ASSESSMENT OF BRAINSTEM DYSFUNCTION IN INDIVIDUALS WHO STUTTER

Reg.No. 02SH0003

An Independent Project as a part fulfillment of
First YearM.Sc, (Speech and Hearing),
Submitted to the University of Mysore,
Mysore.

ALL INDIA INSTITUTE OF SPEECH AND HEARING
NAIMISHAMCAMPUS.MANASAGANGOTHRI,
MYSORE-570006

JUNE-2OO3

DEDICATED TO,

APPA AND AMMA

THANKS MA, PA, FOR BEING THE MOST WONDERFUL PARENTS I COULD EVER HAVE. THIS GOES *OUT TO YOU* AS A SMALL TOKEN OF GRATITUDE FOR ALL THAT *YOU* HAVE DONE FOR ME.

CERTIFICATE

This is to certify that this Independent Project entitled "ASSESSMENT OF BRAINSTEM DYSFUNCTION IN INDIVIDUALS WHO STUTTER " is a bonafide work in part of fulfillment for the degree of Master of Science (Speech and Hearing) of the student **(Register No.02SH0003)**

DIRECTOR

Dr. M.Jayaram

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Mysore

June, 2003

CERTIFICATE

This is to certify that this Independent Project entitled "ASSESSMENT OF BRAINSTEM DYSFUNCTION IN INDIVIDUALS WHO STUTTER" has been prepared under my supervision and guidance. It is also certified that this has not been submitted earlier in any other University for the award of any diploma or degree.

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DECLARATION

This is to certify that this Independent Project entitled **"ASSESSMENT OF** BRAINSTEM DYSFUNCTION IN INDIVIDUALS WHO STUTTER" is the result of my own study under the guidance of **Mr. Animesh Barman,** Lecturer in Audio logy, Department of Audio logy, All India Institute of Speech and Hearing, Mysore and has not been submitted in any other University for the award of any degree or diploma.

Mysore,

June, 2003 **Reg. No.02SH0003**

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INTRODUCTION

Stuttering is a speech disorder, which has been defined as involuntary hesitations, repetitions and prolongation of sounds (Bloodstein, 1993). According to International Classification of Disorders stuttering refers to disorders in the rhythm of speech, in which the individual knows precisely what he wishes to say, but at thattime is unable to say it because of an involuntary repetitive prolongation / cessation of sound (WHO, 1977, cited in Andrews, Craig, Feyer, Hoddinnat, Howaiew & Neilson,1983).

It is seen in about 5% of the population and the incidence is highest during the preschool years. For at least 20% of these children, stuttering would persist (Curlee, 1993, cited in Curlee & Perkins, 1993).

Stuttering is a disorder with a multidimensional perspective. Several theories have been proposed to explain the cause and nature of stuttering. The theories broadly classify stuttering as:

a) a neurotic response (i.e. repressed need theory).
b) communicative failure and anticipatory behaviour,
c) a learned behaviour.

Apart from these, certain physiological deficits have also been attributed to stuttering. These also referred to as breakdown theories, characterize the moment of stuttering as an indication of failure or breakdown in the complex co-ordination required for fluent speech. Most of these theories assume that a person who stutters

has a constitutional predisposition towards stuttering, that is, precipitated by psychosocial or environmental stress and a reduced physiological capacity to co-ordinate speech. Such theories have postulated, perceptual, motor and central deficits (Andrews, Craig, Feyer, Hoddinatt, Howaiew & Neilson, 1983; Nicolosi, Harryman & Kresheck, 1996, cited in Shapiro, 1999).

These theories thus support the concept of organicity in stuttering. Several investigators have reported a number of organic etiological factors (as extracted from the article by Liebetrau & Daly,1981) such as:

- Genetic inheritance (Andrews & Harris, 1964 ; Records, Kidd & Kidd, 1976) unusual latent tetany (Weiss, 1967)
- Difference in neuromuscular control (Schwartz, 1974; Starkweather, Hirschman & Tannebaum, 1976)
- Atypical performance on neuropsychological tests (Daly & Smith, 1976; Daly, Kimbarrow & Smith, 1977)
- Lack of cerebral dominance (Curry & Gregory, 1969; Brady & Berson, 1976)
- Dysfunction of auditory processing and perceptual abilities (Hall & Jerger, 1978; Toscher & Rupp, 1978)

The impetus for comparing the auditory function of stutterers with nonstutterers has arisen from two major theories about etiology and possible sites of lesion for stuttering and stuttering behaviour.

Some researchers have used measures of auditory function to investigate cerebral dominance of language. This pertains to the theory proposed by Orton (1928, cited in Rosenfield & Jerger,1985) and Travis(1931) that stutterers do not develop complete dominance of the left hemisphere for language and/or for control of the motor activity for speech mechanism. Differences between stutterers and nonstutterers would suggest a possible site of lesion in a cortical area. However, some researchers have used measures of auditory function to investigate possible abnormality along the auditory pathway. This pertains to hypothesis that stuttering is related to problems with auditory feedback during speech production. Since majority of stutterers have normal hearing sensitivity, any problems with auditory feedback are more likely to be related to deficits in the central rather than peripheral auditory system (Stager, 1990). Differences between stutterers and nonstutterers on central auditory tests would locate a portion of the auditory system as being abnormal, portion that might then be linked with speech production system.

Investigators using tests of central auditory disorders in adult stutterers have found evidence of abnormality present in some stutterers at the brainstem level (Hall & Jerger, 1978; Hannley & Dorman, 1982; Stromsta, 1972; Toscher & Rupp, 1978), than at cortical level. On the SSI-ICM test, it was shown that stutterers score lower than nonstutterers (Hall & Jerger, 1978; Toscher & Rupp, 1978; Molt & Guildford, 1979). However conflicting results have also been obtained on this test (Hannley & Dorman, 1982; Kramer, Green & Guitar, 1987). It has also been shown that stutterers produce significantly poorer Masking Level Differences (MLDs) than nonstutterers (Kramer, Green & Guitar, 1987; Libetrau & Daly, 1981). Studies

investigating auditory processing dysfunction in stutterers using Auditory Brainstem Response(ABR) have yielded conflicting results. Studies on ABR in stutterers reveal increased interpeak latency differences especially between waves I and V (Blood & Blood, 1984; Stager, 1990). However some investigators have shown no significant differences between stutterers and nonstutterers in terms of interpeak latency intervals (Decker, Healy & Howe, 1982; Newman, Bunderson & Brey, 1985). As a group, stutterers did not show deviancy in latency or amplitude, but individually they differed on at least one measure of Auditory Evoked Potentials (Odekar, 2000; Stager, 1990).

Need for the study:

Thus there is no doubt that some of the stutterers do show organic involvement. Among those, some show abnormalities in auditory processing and perceptual abilities (Hall & Jerger, 1978; Toscher & Rupp, 1978) and some, more specifically show abnormality at the brainstem level (Hall & Jerger 1978 ; Hannley & Dorman, 1981; Stromsta, 1972; Toscher & Rupp, 1978). The auditory tests that have been carried out have mostly used subjective tests where results may be affected by several factors. Few researchers have made attempts to study organicity in stuttering, using objective tests. However the results of these tests are not conclusive. Hence more research is to be done to substantiate the findings and more than one test should be carried out to support results obtained from each test. Hence this study has been taken to probe further into organicity in stuttering, using audiological tests, which basically assess either lower or upper level of brainstem.

Also most of the earlier studies checking for brainstem abnormality in stutterers have used tests such as acoustic reflex threshold (Hall & Jerger, 1978), SSI-ICM (Hall & Jerger, 1978; Toscher & Rupp, 1978) and ABR (Blood & Blood, 1984; Stager, 1990). However in none of the studies, contralateral suppression of transient evoked otoacoustic emissions(TEOAEs) which is a measure to check the intactness of the afferent and efferent auditory system, has been used to assess central auditory processing at the brainstem level, especially in stutterers. Hence this test is included in the study along with two other well-known tests to assess brainstem dysfunction. Thus there is a need for further investigations to contribute in the direction of substantiating findings that there can be brainstem dysfunction in a subgroup of stutterers at least.

***Aim of the study**:*

- To detect a subgroup of stutterers who may have brainstem dysfunction.
- To assess the sensitivity of three tests that check for brainstem functioning, in detecting such a subgroup of stutterers. These include:
    - Masking level difference (MLD)
    - Contralateral suppression of transient evoked otoacoustic emissions (TEOAEs)
    - Auditory brainstem response (ABR).

REVIEW OF LITERATURE

Auditory tests have played two important roles in stuttering research

1. As a sensitive measure of cerebral dominance for language.
2. In the search for abnormalities of auditory feedback mechanism subserving speech production.

The following review gives evidence both positive and negative relating stuttering to abnormalities in each of these areas.

STUTTERING AND CEREBRAL DOMINANCE

Orton and Travis thesis

Orton (1928, cited in Rosenfield & Jerger, 1985) and Travis (1931) proposed that individuals stutter due to incomplete cerebral lateralization of language i.e., lack of lateralization to the appropriate hemisphere. As the child grows older, the language lateralization process becomes more complete and the disfluency disappears. However, some subjects retain their abnormal bilateral representation and continue to stutter.

As a result of the Orton Travis observation, many investigators addressed the prevalence of right and left-handedness among stutterers. Investigators derived conflicting data and arrived at disparate results (Bryngelson, 1935, 1939; Daniels, 1990; Despert, 1946, cited in Rosenfield & Jerger, 1985; Mc Allister, 1937, cited in

Rosenfield & Jerger, 1985; Meyer, 1945, cited in Rosenfield & Jerger, 1985; Milisen & Johnson, 1936; Spadino, 1941, cited in Rosenfield & Jerger,1985).

Tachistoscopic visual procedures:

All investigations with stutterers using this method (Hand and Haynes, 1983; Moore, 1976; Plakosh, 1978, cited in Moore, 1985), revealed that significantly larger proportion of stutterers had a left field (right hemisphere) preference.

Cerebral blood flow

Wood, Stumps, Mckeehan, Sheldon and Protor (1980) subjected two stutterers to cerebral blood flow measurements while reading aloud. They reported that both stutterers showed higher cortical blood flow in Broca's area on the right compared to the left hemisphere during stuttering. With regard to the posterior areas, greater cerebral blood flow was found in Wernicke's area in the left, compared to the right hemisphere, during stuttering. Interestingly, when subjects were fluent, greater flow was observed in the left hemisphere compared to the right. Finitzo, Pool, Freeman, Devous, & Watson(1991) found reduced blood flow in the frontal lobes of twenty stutterers in "recognized cortical regions of speech - motor control" as well as in left temporal regions of the brain.

Hemispheric alpha asymmetries

Moore and Lang (1977) reported a suppression of alpha waves over the right hemisphere in eight stutterers before each of several oral readings of a passage. In

nonstutterers, on the other hand, they noted less activity in the left hemisphere which accords with past findings on normal speakers engaged in linguistic processing.

Moore and Haynes (1980a & b) found reduced alpha in stutterers' right hemisphere for speech or pure tones. They suggested that stutterers demonstrate, not reversed cerebral dominance for speech, but right hemisphere processing for both verbal and non-verbal stimuli.

Mc Farland and Moore (1982, cited in Moore, 1985) and Boberg, Yeudall, Schopflocher and Bo-Lassen (1983) gathered hemispheric alpha asymmetry data from anterior and posterior brain sites before and after treatment. Prior to treatment, stutterers showed less alpha over right posterior frontal region while after treatment, there was less alpha over left posterior frontal regions. It was concluded that fluency accompanying treatment shifts alpha suppression from the right to left hemisphere.

In a further study of alpha asymmetry, Wells and Moore (1990) again found evidence of right hemisphere activation in stutterers during speech, using a sentence repetition task.

However, Pinsky and Mc Adam (1980) and Fitch and Batson (1989) reported that stutterers did not differ form their controls in alpha asymmetry during verbal processing tasks.

CT scan and PET scan

Strub and Black (1987) subjected two siblings with stuttering to a number of investigations including CT scan asymmetry measurements. Data showed abnormal cerebral dominance on variables investigated. CT scan showed atypical asymmetries especially in occipital regions.

Using PET, De Nil, Krol, Kapur and Houle (1995, cited in Guitar, 1998) found that when reading aloud, adults who stutter activated areas in the right hemisphere that were "mirror images" of left hemisphere areas used by non stutterers for the same purpose.

Braun and associates (1996, cited in Guitar, 1998) found in their PET study of people who stutter and controls, that when disfluent, stuttering subjects did not activate the left hemisphere areas that controls did. Instead, left hemisphere activity was absent and present in homologous areas of the right hemisphere or activity was seen in both hemispheres.

Fox et al. (1996) using PET scan, showed that stuttering induced widespread over activation of the motor system in both cerebrum and cerebellum, with right cerebral dominance.

Wu et al. (1997) using PET scan showed that stutterers showed significantly higher 6FDOPA uptake than normal controls in medial prefrontal cortex, deep orbital cortex, insular cortex, extended amygdala, auditory cortex and caudate tail supporting

the hypothesis that stuttering is associated with an overactive presynaptic dopamine system in the brain region that modulate verbalization.

Auditory evoked potentials

AEPs provide an objective measure to assess hemispheric processing.

Ponsford, Brown,Marsh and Travis (1975) used Auditory Evoked Responses (AERs) to investigate hemispheric differences between stutterers and non-stutterers. Potentials were evoked with meaningful words embedded in phrases. Stutterers showed a reversal of the normal trend, with greater differences in the right hemisphere and greater variance among subjects.

Zimmerman and Knott (1974) used the Contingent Negative Variation(CNV) method to investigate hemispheric differences in stutterers and nonstutterers. Results revealed differences between groups for frontal electrodes placed over Broca's area on the left and its contralateral homologue on the right. They stated that while processing verbal stimuli, stutterers appear to show more variable inter hemispheric relationships and do not show a shift that is consistently larger in the left hemisphere than the right.

This findings was not confirmed however, by Pinsky and Me Adam (1980) or by Prescott and Andrews (1984, cited in Bloodstein, 1995) and Prescott (1988, cited in Bloodstein, 1995), who found few differences between stutterers and nonstuttereres in the CNV.

Molt and Brading (1994) used a sixteen channel topographic brain mapping procedure to examine hemispheric patterns for dichotically presented consonant - vowel stimuli and noted the P300 and N200 components. No ear advantage was found between stutterers and nonstutterers. Stuttering subjects demonstrated significantly less cross-hemispheric amplitude difference for the P300 and N200 component. Pool, Freeman and Finitzo (1987) identified cortical dysfunction over the medial prefrontal and left temporal cortex in three stutterers using multichannel long latency evoked potential recording.

Finitzo, Pool, Freeman, Devous and Watson (1991) observed lower amplitudes in stutterers than nonstutterers, for all the three major components of the auditory evoked potentials.

Dietrich, Bary & Parker (1995) recorded middle latency responses from ten male stutterers and ten controls using a variety of filter pass bands in response to clicks presented binaurally at various rates. The latency of Pb wave was found to be significantly shorter in groups of subjects who stuttered. Hood (1987, cited in Dietrich, Bary & Parker, 1995) reported increased Pb latencies in the stuttering group.

Dichotic listening

Dichotic listening methods have generated the largest number of investigations in exploring hemispheric processing in stutterers.

Curry and Gregory (1969) used dichotic listening paradigm with twenty adult right-handed stutterers and twenty appropriate controls. The anticipated right ear

superiority was significantly less for stutterers than for non-stutterers. 75% of the non-stutterers had right ear scores that were higher than their left; this was true only for 45% of the stutterers. Quinn (1972, cited in Rosenfield & Goodglass, 1980) detected no reliable differences between the right-handed stutterers and matched controls.

Slorack and Noehr (1973, cited in Rosenfield & Goodglass, 1980) examined fifteen stutterers aged 6 to 9 years. They presented dichotic digit pairs and tested not only the free recall of digits, but also the performance on the instructed order of report from particular ears. The stutterers' scores were similar to those of controls. Gruber and Powell (1974) also failed to find significant interear differences for either stutterers' or controls' free recall reports.

Dorman and Porter (1975) evaluated sixteen right-handed adult stutterers and compared them to twenty controls. Subjects had to write down responses to synthetically generated consonant vowel dichotic stimuli. There was no significant difference between stutterers and nonstutterers.

Pinsky and Mc Adam (1980) tested five adult stutterers and five fluent speakers in a dichotic listening paradigm. All individuals were right handed except one, who was stated to be "weakly right-handed". The authors failed to find a significant difference between stutterers and nonstutterers.

Sussman and Mac Neilage (1975) employed a dichotic test paradigm and pursuit auditory tracking. The authors noted a right ear advantage for both

nonstutterers and stutterers on dichotic studies. In tracking paradigm however, normals had a right ear advantage whereas stutterers did not. Neilson, Quinn and Neilson (1976, cited in Bloodstein, 1995) subsequently confirmed the observations of Sussman and Mac Neilage(1975) in the pursuit auditory tracking for normal speakers, but found no differences between normal speakers and stutterers.

Bhat (1999) tested twenty adult male stutterers in the age group of 17-30 years on the dichotic CV paradigm at various lag times of 0, 30 and 90 msecs. Of the twenty stutterers, five were mild, nine moderate and six severe grade stutterers. Significant right ear preference was not demonstrated by stutterers at 0 and 30 msec lag time. Also, scores were seen to diminish with increase in severity of stuttering.

STUTTERING AND AUDITORY FEEDBACK

The notion that stuttering might be due to a defect in the auditory feedback mechanism subserving speech production has been widely researched upon.

The acoustic reflex

Shearer and Simmons (1965) investigated the stapedius muscle activity in stutterers and non-stutterers during ongoing speech. They observed that stapedius muscle activity tended to parallel vocalization in non stutterers. In stutterers, however, parallelism was less consistent. At times the onset of stapedius activity seemed to be delayed relative to the onset of vocalization. In general, however, differences between the groups were not striking. Hall and Jerger (1978) compared the acoustic reflex to external sound in stutterers and controls. Reflex threshold was equivalent in the two groups, but reflex amplitude was smaller in the stuttering group.

Hannley and Dorman (1982) however failed to note any differences between the stutterers and non stutterers.

Phase Disparity

Another approach to the question of intrinsic abnormality in the stutterers' auditory monitoring system is to study phase disparities between air and bone conducted tones. Stromsta(1957, cited in Rosenfield & Jerger, 1985) conducted a study in which stutterers and non stutterers listened to an air conduction tone introduced to the ear and to a bone conduction tone of the same frequency introduced at the teeth. Subjects were asked to vary the phase and amplitude of the air conducted tone until a critical adjustment was achieved at which, no sound was audible to them. There was significant difference observed between stutterers and non stutterers in the relative phase angle of air and bone conducted sounds at 2 kHz. Using a similar method, Stromsta (1972) noted an unusual phase disparity between stutterers and left and right ears.

Mangan (cited in Gregory & Mangan, 1982) replicated Stromsta's earlier study and failed to find a difference between stutterers and non-stutterers in the phase and amplitude adjustments of air and bone conducted sounds needed to obtain a null.

CENTRAL AUDITORY DYSFUNCTION:

At an early date, theories linking stuttering and defect in auditory feedback mechanism focussed the attention of researchers on the clinical integrity of the stutterers' central auditory system. The available diagnostic tests were soon put to use. It was assumed that stutterers have disturbed auditory feedback loops that occur

as a result of Central Auditory Processing deficit at some level of auditory functioning.

Rousey, Goetzinger and Dirks (1959, cited in Rosenfield & Jerger, 1985) reported that stuttering children did not perform as well as nonstutterers in making median plane sound localization responses.

Gregory (1964) found that adult stutterers did not differ for the most part from non-stutterers on tests of sound localization, binaural loudness balance and discrimination of speech distorted by frequency filtering.

Hall and Jerger (1978) compared the performance of stutterers and nonstutterers on a battery of seven tests of central auditory processing - the acoustic reflex threshold, performance intensity function for monosyllabic phonetically balanced (PB) word lists (PI-PB), performance-intensity functions for synthetic sentences (PI-SSI) synthetic sentence identification with ipsilateral and contralateral competing message (SSI-ICM), (SSI-CCM) and the staggered spondiac word test (SSW). On most of these, the stutterers' responses were normal. There were small differences, however on three tests that are especially sensitive in detecting central auditory processing deficit- the acoustic reflex amplitude function, SSI-ICM and the SSW test. Although the overall pattern of the stutterers' test findings failed to suggest substantial central auditory disorders' Hall and Jerger stated that the pattern was suggestive of subtle central auditory disorders at the level of the brainstem.

A study by Toscher and Rupp (1978) using identification of synthetic sentences with ipsilateral competing message (SSI-ICM) corroborated the findings of Hall and Jerger. The SSI-ICM test is a sensitive index of brainstem auditory function.

Molt and Guilford (1979) also obtained findings essentially identical to those of Hall and Jerger on the SSI test. With contralateral competing message the stutterers and nonstutterers did not differ but with ipsilateral competing message, stutterers scored lower than nonstutterers. Only Hannley and Dorman (1982) and Kramer, Green and Guitar (1987) found no difference on this test in their studies.

A variety of other tests of central auditory function have also been employed. On a sound fusion task, Bonin, Ramig and Prescott (1985) found that stutterers appeared to require longer time interval between sounds before they heard them as different sounds.

Anderson, Hood and Sellers (1988) obtained negative results with phonemic synthesis test which evaluates a subject's ability to fuse separate phonemes into words and on Binaural Fusion test, in which high and low frequency components of a spondaic word are presented simultaneously to different ears.

Meyers, Hughes and Schoeny (1989) found that stutterers did not differ from controls in judging which ear received the stimulus first when the syllables were presented in pairs to both the ears with different degrees of asynchronization between the ears.

Harris, Fucci and Petrosino (1991) found that in scaling the magnitude of tones of different intensities, stutterers tended to use a restricted range of numerical values.

A great focus of research interest has centered on the brainstem as a possible site of central auditory system dysfunction in stutterers. An innovative technique for detecting central auditory brainstem pathology is a psychoacoustic phenomenon known as MLD. Liebetrau and Daly (1981) examined the central auditory processing abilities of groups of "organic" and "functional" stutterers and control subjects with MLD task. They found that "organic"stutterers (those who evidence some neurophysiological deficits) performed significantly poorer than the control group and functional stutterers (those showing no evidence of organicity) performed similar to normal speakers.

Kramer, Green and Guitar (1987), found that stutterers produced significantly ($p<0.01$) poorer MLDs than the nonstutterers. This may be interpreted as support for the hypothesis put forth by Kent (1983, cited in Kent,1984), that stutterers may be poorer at temporal processing.

Researchers have speculated that temporal processing of incoming signals is a particular weakness of stutterers who perform poorly on central auditory tests. They have tried to link this to stuttering by suggesting that a single mechanism in the brain may control functions for both incoming and outgoing signals (Kent, 1983,cited in Kent ,1984). Faulty processing of the temporal dimensions of incoming signals would

give rise to stutterers' poorer performance on central auditory tests. Faulty processing of out going signals would result in stuttering.

Auditory brainstem response

Since a great focus has been laid upon brainstem being the possible site of lesion in stutterers, more research has been done on early latency potentials known as auditory brainstem response, with conflicting results. Blood and Blood (1984) performed brainstem evoked response testing on eight adult stutterers (four severe and four moderate) and eight nonstutterers. Stutterers demonstrated prolonged central conduction time as measured by inter peak latency (IPL) differences between waves I to V. Five of the stutterers manifested abnormalities unilaterally while three showed abnormal responses bilaterally. No relationship was found between brainstem evoked response testing and severity of stuttering.

Newman, Bunderson and Brey (1985) obtained auditory brainstem electrical responses of right and left ears of active stutterers, recovered stutterers and nonstutterers, both male and female adults at click rates of 11.1 and 71.1/sec. Latency intervals of waves I, III and V were measured. No significant differences were obtained between stutterers and nonstutterers. However female subjects (stutterers and nonstutterers), showed faster neural conduction times than males.

Decker, Healy and Howe (1982) compared between stutterers and nonstutterers, latencies of waves I, III, and V, interepeak latency differences between waves I and V, the amplitude of wave V and the comparison between right and left

monoaural stimulation waveforms with binaural stimulation waveforms. No abnormality in the response of stutterers was observed.

Smith, Blood and Blood (1990), recorded the brainstem-evoked responses when subjects were engaged in overt speech, whispering, silent articulation and covert verbal rehearsal tasks. Results revealed that stutterers demonstrated significantly larger wave V to wave I amplitude ratio than nonstutterers. However no significant differences were found between stuttering and nonstuttering subjects for absolute/interpeak latencies of the waves during the verbal rehearsal tasks.

Stager (1990) measured interpeak latency differences between waves I and V, amplitude ratios between waves V and I and latency shifts in wave V between low and high stimulus repetition rates in ten male stutterers and twelve male nonstutterers (with normal hearing sensitivity). As a group stutterers did not differ significantly from nonstutterers on any of the measures. Individually half the stutterers demonstrated latencies greater than 2 SD from nonstutterers' means on at least one measure.

Odekar (2000) carried out an investigation aimed to study the evoked potentials in stutterers. Sixteen stutterers, ten males and six females between the ages six to thirty years participated in the study. The results obtained did not indicate deviant amplitude and latency measures on any of the evoked potentials recorded for stutterers as a group. However inspection of individual data revealed that nine out of sixteen stutterers who participated in the study showed deviancy on at least one

measure of AEPs. With respect to ABR, two subjects showed reduced V/I amplitude ratio. One subject did not demonstrate ABR peaks at higher repetition rates.

METHOD

The present investigation aimed to study brainstem dysfunction in stutterers using the following tests.

- Masking level difference (MLD)
- Contralateral suppression of transient evoked otoacoustic emissions (TEOAEs)
- Auditory brainstem response (ABR)

SUBJECTS:

Sixteen male stutterers in the age range of 18-35 years participated in the study. For comparision , sixteen nonstutterers matched for age and sex were taken as the control group.

Selection criteria:

No history or present complaint of hearing loss or any other otological problem.

No concommitant speech and language problems.

No history of any neurological disease or gross neurological problems.

No intellectual deficits.

No difficulty in understanding speech in the presence of noise.

Hearing within normal limits (thresholds within 15 dBHL at octave frequencies between 250 Hz to 8 kHz).

"A" type tympanogram with normal reflexes on immittance.

INSTRUMENTATION:

PURE TONE AUDIOMETRY: A calibrated Madsen OB822 audiometer with TDH-39 earphones lodged in MX-41 AR ear cushions was used to assess hearing sensitivity.

IMMITTANCE AUDIOMETRY: A calibrated Grasen and Stadler-33(version 2) middle ear analyser was used to find out the middle ear status.

MASKING LEVEL DIFFERENCE: A calibrated Grasen and Stadler audiometer (GSI-10) with TDH-50 earphones mounted in supra-aural ear cushions was used.

OTOACOUSTIC EMISSIONS: TEOAEs were measured using ILO-292 DP Echoport plus. For contralateral suppression of TEOAEs, noise was presented to the contralateral ear using the insert reciever of a calibrated OB822 audiometer.

AUDITORY BRAINSTEM RESPONSE: The electrophysiological unit,Nicolet Bravo,with the following accessories was used to record ABR.

- Disc electrodes for recording the potentials.
- TDH-39 earphones with MX-41 AR ear cushions to present the stimulus.

TEST PROCEDURE:

Pure tone audlometry was conducted to ensure normal hering sensitivity at octave frequencies between 250Hz to 8kHz.

Immittance evaluation was then performed on the subjects to check for normal middle ear functioning indicated by a static compliance value between 0.5-1.75ml, peak pressure, -100 daPa to +60 daPa and presence of both ipsilateral and contralateral reflexes with in 100 dBHL for frequencies 500Hz,1000Hz and 2000Hz.

Masking level difference:

To obtain masking level difference values,each subject was presented binaurally with a narrow band noise of 50dBHL centred around 500Hz (as ear is more sensitive to phase differences at low frequencies) and 500 Hz pulsed tones with on and off time of 200msec under the following conditions.

(i) Homophasic (NoSo)- when both noise and signal are in phase at the two ears.

(ii) Antiphasic(NoSπ)- when phase of the signal is reversed at the two ears.,

(iii) Antiphasic (NπSo)-when the phase of the noise is reversed at the two ears.

Difference between the amount of noise required to mask the signal in homophasic and antiphasic conditions which gives the masking level difference was compared between stutterers and non stutterers.

Contralateral suppression of transient evoked otoacoustic emissions:

Patients were made to sit comfortably on a chair in a sound treated room. The probe with a tip was positioned in the external ear canal and adjusted to obtain almost flat frequency stimulus spectrum across the frquency range.

Data was obtained in two phases.

1. TEOAE response for 260 sweeps of clicks was averaged at intensity between 75-80dB SPL. This was considered as the baseline TEOAE response.

2. TEOAE was measured once again by presenting WBN in the contralateral ear at 40dBSL without altering the position of the probe, through the insert reciever to avoid any chances of cross over.

The difference between the baseline TEOAE amplitude and the TEOAE amplitude measured in the presence of contralateral noise was considered as the the amount of TEOAE suppression.

A minimum of one minute time gap was given between any two recordings to reduce the influence of one recording over another.

For a few subjects,the order was reversed with OAEs being measured in the presence of noise first and then taking baseline.

Auditory brainstem response:

ABR was recorded for each ear at three repitition rates. The electrode sites chosen were :

SITE	POSITION	TYPE OF ELECTRODE
Forehead	F2	common
Left mastoid	Al	inverting
Right mastoid	A2	inverting
Vertex	Cz	noninverting

The following two conditions were taken care of:

Electrode impedance at each electrode site-<5Kohms

Interelectrode impedance-<2Kohms , to arrive at a better response.

Stimulus parameters:

Stimulus: click

Polarity: rarefaction

Repitition rate: 11.1/s , 65.1/s , 90.1/s

Filter setting: 100 Hz - 3 KHz

Montage: Cz/Al ; Cz/A2

Transducer: headphones

No.of sweeps: 1500

Intensity: 80dBnHL

Instuction: Subjects were asked to sit comfortably on the chair and relax. They were instructed to avoid extraneous movements of head,neck and jaw during the recording of potentials.

The following parameters were studied from ABR

Absolute latencies for waves I,III and V

Interpeak latency difference between waves I-III, III-V and I-V

Amplitude ratio between waves V and I.

These were measured at the three repitition rates.

Data obtained from all these tests were then subjected to suitable statistical analysis to reach the aim.

RESULTS AND DISCUSSION

Data obtained from the tests were analysed in two steps.

1. Comparision of data obtained between stutterers and non stutterers.

MLD values, amount of contralateral suppression and the ABR results, obtained from stutterers and nonstutterers were subjected to analysis using paired "t" test, to check for the significance of difference between the data obtained by the two groups.

2. Estimation of the sensitivity of the three tests in detecting brainstem dysfunction in a subgroup of stutterers.

Scores obtained by each subject were compared with age and sex matched nonstutterer. The performance of the subjects across the three tests was descriptively analysed to determine which test was more sensitive in identifying brainstem pathology.

Masking level difference

MLD was computed for each of sixteen stutterers and sixteen nonstutterers. Values obtained are as follows.

Table 1: Depicts the Mean (M), Standard Deviation (SD) and 't' values of MLD obtained by stutterers and nonstutterers.

	M	SD	t
MLD(s)	11.7188	1.5052	1.159 NS
MLD(ns)	12.5000	1.8257	

s - stutterers ns - nonstutterers NS - not significant

Values obtained from stutterers were lesser than those obtained from nonstutterers. However this difference was not statistically significant. Liebetrau and Daly (1981), observed a similar pattern of results in functional stutterers. However they noted a significant reduction in MLD values in organic stutterers (those with neurophisiologicaldeficits). The findings of the present study are however in contrast to the findings of Kramer, Green and Guitar (1987) who found statistically significant difference on MLD measurements between stutterers and non stutterers.

MLD is a direct measure of the intactness of temporal processing. The release from masking phenomena is mediated at the level of lower brainstem. Thus MLD indirectly reflects the functioning of the lower brainstem. Hence the findings obtained in the present study contradict the hypothesis put forth by Kent (1983, cited in Kent, 1984) that stutterers may be inherently poor at central temporal processing. Also stutterers may not have abnormalities at the level of the lower brainstem. The anomaly could be at a higher level.

Contralateral suppression of TEOAEs:

The results obtained by stutterers and nonstutterers are shown in the table below.

Table 2: Depicts the Mean (M), Standard Deviation (S.D) and 't' values on contralateral suppression of TEOAEs obtained in stutterers and non-stutterers.

	Ear	Subjects	M	SD	t
Overall suppression	R	s	1.53	1.08	0.145 (NS)
		ns	1.59	0.84	
	L	s	1.17	0.86	1.254 (NS)
		ns	1.49	0.93	

s - stutterers : ns - nonstutterers NS - not significant

TEOAE amplitudes were measured both in with and without noise (baseline response) conditions. The overall suppression in TEOAE amplitude was measured. Stutterers showed relatively lesser suppression than non stutterers.However this difference was not statistically significant.

Sound induced suppression of OAEs is a normal phenomenon mediated by the efferent auditory system. The stimulus is presented to the contralateral ear, to observe the suppression in the probe ear. The stimulus passes through the cochlea,auditory nerve and reaches to the Superior Olivary Complex which is situated in the lower brainstem. Impulses then activate the contralateral efferent Medial Superior Olivary

Complex bundle, thus resulting in reduction in the activity of Outer Hair Cells in the probe ear and hence suppression in OAE amplitude is seen.

The present study did not show any significant difference in contralateral suppression when stutterers taken as a whole were compared to non stutterers. Hence this suggests that stutterers as a group may not show any abnormality related to the lower brainstem with respect to both efferent and afferent systems as discussed above.

Auditory brainstem response

The results are tabulated as shown in the following page.

Table 3: Depicts the Mean, Standard Deviation (SD) and 't' values of ABR obtained in stutterers and non-stutterers at three repetiton rates.

	11.1				65.1				90.1			
	R		L		R		L		R		L	
	s	**ns**	s	ns	s	**ns**	s	ns	s	ns	s	ns
Peak I												
Mean	1.67	1.79	1.67	1.77	1.77	1.85	1.74	1.80	1.79	1.73	1.88	1.96
SD	**0.18**	0.21	0.13	0.17	0.14	0.16	0.13	0.13	0.08	0.08	0.14	0.13
t	2.07**		3.40*		1.28 (NS)		1.10 (NS)		0.28 (NS)		1.09(NS)	
Peak III												
Mean	3.71	3.81	3.70	3.78	3.85	4.01	3.91	3.95	4.06	4.14	4.01	4.51
SD	1.27	0.17	0.14	0.15	0.18	0.18	0.16	0.14	0.32	0.20	0.18	0.84
t	1.64 (NS)		1.46 (NS)		2.58**		0.56 (NS)		0.93 (NS)		2.02 (NS)	
PeakV												
Mean	5.63	5.63	5.54	5.52	5.96	5.96	5.97	5.89	6.13	6.14	6.16	6.09
SD	0.17	0.25	0.19	0.26	0.19	0.21	0.18	0.20	0.22	0.23	0.22	0.28
t	0.03 (NS)		0.28 (NS)		1.39 (NS)		0.07 (NS)		0.17 (NS)		0.83 (NS)	
IPL I-III												
Mean	2.03	2.02	2.04	2.02	2.02	2.19	2.10	2.11	2.05	2.29	2.10	2.14
SD	0.20	0.20	0.17	0.13	0.14	0.19	0.20	0.14	0.05	0.33	0.22	0.13
t	0.03 (NS)		0.43 (NS)		1.64 (NS)		0.13 (NS)		1.37 (NS)		0.62 (NS)	
IPL III-V												
Mean	1.91	1.81	1.74	1.85	2.06	1.95	1.99	1.91	2.10	2.00	2.09	1.96
SD	0.17	0.18	0.16	0.21	0.13	0.11	0.15	0.13	0.14	0.13	0.19	0.16
t	1.70 (NS)		1.73 (NS)		3.09*		2.27**		3.12*		2.03 (NS)	
IPL I-V												
Mean	3.94	3.84	3.89	3.73	4.08	4.17	4.16	4.06	4.16	4.28	4.16	4.10
SD	0.27	0.23	0.27	0.30	0.16	0.29	0.27	0.21	0.11	0.34	0.29	0.25
t	1.28 (NS)		1.66 (NS)		0.69 (NS)		1.40 (NS)		0.78 (NS)		0.59 (NS)	
Amplitude ratio												
Mean	2.12	2.07	2.08	1.84	3.74	1.86	2.51	2.77	3.09	2.35	2.89	2.92
SD	1.50	1.13	1.16	0.84	2.82	0.34	1.26	1.69	1.72	0.48	2.00	1.29
t	0.10(NS)		0.56(NS)		1.53(NS)		0.38(NS)		0.77(NS)		0.03(NS)	

*p<0.01 **p<0.05 NS Not significant

It can be seen from the table that stutterers as a group did not differ from nonstutterers on most of the parmeters of ABR. At lower repitition rates statistically significant difference ($p<0.05$) was found between the wave I latencies of stutterers and nonstutteres. Stutterers showed shorter wave I latency when compared to that of the nonstutterers. At higher repitition rates,the difference was mainly with respect to interpeak latency differences more so between waves III and V and this was statistically significant ($p<0.05$) with stutterers showing greater III - V interpeak latencies than nonstutterers. However the absolute latencies of waves III and V did not show statistically significant differences between stutterers and nonstutterers. Hence clinical importance of the statistically significant difference in III -V interpeak latencies at higher repition rates is questioned. Also no statistically significant difference was found in other absolute or interpeak latencies. The V/I amp lite ratio also did not reveal any statistically significant difference at all the three repitition rates.

ABR measures the functioning of afferent auditory brainstem pathway. It represents electrical activity generated in response to auditory stimulation, by the eighth cranial nerve, within the brainstem, upto the level of lateral lemniscus and inferior colliculus. The findings here do not clearly reveal any abnormality in this pathway in stutterers.

Hence when stutterers were compared as a whole with nonstutterers, statistically significant difference was not obtained. Similar results were obtained by Stager (1990), who attributed this finding to heteroginity seen in stuttering . Newman, Bunderson and Brey (1985) also did not find statistically significant

difference between stutterers and nonstutterers,in the interpeak latencies, at both lower and higher repitition rates. Even Decker, Healy and Howe (1982), in their study did not find statistically significant difference in interpeak latencies and amplitude of wave V between stutterers and non stutterers.

Thus from the above discussion, it is clear that stutterers as a group did not show statistically significant abnormalities on all the three tests.

Hence to reach the aim of finding a subgroup of stutterers having brainstem dysfunction, results obtained by each stutterer were discussed in comparison to the data obtained by age and sex matched nonstutterer.

The results of such an analysis are tabulated as shown.

Table 4: Depicts the severity of stuttering and values obtained by stutterers and non-stutterers on all the three tests

Subject	Severity	Contralateral suppression of TEOAEs				MLD				ABR(I-V IPL) at 11.1, 65.1 & 90.1 repetition rates			
		NS		S		NS		S		NS		S	
		R	L	R	L	NoS_o-NoSπ	N_0So-NπSo	N_oSo-NoSπ	NoSo-NπSo	R	L	R	L
1	Mild	0.6	2.8	1.9	0.9	12.5	12.5	10	7.5	3.86	3.86	4.00	3.74
										-	4.12	4.28	
											4.02	-	
2.	Mild	1. 5	0.5	**0**	0.9	12.5	10	12.5	10	4.20	4.08	4.02	3.48
										4.28	4.26	4.12	4.08
										4.42	4.30	4.12	3.78
3.	Severe	3.3	2.7	3.1	2.5	12.5	12.5	12.5	10	3.88	3.86	4.38	4.30
										4.20	4.18		4.60
											4.42	-	-
4.	Mild	0.5	0.3	0.4		12.5	12.5	10	10	3.80	4.00	4.24	4.10
										3.96	4.06	4.32	4.28
										3.98	4.06	4.24	-
5.	Moderate	2.8	1.7	0.5	0.1	12.5	12.5	12.5	12.5	4.22	4.18	4.00	3.98
										4.34	4.32	3.94	4.22
										4.48	4.56		4.38
6.	Mild	1.5	1.2	2.9	2.7	10	10	12.5	12.5	3.72	3.68	3.82	3.78
										3.96	4.08	-	3.82
										4.22	4.04	-	4.34
7.	Moderate	1.7	0.4	1.6	1.1	12.5	10	10	10	3.80	3.72	4.22	4.20
										4.12	3.92		4.26
										4.20	3.88		

8.	Severe	2.7	2.2	1.1	0.9	12.5	12.5	12.5	12.5	3.80	3.72	3.60	3.70
										3.84		3.88	4.00
										4.02		4.02	4.08
9.	Mild	2.0	0.6	2.6	1.3	12.5	10	12.5	12.5	3.98	3.68	3.84	3.82
												4.08	3.96
													4.16
10.	Moderate	0.4	0.8	3	1.5	15	12.5	10	10	3.62	3.26	4.16	3.88
										3.76	3.80	-	4.14
										3.80	3.14	-	4.26
11.	Moderate	1.5	1.9	1.7	1.1	15	12.5	12.5	10	3.68	3.72	3.88	3.94
												4.00	
12.	Severe	0.9	0.7	0.6	0.2	10	10	15	12.5	3.74	3.64	3.34	3.38
										3.90	3.72	-	3.76
										3.98	3.76	-	3.78
13.	Moderate	0.8	1.0	2.2	0.7	10	10	10	10	3.52	3.92	4.02	3.74
										3.78	4.04	-	
										4.14	3.94	-	-
14.	Severe	1.5	3.2	2.3	2.7	15	12.5	10	10	3.76	3.62	3.68	3.92
										3.92	3.98	4.12	4.00
											4.00		4.10
15.	Severe	1.7	1.2	0.6	0.2	15	12.5	12.5	10	3.58	3.52	3.72	3.82
										3.98	3.94	4.00	
											4.08		-
16.	Moderate	2	1.3	0.1	0.5	10	10	12.5	12.5	4.30	3.98	4.12	4.44
										4.60	4.28	4.12	4.48
										4.70	4.22	4.24	4.58

On the MLD task, no abnormality was seen in stutterers. Results on contralateral suppression of TEAOEs showed reduced suppression for six stutterers in comparision with nonstutterers. Among them, four showed abnormal suppression bilaterally, and one stutterer showed no suppression only in the right ear and one, no suppression only in the left ear, when compared to normals.

With respect to ABR, nine stutterers showed clinically abnormal ABR, in comparison with nonstutterers. Three stutterers showed absence of peaks at higher repetition rates only in the right ear and two only in the left ear. The remaining four stutterers revealed both prolonged I-V interpeak latencies at lower repetition rates as well as disappearance of peaks at higher rates in both ears.

Among the six stutterers who showed no or very less suppression, three had abnormal ABR;. The remaining three stutterers showed abnormal suppression but normal ABR. Six stutterers showed prolonged latencies and absent peaks at higher repitition rates, but showed normal contralateral suppression.

An attempt was also made to compare the severity of stuttering and the abnormality observed in contralateral suppression of TEAOEs and ABR. Severity of stuttering ranged from mild to severe as determined by, Stuttering Severity Index (SSI). It is evident from the table that there is no one-one correlation between severity of stuttering and results abtained on these tests. Blood and Blood (1984) also, in their study reported no relationship between severity of stuttering and brainstem evoked response testing.

Normal results on MLD reveal that it is a less sensitive test than the other two tests, which have shown abnormalities in some stutterers. This could be because MLD is a binaural response phenomenon, while the other two tests check for sensitivity with monoaural stimulation.

Contralateral suppression of TEOAEs checks for the intactness of both afferent and efferent systems at the lower brainstem level. Hence abnormal suppression will not localise the lesion to the afferent or efferent systems. However it will definitely assess the functioning of the lower brainstem. Similarly ABR checks for the intactness of both the lower and upper brainstem. Abnormalities at either or both these levels would result in abnormal ABR.

Deviant results obtained by subjects on both contralateral suppression of TEOAEs and ABR,would reflect on the afferent pathways at the level of lower brainstem being affected. Abnormality seen only with reference to suppression with normal ABR would suggest the the possible site of anomalies at the efferent pathways of the lower brainstem. If only ABR is abnormal, then only the upper brainstem can be suspected to be affected.

In the present study, results on both ABR and contralateral suppression of TEOAEs suggest that three stutterers may be manifesting afferent lower brainstem lesion, three efferent lower brainstem abnormality and six stutterers, upper brainstem anomalies.

Thus by comparing between stutterers as a group with non stutterers,no statistically significant difference was obtained on all the three tests. However descriptive analysis of each stutterer in comparison with age and sex matched non stutterer, has revealed differences suggestive of a possible lower or upper brainstem pathology. This supports findings of Stager (1990) and Odekar (2000) who also failed to report statistically significant differences,but on inspection of individual data found abnormalities on atleast one measure of Auditory Evoked Potentials.

From the above discussion,it is clear that there is a subgroup of stutterers manifesting brainstem abnormalities. Also, ABR or contralateral suppression of TEOAEs when used alone may not reveal the exact site of brainstem dysfunction. A combination of the two would be more sensitive in identifying brainstem pathology especially in a certain subgroup of stutterers.

SUMMARY AND CONCLUSIONS

A number of studies have been carried out in the past exploring auditory function in stutterers. Some have been done to determine cerebral dominance for language, some to check for auditory feedback and others to look for possible anomalies along the central auditory pathway. The results of many studies using both subjective and objective tests in investigating central auditory pathway till the level of the brainstem, have been inconclusive. Thus the present study was undertaken to contribute in the direction of substantiating earlier findings that some of the stutterers i.e., a subgroup of stutterers may be manifesting brainstem dysfunction. Also, in none of the earlier studies, contralateral suppression of TEOAEs, which is a measure of the intactness of the afferent and efferent systems, was used to assess central auditory processing at the brainstem level especially in stutterers. Hence this test was included in the study along with two other well-known tests to assess brainstem dysfunction i.e. ABR and masking level difference.

The present investigation aimed at:

- Detecting a subgroup of stutterers who may have brainstem dysfunction
- Finding sensitivity of three tests that check for brainstem functioning in detecting such a subgroup of stutterers. These include

    Masking level difference

    Contralateral suppression of TEOAEs

    Auditory brainstem response

Sixteen male stutterers in the age range of 18 to 35 years participated in the study. For comparison, sixteen nonstutterers matched for age and sex were taken as the control group. The range of stuttering was from mild to severe as determined by Stuttering severity index (SSI).

The following instruments were used in the study.

- A calibrated OB822 audiometer with TDH-39 earphones lodged in MX-41AR ear cushions, to assess hearing sensitivity at different octave frequencies.
- A calibrated Grasen and Stadler -3 3 (version 2) middle ear analyzer to find out the middle ear status.
- A calibrated Grasen-Stadler audiometer (GSI-10) with TDH-50 earphones lodged in MX-41AR ear cushions to obtain MLD values.
- ILO-292 DP Echoport plus to measure the TEOAEs. For contralateral suppression, noise was presented to the contralateral ear using the insert receiver of a calibrated OB822 audiometer.
- Nicolet Bravo with disc electrodes (to record the potentials) and TDH-39 earphones, with MX-41 AR ear cushions (to present the stimulus), to record ABR.

The results obtained did not reveal statistically significant difference on the three tests between the stutterers and nonstutterers. Thus to reach the aim of finding a subgroup of stutterers, descriptive analysis was done, where results obtained by each

stutterer were discussed in relation to the data obtained by age and sex matched non stutterer.

On the MLD task, stutterers, in comparison with nonstutterers, showed no clinical abnormality. Results on contralateral suppression showed reduced suppression for four stutterers bilaterally, one stutterer showed no suppression only in the right ear and one showed no suppression in the left ear only, when compared with the age and sex matched nonstutterer. With respect to ABR, nine in comparison with nonstutterers, showed clinically abnormal ABR, where three stutterers showed absence of peaks at higher repetition rates only in the right ear and two only in the left ear. The remaining four stutterers revealed both prolonged I-V interpeak latencies at lower repetition rates as well as disappearance of peaks at higher rates.

The clinically abnormal results obtained on the above measures thus suggest that some stutterers may be manifesting central auditory processing problem at the level of the brainstem.

In the present study, three stutterers manifested abnormalities on both contralateral suppression of TEOAEs and ABR, thus suggesting afferent lower brainstem pathology. Three stutterers had reduced suppression, but normal ABR suggesting a possible efferent lower brainstem abnormality. Six showed only abnormal ABR indicating a possible upper brainstem abnormality.

To conclude we can say that group comparison between stutterers and non stutterers may not clearly reveal statistically significant differences. Hence individual analysis throws more light on the subtle abnormalities manifested by a heterogenous

disorder like stuttering. The results clearly show that there may be a subgroup of stutterers manifesting brainstem abnormalities. Also with respect to the sensitivity of the tests, a combination of OAE and ABR would be more sensitive in identifying abnormalities in lower or upper brainstem in a subgroup of stutterers.

IMPLICATION

The present study visualizes the fact that descriptive analysis is always advisable when research is on to find a subgroup of a certain disorder. This also suggests that contralateral suppression of TEOAEs and ABR together will be a better tool to assess brainstem dysfunction in a given population. It paves the path for further research regarding how the results obtained in the present study can be applied in rehabilitation.

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