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I. COMMUNICATION DISORDERS

A. STUTTERING EXPLAINED AS A PHYSIOLOGICAL TREMOR*

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Definition

Stuttering is a communication disorder characterized by excessive involuntary disruptions or blockings in the flow of speech, particularly when such disruptions consist of repetitions or prolongations of a sound or a syllable, and when they are accompanied by avoidance struggle behavior (from the Speech Foundation of America, 1970).

Etymology

The term stuttering is an onomatopoeia; so also the corresponding terms in most of the other languages. One of the most onomatopoetic word for stuttering is "kohindahinda" which occurs in the Bantu language.

The Greek term "battarismos" is, according to Hunt (1861), derived from Battos. Herodotus (484 B.C.) says that the Therean Battos, who had been a stutterer from his youth, consulted the oracle at Delphi. The oracle said: "Battos, thou comest on account of thy speech, but King Phæbus Appollo sends thee to Libya, in the land of sheep to dwell". After having founded the colony Cyrene, he was, according to Pausanias (born 175 A.C.), cured of stuttering by the unexpected sight of a lion.

Introduction

Since the days of Moses** (The Holy Bible, Exodus, Chapter IV, verse 10) man has been writing about the speech disorder called "stuttering", which is the most complicated and integrated speech disorder we know. It has been the subject of much wonder and research since the time of Hippocrates (dead 377 B.C.), but we still know very little about the nature of this phenomenon.

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** Moses was a stutterer himself.
In non-disordered communication, i.e. normal speech, there is the potential for the individual to be relatively free and consciously aware of his feelings and thoughts as they relate to his environment. This fluent freedom of expression gives rise to the feeling of the unity and autonomy of the self, thus facilitating the ability to communicate freely with others. The stutterer lacks this feeling of freedom in the speech situation precisely because stuttering is a functional disorder in the communication situation. Therefore, stuttering provides one of the most socially disabling afflictions to which a human being can be subject.

There are quite a number of theories about the nature and origin of stuttering, explaining this defect from different points of view, but the author of this study is anxious to point out that stuttering is not only a medical problem, not only a phonetic problem, and not only a psychological problem, but a problem which involves all branches of knowledge with relation to the human being. Many different theories about the physiology and psychology of stuttering have been elaborated. Without derogating these dedicated and quite brilliant stuttering research workers, the author of this study does not believe that they have as yet come up with the answer of the problems of stuttering and most of them have not taken into account that stuttering is an integrative whole and cannot be studied in parts as the blind men in the story studied the elephant.

Examinations about stutterers' muscle coordination, laterality, and cerebral dominance have been done throughout this century, but according to my impression the conclusion is that stutterers do not significantly differ from the normal population organically, although some interesting observations have been made on that one percent of the human population who are stutterers. However, no one will deny that the speech performance is acoustically different in stutterers versus normal speakers. Because all speech generated by man is a result of muscle activity coordinated in time and space, the muscle activity must also be different in stutterers and in normal speakers. But when the stutterer does not stutter*, the speech performance is very like - if not identical to - the speech performance of a normal speaker.

* Most stutterers do not stutter when they are talking to themselves, speaking in chorus, or singing (von Wiechmann and Richter (1966) and Morávek and Langová (1967)).
How can we progress in stuttering research? We can, for example, try to cross traditional vocational boundaries and knowing that the overt symptom of stuttering often changes, when the social environment or the psychological factors are changed, any hypothesized mechanism of stuttering must be shown to vary with emotional states. Hence we can ask ourselves: "Which physiological phenomena can undergo changes when the psychological factors are changed?"

**Physiological tremor**

One physiological phenomenon which undergoes change under stress is the physiological tremor* (Lippold, Redfearn, and Vučo (1959); Graham (1945); Clare and Bishop (1949); Sugano and Inanaga (1960); Dossytchev, Vojava, and Nemtchine (1967)).

Under a variety of circumstances, normal or pathological, motor activity may take the form of periodic pulses of contraction instead of the normal smooth tetanus. In normal persons it is often seen in connection with fatigue or excitement (Bishop, Clare, and Price (1948)). They have also noted that under the influence of adrenaline or fatigue, tremor may be merely easier to elict, or involuntarily present and even difficult to suppress. Tremors most commonly found can be divided into two groups: those in which opposing muscles contract in alternate bursts, of which Parkinsonian tremor is the classical example, and those in which the bursts are synchronous in opposing muscles. The latter type shows increasing amplitude under condition of nervous tension. These two types of tremors appear overtly as "coarse" and "fine", respectively, although various complications of pattern, frequency, and intensity confuse this correlation. All normal subjects can induce an alternating, or clonic, tremor of the arm by clenching arm and shoulder muscles and voluntarily initiating a reciprocating movement. Many normal subjects can induce a synchronous tremor, particularly in forearm muscles, by simple clenching without voluntary movement, and under certain situations they may show involuntary tremor.

It is a common experience that fatigue, prolonged stress, either physical or emotional, and certain drugs induce or accentuate tremor. For instance, Clare and Bishop (1949), note that an involuntary tremor appeared at irregular intervals, during rest or with minimal contraction of muscles in a

* Also called: normal tremor, minor tremor, micro-tremor, micro-movement, micro-vibration, or body-vibration.
normal subject, a student, who served as a test subject, shortly after a final examination. In another subject, examined 24 hours after a tennis game, when the arm felt fatigued and uncomfortable, such a tremor appeared.

Physiological tremor of voluntary contraction limits our finesse of motor control as, for instance, is apparent when we attempt to thread a needle or wield a soldering iron, and it has been recognized and recorded since 1886 (Schäfer, Canney, and Tunstall (1886)). Despite many investigations, however, the mechanisms producing this tremor are still uncertain, but we know at all events that they are involuntary movements resulting from the synchronous contraction of muscle groups (Awazu (1965)) which produce rhythmic or alternating movements with a main frequency at 8-12 Hz. Tremor varies considerably in different subjects and in the same subject at different times. Experimental studies on tremor must take into account such normal variations.

The phenomenon of physiological tremor can be broken down into a number of physiological factors, or components, of which the principal ones are the periodic action and inaction in a given muscle involving mutual facilitation of motor units; synchronous innervation of the adjacent and opposing muscles. These factors can be detected in the activity pattern of many normal subjects who show varying degrees of tendency toward tremor, especially under nervous tension, stress, excitement, or fatigue, or during considerable effort. Tremors tend to manifest themselves at the start and particularly during the relaxation of effort, especially if relaxation is carried out gradually (Clare and Bishop (1949)). Tremor of the skeletal muscles is only one of the rhythmic, cyclic phenomena which occur in the human organism. Biologic oscillation may be mechanical, as in the case of the cardiac impulse, or bioelectric, as the \( \alpha \)-rhythm of the cerebral cortex. It may be extremely rapid, as in the electromyogram, or quite slow, as in the menstrual cycle of the human female.

Physiological tremor at rest is invisible to the naked eye, lies entirely in the micron range, and must be detected and amplified electronically for study. Physiological tremor on intention is likewise difficult to see but is grossly apparent in such states as anxiety and hyperthyroidism. Brumlik (1962) defines the physiological tremor at rest in the 3 to 80 \( \mu \) range and physiological tremor on intention as ranging from 100 to 1000 \( \mu \). There is

* Intention: The act or an instance of determining mentally upon some action or result.
considerable evidence that the increased tension of the skeletal muscles constitutes one of the important physiological manifestations of emotion, and the association of tremor and emotion has long been recognized. Phrases such as "shaking with fear" and "trembling with emotion" are familiar in everyday life. The occurrence of tremor as a result of exertion or physiological fatigue is also frequently observed clinically. Graham (1945), Clare and Bishop (1949), Friedlander (1956), Lippold, Redfearn, and Vučo (1959), and Dossyachev, Vojava, and Nemtchine (1967) have stressed that the amplitude of the physiological tremor - which, as previously noted, seldom is visible with the naked eye under optimal psychological conditions - increases in connection with the following factors: stress, anxiety, nervousness, mental effort, physical exertion, pain, and respiration. According to Eagles, Halliday, and Redfearn (1953) the medium frequency band of the tremor (6-12 Hz) is the band which is chiefly influenced by emotion and fluctuating "nervous tension". Lippold, Redfearn, and Vučo (1959) have shown that the tremor in stress situations - in casu: when normal subjects were anticipating the entrance interview to Medical School - is very alike the tremor in anxiety patients, which means that the tremor frequency is the same as in normal subjects but that the amplitude of the tremor is greatly increased.

Stress

Many investigators have drawn the conclusion that stutterers have a high degree of anxiety, shyness, and insecurity (Santostefano (1960) loc. cit.), and Santostefano (1960) say that stuttering and being a stutterer places an individual in a fairly constant state of stress because of actual and continually imminent negative reactions by the environment and because of the stutterer's own evaluation and interpretation of the handicap in terms of his self-esteem, security, and identity. Also van Riper (1963) writes about stress and stuttering and Edgren, Leanderson, and Levi (1969 and 1970), and Edgren (1969) point out that stuttering is precisely a form of "speech stress". Wilson Jr. and Kunkle (1953), Wright, Keele, Neil, and Jepson (1965), Foley, Marsden, and Owen (1967), Marsden, Foley, Owen, and McAllister (1967), Marsden and Owen (1967), and Marsden and Meadows (1968) have shown that the amplitude of the physiological tremor increases to a concentration of 1-10 µg/l after adrenaline has been injected into the blood, and it has been calculated (Wright, Keele, Neil, and Jepson (1965)) that under stress the adrenal medulla secretes adrenaline at a rate of about 10 µg per minute, which according to
Foley, Marsden, and Owen (1967) is the optimal quantity for raising the amplitude of the physiological tremor. Furthermore, Leanderson and Levi (1967) and Edgren, Leanderson, and Levi (1969) have found that the speech situation for stutterers is accompanied by a rise in the excreation of adrenaline by almost 300%. Confronted with all these facts about stuttering, stress, adrenaline, and physiological tremor*, an examination of the motor speech apparatus of stutterers in a speech situation with respect to physiological tremor would be motivated.

Methods and Materials

The physiological tremor can be recorded in different ways. The method applied in this study is the electromyography (EMG). In this technique the action potentials of muscle fibers - which originate at the motor end-plates and are triggered by incoming nerve impulses at the myoneural junction - are amplified and can be registered on cathode-ray oscilloscope, film, tape, and/or paper.

EMG-studies of the activity in the motor speech apparatus are of great interest to persons who stutter, inasmuch as oral speech consists of sequences of muscle contractions which are highly integrated in time and space, and which likely are effectuated by means of a coordination between phonetic rules and usual neurophysiological activities. The sequences of activity in the speech apparatus must be much more exact than any other voluntary action, and for this reason it is very easy to disrupt this sequence. This circumstance gives the speech scientist the possibility of making reproducible recordings of very complex sequences of human voluntary activities, as well as the possibility of recording and analyzing the muscular dyscoordination when a person is stuttering.

The EMG-technique used in this study is close to the method described by Öhman, Leanderson, and Persson (1965 and 1966), Öhman (1967), Leanderson, Öhman, and Persson (1967), Persson, Öhman, and Leanderson (1965 and 1968), Persson, Leanderson, and Öhman (1969), Leanderson, Persson, and Öhman (1970), and Fibiger (1971b). The EMG-amplifiers are constructed by J. Liljencrants at this laboratory. In most cases a 60 dB EMG-preamplifier was used in combination with a Grass Polygraph D.C. Driver Amplifier, model

* Van Riper (1963) writes about something he calls the "Stuttering Tremor" when he describes the "Third Stage of the Development of Stuttering".
7DAC, adjusted to amplify 20 dB, and in the last experiments an EMG-amplifier which amplifies 80 dB was used.

The EMG-electrodes were concentric needle electrodes, which consist of an insulated platinum wire located inside a 0.3 mm steel cannula embedded in Araldite. The platinum surface was 0.015 sq. mm and the needle length 20 mm. These electrodes have low noise level. The noise from the platinum surface was approx. $10 \mu V_{p-p}$ with the electrode inserted in the muscle. These electrodes are constructed by DISA in Denmark under ordering code: 9013 K 0593. The surface of the electrodes was cleaned in 0.9% saline by applying a few volt AC at 50 Hz across core and shaft. The preparation was stopped when one or two bubbles had formed on the surface of the core. Just before the initiation of the experiments the electrodes were sterilized in boiling water for 20 minutes. The EMG-signals were amplified and stored with a 14-channel FM-tape recorder, model Ampex FR-1300.

The examined muscles were the facial articulatory muscles, i.e. M. orbicularis oris sup., M. orbicularis oris inf., M. levator labii superioris, M. depressor labii inferioris, and M. buccinator (Figs. 1-A-1 and 1-A-2). Both unilateral and bilateral recordings were made. Note that M. orbicularis oris versus M. buccinator, M. orbicularis oris sup. versus M. levator labii superioris, and M. orbicularis oris inf. versus M. depressor labii inferioris are antagonistic pairs. These are also the most important muscles for oral articulation.

When investigating the physiological tremor, it is very important to examine muscle groups in which one muscle is antagonist to the other, since the physiological tremor is characterized by the synchronous activity in such muscle groups. The bilateral recordings - where the electrodes are placed as symmetrically as possible - are initiated by the fact, that physiological tremor is bilaterally synchronous in 66% of normal subjects (van Buskirk (1962)).

It is relevant to ask why we are interested in the facial muscles. In principle, we are also interested in the other muscles in the speech apparatus, but it is worthwhile to remember, that the stuttering symptom is changed with the speech situation. The only muscles in the speech apparatus which are easily accessible for injection of needle electrodes without anesthesia and pain are the facial muscles. It is very important when examining the stuttering symptom to encroach as little as possible upon the subjects, and therefore EMG-
Fig. I-A-1. Muscles of facial expression.
Fig. I-A-2. Electrode positions.
examinations of the articulatory facial muscles are appropriate. But these muscles are by no means the least interesting ones; they transmit a great deal of phonetic and linguistic information, which is of especially great value for deaf persons with some degree of hearing loss.

The correct placement of the needle electrode in the muscle is controlled by a cathode-ray oscilloscope or a Mingograph. In this connection it is worthwhile to emphasize that the needle electrode does not cause pain after the skin is penetrated, and the cannula can be moved in and out of the muscle with no reaction at all. If necessary, the skin surface is anaesthetized with lidocaine-aerosol. The speech signals are recorded using a Sennheiser microphone, model MD 421, at a distance from the subject of ca. 25 cm, and the FM-tape recorder. In one subject the speech signals were also recorded with a contact microphone, developed at the Department of Speech Communication from the sub-minor earphone SM-HA, made by Danavox International, Denmark. This microphone was attached on the outside of larynx with an adhesive disc, Stormaseal 1500, made by 3M Company, Minnesota, U.S.A.

During the experiment the subject was sitting as comfortable as possible. The subject read words from the dictionary of the Swedish Academy (Svenska Akademiens Ordbok), connected text, or he spoke spontaneously. It was considered important, to employ meaningful words instead of nonsense-words to secure a more natural speech situation. Each test session lasted about one hour. Recordings from four stutterers were studied in detail. All of them were adult males between the ages of twenty and thirty; the youngest one, a stutterer, was 23 years old. All the subjects had suffered from stuttering since childhood and had received logopedic therapy - with poor therapeutic results - during long periods. All of the subjects belong to the P-Club, a lay association of chronic stutterers.

Results

The electromyograms (EMG) have been studied in every detail and no neuro-pathological deviations have been observed. But it is obvious from the EMG-recordings that the muscle activity in the facial muscles during stuttering is an activity which arises from a physiological tremor with exaggerated amplitude (Fig. I-A-3); thus the EMG-activity is of the same intensity during physiological tremor as it is when producing articulatory movements. But it is also obvious that no tremor is seen in stutterers during fluent speech.
nor in normal speakers (Figs. I-A-4 and I-A-5). The main frequency band in the subjects studied here was 7-10 Hz, with the main frequency 8 Hz, and the synchronous activity in the antagonists was very well established. However, the tremor activity often starts in only one or some of the muscles and then after some activity-bursts in these muscles all of the muscles in consideration - in casu four muscles - are involved in the tremor activity (Fig. I-A-6). In the subjects examined here, the tremor activity in the symmetrical pairs of muscles is synchronous (Fig. I-A-7). It is, however, often the case that the muscle on one side starts one burst earlier than in the symmetrical one (Fig. I-A-8), but no general statement about the predominating side can be made.

Inspection of the electromyograms can sometimes reveal that only every second of the synchronous tremor bursts is seen or that every second burst has a lower intensity. This phenomenon is especially prevalent during phonation, and because of this, a careful investigation of the interaction between tremor activity and phonation was made. Inspections of the electromyograms give rise to the assumption that the tremor often lies latent but is suppressed or interacted upon by phonation. The interaction between tremor and phonation is sometimes manifest in that the phonation only suppresses the tremor in some of the muscles, while the tremor is still recognized in the other muscles (Fig. I-A-9).

As stated by Langová (1967), Morávek and Langová (1967), and Wingate (1967) the main problem of stuttering is the initial tonus or the prephonation. This is also seen in this study; the tremor activity is much more marked and frequent under prephonation (Fig. I-A-6 and I-A-10).

But how does the tremor decrease and cease, and how does normal articulation start? Analysis of the electromyograms shows that here the phonatory attempts are very important, because the acoustic output (unsuccessfully articulated speech elements) suppresses the tremor activity in the muscles. The recordings show that about 100 msec without tremor activity must precede before normal articulation can take place (Figs. I-A-8 and I-A-10). It thus seems that the condition for normal articulated speech is that there is no tremor activity during the time before the acoustic onset of a phoneme, where muscle activity in the articulatory apparatus is necessary for normal fluent speech. A following course of events is seen in the EMG: The tremor activity stops and the normal muscle activity for the
articulation starts, but before the acoustic onset, a new tremor in the facial articulatory muscles has started, the articulatory muscle activity ceases, and the stuttering continues (Fig. I-A-11).

An activity of tremor bursts starting under normal articulated phonation influences the intensity of the phonation which is decreased or ceased depending on the intensity of the tremor and the intensity of the phonation. This effect on the speech is effectuated with a delay of about 50-100 msec after the first tremor burst, depending on the amplitude of the tremor (Fig. I-A-12). Generally, phonation of low intensity is easier to interrupt than one of greater intensity. It can be noted that if an articulated phonatory attempt coincides with a tremor burst, the phonation will decrease in intensity or cease 50-100 msec after the tremor burst has started, depending on the amplitude of the tremor (Fig. I-A-13).

One of the subjects (LSA) tried to modify his stuttering symptom in a way described by Sheehan (1970). This modification of the stuttering symptom involves inter alia a smooth syllable prolongation and is called a "slide". On the electromyograms recorded from LSA it was seen that the amplitude of the tremor activity was suppressed under continuous phonation (Fig. I-A-14). In the recordings of the other subjects it was also seen that the phonation or the phonatory attempts suppress the activity of the tremor; in other words, if the stutterer can maintain an acoustic output the tremor activity will very soon cease.

It is difficult to observe the correlation between the suppression of the tremor amplitude and the sound frequency of the acoustic output of the stutterers, because the intensity of high-frequency consonants of the unsuccessfully articulated speech elements is very low, but it is obvious that there exists some kind of proportionality between the suppression of the tremor and the intensity of the acoustic output from the subjects.

General discussion

Redfearn (1957) found that the tremor amplitude is greatly increased especially in the 8-10 Hz band - in morbidly anxious persons, and Lippold, Redfearn, and Vučo (1959) stated that this was also the case in normal subjects under stress.* Carrie (1967b) found that the tremor of anxious subjects

* As noted under "Physiological tremor" also hyperthyroidism elevates the tremor amplitude (Brumlik (1962)). This fact is of special interest because Gordon (1928) has shown that stuttering may be produced by thyroid medication.
Fig. I-A-3. Mingogram. Stuttering on [ʃ] in the Swedish word "själfrid" [ʃɛːls,friːd] * (peace of mind) under the first reading trial. The tremor frequency is about 8 Hz.

Fig. I-A-4. Mingogram. Normal fluency on [ʃ] in the Swedish word "själfrid" [ʃɛːls,friːd] (peace of mind) under the 60th reading trial. This mingogram is taken from the same recording as in Fig. I-A-3 and the subject and the electrode placement are unchanged. The adaptation** is well established.

Fig. I-A-5. EMG/speech oscillogram - polygram from a normal subject. No tremor activity is seen. With the kind and most generous permission from Öhman (1967).

Fig. I-A-6. Mingogram. Stuttering on the initial consonant [ʃ] in the Swedish word "själfrid" [ʃɛːls,friːd] (peace of mind) under the first reading trial. Note that the tremor activity starts successively in M. depressor labii inferioris, M. orbicularis oris inferior, M. orbicularis oris superior, and M. levator labii superioris.

Fig. I-A-7. Mingogram. Stuttering on [ʃ] in the Swedish word "frackskört" [ʃrakskɔːrt] (tail of the tails). Note that the tremor activity in symmetrical pairs of muscles is synchronous.

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* This subject is from Gothenburg, where the [ʃ] is more frequency than [ʃ]. In Stockholm [ʃ] is more frequent than [ʃ].

** Adaptation: The reduction in frequency or severity of stuttering as a function of repeated utterances in a relatively constant speaking situation; the stutterer's measured reaction to a constant situation. Adaptation is often measured in terms of the observed frequency of stuttering during successive readings of the same passage (from the Speech Foundation of America, 1970).
Fig. I-A-8. Mingogram of spontaneous speech. Note especially that the tremor activity starts in M. depressor labii inferioris d. In the next period the activity is also seen in the two symmetrical muscles, M. orbicularis oris inferior d. & l. In the third period the tremor activity is seen in all four muscles. Note also that about 100 msec without tremor activity must precede the vowel which opens a passage with fluent speech.

Fig. I-A-9. Mingogram. Stuttering on \( [\text{j} \text{f}] \) and \( [\text{f} \text{e}:] \) in the Swedish word "själfrid" \( [\text{x} \text{f} \text{e}:\text{l}s, \text{fri:d}] \) (peace of mind). Note that the intensity wax and wane in relation to the acoustic output from the subject in such a way that only every second tremor burst - especially in M. levator labii superioris d. and M. orbicularis oris superior d. - is seen.

Fig. I-A-10. Mingogram of spontaneous speech. Note especially the tremor activity under prephonation. Note also that about 100 msec without tremor activity must precede the vowel which opens a passage with fluent speech.

Fig. I-A-11. Mingogram. Stuttering on \( [\text{f}] \) in the Swedish word "själfrid" \( [\text{x} \text{f} \text{e}:\text{l}s, \text{fri:d}] \) (peace of mind). Note that the articulatory muscle activity in M. depressor labii inferioris d. twice is interrupted by tremor activity before the acoustic onset of \( [\text{ri:d}] \).

Fig. I-A-12. Mingogram of spontaneous speech. Note that the phonation is inhibited by the tremor activity.

Fig. I-A-13. Mingogram. Stuttering on \( [\text{j} \text{f}] \) and \( [\text{f} \text{e}:] \) in the non-fluent Swedish word "självskirad"* \( [\text{x} \text{f} \text{e}:\text{lv}, \text{fji:d}] \). Note that the tremor which starts simultaneously with a phonatory attempt of \( [\text{e}] \) inhibits the phonation totally about 100 msec after the start of the tremor burst.

Fig. I-A-14. Mingogram of "sliding" spontaneous speech. Note the inhibition of exaggerated tremor activity in connection with the smooth syllable prolongation.

* "självsirk (or självsirkad) honung": an old Swedish term for "selfdrained honey".
Fig. I-A-4.
M. ORBICULARIS ORIS INF. L.
M. DEPRESSOR LABII INF. L.
M. ORBICULARIS ORIS INF. D.
M. DEPRESSOR LABII INF. D.

SPEECH OSCILLOGRAM

Fig. I-A-7.
1/32 sec

SPEECH OSCILLOGRAM
FREE FIELD MIC.

SPEECH OSCILLOGRAM
CONTACT MIC.

M. ORBICULARIS ORIS INF. D.

M. DEPRESSOR LABII INF. D.

M. ORBICULARIS ORIS INF. L.

M. DEPRESSOR LABII INF. L.

Fig. I-A-8.
SPEECH OSCILLOGRAM

M. LEVATOR LABII SUP. D.

M. DEPRESSOR LABII INF. D.

M. ORBICULARIS ORIS SUP. D.

M. ORBICULARIS ORIS ORIS INF. D.

1 SEC
SPEECH OSCILLOGRAM

fffr i:

M. LEVATOR LABII SUP., D.

M. DEPRESSOR LABII INF., D.

M. ORBICULARIS ORIS SUP., D.

M. ORBICULARIS ORIS INF., D.

1 SEC

Fig. I-A-11.
Fig. I-A-12.
Fig. 1-A-14. This figure continues on next page.

M. DEPRESSOR LABII INF. D.

M. ORBICULARIS ORIS INF. L.

M. DEPRESSOR LABII INF. D.

M. ORBICULARIS ORIS INF. L.

100 MSEC
differs from that of the controls in that 1) there was a greater average amplitude, and 2) the 7-10 cps components were relatively more prominent than other components in the 4-12 cps range. The frequency found in those studies coincides very well with the tremor frequency which is found in this study, and the author therefore concludes that the overt symptom of stuttering is precisely a physiological tremor in the motor speech apparatus with an exaggerated amplitude, induced indirectly because the speech situation is a stress situation for the stutterer, and more directly by the increased adrenaline excretion.

The number of subjects in this study is too small to convincing prove that the stuttering symptom manifests itself more readily at the 8 Hz tremor frequency than at the 10 Hz frequency which is the mean frequency of physiological tremor in normal adult subjects. This hypothesis, however, is of considerable interest. First of all, it is important to point out that a tremor frequency of 8 Hz is within the normal range of 8-12 Hz. Furthermore, the tremor frequency in children - where stuttering generally is more frequent* - is according to Marshall (1959) 5-6 Hz and increases abruptly about the 10th year, and Zdunkiewicz and Gorynski (1968) found that the physiological tremor decreased as age increased, particularly from the 15th year. The fact that many children recover from the stuttering symptom may also be seen in the general connection that children are apparently clumsy in performing fine movements, which may be related to the more random nature of their motor activity. It is well known that normal fluent speech involves a very fine motor coordination in time and space with as many as 10,000 to 15,000 neuromuscular events per minute (Lenneberg (1967)), which only Homo sapiens can perform in spite of the fact that most mammalians have lungs, larynx, tongue, lips, and all other anatomical details which are necessary for production of sounds. The nature of the neural pathways responsible for adult motor finesse is unknown.

Graham (1945), Friedlander (1956), and Marsden, Meadows, Lange, and Watson (1969) point out that the shape of the physiological tremor spectrum is very characteristic and rather constant for an individual, but that it varies in different subjects in the same way as voice and the stuttering symptom do.

* There is a great spontaneous recovery from stuttering - according to Sheehan and Martyn (1970): 80 % - mainly in the two periods of life, 11-14 years and 18-22 years.
Only the amplitude varies within great ranges, accentuated by stress, anxiety, fatigue, hunger, and senility. The predominating frequency found here (8 Hz) may also be seen in connection with the fact that there is an inverse relationship between frequency and amplitude. Graham (1945) and Friedlander (1956), and Lippold, Redfearn, and Vuču (1959) note that the most striking feature of physiological tremor is the fact that it has components in all the frequencies at least up to 30 Hz, and that under isometric recordings the amplitude is large in low frequencies, gradually diminishing as the frequency increases.

Brumlik (1962) has remarked that the peak frequency of the tremor is elevated from 7-8 Hz to 9-10 Hz when the muscles are activated maximally, which is in accordance with a tendency noted in this study. According to Clare and Bishop (1949) the physiological tremor, typically synchronous in antagonistic muscles does not build up in amplitude as the contraction continues, as is often observed in the Parkinsonian tremor, in which gross movement is adequate to set up reciprocal stretch stimuli reinforcing the initial periodicity. If antagonists are involved synchronously at a higher frequency than 10 Hz, no visible tremor movement may result.

The observation made in this study, that the overt symptom of stuttering is established with a tremor frequency of about 8 Hz, and not with the mean frequency for physiological tremor, 10 Hz, may also be viewed in light of the fact that stuttering often is a repetition of syllables. But how can the tremor frequency in the articulatory apparatus be effected by the repetition of syllables? To answer this question we must keep in mind the rate of repetition of syllables or the diadochokinetic speed, since the syllable is the "true basic unit" in speech (Stetson (1951)) and because the physiological correlate to the syllable is the breath pulse. Lenneberg (1967) and Lehtonen (1971) have stated that the diadochokinetic speed normally is about 6 syllables per second and that it can increase to about 8 syllables per second, and occasionally even 9 syllables per second for the duration of a few seconds. Also Hudgins and Stetson (1937) found that the relative speed of articulatory movements lies in the interval 5.7 to 7.7 per second. Lenneberg (1967) also stated that a temporal unit of 160 msec ± 20 msec (~ 7.1 Hz, 5.5 Hz) appears to play a role in general psychological

* Diadochocinesis is making of repetitive alternating muscular movements, such as tapping the foot, tapping the finger, raising and lowering the brows, and opening and closing the jaw.
and neurophysiological processes. This correlated to the previously mentioned fact that the phonatory attempts influence the tremor bursts and may lead to the assumption that the frequency of the tremor is established at the maximum rate of repetition of syllables. The fact that this is the maximum rate of repetition of syllables may have a connection with the well-known fact that stuttering often appears in connection with cluttering, where the rate of repetition of syllables is higher than with normal speech (von Seeman and Novák (1963)).

As noted previously sometimes the left muscle has the greatest amplitude and sometimes it is the (symmetrical) right muscle. It may be a consequence of the placement of the electrode, but Brumlik (1962) notes that the amplitude of the tremor of the left upper limb was of greater magnitude than the right one in some of the subjects he studied, but in others, the tremor of both the upper limbs were of the same amplitude. Zdunkiewicz and Goryński (1968) report that in all right-handed children examined, the left hand was found to have greater tremor than the right one, and the opposite was true in left-handed children.

As noted under "Results" all the subjects examined in this study have similar frequency on both sides of the face, which may be seen in connection with the findings of van Buskirk (1962) which indicates that when comparing the two sides of the subjects, 34% had frequencies differing from one side to the other.

It has been postulated by many authors that the electroencephalogram (EEG) of stutterers is not fully normal in every aspect. For a review: see Langová and Morávek (1966) and Beech and Fransella (1968). The EEG changes in stutterers are mainly diffuse and unspecific. Some authors postulate a slower α-rhythm (e.g. Fritzell, Petersén, and Selldén (1965), and Langová and Morávek (1966)), and Morávek and Langová (1962) say that stutterers have a greater amplitude of the α-rhythm. These two attributes of the α-rhythm in stutterers are the same attributes as we find in the physiological tremor of stutterers. Therefore the relations between physiological tremor and EEG are of considerable interest.

Ozaki, Sato, Awazu, Mimura, Honda, Teramoto, and Kitajima (1962) stress the correlation between the tremor and EEG, and according to Lippold (1970), and Lippold and Novotny (1970), the α-rhythm in the EEG is an artefact, generated by the physiological tremor. Lippold and Novotny have also
stated that curves for tremor frequency against age and $\alpha$-frequency against age coincide. They also mention that fatigue and a greater tremor amplitude of the extraocular muscles can be produced experimentally by asking the subject to follow a moving object with his eyes; after some time the amount and amplitude of the $\alpha$-rhythm increases. The control experiment which involves a steady fixation of an object while the background is moved (in that way producing the same retinal stimulation) does not result in an increase of $\alpha$-rhythm.

These facts are well in accordance with the above-mentioned changes of EEG in stutterers and also the assumption of Sheehan (1970) that the stuttering symptom is improved when the stutterer has eye-contact with the interlocutor. It is normally very difficult for a stuttering person to have eye-contact with the communicated subject; normally the stutterer averts and roves his eyes under speech communication situations. But if the stutterer concentrates on visually fixating on the interlocutor’s eyes, his eyes can be at rest, the tremor amplitude decreases, and the stuttering symptom is improved to some degree.

Exaggerated tremor amplitude in the speech apparatus of a stutterer in a speech situation may be seen in connection to the well-known fact that the proportion of male stutterers to female stutterers is about 4 to 1. Carrie (1967a) has shown that the amplitude of the tremor in anxious males is the double of the tremor amplitude in anxious females, and as previously noted, Lippold, Redfearn, and Vučo (1959) have stated that the tremor in stress situations is very alike the tremor in anxiety patients.

As described under "Results" phonation and phonatory attempts diminish the tremor. It is well-known from stuttering therapy that a change in the acoustic stimulus alters the stuttering symptom, as is seen during a "slide" or in experiments with delayed auditory feedback (DAF), while noise stimulation or when singing or speaking in chorus. This fact only underlines the well established phenomenon that auditory feedback and other acoustic phenomena produce changes in the performance of different types of motor activity (Chase, Sutton, and Rapin (1961)). According to Awazu (1965) rhythmic visual and acoustic stimulations increase the physiological tremor, whereas irregular stimulation causes depression of the physiological tremor.

* "Slide" means a smooth syllable prolongation (Sheehan (1970)).
It may be of interest to study this problem in more details, and we will first examine singing. In relating speech and singing, it is important to note that the speech melody of stutterers is generally inflexible. The stutterers speak monotonously (von Weichmann and Richter (1966)), exhibit a poorer rate of speaking, speak with greater force or straine, and are judged to use less rhythmical speech than the nonstutterers (Wendahl and Cole (1961)), whereas in singing vowels are prolonged and the phonation more continuous. Accordingly, the movements of the articulatory muscles become slower and easier to control. The more continuous phonation when speaking in chorus, singing, or "sliding" is, according to the author of this study, the cause of the improvement of the stuttering symptom in these situations.

The effects of DAF and white noise will now be discussed in more detail. According to many authors (see e.g. Stromsta (1956), Stensland (1966), Beech and Fransella (1968), and Burke (1971)), which have investigated the effect of DAF on normal speakers and stutterers, stutterers have an auditory central nervous system disorder. But the results which have led to that hypothesis are not very significant and the hypothesis is strongly rejected by many other authors, e.g. Gregory (1964) and Langová and Morávek (1969). In connection with the view that the cause of stuttering is anxiety, it is relevant to note, that Dinnerstein and Lowenthal (1966) mentioned that one possible way in which anxiety disrupts a mode of normal behavior should be the producing of an increased latency in the perceptual systems which govern that behavior. According to my opinion it is likely that the effect of DAF and white noise can be explained as an effect on the tremor amplitude, but further investigations in this field are needed.

But how can we explain this interaction between stuttering, auditory stimulation, and tremor? The mechanisms determining the amplitude of physiological tremor are obscure. However, these mechanisms may include a common pathway through which the changes in tremor characteristics, due to stress and auditory effects, are mediated. A great deal of the stuttering therapy which is given today is psychologically oriented. In this connection it is important to underline that emotional manifestations are thought to initiate in the hypothalamus. Psychologically oriented therapy could accordingly affect the hypothalamic areas, providing a connection link between neurological and psychological fields (Kraft (1969)). The exaggerated excretion of adrenaline could be caused by the emotional stimulation of the
hypothalamus (see Fig. I-A-15), and Birzis and Herningway (1957) have generated tremor in a cat by direct electrical stimulation of the hypothalamus.

The inhibition of tremor after the acoustic onset of the phonation or the phonatory attempts is well explained from the findings of Sugano and Inanaga (1960) and Sugano (1963), which indicated that stimulation of the inferior colliculi - the centers for auditory reflexes - (see Fig. I-A-15) and the auditory areas on the cortex (see Fig. I-A-15) inhibit the physiological tremor.

Speculations

In view of the fact that children are clumsier than adults it is likely that they also have difficulties with the complex time, space, programming of normal speech*. If the interlocutors concerned about the child’s speech**, the child, if he is genetically prone to "speech stress", can be stressed in the speech situations, the excretion of adrenaline is in its turn elevated, tremor with an exaggerated amplitude is generated in the motor speech apparatus in speech situations, the stuttering symptom is manifested, and the "vicious spiral" is generated.

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* e.g., Irwin and Becklund (1953) note that the mean rate of repetition of syllables varies from 3 per seconds at the age of 6 to 6 per second at the age of 15, but in connection with the sex dependency of stuttering, it may be noted that at the age of 6 girls reach a higher rate of repetition of syllables than boys. Around the ages of 13-14 years the speed is equal for both sexes, and in adults the diadochokinetic rate is higher in males than in females (Fairbanks and Spriestersbach (1950)).

** According to many authors, e.g. Johnson (1942), Bloodstein, Jaeger, and Tureen (1952), and Glasner and Rosenthal (1957), parents of stuttering children may be unusually intolerant of childhood nonfluency, such as, according to Johnson (1942) approximately 92% of the parents of stutterers misdiagnosed normal nonfluency as stuttering. Glasner and Rosenthal (1957) report that parents attempt to reduce nonfluencies by suggestions as for instance: 'told him to speak more slowly and to take his time; made him repeat; made him stop and start over; said the word for him; reminded him not to stutter; told him to speak more softly; got angry; corrected him; emphasized the sound of the proper speech etc." A significant relationship between active correction of nonfluency and the perpetuation of the stuttering was found.
Fig. 1-A-15. Feedback model of the speech production. (Based in part on Dalby (1970) and Fibiger (1971a).)

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It is hoped that this paper may add to a better understanding of the nature of stuttering. However, much more research is needed, especially with respect to the interaction between tremor and auditory stimulation. A better understanding of the nature of stuttering is of basic importance for an improved stuttering therapy. One possible outcome of this study is that the research of therapy of the overt symptom of stuttering may be concentrated within the research field of the treatment of tremor.

Summary

The facial muscles are studied electromyographically in speech situations at stutterers. It has been found that during stuttering there is a physiological (normal) tremor with an exaggerated amplitude in the articulatory muscles. It has further been found that phonation or phonatory attempts inhibit the tremor. The author concludes that the overt symptom of stuttering is precisely a physiological tremor in the motor speech apparatus with an exaggerated amplitude, induced indirectly because the speech situation is a stress situation for the stutterer, and more directly by the increased adrenaline excretion. The findings are discussed generally, inter alia in connection with neurophysiology, adrenaline secretion, and emotional stress.

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