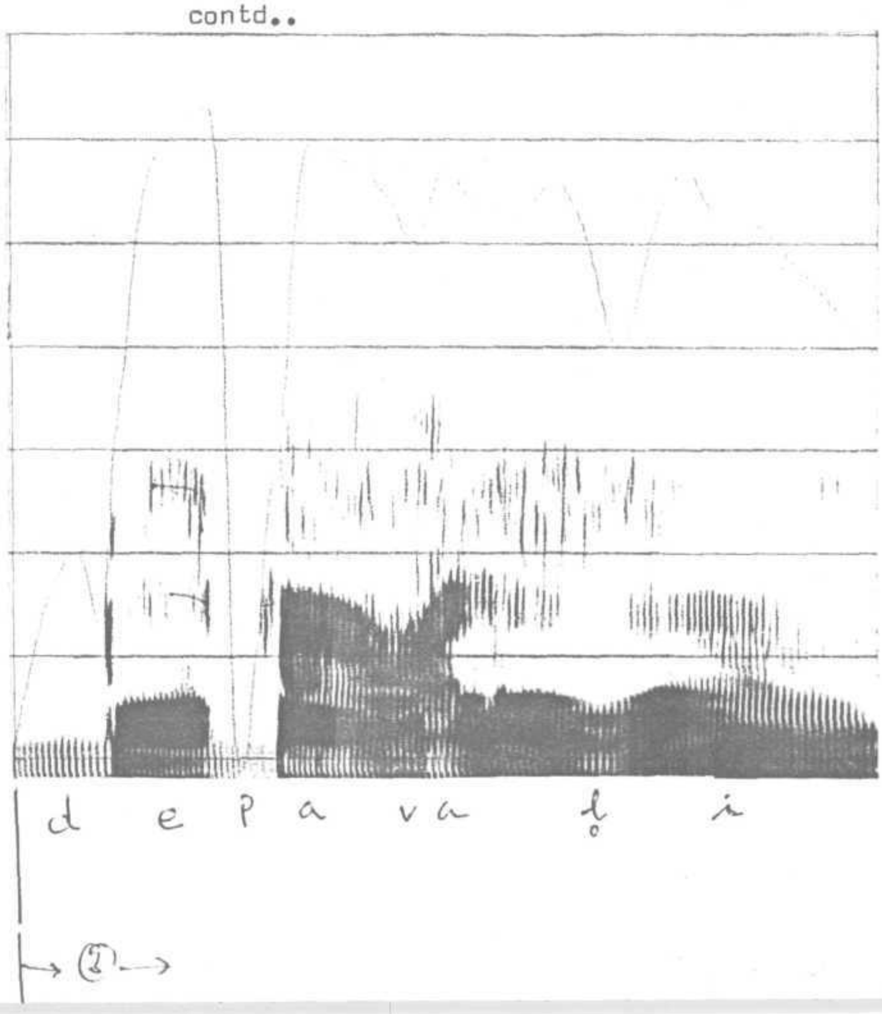
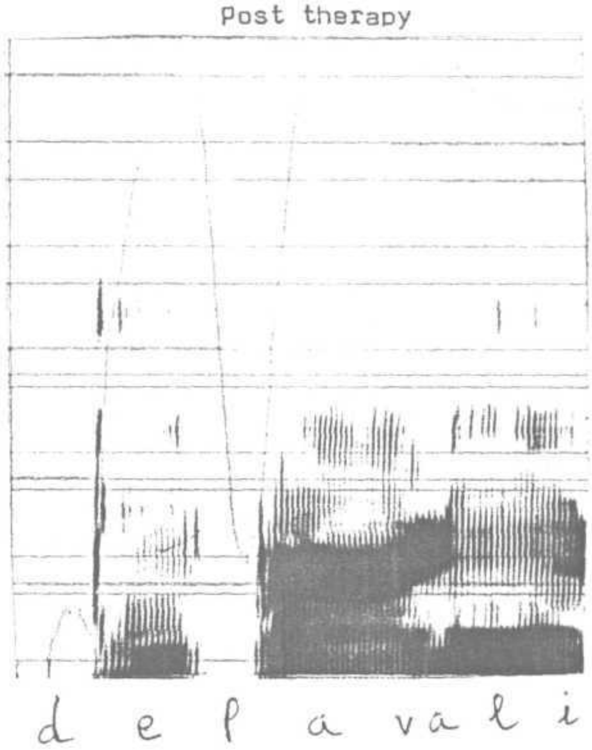


Post therapy



3.

1) /jana/ (If j If j If j If ... jana)

	Pre-therapy	Post-therapy
duration of / j /	30 (2)	50
F- transition from / j / to /a/	absent (x) Absent (y) falls at a speed of 25 hz/ms (2)	
duration of /a/	20 (2)	50

Table 8: Durations and speed of transitions in the word /jana/ (in m.secs & Hz/m.sec respectively).

The pre-therapy utterance started with a inspiratory friction and was followed by several attempts to utter / j / . However the frictions in the supraglottal region were not accompanied by voicing.

Similar resonance patterns in all the inspiratory frications perhaps indicates that the tongue assumes the same position after each unsuccessful attempt of / j / .

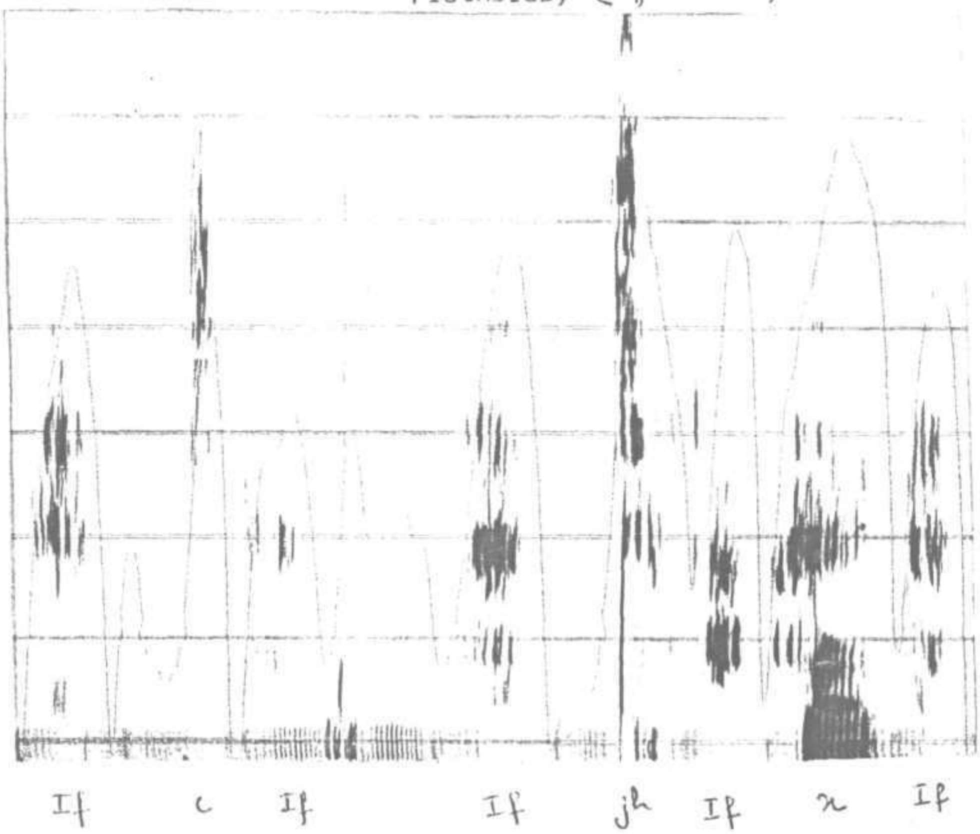
F_2 transitions were absent in nonfluent pre-therapy utterances.

2) /jagala/ (j If If If j If If If jagada)

The pre-therapy utterance showed repeated 'inspiratory frication' and unsuccessful attempts to say / j / accompanied with an absence of vocal fold vibration proceeding affrication.

F- transition from / j / to /a/ was absent in the pre-therapy utterance, whereas in the post therapy utterance it is falling at a rate of 4.6 hz/ms.

Pretherapy (jana)



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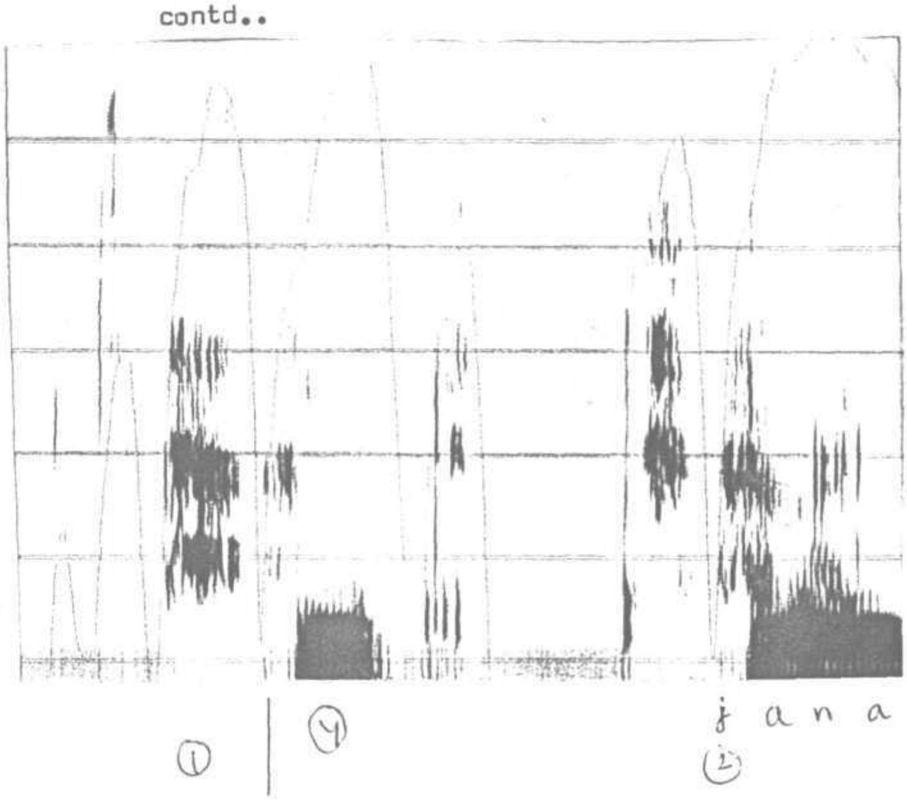
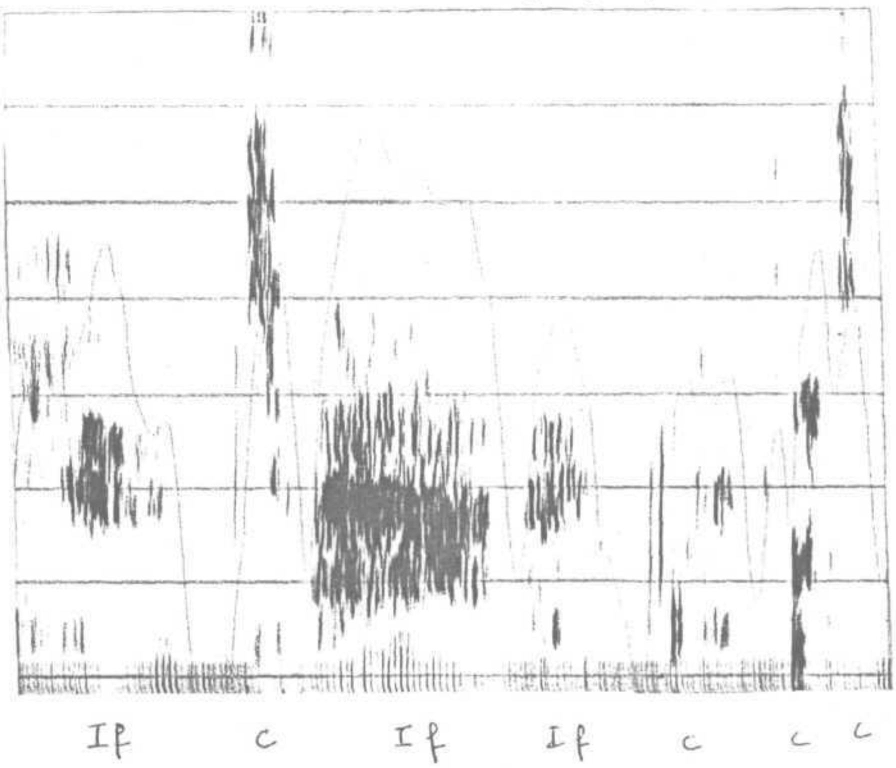


Note: Post therapy spectrogram not presented.

Pretherapy (ja-gaḍa)



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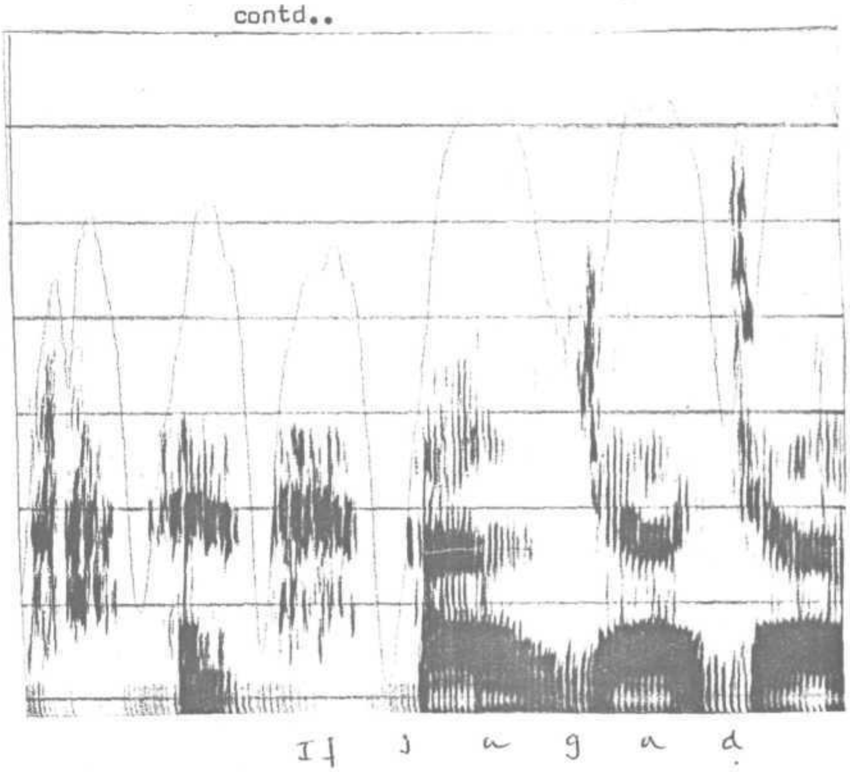
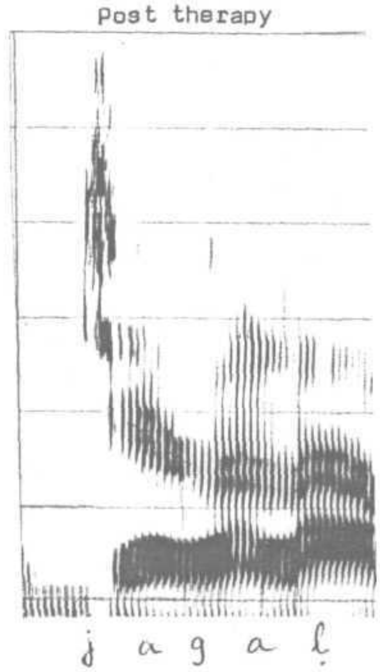


Post therapy



4.

1) /māḍidə/ (mā If If māḍi māḍidə)

	Pre-therapy	Post-therapy
duration of /m/	70 (1) 20 (2) 80 (3)	70
duration of /a/	110 (1) 30 (2) 30 (3)	65
F_2 transition from /a/ to /d/	6.7 hz/ms (1) absent (2) 17.5hz/ms (3)	
If	155ms & 95 ms (1)	
duration if ḍ	35 ms (2) 30 (3)	

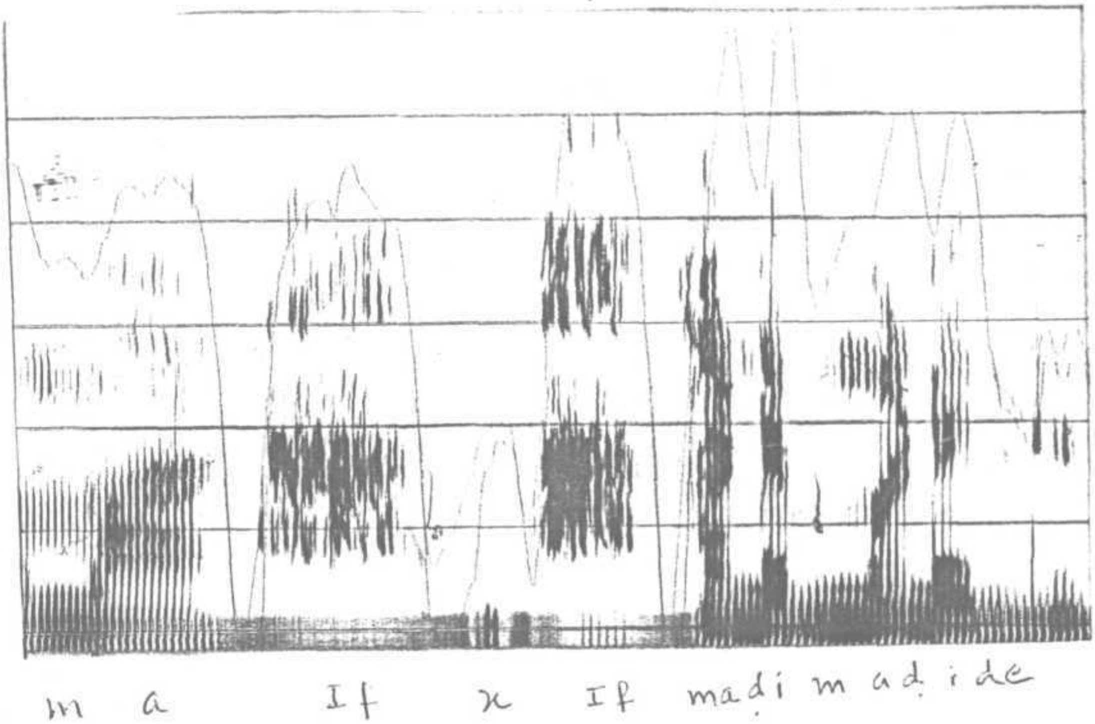
Table 9: Durations and speed of transitions in the word /madide/ (in m.secs and Hz/m.secs respectively).

In the pre-therapy utterance the durations of some segements were longer (/m/ & /a/), duration of /d/ was shorter and for a given segment the duration varied with in the nonfluent utterance.

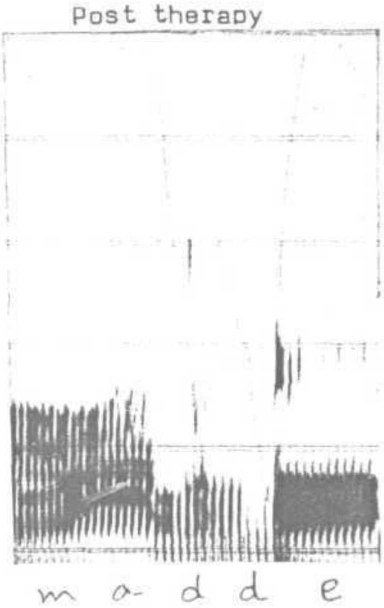
F_2 transitions had higher speed in the pre-therapy utterance.

The subregion 'x' showed strong periodic vocal cord activity for 20 ms higher formants were not visible at this region.

Pretherapy /māḍide/



Post therapy



Inappropriate (absent) F_2 transition indicating improper articulatory positioning seemed to trigger inhalatory friction and abnormal vocal cord activity (region x).

2) /nōḍilla/ (If o If If ho If nhōḍilla)

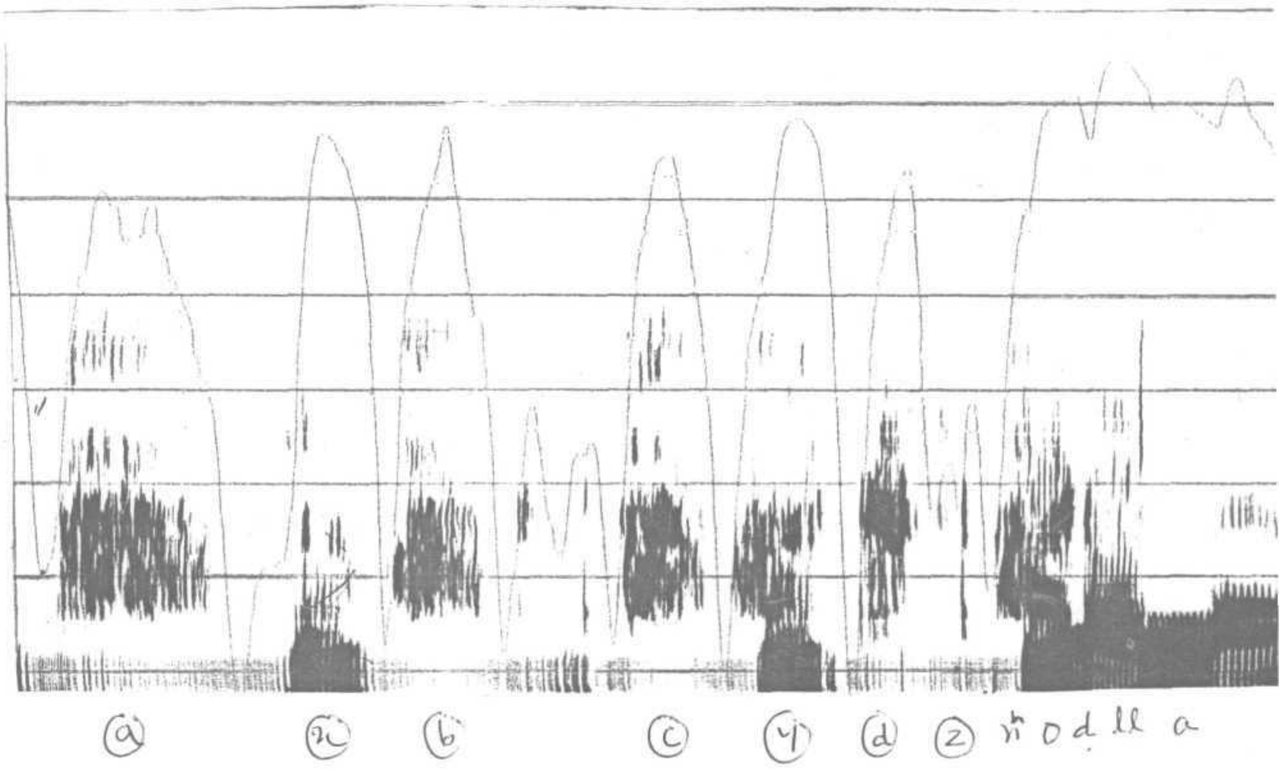
	P1re-therapy	Post-therapy
duration of /n/	/n/ was absent (1) 40 (2)	40
duration of 'If	150 (a) 100 (b) 90 (c) 50 (d)	
F_2 transition /o/ to /d/	6.7 hz/ms (x) absent (y) rising (z)	rising at a rate of 10 hz/ms

Table 10: Durations and transition speeds in the word /nodilla/ (in m.secs & Hz/m.secs respectively)

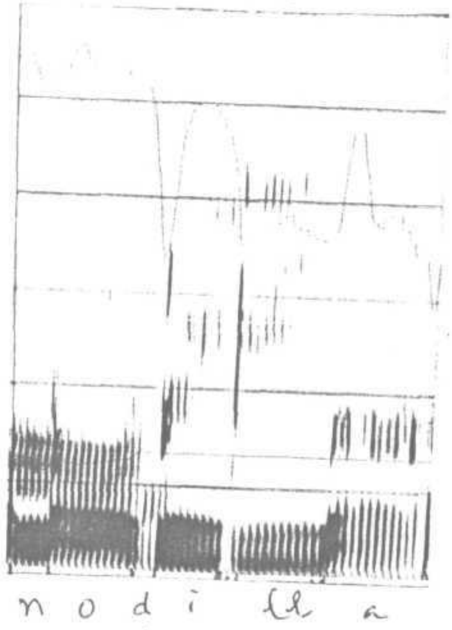
In the pre-therapy utterance, /n/ was omitted. The duration of /n/ was the safe as in post therapy utterance when it is produced. (in region z). Inspiratory frications were reducing in durations in successive attempts.

In the Pre-therapy utterance, the F_2 transition is absent or slower in speed compared to the post therapy utterance.

Pretherapy /nōḍilla/



Post therapy



3) /nō ḍi dɪvi/ (If id If nōḍidɪvi)

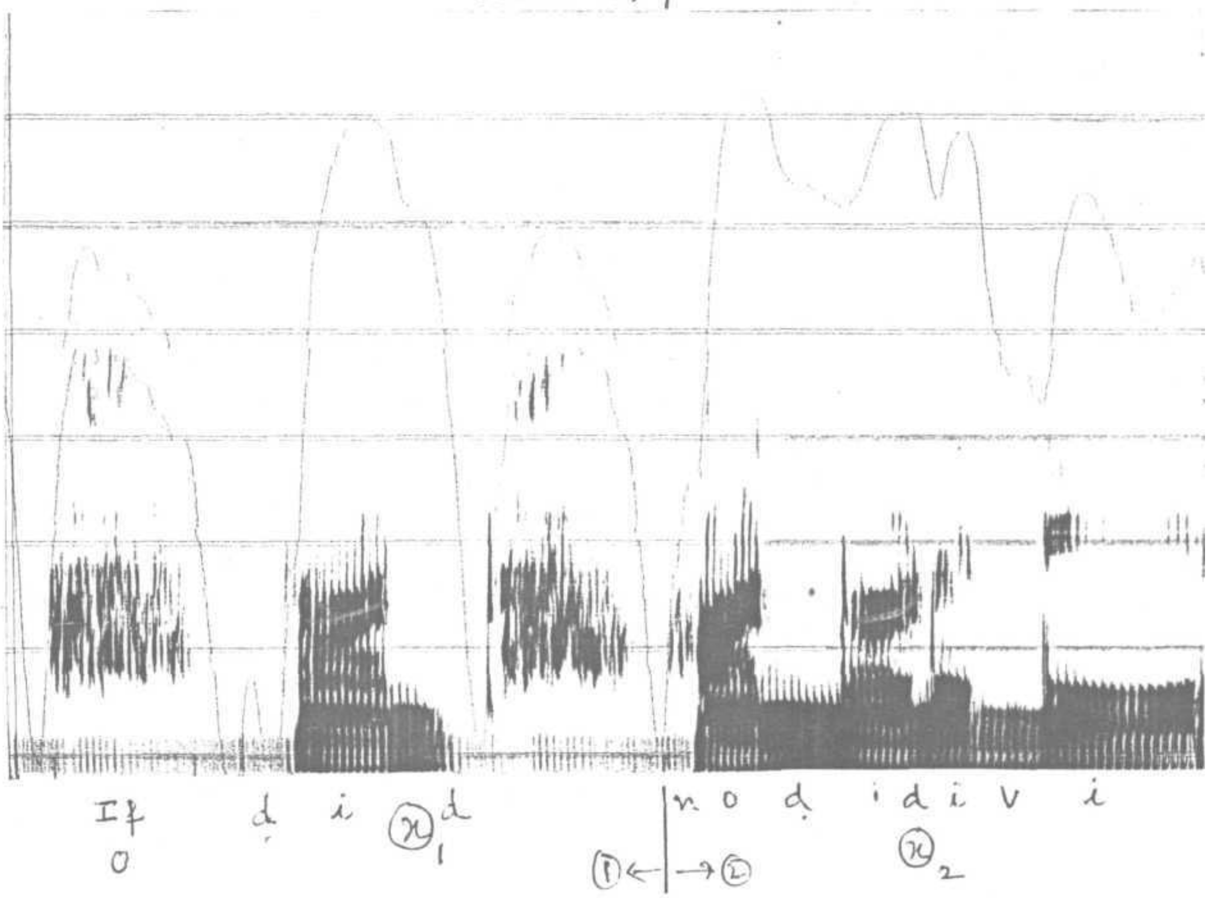
S.Q. of vocal fold cycles of /d/ in region (1)	S.Q. of vocal fold cycles of /d/ in region(2)
1.0	1.1
O.8	1.2
0.6	
0.5	
0.6	
1.1	
0.9	

Table 11: Speed quotients of the vocal fold vibrations of /d/ in the uord /nodidivi/

This word could not be elicited in the post therapy spontaneous speech recording. The Pre-therapy spectrogram depicted two important features, /n/ was absent. While the spectrogram indicated a gap between the vocal cord vibrations (voice bars) for /d/, the laryngograms showed period cycles for the entire period.

S.Q. of vocal cord vibrations seemed to vary in the segment /d/ in the nonfluent pre-therapy utterance in region (1). But not much variation was found in the segment /d/ of fluent pre-therapy utterance in region (2). The former also depicted clear kneepoints which were not observed in the latter.

Pretherapy /nōḍidīvi/



Note: Post therapy spectrogram not presented.

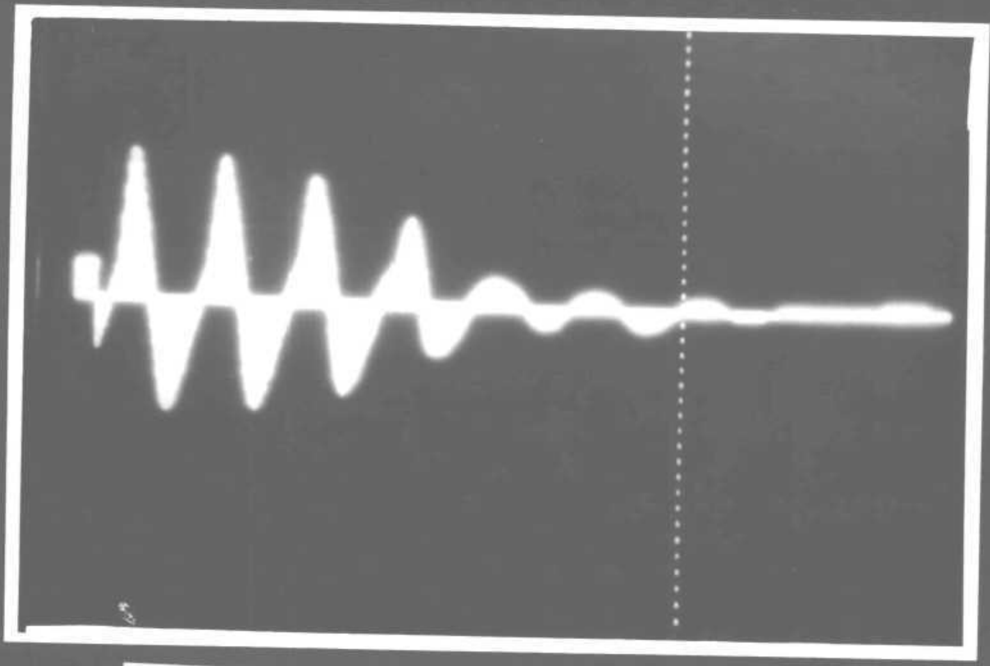


fig (11) noddivi: Lx cycles at region x_1

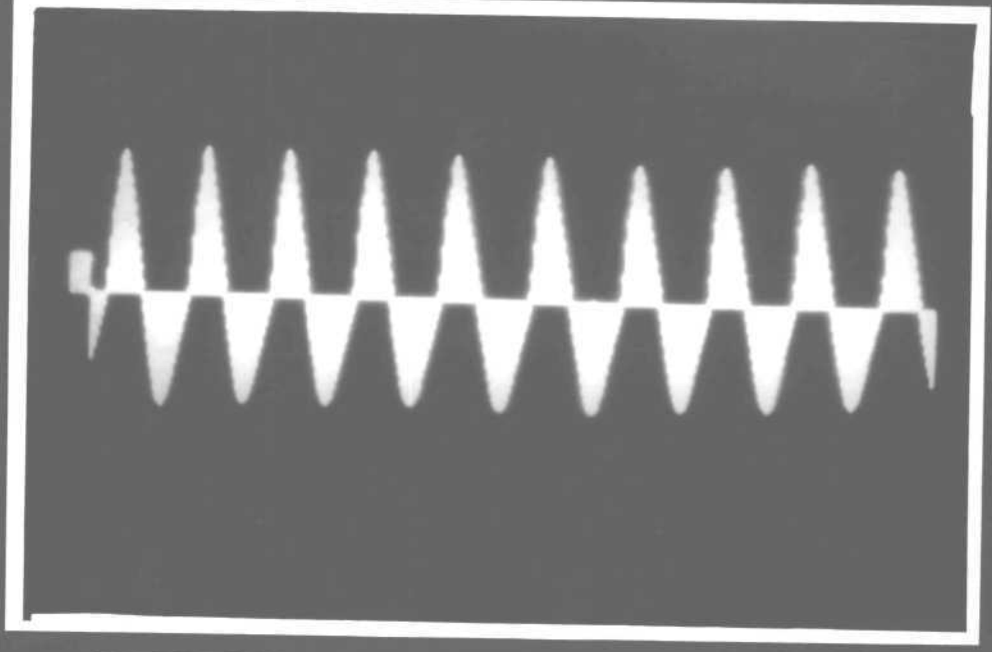


fig (12) noddivi: Lx cycles at region x_2

5.

1) /r ā j a m m a/ (rā If rhā If rhājamma)

S.Q. of vocal fold vibrations			
during If in region(1) Pre-therapy Utterance	during If in region (2) Pre-therapy Utterance	of /a/ in syllable /ra/ in region (3) pre-therapy Utterance	of /a/ in /ra/ in Post-therapy Utterance
0.5	0.8	1.1	1.2
0.3	1.1	1.1	1.1
0.9	0.8	1.1	1.0
0.8	1.3	1.2	0.8
0.8	0.5	1.1	0.7
1.3		1.2	0.7
1.3		1.2	0.7
1.1		1.2	0.7
		1.3	
		1.2	
		1.1	
		1.1	
		1.1	
		1.1	
		1.0	

Table 12: Speed quotients of vocal fold vibrations for 'If' and /a/ in the word /rājamma/

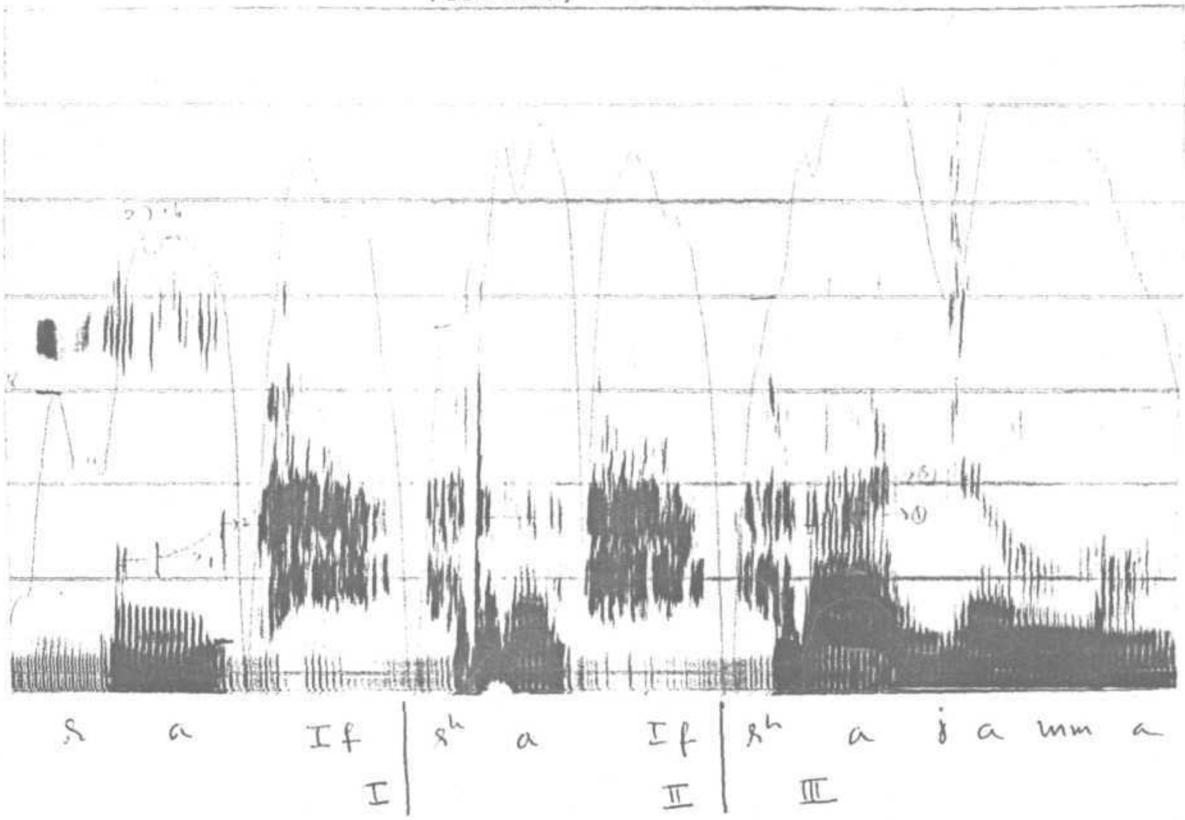
	Pre-therapy Utterancs	Post-therapy Utterance
duration of /r/	125 (1) 55 (20 m.secs of aspiration is also present) (2) 70 (30 m.secs of aspiration is also present) (3)	30
F_1 transition from /a/ to /j/	absent in (1 & 2) rising & falling in(3) rising at a rate of 5.7 hz/ms	rising & falling. rising at a rate of 5.5hz/ms
F_2 transition from/a/ to /j/	(1) absent (2) 8.9 hz/ms (3)	
duration of If	160 (1) 130 (2)	

Table 13: Durations and transition speeds in the word /rajamma/ (in m.secs & Hz/m.sec. respectively)

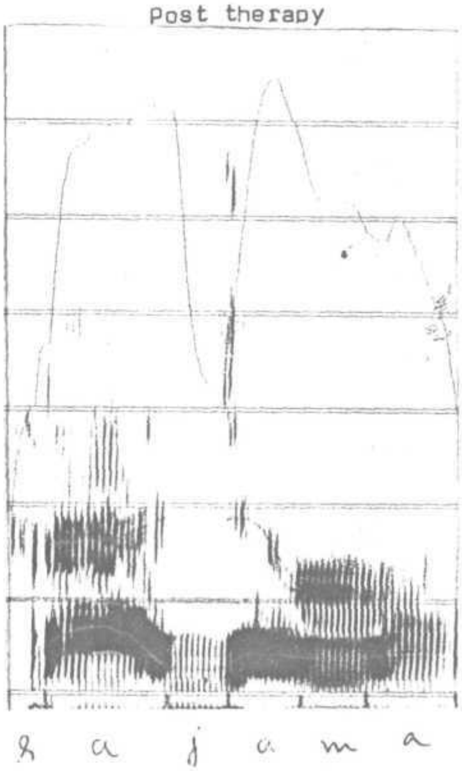
Duration of pre-therapy /r/ was longer compared to that of post therapy utterance.

In the pre-therapy utterance, F_2 transition was absent or had a higher speed compared to that of the cost therapy utterance. In the post therapy utterance /a/ to /j / transition showed falling F_1 and rising F_2.
In the pre-therapy utterance such F_1 and F_2 transitions were observed only in region 3. In 1, F_2 was rising but F_1, was flat. In 2, both F_1 and F_2 were flat. Thus simultaneous and appropriate F_1, & F_2 transitions were

Pretherapy /rājamma/



Post therapy



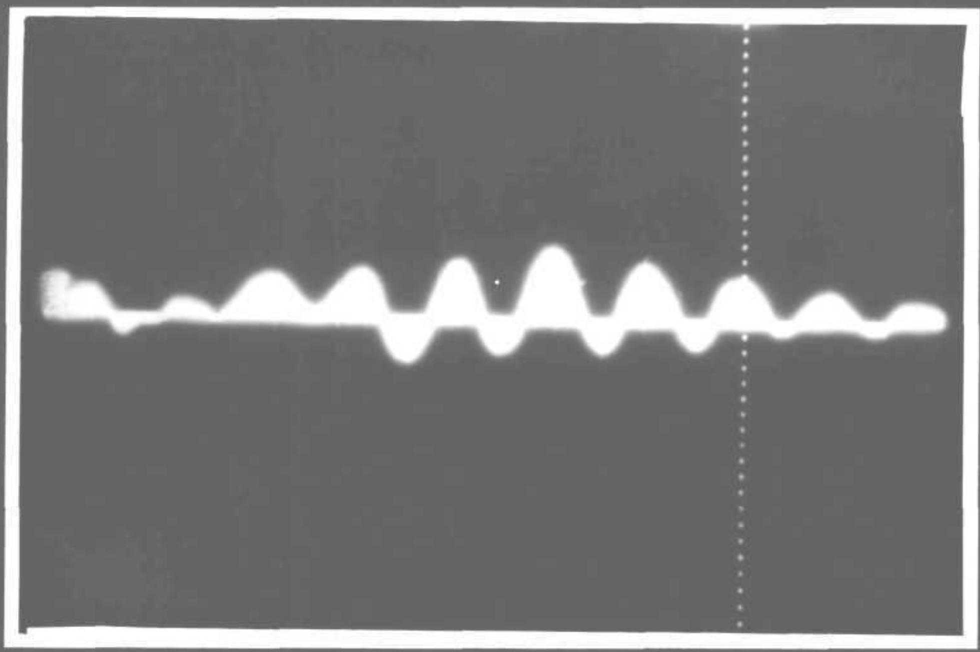


fig (13) Rajamma: Lx cycles during 'If' in region I

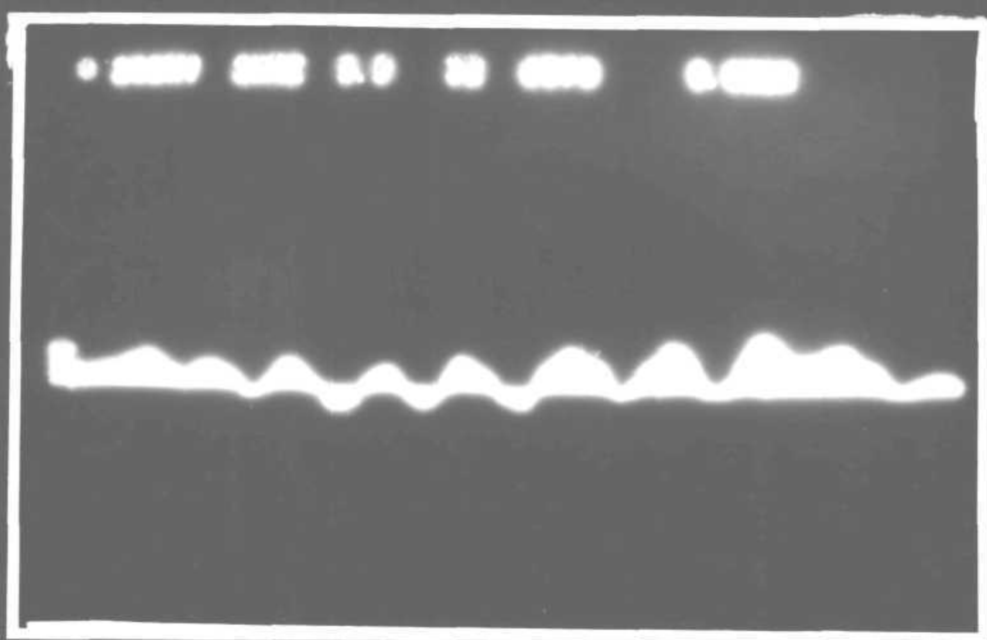


fig (14) Rajamma: Lx cycles during 'If' in region II

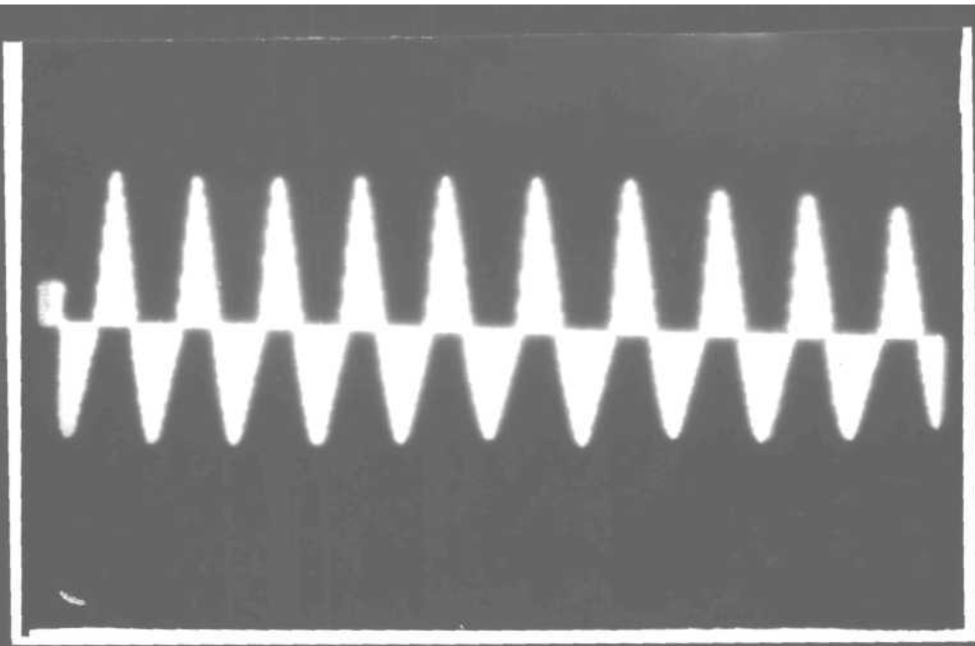


fig (15) Rajamma: Lx cycles during /a/ in syllable /ra/ in region III in pre therapy utterance

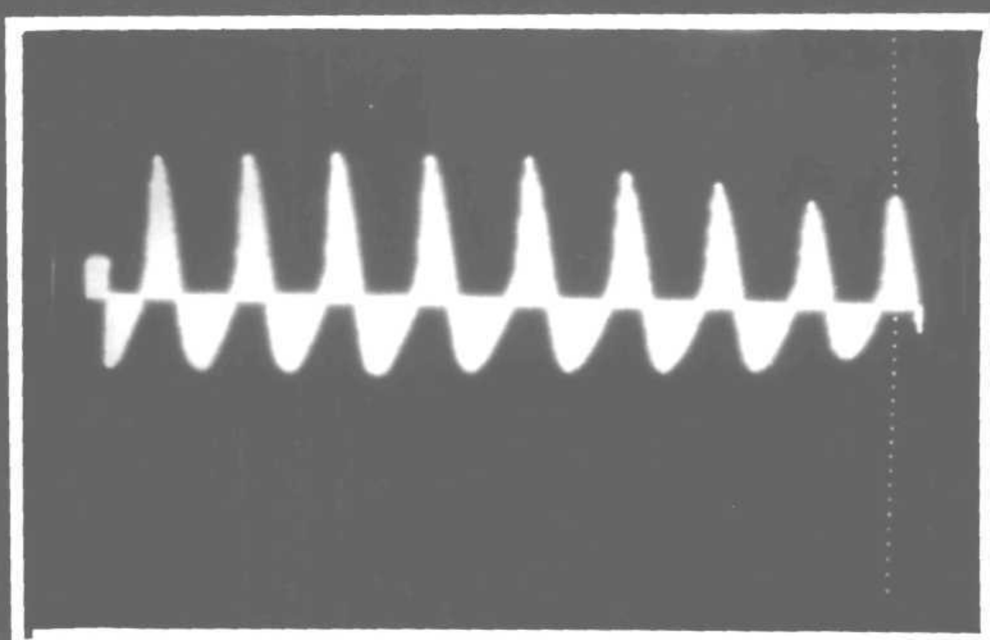
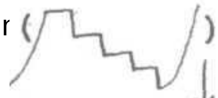


fig (16) Rajamma: Lx cycles during /a/ in syllable /ra/ in post therapy utterance lacking in the pre-therapy nonfluencies.

Inspiratory frication was accompanied by vocal fold fibrations. S.Q. of these vibrations varied from cycle to cycle in both region 1 and 2. In region 2, stairstep pattern () in the opening phase was observed in two of the cycles. ↓ increasing glotta impedance.

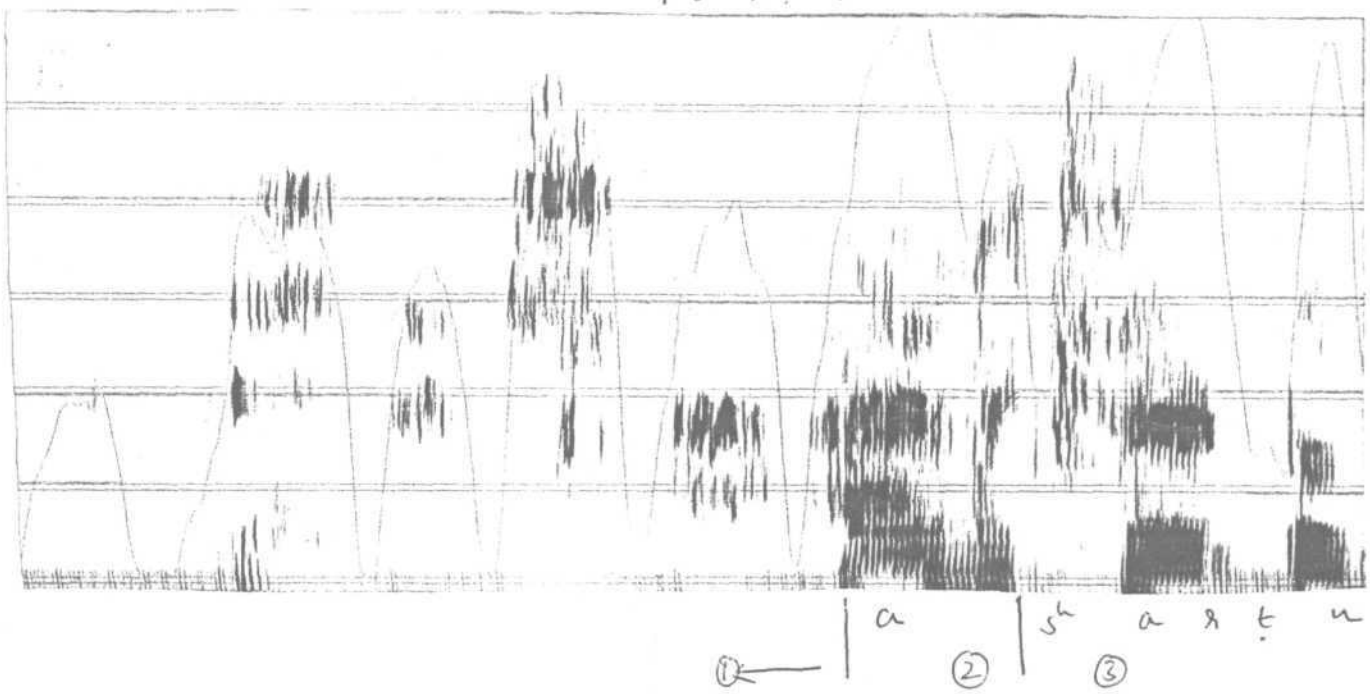
S.Q. of the vocal fold vibrations in the vowel /a/ of the syllable /ra/ in the pre-therapy fluent utterance (Region 3) was constant and was always greater than one S.Q. decreased steadily in /a/ of the cost therapy utterance and reached 0.7 in the last few cycles.

6) 1) Śarṭu (S If If s If sarṭu sarṭu)

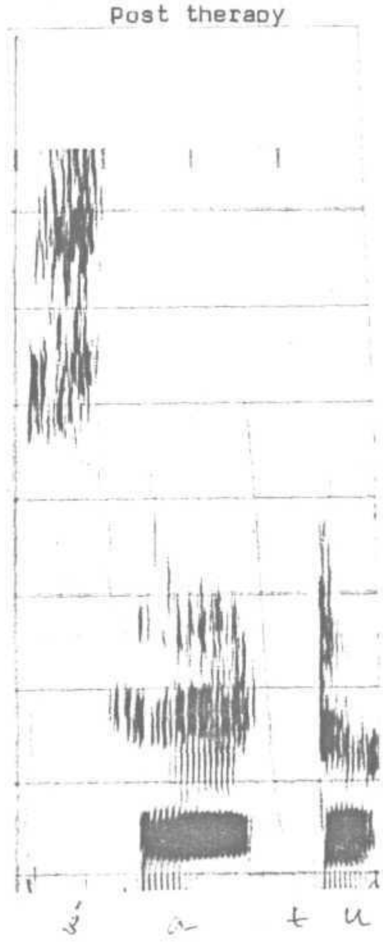
	Pre-therapy Utterance	Post-therapy Utterance
Voicing	was present in the preburst period of /ṭ/ (2) was present for preburst period of /ṭ/ (3)	was absent in the preburst period of /ṭ/.
F_2 transition from /ṭ/ to /u/ hz/ms	It was rising at a speed of 10 hz/ms (2) It was falling at a speed of 7.5 (3)	It was falling with a speed of 6.7 hz/ms

Table 14: Voicing & transition speeds in the word /sarṭu/ (in Hz/m.secs)

Pretherapy /saṛṭu/



Post therapy



The pre-therapy utterance showed a distorted /a/ indicating an incorrect constriction. This may trigger repeated inspiratory frications, with different articulatory constrictions. Vocal tract shaping seems to be controlled independant of the airflows.

In region (2) & (3) voicing occurred at an inappropriate time.

F_2 transition was opposite in direction and when the direction was correct the speed was faster compared to that of the post therapy utterance.

II. Intraword silent pause

In this, the first phoneme was produced which was followed by a pause. There were two words in this category.

1. /jāli/
2. /hesaru/

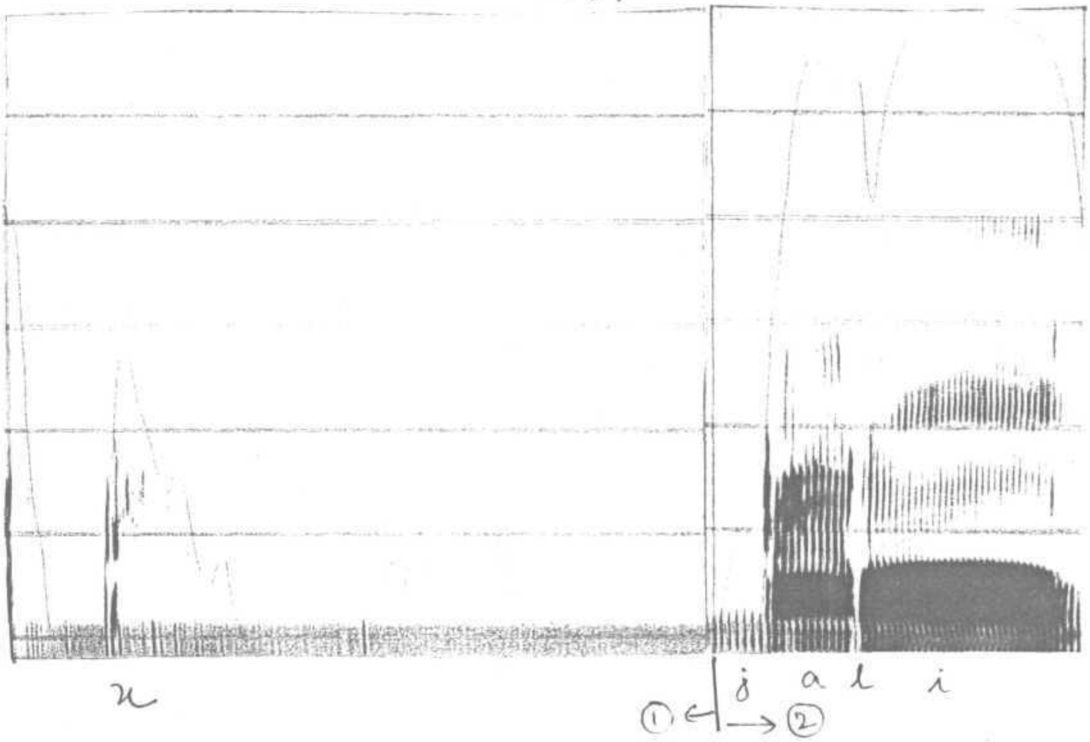
The perceptually silent pause sometimes showed vocal fold vibrations on the spectrogram. These vibrations were also analysed.

1. /jāli/ (j - jāli)

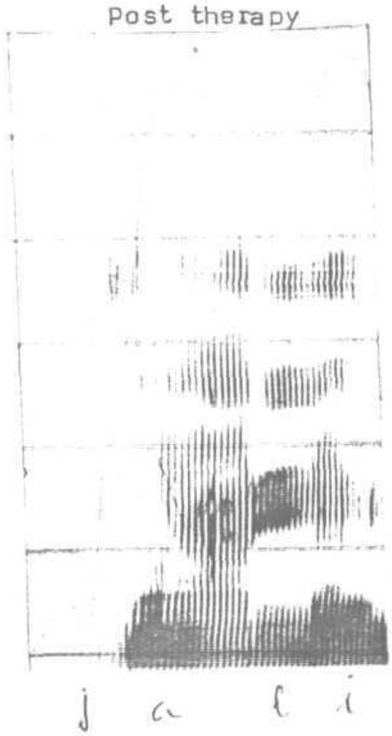
Speech sounds	Duration in the	
	Pre-therapy Utterance	Post-therapy Utterance
/j/	50 (2)	30
/a/	60 (2)	120
/l/	10 (2)	65

Table 15: Durations of segments in the word /jāli/ (in m.secs)

Pretherapy(jāli)



Post therapy



Duration of / j / was longer and that of /a/ & /l/ was shorter in the pre-therapy utterance.

In the pretherapy utterance an attempt to produce /jāli/ seemd to cease after / j / . This might be because of the simultaneous voicing with frication. Following this was a pause of 625 ms.

2. /hesaru/ (e - hesaru)

Some peculiar electrolaryngographic (lx) waveforms were observed during the pre-therapy utterance. The point marked x is shown in figure. 17 It might be possible that the vocal folds are vibrating under erratic expiratory airflows and/or the folds themselves might be under the influence of abnormal laryngeal muscle contractions.

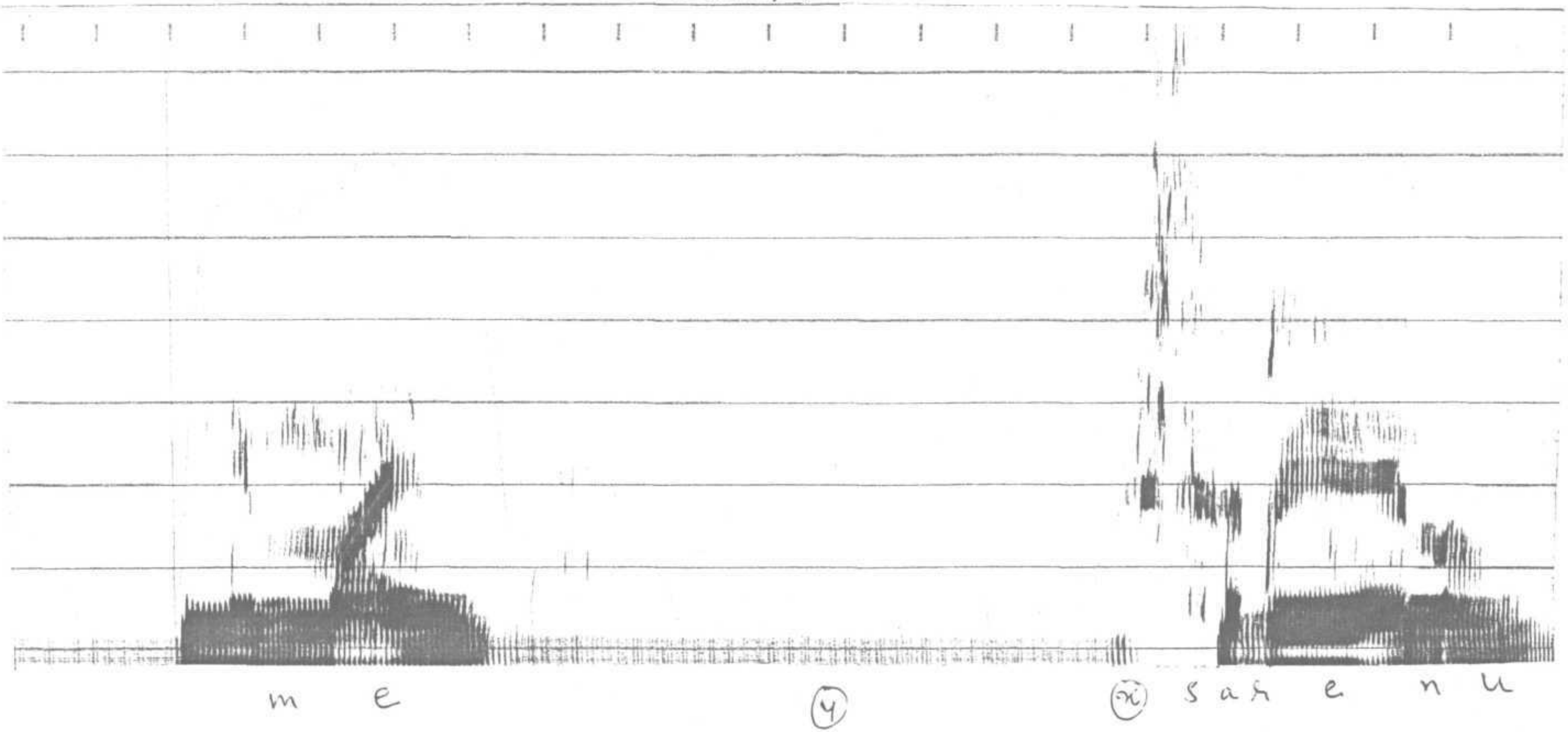
At point (y) a similar abnormal lx waveform was noticed. In the figure (18) the cursor is placed at the end of one of the prominent cycle.

The 820 ms long silent pause on spectrogram showed very weak voice bars indicating some vocal fold vibrations. Absence of formants cued a suoraglottal constriction. However, no clear lx waveforms during this 820 ms was observed.

III. Whole word repetition

In this, when a word was followed by a case marker, the word was repeated several times. Only one word was found in this category.

Pretherapy /hesaru/



(nimmesarenu)

Note: Post therapy spectrogram not presented.

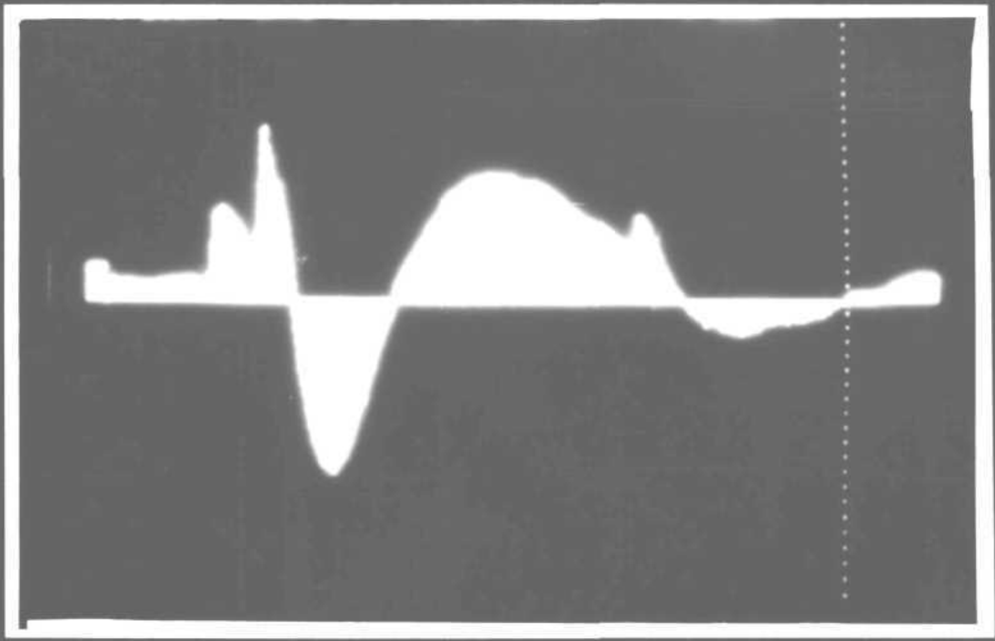


fig (17) hesaru: Lx cycles at region x

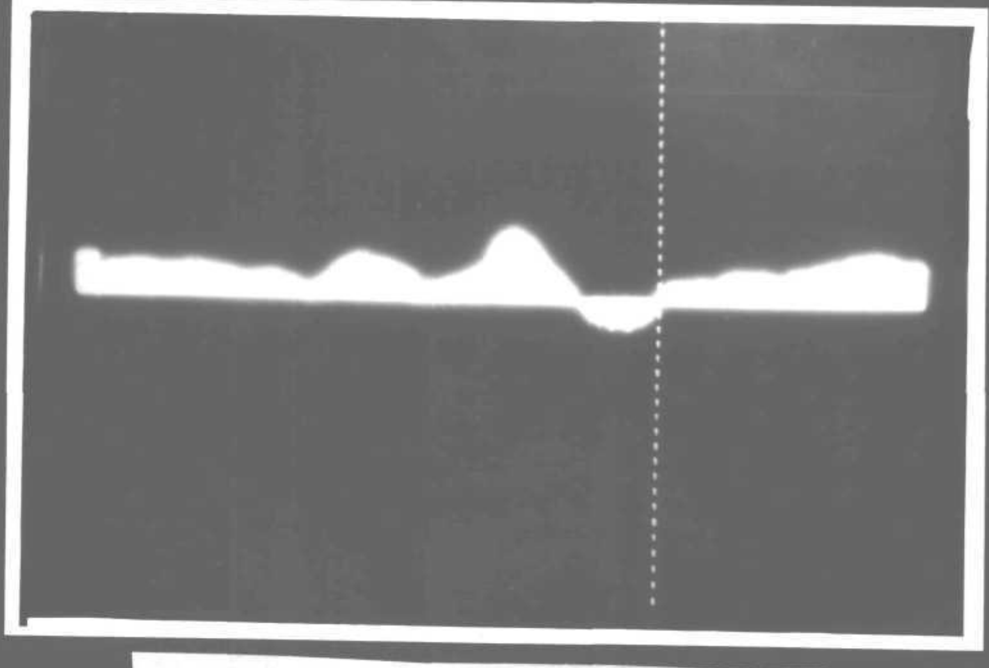


fig (18) hesaru: Lx cycles at region y

1. /mane/ (in /manēlidIni/) (mane mane, manilidIni Sa)

	Pre therapy Utterance			Post therapy Utterance
duration of /m/	80 (1)	60 (2)	65 (3)	60
duration of /a/	65 (1)	135 (2)	45 (3)	145
duration of /n/	55 (1)	70 (2)	80 (3)	110
duration of /e/	40 (1)	60 (2)	30 (3)	80
F_2 transition from/m/ to /a/	20hz/ ms rise (1)	22.5hz/ ms rise (2)	13.3 hz/ms rise (3)	24 hz/ms rise
F_2 transition from /e/ to /l/	17.1 hz/ms fall* (1)	8hz/ms fall* (2)	13.75 hz/ms rise (3)	- * After /e/ the articulator approximated to /l/

Table 16: Durations & Speed transitions in the word /manelidini/ (in m.secs & Hz/m.secs respectively)

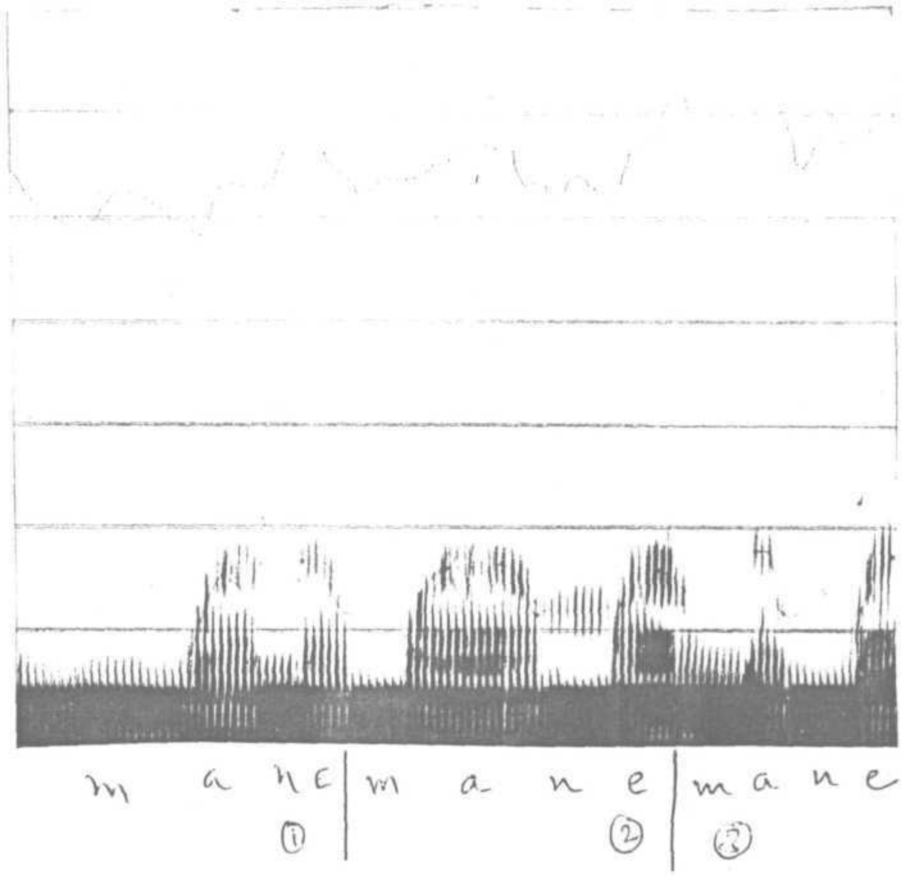
In the pre-therapy utterances durations varied and were generally shorter compared to that of the post therapy utterance.

Speeds of F_2 transitions, varied widely and they had a rise-fall pattern instead of only rising. This indicates that the articulator is transiting back to /m/ instead of getting positioned for /l/.

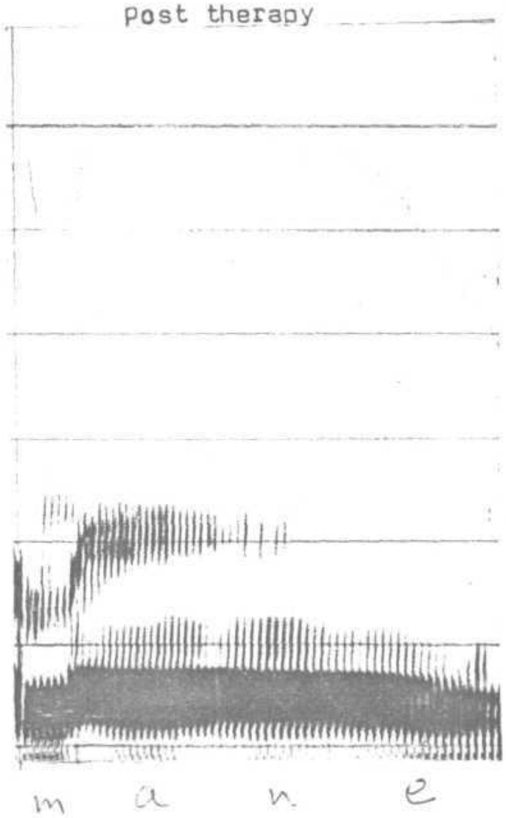
IV. Intraword repetition

In this syllabic/intrasyllabic repetitions were followed by a fluent utterance. There were 8 words under this category.

Pretherapy /mane/



Post therapy



1. Words starting with a plosive.

   1) /bartāide/

   2) /pakṣi/

   3) /barabēku/

   4) /kempu/

   5) /bōrd/

2. Words starting with a nasal continuant

   1) /naḍkonḍu/

   2) /namma/

3. Words starting with a fricative

   1) /hōgbahudu/

Two words showed syllabic repetition. Five words showed intra-syllabic repetition and one word showed both.

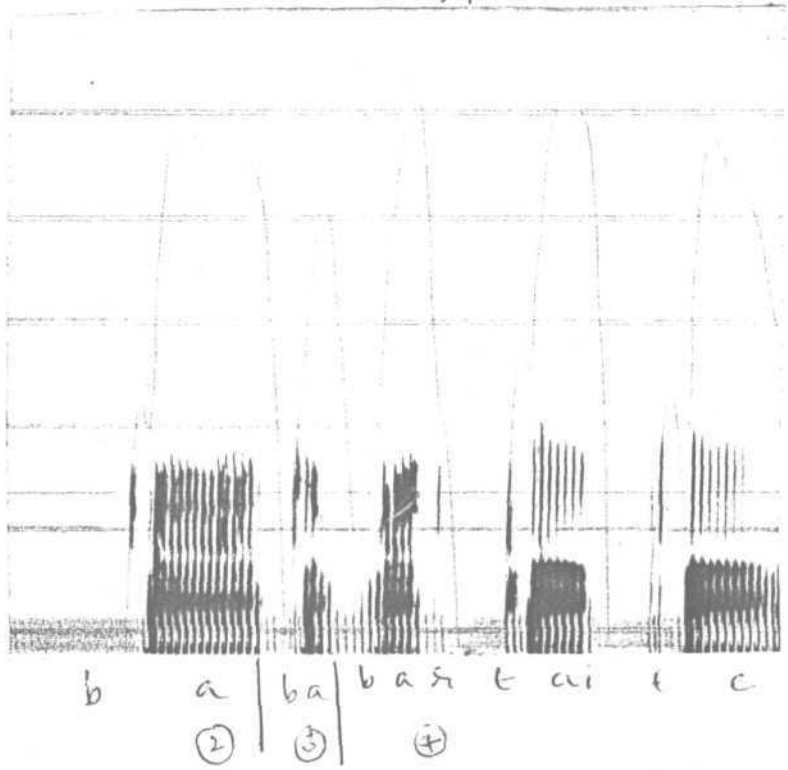
1) 1. /bartaide/ (ba ba ba bartaide)

In this word, ths initial CV sequence /ba/ was repeated.

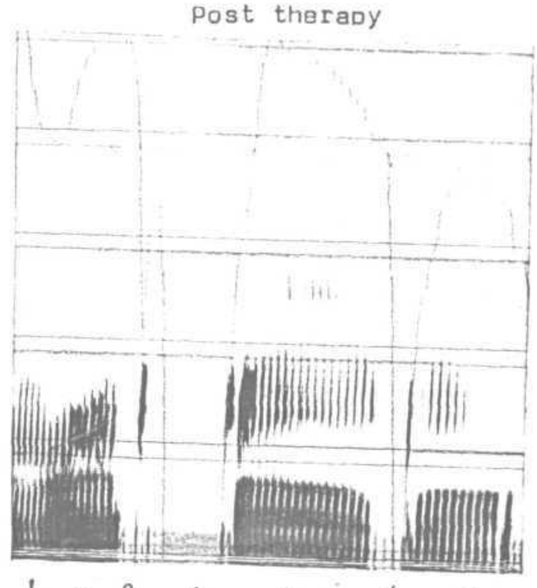
	Pre therapy Utterance	Post therapy Utterance
duration of b	70 (1) 50 (3) 50 (4)	85
duration of a	190 (1) 110 (2) 25 (3) 40 (4)	75
F_2 transition from /a/ to /r/	Flat (1 & 2) 7.5 hz/ms fall (3) 11.43 hz/ms rise	5.4 hz/ms rise

Table 17: Durations & speed of transitions in the word /bartaide/ (in m.secs & Hz/m.secs respectively).

Pretherapy /baɾtaide/



Post therapy



In the pre therapy utterance durations of /b/ was shorter.

F_2 transition in fluent pre therapy utterance and post therapy utterance were different. F_2 transitions from /a/ to /r/ were flat or opposite in direction.

In region (2) voicing was not observed during the production of /b/.

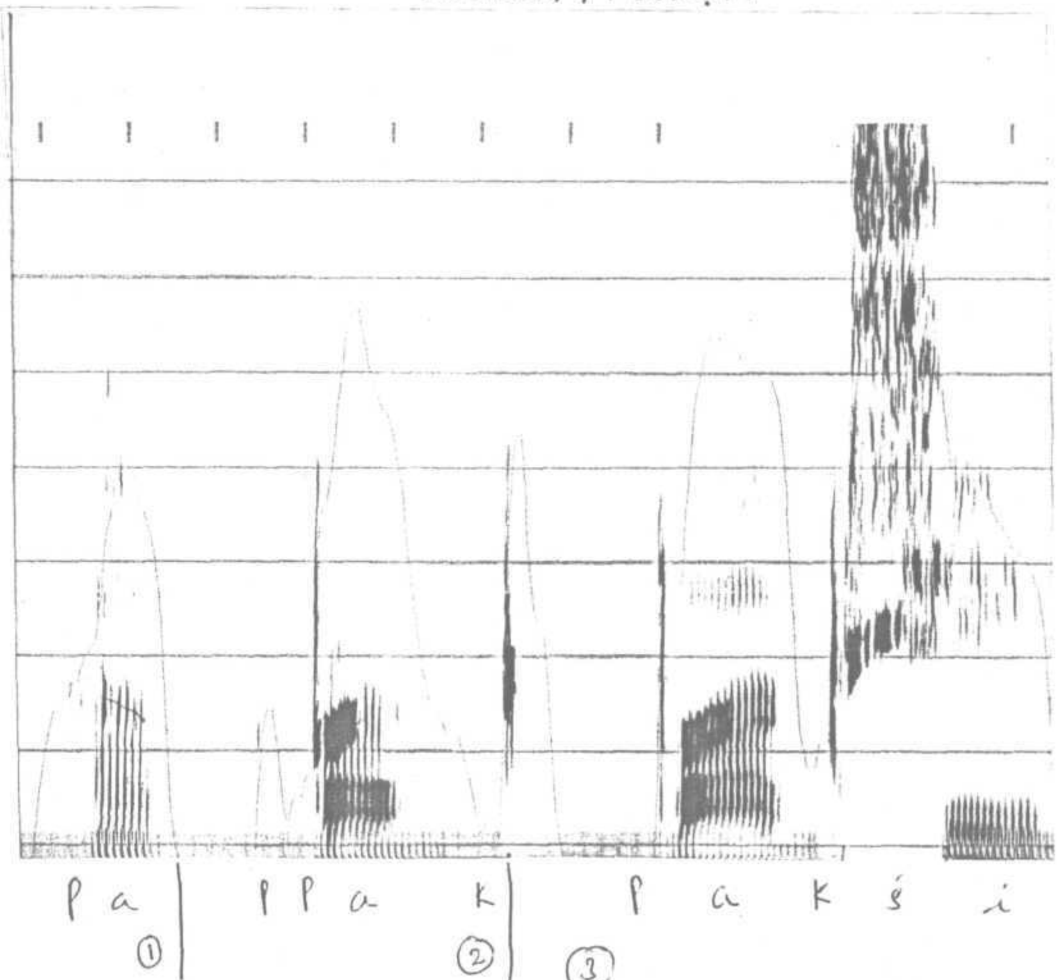
2) /pakṣi/ (pa p pak pakṣi)

	Pre therapy utterance	Post therapy utterance
duration of /o/	70 (1) 160 (2) 165 (3)	90
duration of /a/	60 (1) 80 (2) 105 (3)	45
duration of /k/	80 (2) 60 (3)	60
F_2 transition from /a/ to /k/	5 hz/ms fall (1) 7.1 hz/ms rise (2) 6.2 hz/ms rise (3)	7.8 hz/ms rise
direction of F_1 transition from /a/ to /k/	falling (1) Flat (2) falling (3)	falling

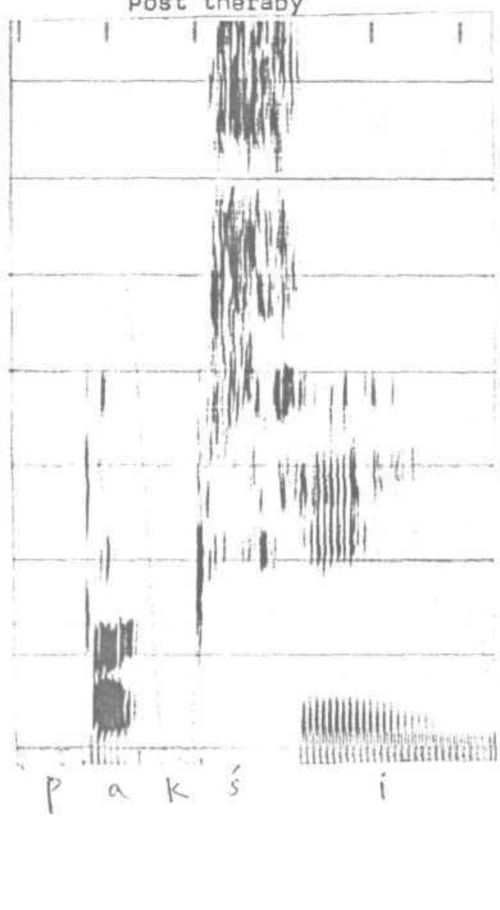
Table 18: Durations & transition speeds in the word /pakṣi/ (in m.secs & Hz/m.secs).

In this word, the nonfluencies were /pa/ and /pak/. In the pre therapy utterance durations of /p/, /a/ and /k/ were longer.

Pretherapy /Pakṣi/



Post therapy



3) /barabēku/ (bara bara barabēku)

	pre therapy utterance	Post therapy utterance
duration of /b/	20 (1) 70 (2) 20 (4)	75
duration of /a/	85 (1) 40 (2) 20 (4)	45
duration of /r/	40 (1) 50 (2) 20 (4)	20
duration of /a/	50 (1) 40 (2) 40 (4)	20
F_2 transition from /b/ ti /a/	5 hz/ms fall (1) 23.3 hz/ms fall (2) 10 hz/ms rise	6 hz/ms rise
F_2 transition from /r/ to /a/	not clear (1 & 2) 5.7 hz/ms fall (4)	

Table 19: Durations & Transition speeds in the word /barabeku/ (in m.secs & Hz/m.secs respectively)

In this word, /bara/ syllable was repeated. Durations of segments in the pre therapy utterance varied.

In the pre therapy utterance the F_2 transitions from /a/ to /b/ were either flat or rising which was falling in the pre therapy fluent (4) as well as the post therapy utterance.

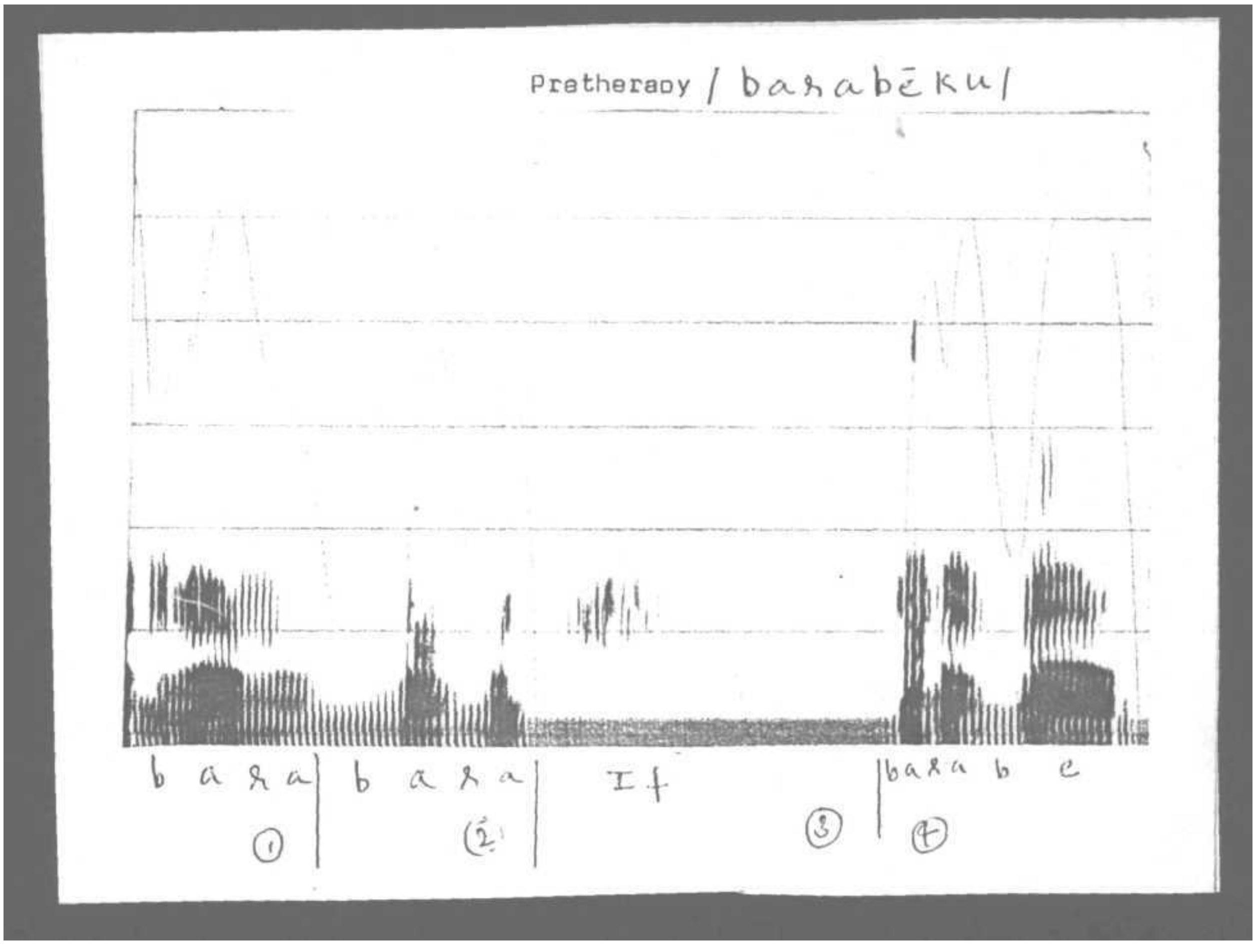
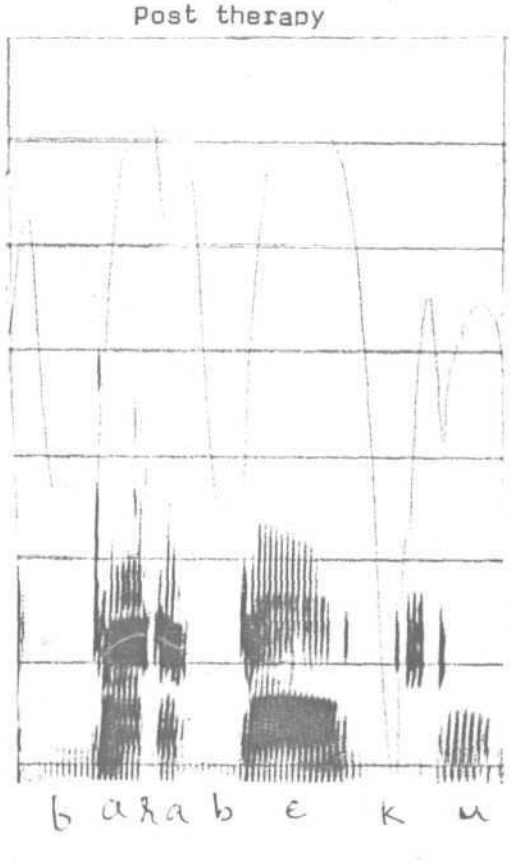


fig (19) Barabeku: Mouth expiratory airflow waveform (MEAW) during 'If' in region (3)

Post therapy



4) /kempu/ (kem kempu)

	Duration in the pre-therapy utterance	Post therapy utterance
/k/	70 (1) 55 (2)	50
/e/	60 (1) 80 (2)	100
/m/	105 (1) 100 (2)	105
/p/	50 (2)	70
/u/	65 (2)	360

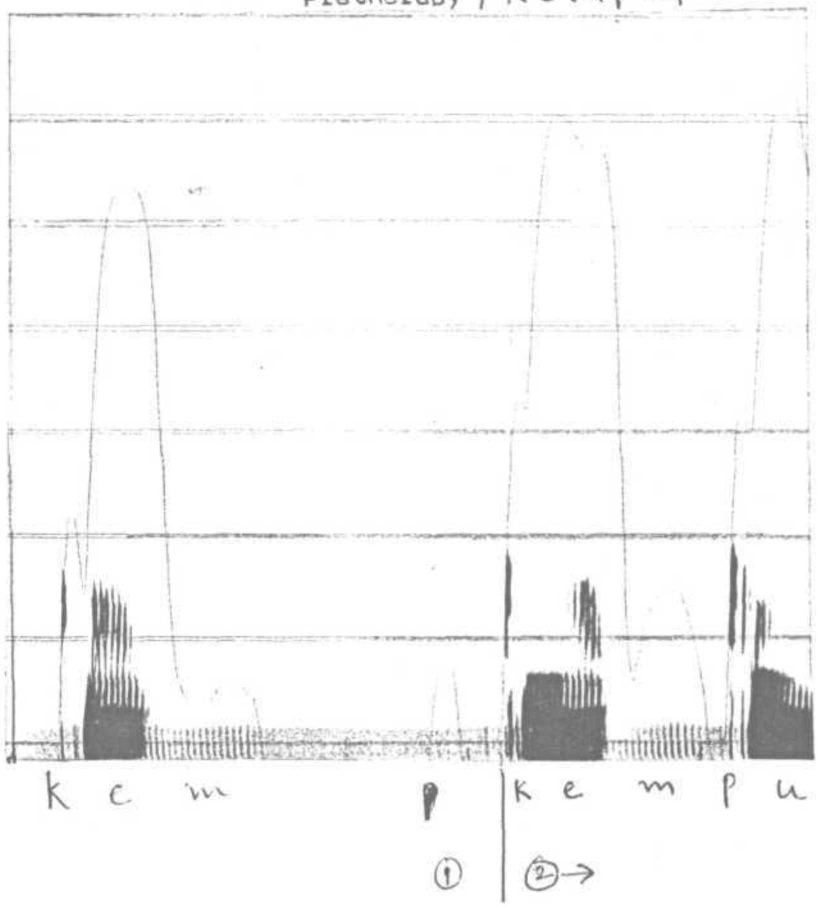
Table 20: Duratipns of the segment in tha word /kempu/ (in m.secs)

In this word /kem/ was repeated.

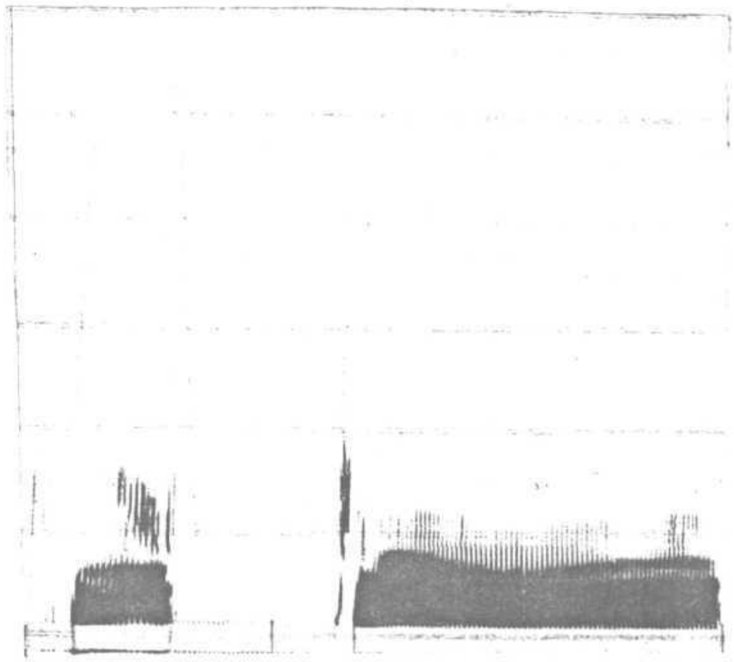
In the pre therapy utterance durations of /k/, /e/, /m/ in the fluent utterance approximated the post therapy durations. However, the duration of /u/ was short in the pre therapy utterance (shorter by 295 ms). This is expected as the post therapy /u/ was in the word end.

Region (2) of ore therapy utterance ahowed voicing in /k/ segment, which is inappropriate.

Pretherapy /Kempu/



Post therapy



5) /bōrḍə/ (in the phrase bōrḍə hakilla) (bo bo bōrḍə)

	Pre theraoy utterance	Post theraoy utterance
duration of /b/	50 (1) 40 (2) 75 (3)	25
duration of /o/	70 (1) 230 (2) 210 (3)	110
duration of /rḍ/	15 (3)	60
duration of /e/	20 (3)	-
F_2 transition from /b/ to /o/	F_2 was absent (1) F_2 rises at a rate of 4.8 hz/ms (2) F_2 was rising (3)	F_2 rises at a rate of 7.3 hz/ms

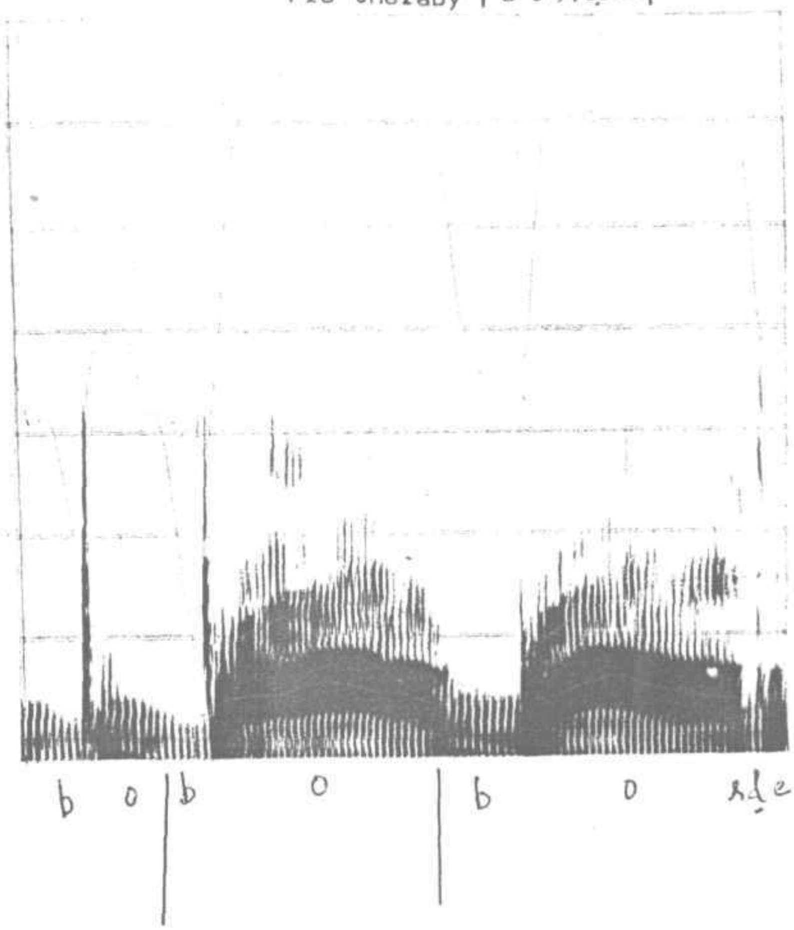
Table 21: Duration & transition spaed3 in the segment /bōrḍə/ (in m.secs & Hz/ m.secs respectively)

In this word, initial CV sequence /bo/ was repeated twice.

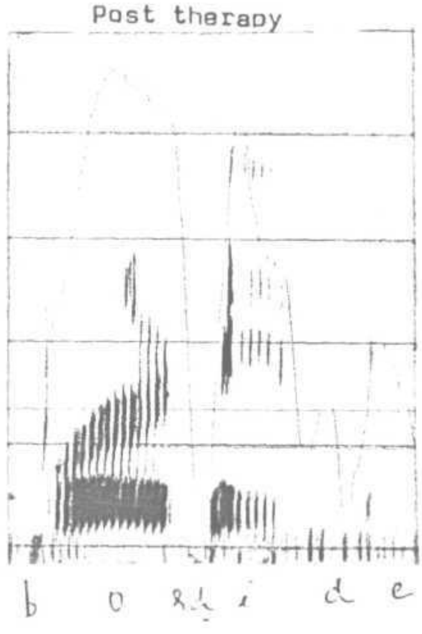
Durations were longer for the pre therapy /b/ and /o/ and shorter for the pre therapy /rd/ cluster.

Pre therapy F_2 transitions were slower or absent when compared to that of the post therapy utterance.

Pre therapy



Post therapy



2. 1) /naḍkoṇḍu/ (na naḍkoṇḍu)

	Pre therapy utterance		Post therapy utterance
duration of /n/	185 (1)	105 (2)	60
duration of /a/	100 (1)	70 (2)	65
F_2 transition from /a/ to /d/	2.3 hz/ ms rise (1)	13.3 hz/ ms rise (2)	10 hz/ms rise

Table 22: Durations & transition seeds in the word /naḍkoṇḍu/ (in m.secs & Hz/m.secs respectively).

In this word the initial CV sequence /na/ was repeated.

In the pretherapy utterance, the segment durations were longer.

2) /nam/ (in tha word nammūru) (na na a nammūr)

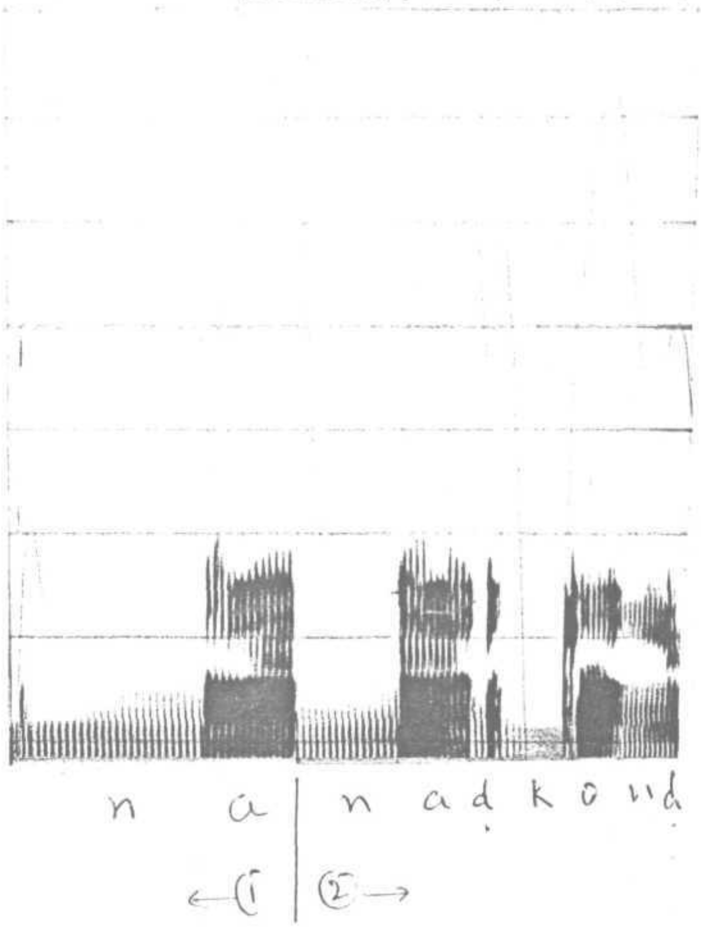
Speech sound	Duration in the			
	pre therapy utterance			Post therapy utterance
/n/	200 (1)	90 (2)	80 (4)	25
/a/	40 (1)	130 (2)		60
	30 (3)	75 (4)		
/m/	70 (4)			105

Table 23: Duration of the segments in the word /nam/ (in m.secs)

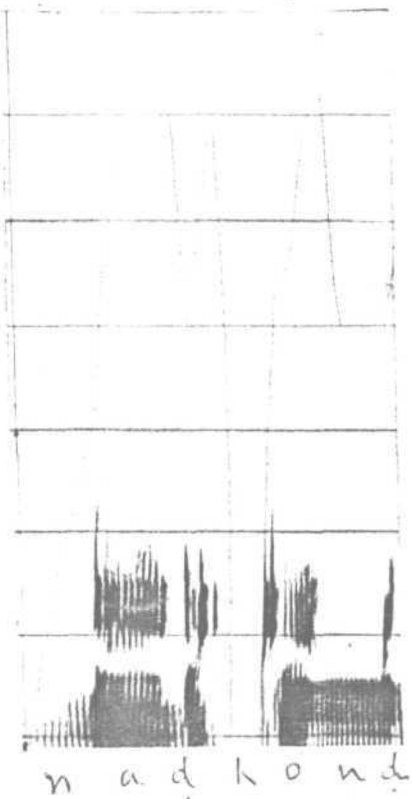
In this word, initial CV sequence /na/ was repeated.

Duration of /n/ was longer in the pre therapy utterance. Duration of /a/ varied and tha duration of /m/ was shorter.

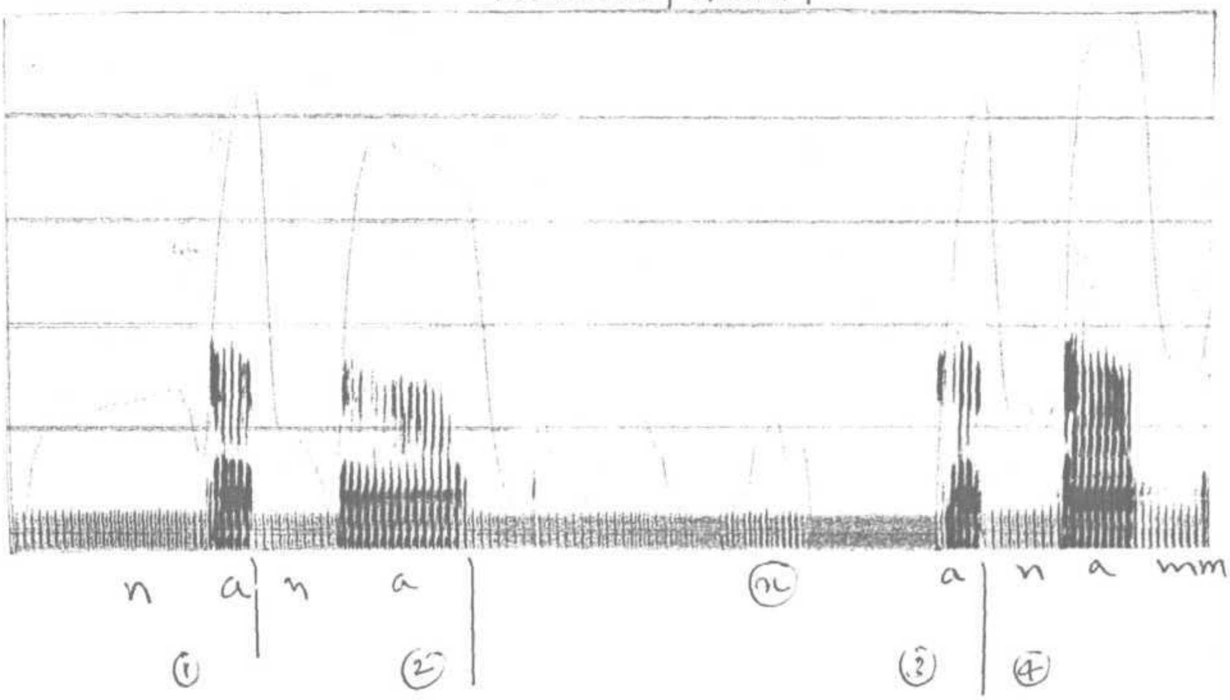
Pretherapy /naḍkoṇḍu/



Post therapy



Pretherapy /nam/



Post therapy

Region (3) showed a few weak vocal fold activity at 'x'. However, there were no formants indicating an abnormal positioning of the articulators.

3\.
1) /hōgbahudu/ (ho ho ho ho hogbahudu)

	Pre therapy utterance	Post therapy utterance
duration of /o/	100 (1) 115 (2) 125 (3) 55 (4) 145 (5)	195
F_2 transition from /o/ to /g/	Flat (2) 40 hz/ms fall (3) Flat (4) flat (5)	5 hz/ms fall

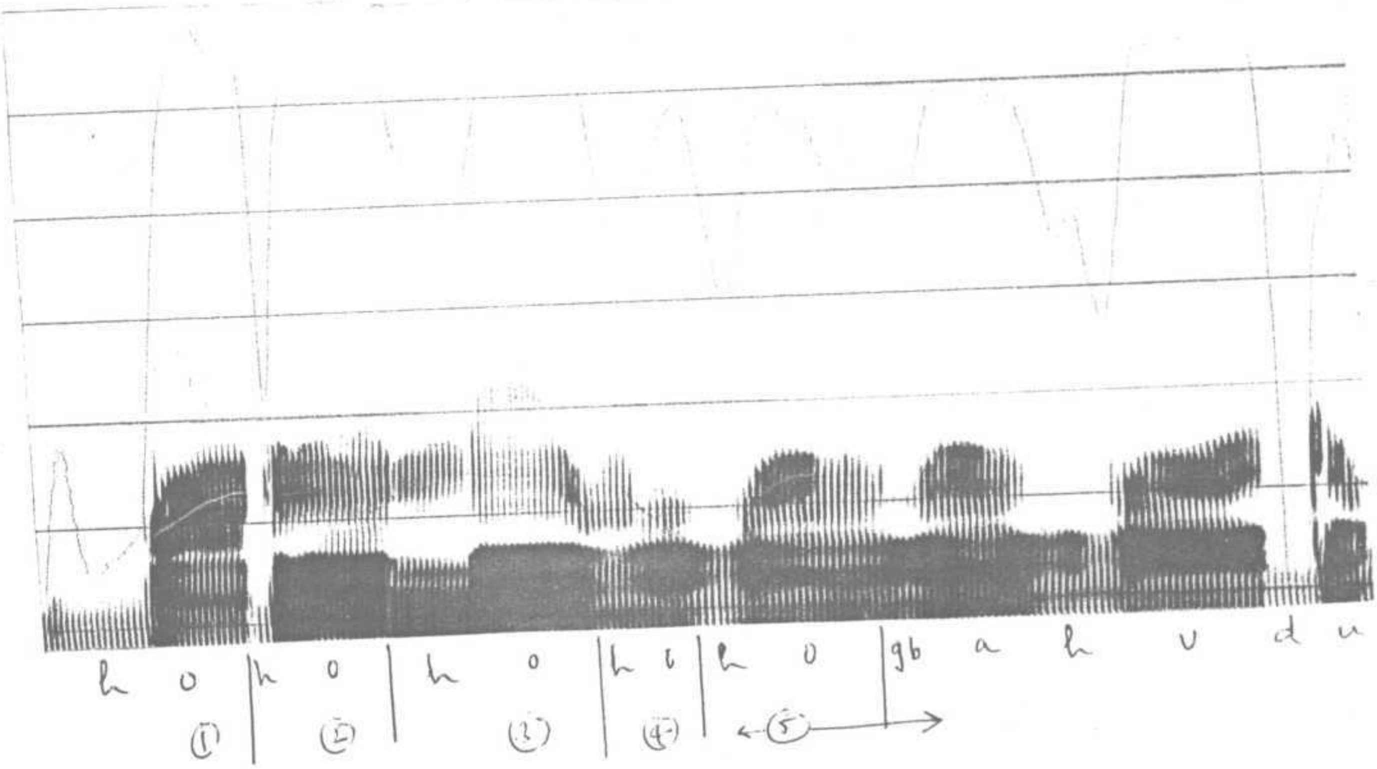
Table 23: Durations & transition speeds in the word /hōgbahudu/ (in m.secs & Hz/m.secs respectively).

In this word the initial CV sequence /hō/ was repeated.

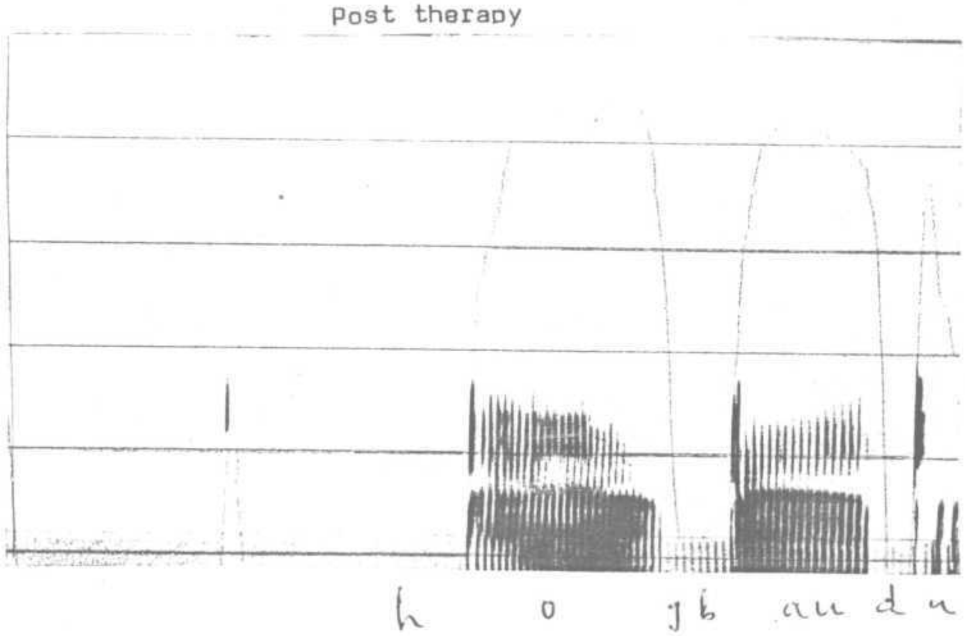
In the pre therapy utterance, the duration of /o/ varied and was shorter when compared to that of the post therapy.

Pre therapy F_2 transition from /o/ to /g/ was absent and when prasent had high speed compared to the post tharapy utterance.

Pretherapy /hõgbahudu/



Post therapy



V. Phoneme prolongation and repetition

Perceptually the phoneme was either prolonged or repeated. In this category there was one word with phoneme prolongation and five words with phoneme repetition.

1. Phoneme prolongation

1) /ombattu/ (ombatt....u)

In the pre therapy utterance the duration of the geminate cluster /tt/ was prolonged (240 ms) compared to that in post therapy utterance (70 ms). It appears as though the muscular constractions that elevated the tongue for /t/ was prolonged for a longer time than required. Vocal fold vibrations were also absent during this period.

2. Phoneme repetition

1) words starting with a plosive

i) /kuḍītāre/

ii) /kiṭaki/

2) words starting with an afflicats

i) /citrānna/

3) words starting with a fricative

i) /sarōja/

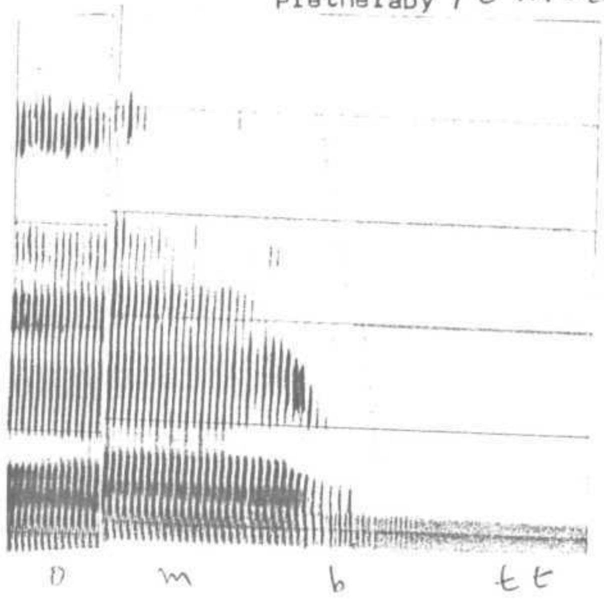
ii) /sirā/

1) i) /kuḍitāre/ (k k k k k kudi kuḍitāre)

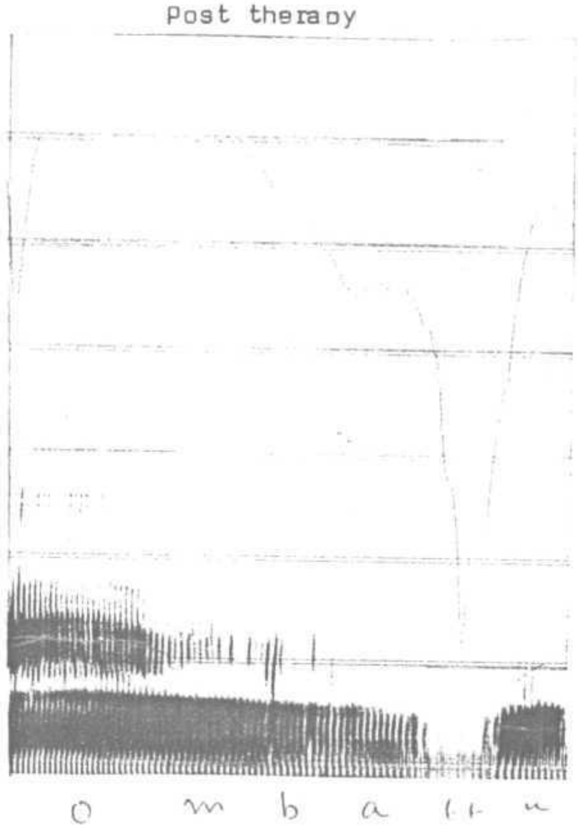
	Pre therapy	Post therapy
F_2 transition from /u/ to /ḍ/	8.9 rise (2)	6.7 rise
F_2 transition from /i/ to /t/	3.3 fail (2) 3.8 rise (3)	Flat

Table 25: Transition speeds in the word /kuḍitāre/ (in hz/m.secs)

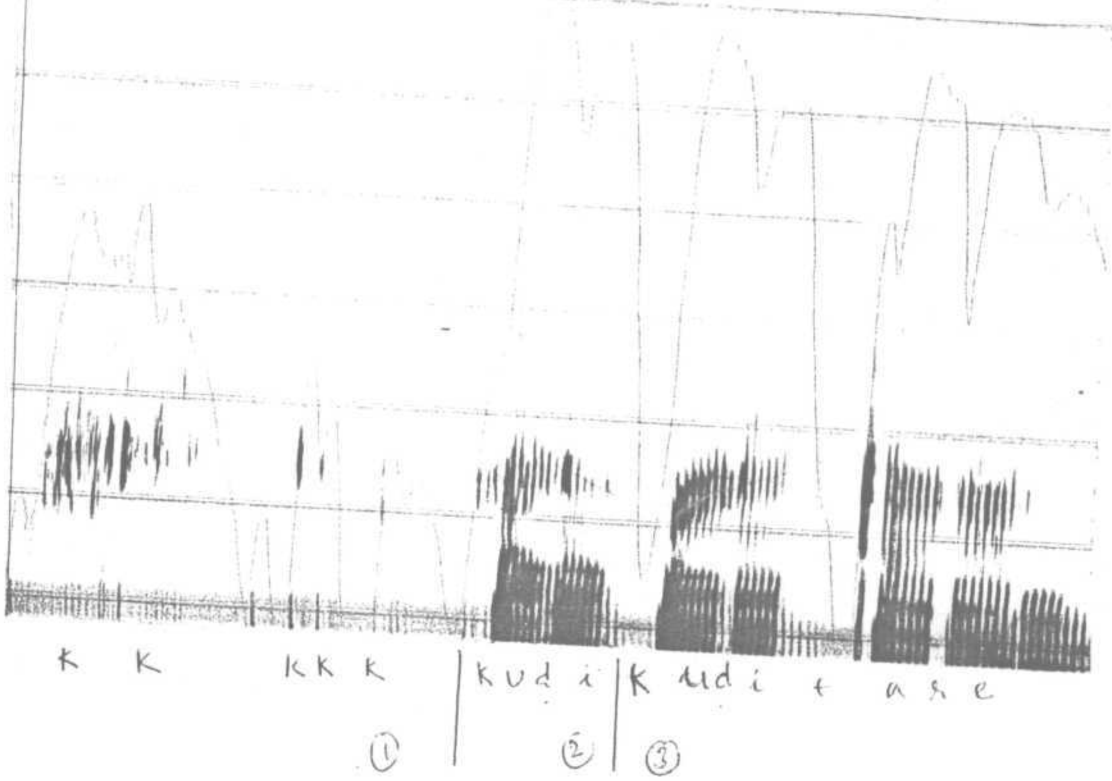
Pretherapy /ombattu/



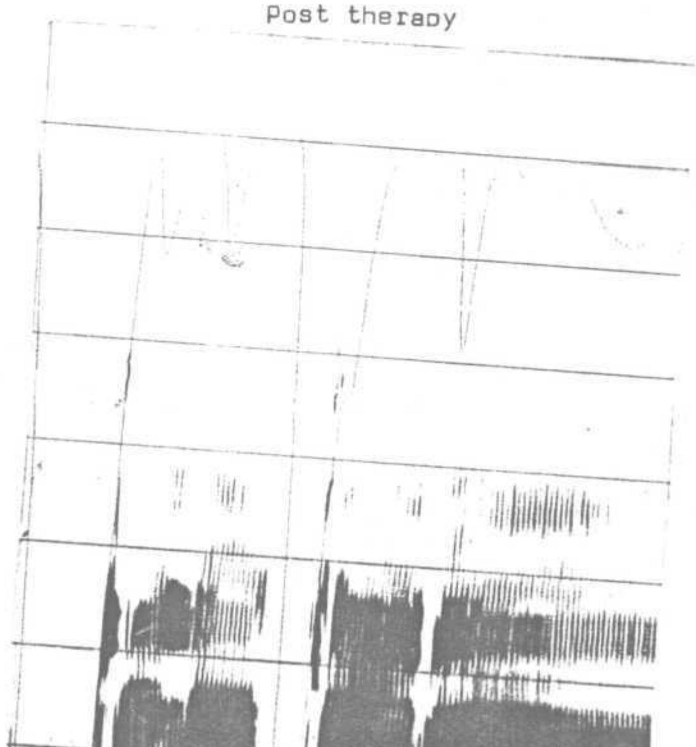
Post therapy



Pretherapy /Kuḍitāṛe/



Post therapy



In the pre therapy utterance phoneme /k/ was repeated. Initial two syllables were also repeated.

In the pre therapy utterance there were multiple bursts and each burst was accompanied by one vocal fold vibration. F_2 transition speed in the pre therapy utterance (u-d) was greater than in the poat therapy utterance.

F_2 transition from /i/ to /t/ was improper and the transitions indicated that the tongue approximated to /k/ again.

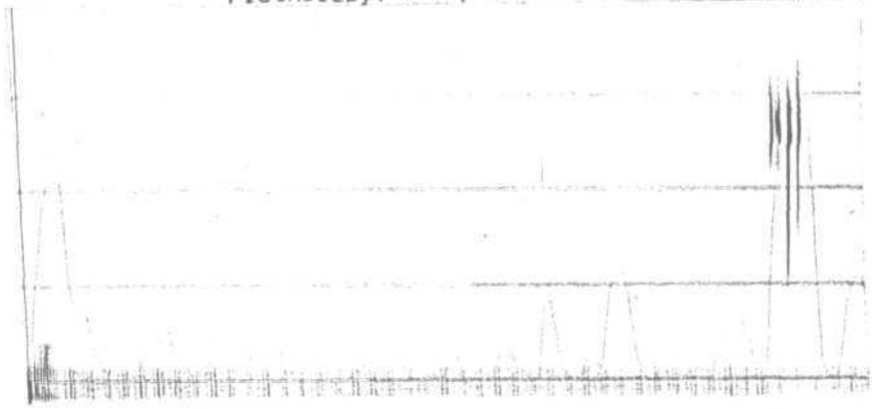
(ii) /kiṭaki/ (kkkkkkkkkkkkkiṭaki)

	Pre therapy	Post therapy
duration of /k/ (in syllable /kiṭa/)	75 (2)	115
duration of /i/	75 (2)	15
duration of /ṭ/	75 (2)	75
duration of /a/	50 (2)	35
duration of /k/ (in syllable /ki/)	120 (2)	60
duration of /i/	80 (2)	50
F_2 transition from /t/ to /a/	8.9hz/ms rise (2)	14.3hz/ms rise

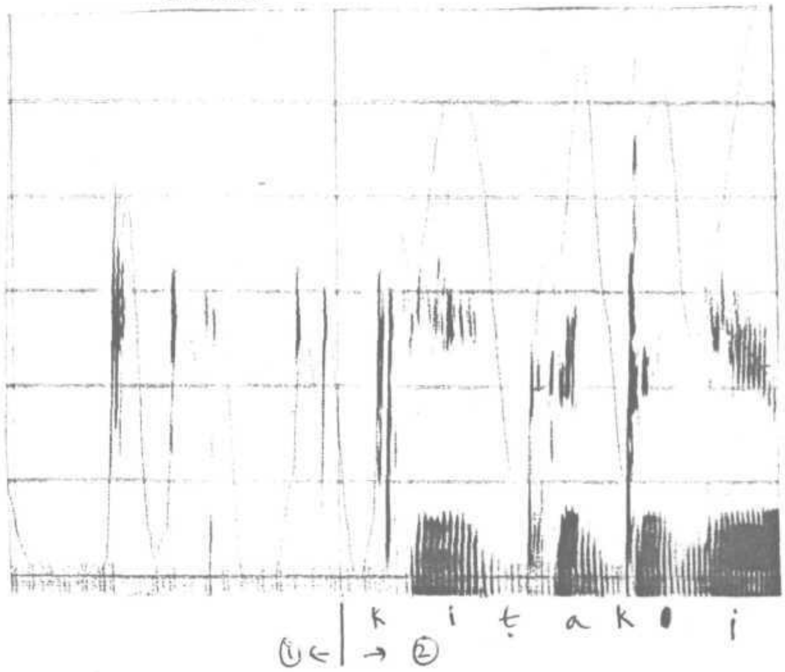
Table 26: Durations and transition speeds in the word /kiṭaki/ (in m.sec & Hz /m.sec respectively).

In the pre therapy utterance, all the segments except the initial /k/ were longer in duration.

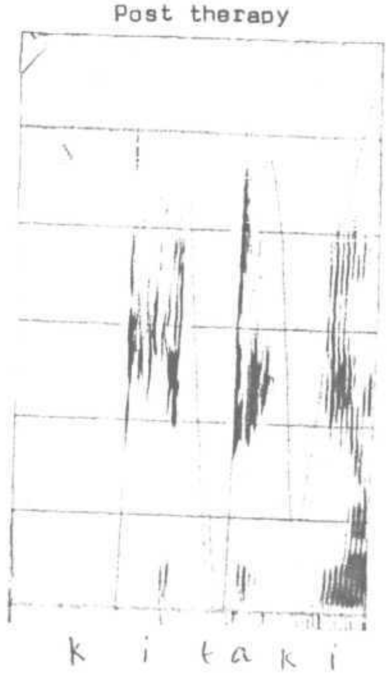
Pretherapy/ kiṭaki/



contd..



Post therapy



Also the /t/ to /a/ transition in the beginning of the word had a raising pattarn. However, the speed was low.

While attempting the initial /k/, for about 800 ms, some abnormal vocal fold activity was visible. Also possibly an abnormal supraglottal constriction occurred.

During this 800 ms there were about 12 uttarances of /k/

2) (i) /citrānna/ (c citrānna)

In the pre therapy utterance it was observed that the necessary F_2 transition from /c/ to /a/ was absent indicating the inability of the tongue to move from /c/ to /i/ position (1). However, this transition was noticed in 2.

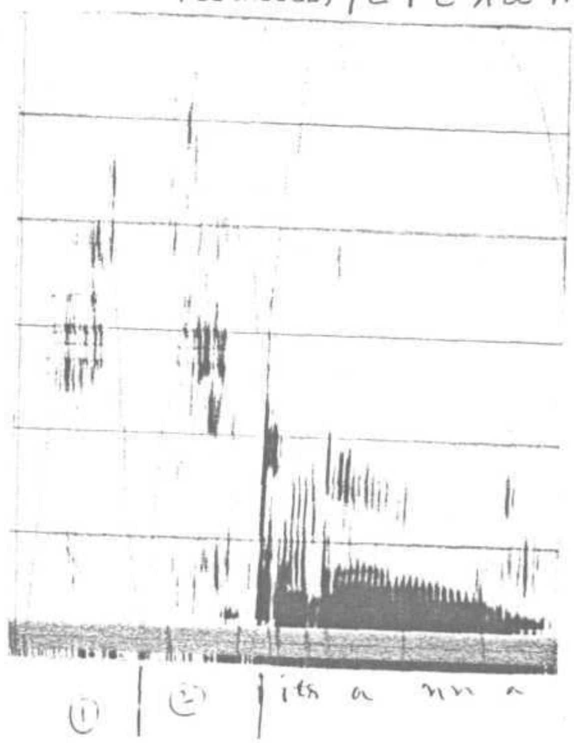
In the post therapy utterance vocal fold activity was noticed prior to application. This seems to be abnormal as /c/ is voiceless.

3) i) /sarōja/ (s sarōja)

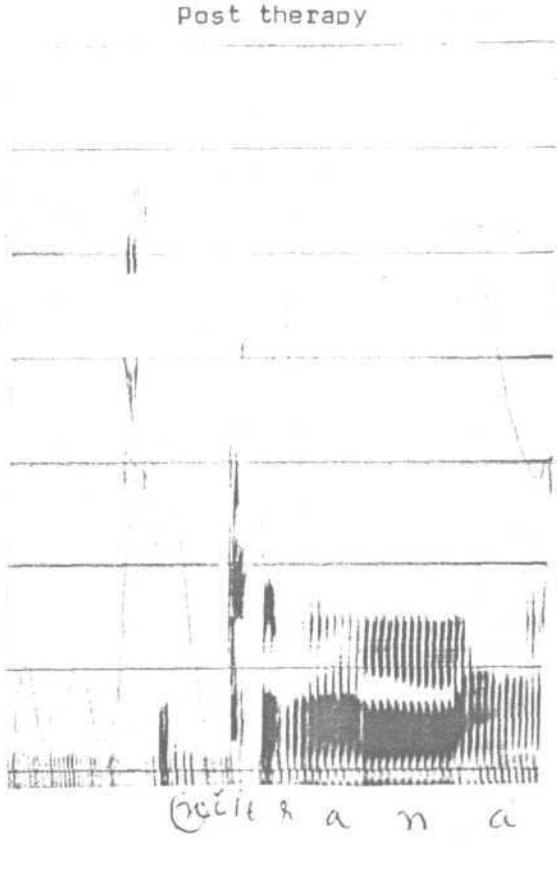
	Pre therapy	Post therapy
duration of /s/	120 (2)	160
duration of /a/	45 (2)	50
energy distribution of /s/	around 5 (1) around 3-6 (2)	5-7 (3)

Table 27: Duration & energy distributions in the word /sarōja/ (in m.secs & kHz respectively)

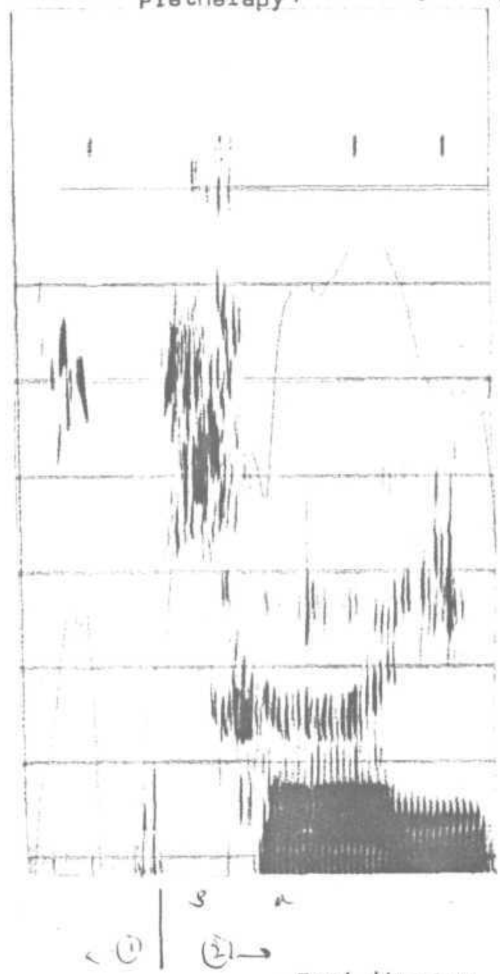
Pretherapy /citrānna/



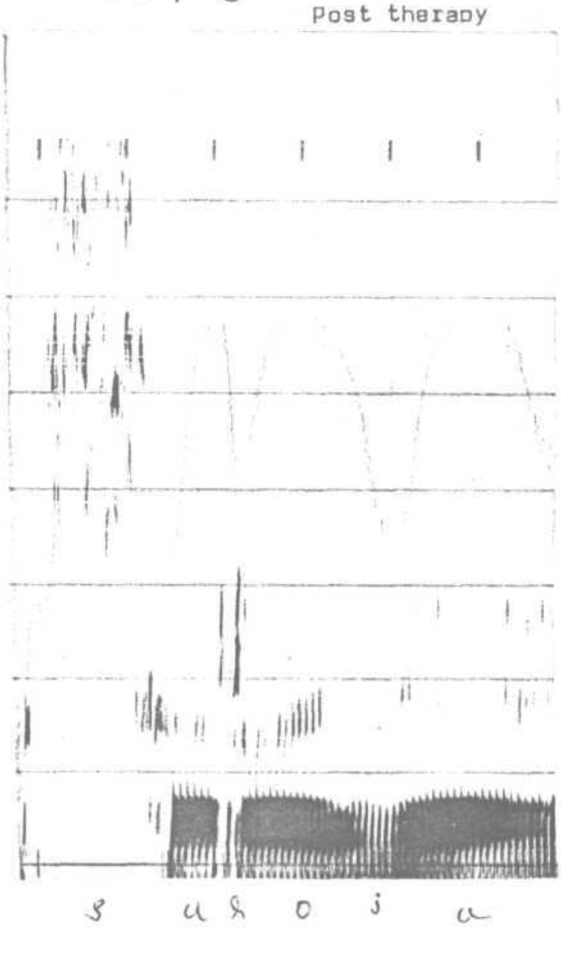
Post therapy



Pretherapy /saɾōja/



Post therapy



In the pre therapy utterance duration of /a/ use shorter and energy spectrum of /s/ was different compared to the post therapy utterance. /s/ in both region (1) and region (2) seem to involve abnormal muscle contractions resulting in abnormal constrictions and resonation.

/s/ in region (1) appears to show energy concentration around 5 KHz which indicates that tha precise constrictions in the oral cavity might not be formed. This could be due to the ballistic nature of the neuro-muscular machinery involved in the production of /s/.

(ii) /sirā/ (s ss ss sirā)

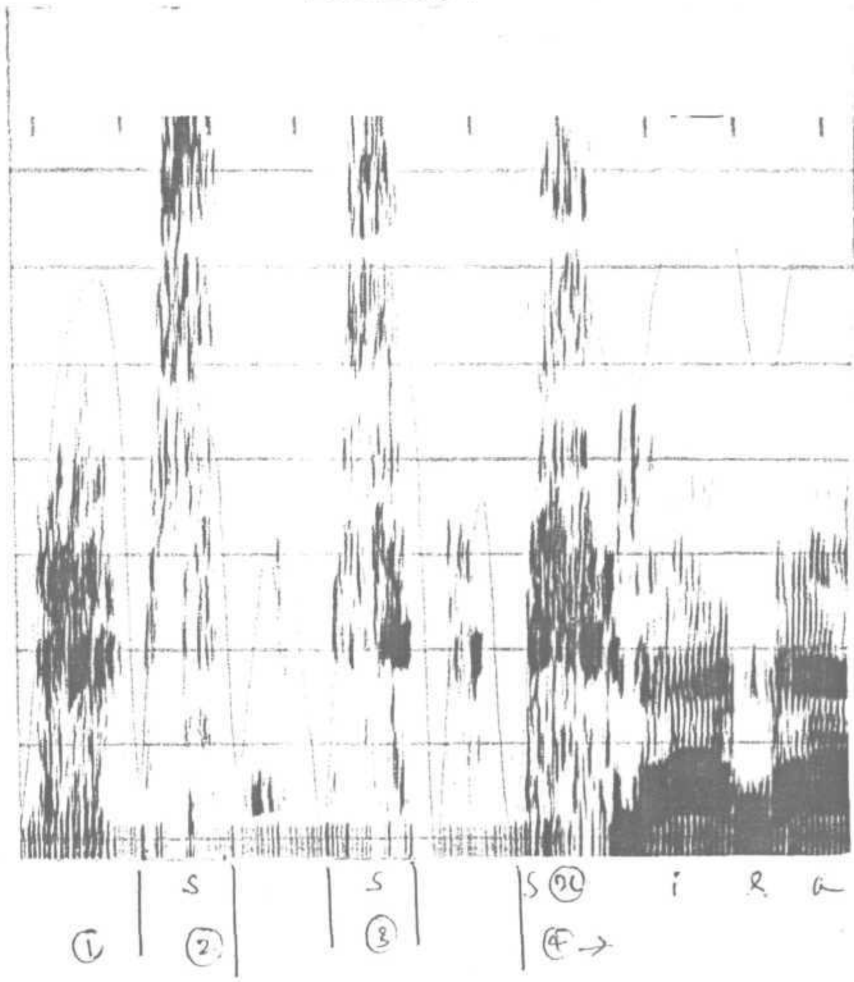
Speech sound	Duration in	
	Pre therapy utterance	Post therapy utterance
/a/	100 (1) 70 (2) 80 (3)	100 (4)
/i/	130 (4)	25
/r/	50 (4)	20
/a/	80 (4)	125

Table 28: Duration of segments in the word /sirā/ (in m.secs)

s.q. of lx waves at region (1)	S.Q. of one wave at point x
1.8 1.6 1.6 1.2 1.5 1.2 1.1 0.8	1.3

Table 29: Speed quotients of lx waves in the word /sirā/

Pretherapy /sira/



Post therapy

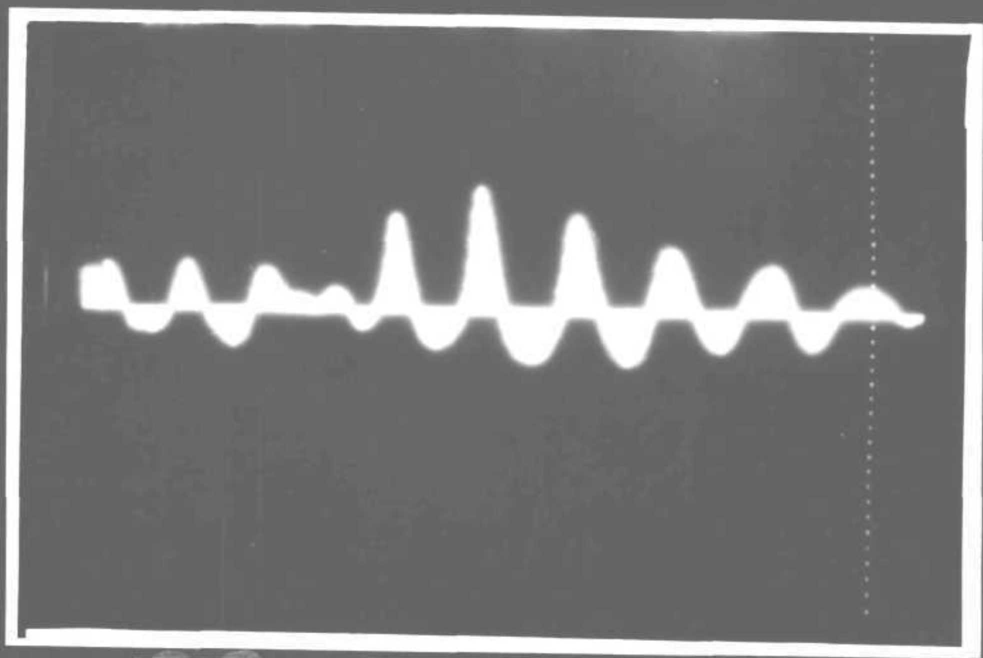


fig (20) sira: Lx cyles at region (1)

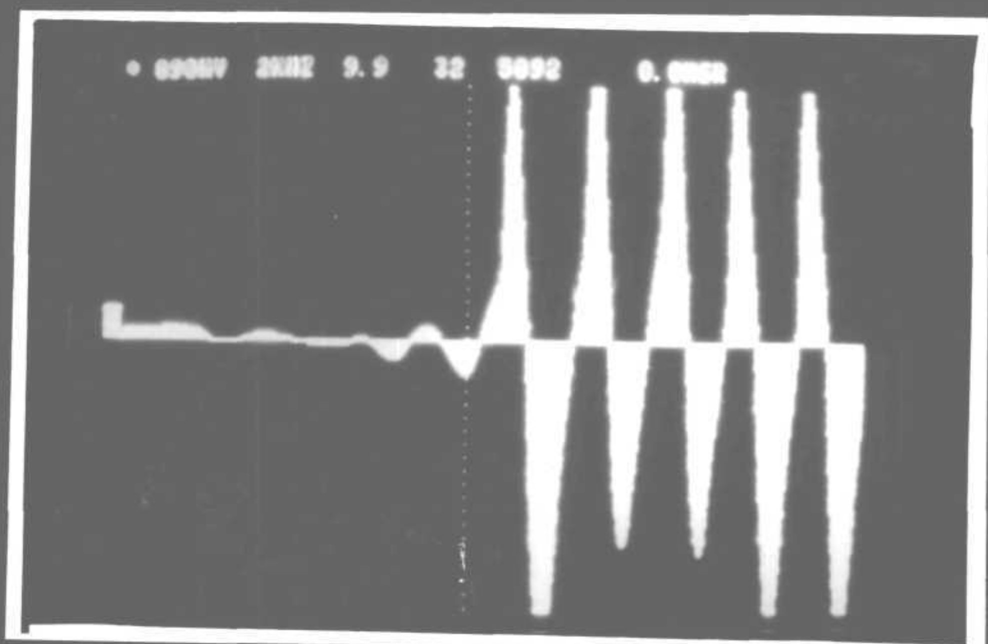


fig (21) sira: Lx cycles at point x in region (4)

In the pre therapy utterance vowel /i/ and trill were longer in duration and /a/ was shorter. Duration varied for /s/.

Energy spectrum of /s/ productions with a low frequency emphasis in the pre therapy utterance indicates an abnormal constriction (when compared to a normal post therapy /s/ production with energy in high frequencies).

In pre therapy utterance, vocal fold activity was present during the production of /s/.

Pre therapy Lx waveforms at region (1) and subregion x in region (4) are shown in figure (20 & 21). From Table 29, it can be observed that the SQ varied from cycle to cylce and was greater than one. Normal average S.Q. for vowel /i/ is 1.8 (Sridhara, 1986) which was observed in three cycles here.

The opening phase of one cycle in region (1) was peculiar. One cycle at point x showed a stairstep pattern in the opening phase.

Interestingly the Lx recording at subregion 'x' showed only one cycle despite several clear voicbars on the baseline on the spectrogram.

The Speech Initiation Time for the pra and the post therapy utterances are given in the table.

In the post therapy utterances, out of the 21 words, 12 words had lesser SIT's and nine words had greater SIT's when compared to the pre therapy utterances (Table 30).

	Word	Speech Initiation times in		Pre Post
		Pre therapy utterances	Post therapy utterances	
1)	inna	332.5	234.4	+ 99.1
2)	endu	291.4	262.5	+ 28.9
3)	ondu	490.6	187.5	+303.1
4)	māke	246.0	215.7	+ 30.3
5)	rāma	415.7	281.3	+134.4
6)	pāndi	339.8	206.3	+133.5
7)	gunda	360.1	196.8	+163.3
8)	bādūr	640.7	220.4	+420.3
9)	bahala	221.0	220.4	+ 0.6
10)	sudha	400	323.4	+ 76.6
11)	hole	473.4	323.5	+149.9
12)	mūru	261.7	257.9	+ 3.8
13)	tammana	139.8	267.1	-127.3
14)	kuri	260.9	375.0	-114.1
15)	bāl	89	290.7	-201.7
16)	jnapnasodu	168.7	1523.5	-1354.8
17)	gōvinda	187.5	286.0	-98.5
10)	smabāshare	191.4	300	-108.6
19)	sirā	185.1	379.7	-194.6
20)	ondu	190.2	290.7	-100.5
21)	eradu	200	239.1	-39.1

Table 30: Speech initiation times for different words (in m.secs)

The detailed analysis of the words has led to several common observations. They are as follows:

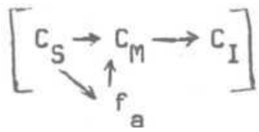
Respiratory

12 Words had a common feature, which was referred to as 'inhalatory frication' (If). 'If seems to appear under the following conditions:

1. Absence of an appropriate F_2 transition - a transition may be flat, speed of F_2 transition may be slow, or the direction of F_2 transition may be opposite, which indicates that the supraglottal positioning or the articulatory speed is in appropriate.
2. Distorted production of /s/.
3. Absence of voicing at required time instant.
4. Omission of a segment.

The duration of 'If ranged from 50 to 260 ms. The durations varied randomly or progressively decreased during subsequent occurrences in a single dysfluent word. 'If seems to cessate immediately after supra-glottal cavities assume a normal pattern for the particular segment.

It may be possible that an inappropriate feedback (f) from the peripheral musculature, as when it occurs for a inappropriate F_2 transition or for an abnormal constriction for /s/, triggers a brief 'surge' of activity in the neural cell-circuit controlling Inhalation (C_1). The peripheral trigger event may operate through a 'mediating cell-circuit' (C_M). Since the durations of 'If seems to vary randomly or in a systematic pattern'. The 'mediating cell-circuit' and 'cell-circuit' for inhalation may function independently from 'cell-circuits controlling the supraglottal muscles (C_S). However, a signal from the latter may affect the activity of the former, by acting as a cueing agent. In short, perhaps f_a may travel to C_M and trigger an unnecessary 'activity' in C_1. when f_a is replaced by normal or appropriate feedback (f_n) due to normal functioning of the C_S, the C_M may cue to assert the inappropriate activity in the C_I.

The fact that every stutterer does not show Inhalatory frications, perhaps suggest that each stutterer may have dis own unique 'neuronal-Information orocessing circuits' such as  ' accounting for the 'unnecessary events during speech that may be overtly apparent to a listner or not.

LARYNGEAL

Lx waveform was studied in 6 conditions:

i) during inhalatory frication

ii) during the production of a fricative

iii) during the production of a vowel (steady state)

iv) in vowel-consonant transition

v) in consonant-vowel transition

vi) When the voicebars on the spectrograms led to a suspicion of abnormal vocal fold activity.

Among the various observations, 3 observations seem to have been made earlier.

(a) Speed-quotient of vocal fold cycles is less than one in majority of the cycles studied. Only 3 cycles during fricative production had near normal (S.Q. = 1.8, Shridhara 1986) S.Q. values, and where S.Q. was greater than one, it was only 1.1 or 1.2 (during the production of the vowel /a/ in pre therapy fluent utterance in the syllable /ra/ in the word /rajamma/. This indicates greater closing phases painting to excessive adduction. Such finding was also reported by Conture, 1984; Conture et al, 1985. These authors had reported greater glottal adduction in the fluent utterances of nontreated young stutterers. The present study indicates higher adduction phases in adults during pre therapy nonfluancy. Pre therapy fluency and post theraoy fluency (Wrt normal value of 1.84, which is average S.Q. for /a/, /i/ and /u/).

(b) Vowel-consonant transition was studied in the word rajamma. The difference in S.Q. cycle to cycle gradation between pre and post therapy transition leads to the speculation that either ore therapy or post therapy transition tray be abnormal. Whether pre therapy pattern is abnormal is difficult to determine with the present data.

Conture et al (1986) compared VC transition of fluent utterances of untreated stutterers and normal speakers and reported a typical VC transitions. In the past, no study has used S.Q. gradients as an index to differentiate normal fluency from stutterers.

(c) Consonant-vowel transition was compared in a pre therapy, non-fluent utterance and fluent utterance in the word /nōḍidīvi/. In the former S.Q varied randomly but in the latter it remained constant. Here again, it is difficult to determine which is normal. Conture et al (1986) reported a typical CV transition for young untreated stutterer's fluent utterances.

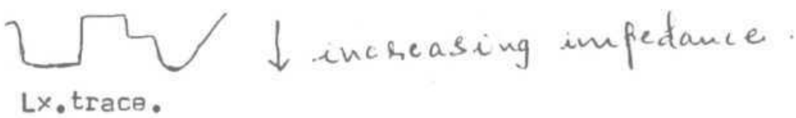
Other interesting observations not reported earlier are as follows:

1. S.Q of cycles varied randomly, when vocal fold vibrations were observed

during inhalatory frication. S.Q showed random variations during voiceless fricative production (an instance of inappropriate timing of voicing) and voiceless consonant during a pre therapy nonfluent utterance.

2. Opening phase of a few cycles during inhalatory frication and fricative production (on an exhalatory airflow) showed a peculiar stairstep pattern:

3. Opening phase in one cycle during exhalatory fricative/production showed a peculiar pattern (see fig.)



4. Few cycles during the expiratory frication production showed near normal S.Q's.

5. Vowel in the cost therapy utterance and consonant during the pre therapy nonfluent utterance showed kneepoints during closing phase, although only the former showed regular kneepoints (kneepoint always occurrad at 3.5 ms, Re: onset of closing phase).

6. a) weak voicebars on tha baseline of the spectrogram corresponded to total absence of 1x waveform.

   b) absence of acoustic energy as evidenced by gap on the spectrogram corresponded to clear properly shaped lx waveforms.

   c) several clear voicebars on the baseline of the spectrogram corresponded to only a single 1x. cycle.

These findings, possibly relate to the recently debated issue that speech production is a nonlinear acoustic generation system.

(Teager H. M. & Teager S.M. 1985)

7. Inappropriate timing of voicing was noted at various instances of ore therapy nonfluent utterances.

a) voicing in the post burst period of /k/

b) voicing in the gap period of /t/

c) voicing simultaneous with the burst for a plosive

d) voicing during an unvoiced frication /s/.

e) voicing simultaneously with the affrication instead of occuring a few milliseconds before for the voiced affricate /j/

f) absence of voicing during a voiced fricative.

g) absence of voicing during the production of /b/.

Disruption of temporal coordination between glottal and supraglottal events has been noted by several previous researchers. (Van Riper 1982, Mc Kay & Mc Donald, 1984).

SUPRALARYNGEAL

(1) In the nonfluent and fluent ore therapy utterances segmental durations

a) Were longer than the post therapy durations

b) Were shorter than post therapy durations

c) approximated progressively to the post therapy durations

d) deviated away progressively from the cost therapy durations.

e) varied in a random manner during the subsequent attempts within a single word, before uttering the fluent utterance.

Among the previous studied Metz et al, 1979; Metz et al, 1983; Prosek & Runyan,1983, and Ranig, 1984 reported shorter segmental durations in the pre therapy fluent utterances when compared to the post therapy utterances.

(2) Speeds of F_2 transitions were fastar or slower, equivalent or variable during the pre therapy fluent and nonfluent utterances when compared to the post therapy utterance. Some times transition directions were opposite in the pre therapy utterance. Also the F_2 transitions were flat when it was to be either rising or falling.

It appears that the articulator seems to move at inappropriate speeds, in wrong directions and becomes immobile sometimes.

On the basis of the acoustic studies of fluency of nontreated stutterers, Pindzola (1987) notes that as the stutterers shout faster articulatory movements, rate control methods seam logical. From the present study a clear pattern of faster pre therapy transitions becoming slower following therapy is not evident.

(3) Several instances of clear voicabars with an absence of energy in the higher frequencies perhaps points to

i) a supraglottal momentary obstruction that filters out all the high frequency energy. Such an obstruction also seems to act as an aperiodic energy source causing noisy additions to formant regions.

ii) this tissue obstruction might completely onstruct pulmonary aggressive airstream or open intermittently, resulting in abnormal airflows for brief periods.

iii) A third possibility is that there could ba an abnormal vocal fold vibration mode itself that generates very low energy harmonics.

iv) An explanation for the total absence of higher formants may also rest in the possible nonlinear speech production acoustics (Teager H.M. & Teager S.M., 1985).

A possibility of abnormal supraglottal constrictions was raised by Conture et al (1985), When thay noted that vocal folds were occluded from view during fiberscopy.

(4) Multiple bursts were observed for /p/ and /k/. Similar observation was reported by Conture (1984) in young preschool stutterers. However, this is to be expected as the production of /k/ and /p/ involves large articulators, the release of which may be part by part.

(5) Articulatory fixations occurred during Inspiratory frication.

(6) Several instances where, one out of the first two formants was inappropriate were observed. This leads us to speculate that tongue is perhaos unable to exert two vector forces in two different directions simultaneously. A forward movement may be appropriate or an upward movement of tongue may be appropriate but both movements may not be occuring simultaneously as required and as seen in cost therapy utterances.

(7) Articulatory constrictions for the production of /s/ were abnormal perhaps in location and absolute dimensions which resulted in low frequency emphasis in the spectrum of /s/.

These observations leads to hypothesize something about the neural-cell circuits operating during dysfluencies.

(1) Inappropriate F_2 transitions, which perhaps mean absence of particular displacement tongue at a particular time instant, may trigger a 'random surge of neuronal activity in the speech centers, causing inapiratory frications, abnormal vocal fold activity and supraglottal tissue obstructions'. With this in view, Valsalva hypothesis given by Parry (1985) appears possible at least in certain dysfluencies in some people if not in all the stutterers.

(2) A segment seems to be omitted when the speech mechanism is unable to produce that segment after repeated attempts.

(3) Stutterers might show omissions of segments just as those seen in functional misarticulations.

(4) Mistimed voicing tray trigger a silent pause, during which there is no voicing and no supraglottal effort to produce speech. The speech mechanism appears to be locked.

(5) Inappropriate formant transitions/articulatory gestures may arrest speech totally and fluent speech way follow immediately.

(6) Even when a vowel sounds perceptually normal if formant transitions are not of appropriate speed it is produced again till the appropriate speeds are attained. This observation is apparently conflicting with a previous observation where it was reported that fluent utterances prior to and after therapy may have widely different transition speeds.

(7) Articulatory and laryngeal events seem to occur even in the absence of perceived nonfluency.

All these are speculative in nature and are based on the observation of nonfluency in one patient and needs to be experimented in detail. Simultaneous research on the activity of the higher speech centres would also be of immense help in the understanding of stuttering.

SUMMARY AND CONCLUSION

SUMMARY AND CONCLUSIONS

A single case study was done on a 17 year old male stutterer. Speech dynamics during pre and post therapy utterances were studied and compared. Speech therapy consisted of attitude changing, initial segments, prolongation, use of soft contacts and airflow technique (Schwartz, M, 1976). Transfer phase was also carried out.

Totally 29 dysfluent words were analysed. Perceptually dysfluencias were of 5 types:

1) Intraword audible pause (Inhalatory frication)
2) Intraword silent pause
3) Whole word repetition
4) Intraword repetition
5) Phoneme repetition and prolongation

These words were elicited in the speech tasks of conversational speech, picture description, sentence and word-repetition. Concurrent audio and electrolaryngographic (Lx) recordings and concurrent audio and electroaerometric (mouth expiration) recordings were Tade. Wide-band bar type spectrograms with average amplitude function were taken for the audio recordings. Segment durations and formant transitions were measured at the apparently disrupted instances. When airflow patterns and/or voice bars on the baseline of the spectrogram were appearing abnormal, corresponding Lx and aerometric recordings were analysed for better understanding of speech dynamics at a particular time instance. Certain observations on the nonfluent pre therapy utterances are presented below:

(1) Inhalatory frications of varying durations (50 to 260 ms) and spectral characteristics was present.

(2) Speed quotient of vocal fold cycles was less than one in the majority of cycles studied. This indicated inappropriately longer closing phases pointing to excessive adduction.

(3) A typical CV and VC transitions of vocal fold cycles were observed.

(4) Random variation of S.Q from cycle to cycle was observed for vocal fold cycles during (i) inhalatory friction, (ii) fricatives & consonants produced on exhalatory airstream.

(5) Peculiar stairstep patterns in opening phases of vocal fold cycles during inhalatory frication.

(6) Abnormal Lx patterns during the opening phase of vocal fold cycles during exhalatory fricative production.

(7) Some paradoxical observations where Lx tracing and voice bars on spectrogram did not correlate were observed which perhaps relate to the issue of nonlinear mechanisms in the speech production acoustics.

(8) Inappropriate timing of voicing.

(9) A clear pattern of longer or shorter post therapy segmental durations is not evident.

(10) A pattern of faster pre therapy transitions becoming slower following therapy was not evident.

(11) Articulatory fixations occurred during inspiratory frication as indicated by the spectrograms.

(12) Abnormal articulatory constrictions for fricatives were indicated by the spectrograms.

The results seem to support the Valsalva hypothesis. Also they indicate several laryngeal, aerodynamic and articulatory abnormalities during nonfluency, which might be because of the abnormal speech musculature, abnormal neural impulses to the speech musculature or abnormally functioning higher speech centres. These abnormalities if exist, however, are momentary and need further detailed exploration.

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