STUTTERING: A CASE STUDY IN THE SCIENTIFIC METHOD

M. N. HEGDE

Introduction

Stuttering is one of the speech problems on which there is relatively more information in the literature. And it is also a problem that has been studied from different angles and viewpoints. In fact, the different approaches to stuttering are so varied that they are often contradictory. Thus, for an objective examination the literature on stuttering is a 'welter of confusion' (Rathna 1969). This confusion, it seems, is not entirely due to the presence of different approaches, but rather to the weakness and the inherent contradictions of each of these approaches. In other words, there are not only mutually exclusive viewpoints, but each viewpoint suffers from a lack of internal consistency and also of empirical evidences.

It is the purpose of this paper to critically examine two such systems—one of Van Riper's and the other of Robert West and his associates. As far as a theory of stuttering is considered, these two approaches are as varied as they could be; but strangely, they converge in their viewpoints on therapy. That further illustrates a contradiction in the position advocated by West *et al.* It is felt that an objective examination of existing viewpoints is urgent from a practical stand point. From a theoretical standpoint, such an examination could be an excellent case study in the scientific method. Van Riper's system, however, has been examined only partially in this paper.

Van Riper

Van Riper, one of the early and most active workers in the field of speech pathology has published extensively on stuttering and also on other speech problems. The techniques of therapy developed by him seem to have had considerable influence on the practice of speech therapy, although not many well planned scientific studies on the effects of such techniques are available in the literature. In the process of developing such techniques Van Riper borrowed several concepts from experimental and clinical psychology. But Van Riper's subsequent use of these concepts in his theoretical framework has not been quite satisfactory. An attempt is made below to examine the way Van Riper makes use of some of these concepts and also the kinds of deductions he seems to make from them.

Mr. M. N. Hegde is Lecturer in Psychology at **the A.I.I.S.H.**

Stuttering: Neurotic Vs. Learned Response

Van Riper believes that speech problems including stuttering have a multiple origin (Van Riper 1958). However, he also thinks that when organic factors are ruled out, most of the speech problems are due to faulty learning and not due to a neurosis. He writes:

> In our experience the large majority of speech problems have their origin in faulty learning or perceptual or organic factors...Our own experience with thousands of cases just does not jibe with the concept that defective speech is always, or even commonly, a neurotic symptom (1963, p. 55.)

But there still are a small number of cases with a speech defect whose problem is not due to faulty learning, but is due to neurosis. In some individuals at least, stuttering is a symptom of *primary neurosis.* And by primary neurosis, he means:

> ...that the speech disorder was *adopted,* usually *unconsciously* as a device to protect the person from the stress of an *emotional conflict* which he seemed to be unable to solve in any other way. (Van Riper, 1963, p. 52). (Italics added).

The above two quotations make it clear that Van Riper distinguishes between speech problems that are due to faulty learning and others that are due to 'neurosis'. He means accordingly that stuttering is *not* a neurotic response when it is a result of faulty learning and that it is, when it is developed unconsciously as a means to resolve emotional conflicts. This clearly indicates that Van Riper uses the term neurosis strictly in the psychoanalytic sense; for elsewhere he also says that this primary neurosis has symptoms that are 'pregenital conversion reactions with both oral and anal components' (1963a, p. 886). However, stutterers are also supposed to exhibit what Van Riper calls *secondary neurosis* which originates 'as a defensive reaction to the penalties which our highly verbal culture imposes upon those' individuals whose non-fluency is excessive' (1963a, p. 886). That means that neurosis in one instance is a 'conversion reaction with both oral and anal components' and in another, it is simply a reaction to the social consequences of one's own non-fluency. Consequently, Van Riper's use of the term neurosis borrowed from psychoanalytic literature does not seem to be consistent even within such a frame of reference. However, in the light of modern research on neurotic behaviour more serious objection can be raised against Van Riper's distinction between the neurotic reactions and *faulty* but learned responses. Behaviour therapists have clearly demonstrated that *all neurotic* responses are in fact learned and the result of the faulty learning itself is neurosis without any underlying unconscious emotional conflicts. Experimental evidences in favour of a psychoanalytic interpretation of neurotic behaviour are conspicuous by their absence and neurotic behaviour is much better explained by the principles of conditioning and learning. The conclusion seems to be that all stutterers exhibit only a learned maladaptive habit and hence they could be considered to make a neurotic response without

psychoanalytic connotations. So the distinction between neurosis and faulty learning cannot be considered valid.

Psychotherapy Vs. Symptomatic Therapy

In his discussion on stuttering therapy Van Riper makes it clear that since the stutterer comes with a symptom he wants to get rid of, the therapist should invariably show a proper concern with the symptom itself. Only then the initial rapport can be built up. However, by the time a stutterer comes to a therapist, he will have experienced a large variety of symptomatic treatment procedures which have not proved very useful. And hence, he would be sceptic of 'mere' symptomatic methods. But what about the therapist?

> **The** therapist also tends to be sceptical, if he has any background at all in Clinical **Psychology** or Psychiatry or any experience in treating stuttering (1963, p. 879).

Mere symptomatic therapy will not be useful because stuttering is a functional disorder and 'the majority of speech therapists recognize the necessity for some form of psychotherapy as basic in the treatment of stuttering' (1963 b, p. 880). And hence, 'We must treat the stutterer as well as stuttering' (1963 b, p. 880). Since stuttering as a symptom and stutterer as a person are both to be treated, Van Riper advocates a certain place for the symptomatic therapy also. '. . . in order to gain time enough to teach him to control not only his stuttering but himself symptomatic therapy is vital' (1963 b, p. 879). In other words, Van Riper advocates both symptomatic therapy and psychotherapy for stutterers.

But what kind of symptomatic therapy? Van Riper classified symptomatic therapies into two 'natural'(?) classes:

> . . . those which have their goal the prevention of the occurrence of stuttering and those which have as their goal the modification of the stuttering symptom. To put it another way, the first tries to teach the stutterer to talk without stuttering; the second tries to teach the case to stutter in a fashion tolerable to both society and himself. The one stresses controlled inhibition; the other controlled exhibition. The basic contrast is between repression and expression as fundamental therapeutic philosophies (1963 b, p. 880).

Van Riper then goes on to cite a number of 'techniques' like solo speech, memorization, choral speaking, singing, dramatics and debate, etc., as examples of *repressive symptomatic therapy.* When these techniques are used alone and not along with psychotherapy, they are bound to fail because the therapeutic goal set by these techniques is the *repression* of stuttering symptom. Since all symptomatic therapies are to be used along with psychotherapy, the best technique of symptomatic therapy should be one that helped rather than interfered with the essentials of psychotherapy. Furthermore, stuttering in the majority is only a learned and not a neurotic response and consequently the effective symptomatic therapy should be relevant also to learning theory. Van Riper solved the problem

in his own way. He tried to develop 'a therapy which, while masquerading as symptomatic therapy would actually be a psychotherapy'! (1963 b, p. 887).

The technique that Van Riper developed, *expressive or dynamic* type of symptomatic therapy, 'had its origin in Freud's early interest in getting the patient to express rather than to inhibit his symptoms and in using the symptoms as barometers of the basic conflict' (1963 b, p. 885). In this therapy, the psychotherapist was fundamentally permissive and this led to significant changes in the personality of the case. The stutterers were urged to exhibit their symptoms as did psychoanalysts. Symptoms were attended to, but only as a means to induce more significant changes in the personality; this was necessary because the stutterer as a person was also to be treated. So it is evident that the new psychotherapy 'masquerading as symptomatic therapy' was inspired by psychoanalysis. But strangely Van Riper asserts that the development of some aspects of his technique was also helped by Dunlap's (1932) beta hypothesis.

Negative practice: Dunlap (1932) was the one who first proposed that under certain specified conditions repeated practice of a habit will result in its extinction. The term negative practice was used to describe his technique. Dunlap believed that neurotic symptoms can be considered as undesirable habits and they can be extinguished by specific operations. Considered in this theoretical framework, it is evident that psychoanalysis-psychotherapy and negative practice have nothing in common. But for Van Riper:

> **Although Dunlap conceived the stuttering symptoms to be *merely* habits which, when the stutterer became highly aware of them, could be brought under voluntary control and discarded, *a good amount of psychotherapy was inherent in the process* (1963 b, p. 885). (Italics added).**

It is not made clear how, in the repeated massed practice of a habit, psychotherapy was inherent. However, Dunlap's beta hypothesis was puzzling from the standpoint of the then learning theory but has subsequently been explained on the basis of the conditioned inhibition cnstruct within the framework of modern learning theory (Kendrick 1960;Eysenck 1964). Negative practice is an operationally defined procedure and is included under behaviour therapy techniques, which, for all practical and theoretical considerations, are opposed to psychotherapy. In this light it is strange, to say the least, that an expressive type of symptomatic therapy was inspired both by psychoanalysis and negative practice. And it is much more difficult to accept Van Riper's statement that 'a good amount of psychotherapy' was inherent in negative practice.

Apart from the fact that both psychotherapy and negative practice cannot be meaningfully integrated into a single system, Van Riper seems to have misinterpreted the technique of negative practice. In the treatment of articulation disorders he describes negative practice as a technique where the child, after learning the correct sound, is 'required to say it *occasionally* in the wrong way' (1963, p. 296).

Van Riper further thinks that the support for this method has come from experimental psychology:

> ... modern experimental psychology had demonstrated that when one seeks to break a habit that is rather *unconscious* (such as a fingernail-biting or the substitution for sh for s), much more rapid progress is made if the possessor of the habit will *occasionally* (and at appropriate times) use the error deliberately (1963, p. 296). (Italics added).

It is very clear from the above statements that for Van Riper *occasional* use of an error is negative practice. Experimental and clinical studies on negative practice do not, however, support the contention that occasional practice of an error will result in its extinction. It is the deliberate and *massed* practice of an error that leads to its elimination (Kendrick 1960; Eysenck 1964). In articulation therapy when the child after mastering the correct sound uses the wrong one occasionally, he may be benefited because that would highlight the difference between the two; but it cannot be labelled negative practice. His further contention that one of the rationale of negative practice is that 'the voluntary practice of the error acts as a *penalty'* (1963, p. 297) is equally unfounded. He seems to use the technique of negative practice to get the child appreciate the difference between a right and a wrong sound (1963, p. 297). Here, what Van Riper does and his objectives in doing so may be compatible but both are not compatible with the rationale and the procedure of negative practice. Van Riper's model that stuttering theory and stuttering therapy should fulfil both learning theory and psychoanalysis, which is decidedly faulty, is further illustrated in some of the specific techniques he uses in his symptomatic therapy.

Cancellation and Pull-outs. At a certain point in the therapeutic process stutterers are recommended *cancellation.* Immediately after a stuttered word has finally been uttered, the stutterer is asked to reproduce the same block; 'he deliberately does some pseudo-stuttering on the cancellation' (1963 b, p. 889). This, however, will not be an exact duplication of the original block but will include some of the modifications already learned. This technique, according to Van Riper (1963 b) fulfils some of the requirements of both psychotherapy and learning theory:

> From the point of view of Psychotherapy what have we in this cancelling technique? Frist of all we have provided an opportunity for *self-confrontation and evaluation.* We have also prevented the *repression* that usually takes place immediately after a moment of stuttering (p. 890). (Italics added).
>
> From the point of view of learning theory, cancellation has these advantages. It takes advantage of the moment *of reaction inhibition,* the point in time at which the old stuttering response is weakest. It interferes with *self-reinforcing tendency* of stuttering symptoms to terminate fear (p. 890). (Italics added).

Further, when the case has learnt cancellation, he is recommended another technique called pull-outs. In pull-outs the stutterer makes a deliberate attempt to modify the block before the release occurs and before the word is spoken.

What is the rationale for it? Van Riper again tries to justify it both in terms of learning theory and psychotherapy. The technique is effective in terms of learning theory because 'the release (the utterance) rewards not the uncontrolled abnormality which formerly preceded it but the voluntary smoothing out and slowing down of the tremor' (1963 b, p. 893). And the technique is also useful because in terms of psychotherapy we find in it:

> . . . a real battle against his neurotic tendencies. Gradually the *insight* comes to dominate the *resistance,* and a further *resolution of the conflict occurs.* The struggle for control of conflicting impulses is the heart of all *psyhotherapeutic healing.* We have it here. (1963 b, p. 891). (Italics added).

Finally about one more assumption made about cancellation. Although Van Riper does not explicitly equate cancellation with negative practice in the above referred writings, others have done it (Lehner, 1960). However, on careful scrutiny it should be clear that negative practice and cancellation are very different.

Desensitization: Desensitization is still another technique used by Van Riper in his psychotherapy (although called symptomatic therapy). It was Jacobson (1939) who demonstrated that relaxation has an autonomically counteracting effect on anxiety. Even earlier Mary Cover Jones (1924) was able to counter condition fear reactions in a small boy. Based on some of these early experiments Wolpe (1958) developed the technique of systematic desensitization therapy for anxiety and phobic reactions.

Van Riper borrowing the concept of desensitization has integrated it into his therapy for stutterers. In his treatment of secondary stutterers desensitization is the third major phase (Van Riper, 1963). The goal of this phase of treatment, as set out by Van Riper is 'to toughen our case to those factors which normally increase the frequency and the severity of stuttering' (1963, p. 396). Although this goal is compatiable with the concept of desensitization, what is done to achieve it does not seem to be so. In this phase of treatment stutterers are given a series of assignments to carry out. The purpose is to expose the case to as many situations as possible that are likely to create frustration, anxiety, guilt, hostility, communicative stress, situational fears and penalty. Stutterers are asked to indulge in various activities and includes such as these: making phone calls and faking a long repetitive block until the listener hangs up on the case; stutterers asking their friends to laugh at their every block; irritating people until they are being attacked; prewriting everything that stutterers wanted to say; not smoking at all; tapping the toe once for each word; stuttering to people in a hostile fashion; stutterers requesting their friends to heckle them during conversation; making 25 phone calls before going to bed; applying for a job in every store; going to every housedoor in a block and to make certain enquiries. The cases are urged to carry out these instructions with a view to desensitize them to those and such other situations.

It is evident that in this procedure no effort is made to counteract the anxiety, frustration and fears that arise in carrying out those assignments. The implicit assumption here seems to be that a mere exposure to traumatic events is desensitization. This, however, is not a valid assumption. Desensitization, basically, is a counter-conditioning procedure (Wolpe, 1958; London, 1964). A response to be eliminated is systematically evoked by the therapist in a context that is antagonistic to the response. Anxiety responses, for example, are evoked when the patient is in a very relaxed state which are immediately followed by deep relaxation responses. Unless this kind of specific care is taken to counteract the effect of an undesirable response with another that is incompatible with it, the procedure cannot be described as desensitization. However, Van Riper's (1963) use of the term in therapy with child stutterers seems more appropriate which further indicates that he uses the concept inconsistently within even his own conceptual framework.

It should be added, however, that what Van Riper is doing, although desensitization is an inappropriate term for it, is not unlikely to give 'good results'. If it is true that stutterers are avoiding those difficult speech situations which now the therapist is trying to get them to face, these conditioned avoidance responses are likely to be extinguished when they repeatedly face the feared situations but come to realize that most of their fears were rather unrealistic. This then is a matter of extinction and not desensitization. So under desensitization the techniques Van Riper describes are not related to the rationale for it. The other objection that in any psychotherapy the concept of desensitization as developed by Jacobson (1930) Wolpe (1958) and others cannot be meaningfully employed is evident.

This brief review of some aspects of Van Riper's theory of and the therapy for stuttering indicates that almost all the basic assumptions of Van Riper are questionable. The very distinction between repressive and expressive types of symptomatic therapies is not based on scientific evidence. In advocating an expressive type of symptomatic therapy Van Riper clearly indicates that symptom removal is not the goal of therapy at all. Expressive symptomatic therapy 'stresses controlled exhibition', it teaches the stutterer 'to stutter in a fashion tolerable to both society and himself. In contrast, repressive symptomatic therapies are depreciated because they teach the stutterer 'to talk without stuttering'. So any therapy that tries to remove stuttering completely is no therapy at all! The controlled exhibition, to say the least, is a paradoxical goal for a therapy programme. This however, should not be taken for a justification for those 'techniques' listed by Van Riper under 'repressive symptomatic therapy'. That they are not scientific enough is sufficient to dismiss them.

This review also reveals several other sources of confusion and inconsistencies. At least one of the major factors responsible for this confusion is a matter of his-

tory; history of the developments in the scientific study of stuttering. Almost all the early and notable workers in this field were clinical psychologists working at a time when psychoanalysis and psychotherapy based on it were creating a considerable amount of impact on the theory and therapy of 'functional disorders'. Stuttering certainly was a functional disorder and what psychoanalysis said about neurosis seemed relevant to the former as well. So all of them, along with psychoanalysts, agreed that what stutterers needed was psychotherapy. However, when the therapists actually started working with stutterers it was discovered that they were, after all, not as 'abnormal' as the psychoanalysts led them to believe. While believing firmly in the proposition that stutterers needed psychotherapy for a lasting cure (because stuttering *was* functional) early investigators started looking elsewhere for an explanation, as to the why of stuttering. The learning theory that was decidedly in its early stages seemed to fulfil this expectation, and hence it was concluded that stuttering in a 'majority' of cases was a learned habit. However, according to them, there still was a certain number of stutterers who were neurotic in the psychoanalytic sense with oral and anal components. This dual loyalty to psychoanalysis, and learning theory and resulting confusion is very well reflected in Van Riper's writings. It also means that some of these early workers who believed in the application of learning principles to behaviour problems have failed to follow the advances made in experimental clinical psychology and hence the incompatibility of making use of psychoanalytic and learning theory concepts in the same theoretical framework was entirely missed by them. They continued to use the term neurosis strictly in the psychoanalytic sense but they found, in actual practice, that the term was irrelevant at least to a majority of stutterers. Meanwhile what had happened was that the same term was rendered irrelevant to all kinds of functional problems. Behaviour therapists had already redefined neurosis, on the basis of acceptable scientific evidences, in terms of maladaptive learned habits. Van Riper's distinction, consequently, between the neurotic and the learned (maladaptive) responses should be considered unfounded.

The second source of confusion follows the first. When it is believed that stuttering can originate both as a neurosis in the Freudian sense and as a maladaptive learned habit, the therapy for stutterers should be consistent with psychoanalysis as well as with learning theory. Since no serious attention was paid to the logical consequences of the hypothesis that stuttering in the 'majority' was a learned habit, Van Riper set out for himself two rather impossible goals of fulfilling the requirements of psychoanalysis and of some rudimentary constructs of learning theory. As a consequence of the same attitude, psychotherapy was considered to be basic to stutterers in spite of the fact that it was a learned habit. Consistent with the psychoanalytic assumptions any therapy that attacked only the symptoms was considered superficial. However, immediately in contradiction to it, the next assumption that stuttering as a symptom is also to be treated, and therefore symptomatic therapy is also useful, was made. And as a climax to this series an

attempt was made to develop a technique that 'while masquerading as symptomatic therapy, would actually be a psychotherapy' (Van Riper, 1963 b, p. 887).

Thus, Van Riper's therapy for stutterers is actually a psychotherapy. But if only he had followed the assumptions of psychotherapy, his system would at least have been internally consistent although the question of its validity would soon have arisen. Unfortunately, as substantiated in the earlier sections, Van Riper borrows concepts from both learning theory and psychoanalysis and does justice to neither of them. The result is a lot of contradictory and unfounded statements or assumptions: 'a good deal of psychotherapy was inherent' in negative practice; in the simple act of pull out 'insight comes to dominate the resistance; and a further resolution of the conflict occurs'; in cancellation an opportunity is provided for 'self confrontation and evaluation'; 'the repression that usually takes place after a moment of stuttering is prevented' and so on.

But what is more objectionable is the fact that concepts from different sources are used without precisely defining them. Van Riper uses the terms such as negative practice, desensitization, without defining them in the present context. When this is so, the assumption naturally is that the terms are used with their import at their sources. But in no time we start running into irrelevnt deductions made from those concepts. Neither the technique described, nor the rationale offered correspond to the established connotations of those constructs.

The scope of this paper will not permit a consideration of the question of validity of Van Riper's therapy for stutterers. However a technique that includes negative practice, desensitization, psychotherapy and many more things cannot be justified on any theoretical grounds. At best Van Riper's is an awfully uneconomical method in terms of the time and the varied kinds of operations involved. This curious concept-salad that is Van Riper's therapy seems to stem from theoretical weakness.

It should be mentioned, however, that Van Riper is perhaps the most open minded and dynamic of all the therapists. The very fact that he borrowed theoretical constructs from many different sources and tried to develop a technique of his own illustrates this. His technique of therapy has been one of the most widely used with stutterers and theoreticians with entirely different orientation have borrowed it. He was one of the early workers to say that stuttering is a learned habit and that the Freudian concepts are not applicable to a majority of stutterers. He was also one of the early therapists to use learning principles in their therapy work.

Conclusions: A critical review of some aspects of Van Riper's theory of stuttering and stuttering therapy warrants following conclusions. Van Riper borrows concepts from very different schools of thought to explain the same phenomenon and the result is a curious mixture of incompatible hypothetical constructs. Borrowed concepts are not defined in their present context and often irrelevant deductions are made from them. Theoretical constructs like

negative practice and desensitization have been misinterpreted and used in a psychotherapy model without any justification. Van Riper's use of the term neurosis, the distinction he makes between a learned undesirable habit and neurosis, his use of learning principles in his therapy model are scientifically inaccurate.

Robert West and his associates

Robert West and his associates occupy a unique position among the theorists on stuttering. While most of the investigators' basic approach to stuttering theory is psychological, West's approach is predominantly organic. In line with the organic approach West is perhaps the most medically oriented of all the investigators in the field, as far as a theory of stuttering is considered. Since West and his associates have championed an organic viewpoint of stuttering which is in contradistinction to all other approaches, a critical look at it would be of special interest.

'Notions' on the nature of stuttering: West, *et al,* (1968) believe that stuttering has two aspects: the speech defect that we call stuttering and the disease entity that causes stuttering. But what is the stuttering symptom as could be observed? 'It is not easy to define stuttering. It is like trying to define *sneering or smiling'* (West, *et al.,* 1968, p. 118) (Italics added). Since stuttering is as idiosyncratic as sneering or smiling, no effort is made to define stuttering in a precise fashion. However, the authors do specify some five characteristics of stuttering: (1) sudden breaks in the automatic process of speech; (2) facial and bodily tensions when these breaks occur, which are reactions to ones own frustration; (3) extended periods when the case is free from these blocks and also a clustering of these blocks in various social situations; (4) tonic or clonic spasms of the phonatory or articulatory muscles during the breaks;. and (5) the occurrence, during the breaks, of various motor movements like tics all over the body. West, *et al.,* (1968) recommend these five characteristics in the place of a definition of stuttering but they further state that '. . . stuttering is that phenomenon of speech that exists when a speaker and his conversational partner tacitly agree after more or less extended experience of talking together that the speaker stutters' (p. 118).

It is evident that if all the five factors are to be taken as defining characteristics of stuttering, a large number of stutterers who do not show secondary motor movements associated with stuttering have to be excluded from their definition. However, the recent trend is to consider only the prolongations and hesitations to be the primary defining characteristics of stuttering (Brutten and Shoemaker 1966). Secondary motor characteristics may not be present in a number of cases and hence those symptoms cannot be taken as universal characteristics of stuttering. Secondly, the author's assertion that stuttering is like a smile or sneer and that it cannot be defined is to make a measurable aspect of behaviour

esotoric; and hence it is unscientific. And to say that stuttering exists when two persons agree on it, is only slightly better than saying nothing at all.

But the most interesting set of notions is about the aetiology of stuttering. West and his associates believe that stuttering is caused by three factors: (1) Atavistic heredity, (2) Brain injury and (3) Hysteroid disfluencies.

Atavistic Heredity. According to West and his associates, there are three basic *faculties* that are essential for communication: symbolization, abstraction and *automaticity.* Simultaneous development of these three faculties in the evolutionary scale resulted finally in the oral speech in the *homo sapiens.* Stuttering develops when automaticity fails to evolve, and automaticity is considered to be the slower one to develop. Accordingly, stuttering is due to a '.. .residuum of delayed evolution, or an atavism that prevents the development of a feedback, or servosystem, for fluent patterns of oral speech' (1968, p. 116). Since the existence of stuttering indicates an earlier stage of evolution, as '. . .millenniums pass, stuttering should lessen in frequency' (p. 116). For West and his associates what is surprising is not why some show disfluency, but why and how so many achieved fluency so early in the evolutionary scale! When the human assumed an erect posture, his range of vision and knowledge increased, and his vocabulary multiplied. When vocabulary multiplied, his abstractive processes increased. In order to communicate effectively, he had to achieve speed, and speed meant automaticity. Those who were lacking in the capacity for automaticity developed stuttering. And then they started transmitting this defect to their children.

Brain Injury. For West *et al.,* it appeared that stuttering in some individuals may be a form of subclinical cerebral palsy. Stuttering is often exhibited by cases with brain damage.

Hysteroid Disfluencies: Some persons develop stuttering to gain certain advantages like sympathy and attention of others. It may be a pretext to escape from the duty; it may be a justification of their failures.

These three, according to West *et al.,* are the basic causative factors of stuttering. However, there are still four more factors that characterize stuttering as a syndrome: (1) muscular hypertonicity, (2) certain physiological anamolies, and (3) hereditary anamolies.

Muscular hypertonicity. Normal speaking requires constant and rapid change in the neuromascular apparatus. Stutterers, however, are not able to achieve this because their speech musculature is hypertonic. Consequently, stutterers' voice is monotonous. Stutterers' articulation is clumsy, again because of this hypertonicity. Another effect of hypertonicity is the slowness of diadochokinesis. It means that a stutter cannot make simple repetitive tasks in a quick succession because his speech masculature cannot be relaxed readily. This mascular inertia is marked in the tonge and face of the stutterer.

Physiological Anamolies. West *et al.,* state that *'In general* it is found that the blood of the stutterer has a high sugar (glycogen) content than that of the

nonstutterer' (p. 125). This *general* finding is supported by one study by Kopp (1934). Another physiological anamoly is the irregular rate of heart beat in the stutterer. West *et ah,* quote one study by Palmer and Gillett (1938) in support of this statement. Still another physiological anamoly is suggested by the fact that stuttering is related to age and sex: it decreases as age advances and is more prevalent among the male.

Hereditary Anamolies. The stuttering syndrome is biologically transmitted. This hereditary anamoly is suggested because stuttering is more common in families that have 'multiple' births; it is related to left-handedness; and the history of allergy is common among stutterers. The authors find it' . . . easy to contemplate that twinning is a throw-back to a stage of evolution in which the young arrived in litters' (p. 117).

Moral Rigidity. Stutterers are a distinct personalities. Stutterer is a 'good' person; but his goals are unrealistic. 'He is a perfectionist, not only in speech, but in all his conduct' (p, 128). His conscience is rigid and he attaches ethical significance to ordinary events of life. Stutterer is a puritan. This whole constellation can be designated as *moral rigidity.*

Stuttering Therapy. Therapy for stuttering means a basic change in the personality of the stutterer; he should now become 'less of an introvert' (p. 349); overcoming stuttering is a 'Struggle which requires courage on the part of the patient and sympathetic understanding on the part of the clinician' (p. 349). The goals of stuttering therapy differ depending on the stage of stuttering—primary or secondary. In case of stutterers with primary stuttering following goals of therapy are set out by West *et ah:* (1) develop confidence in his ability to speak; (2) prevent the development of secondary stuttering; (3) establish normal speech habits. In case of stutters in the secondary stage: (1) get the individual to face his problem honestly; (2) eliminate overt physical mannerisms which accompany the act of stuttering; (3) build confidence in the ability to speak, thus eliminating the extreme anxiety present in the speech situation; (4) develop a realistic social, educational, and vocational adjustment; and (5) develop the ability to speak in any situation either normally or as a stutterer.

With these therapeutic goals, what are the actual operations involved in stuttering therapy? For a child with primary stuttering West *et ah,* recommend the following principles: (1) Direct attention to the speech should be avoided; (2) environmental tensions in the home, school, and in social contacts should be eliminated; (3) the child should be given opportunities for pleasing and successful speech experiences; (4) health of the child should be built up and maintained; and (5) abilities other than speech should be developed.

The therapy for a secondary stutterer'. . . should probably never be considered in terms of a 'cure', (p. 356). (Apparently because it is impossible to cure stuttering). If a stutterer is taught to control his anxiety, then he has been treated

successfully. The first step in the therapy programme is to analyse the stuttering in an objective way. This enables the stutterer to look at his own problem objectively and he would then be able to control his problem better. The second step in therapy is to eliminate the overt acts that accompany stuttering. If the stutterer, by the help of a mirror, observes his own symptoms, these symptoms then would be eliminated quickly. The third step is to analyse the fears that are associated with situations and to desensitize the stutterer to those situations. Whatever is done to achieve this, the goal is important: Speaking should be accompanied by less tension. The fourth step is to introduce difficult communicative situations which may include such activities as pantomime without any speech, reading in unison, *negative practice* of 'faking '; and so on. Group therapy should not be ignored because 'Stuttering is social in nature . . . .' (p. 360).

The Theory of Stuttering: What is objectionable in these notions? Almost everything there seems to be objectionable. Starting from the authors' attitude towards a definition of stuttering, through their notions about the causation of and finally to the therapy of it, everything seems to be based not on empirical evidences, but on their own personal beliefs. For this and few more reasons to-follow, one cannot help calling them notions.

To begin with, their notions about the causation of stuttering. Strictly speaking, there can be only one statement about them: not proven, or no evidences that might even suggest such a set of notions, let alone confirming them. But still, it is perhaps necessary to go into some detail about the bases of their statements.

The concept of atavistic heredity is probably the best example of West and his associates' personal scientific method of building hypotheses. Throughout a page and a half that they write on the atavistic heredity *not a single empirical study is quoted in support of it.* When the reader looks for some kind of evidences, he is thoroughly disappointed. Statements are made with authority and conviction; evidences are taken for granted even when they are totally absent; much worse, statements are made in the face of contrary evidences. If stuttering indicates a failure of evolution in some individuals, then at least in the ontogeny of those individuals it would never be possible ro eliminate stuttering. Apart from the fact that stuttering can be eliminated through therapeutic procedures, there is considerable evidence to show that individuals with stuttering are able to recover spontaneously from their symptom (Sheehan and Martyn 1966). If it should take millenniums for the mankind to outgrow stuttering, as the authors grimly predict it, how is that ever possible for some stutterers, who are on the lower level of evolution, to outgrow it within the span of a couple of years? How could they, within the matter of a few years, be able to come to the level of other fluent members of the society, who were, to begin with, millenniums ahead in evolution? Above all to suppose that stutterers are primitive is scientific banality.

In the development of a theory it is possible that in the early stages not enough evidences are marshalled. Considering this possibility, can we think of atavistic heredity to be a *possible* explanation of stuttering? It looks very unlikely because atavistic heredity as a notion does not even attain the status of a hypothesis that is in need of confirmation. A scientifically acceptable hypothesis should be *verifiable.* Statements that cannot be verified through the accepted methods of science fall outside the range of scientific enquiry. Atavistic heredity is such a statement that cannot be verified, and hence it is a statement of personal belief and it cannot be considered for further scientific investigation.

The second assumption made by the authors on the aetiology of stuttering is that it may be due to brain damage and is related to epilepsy. This important causative factor is being described in three sentences. Only one study, carried out in 1947, is quoted in support of this. Harrison (1947) found that stutterers are more common among epileptics. No other investigators seem to have confirmed this view. And even that single study seems to have shown only a correlation between stuttering and epilepsy. That does not, however, support the statement that stuttering was *caused* by epilepsy or by the underlying brain damage. In addition, there are a majority of stutterers who do not show either epilepsy or any other signs of brain damage.

The third causative factor, according to West *et ah,* is hysteria. Result is a psychogenic stuttering. Again, absolutely no evidences are quoted in support of this statement. The mere observation that some stutterers may use their defect to gain some advantage in society will not make stuttering a hysterical symptom. An individual who is *crippled* may be able to derive some benefits out of his condition, but that does not make his physical handicap *psychogenic.* Some stutterers may blame their stuttering for their failures and even may derive benefits out of their problem, but such a tendency depends largely on the type of personalities they are. Apart from this, to assume that such a tendency is of aetiological significance to stuttering is unwarranted. Further, there is general agreement to the effect that stuttering is more often accompanied by tension and anxiety and it is also known that hysterical manifestations are rare among persons with anxiety reactions (Eysenck 1957).

The further physiological and hereditary factors that are said to characterize stutterers are also equally unfounded. The authors' assertion that stutterers suffer from muscular hypertonicity does not seem to be supported by a single study. However, in support of diadochokinesis in stutterer, there is one study cited that was carried out by West in 1929. Later studies by a number of investigators (Strother and Kreigman 1943; Strother and Kreigman 1944 for a review) have shown no differences between stutterers and non-stutterers on the ability to do rhythmic and rapid movements. Hence, it should be concluded that there exists no satisfacotry evidence in favour of muscular hypertonicity of stutterer and that in fact, there are contrary evidences to that effect cited in the literature.

The next assertion that stutterers' blood sugar level is high is also a private notion of the authors. West *et ah,* think that it is a *general finding* but they themselves quote only one study (Kopp 1934). Similarly they quote one study in support of increased heart rate in stutterers. Again, the later studies have shown that on both these variables stutterers and non-stutterers do not differ significantly (Ritzman 1943; Golub 1952).

That stuttering is related to age and sex may not necessarily indicate a basic physiological anomaly in stutterers. Certain interests and attitudes are also related to age and sex but that does not mean they are physiological phenomena. That stuttering decreases as age advances can be interpreted in terms of spontaneous recovery based on the process of extinction of conditioned responses. There are other kinds of evidences in support of such an interpretation (Hegde 1969). The reported sex difference in the incidence and prevalence of stuttering also does not, on its own accord, prove that stuttering is physiological. Additional evidences are needed to make such an assertion, but they are lacking. Although several hypothesis have been advanced to account for the greater prevalence of stuttering in the male, none have proved very meaningful. This sex difference is quite often interpreted in favour of an organic basis for stuttering but all explanations stop at that. It might *suggest* a physiological basis, but it does not confirm it. However, it seems to the present writer that the modern learning theory can adequately account for the greater incidence of stuttering in the males. Several studies have shown (Hurlock 1964) that the female at all ages excels the male in speech and language skills, provided the other relevant factors are held constant. The male's verbal ability is relatively inferior to that of the female. This may be a matter of constitution or physiology but this in itself will not *cause* stuttering, because all the males are inferior verbally, but all of them do not develop stuttering. Consequently it can be stated that the male, with a basically inferior verbal ability runs the risk of greater chances of developing stuttering. The female, with a basically superior verbal apparatus does not have the same chances of developing it. In any case, the immediate *cause* of stuttering lies in the faulty learning. In this sense, the sex difference in stuttering perhaps indicates differential *predisposing* factor, and does not imply an immediate physiological causation. It has been shown very well, that individuals who are born with a labile sympathetic nervous system in terms of overactivity are more likely to develop anxiety and phobic reactions under conditions of stress (Eysenck 1957; Franks 1956). Similarly under stressful situations male has a greater chance of developing stuttering than the female, and this is reflected in the differential sex ratio in prevalence of stuttering.

The next assumption of the authors that stuttering is hereditarily transmitted because it is related to multiple births, left handedness and allergic problems is also not supported by empirical evidences. For West *et al.,* it is *easy to contemplate* that twinning is a throw-back to an earlier stage of evolution; unfortunately,

the geneticists or embryologists have not found it that easy. It is perhaps true that in general, stutterers tend to have relatives who also stutter (Johnson 1967 for a review) but this in itself is no evidence of biological transmission. Johnson (1967) has shown that although heredity is constant, a change in social and psychological conditions can result in a decrease in the incidence of stuttering. Again in favour of allergy being related to stuttering, no considerable amount of evidence is cited. In case of handedness, a number of studies have reported no significant association with that phenomenon and stuttering (Heltman 1940; Johnson 1967; Sheehan and Martyn 1966).

Finally, the moral rigidity that is said to be the characteristic of stutterer's personality. West *et al.,* do not seem to have noticed the fact that psychologists gave up the impressionistic method of studying personality more than four to five decades ago and that personality, since then, has been studied and being studied through empirically validated objective questionnaires. Moral rigidity, as described by the authors is a totally fictitious trait. Consistent with their tradition, they do not report any studies that have shown this trait in stutterers to a measurable degree. This alleged trait is not even precisely defined, and the authors' discussion on the question is full of unscientific statements like the following: 'The Stutterer is basically a 'good person'; 'If he is 'tempted' or 'falls', he exaggerates the significance of his sin', etc. (p. 128).

The therapy of stuttering : The therapy of stuttering recommended by West *et al.,* evidences a great contradiction in their total approach to stuttering. The author's approach to stuttering theory is *entirely physiological;* but their approach to stuttering therapy is *entirely psychological.* The goals of therapy are psychological (to develop confidence in the stutterer, etc.), and the actual operations of therapy are also psychological from the beginning to end (remove the environmental tensions, effect better adjustment etc.). So when the authors enter into a discussion on stuttering therapy they seem to forget their own theory of stuttering. There is no mention of atavism, heredity, brain damage, blood sugar level. There is a yawning gap, an irreversible logical falacy, an inherent contradiction between their 'theory' and therapy of stuttering. After presenting their notions of stuttering as a physiologically based *disease,* West *et al.,* recommend Van Riper's book 'For a splendid discussion of the therapy of stuttering . . . ' ! (p. 130). If Van Riper's approach, which does not believe in a physiological basis of stuttering, is useful, then the authors' notions on stuttering become totally irrelevant to the therapy they recommend.

Afterall, the two set of principles, one on the nature of the disorder, the other on the therapy for the same, should be complimentary to each other. Infact, therapy of any disorder is nothing but a set of deductions made from the theory of that disorder. In other words, the basic principles of a therapy for a disorder are dictated by the theory of the nature of that disorder. The two can never be unrelated. If they are unrelated, however, at least one of them is false. On the

other hand, it is also possible to work backwards to a theory of **a** disorder, if it is shown clearly that a certain operation is effective in eliminating that disorder. Accordingly, if we make a set of deductions from the West *et at,* theory of stuttering it would immediately contradict the therapy they advocate. If stuttering is an organic fault there is no other way of eliminating this disease except by correcting that organic fault; if stuttering is caused by an error in the metabolism, this should be corrected and nothing else. If stutterers are in the lower level on the evolutionary ladder, well there is no therapy for this and the therapist can only sit, watch and die with a hope that they would evolve some day. If it is a matter of gene transmission the best that can be done is to try to isolate the nature of such a transmission and see whether this can be prevented; nothing else can be expected to cure the condition. In this way, a physiological theory of stuttering dictates a physiological treatment of it. One cannot hold that stuttering is due to brain damage or faulty metabolism and then recommend a change in the stutterer's attitude for therapy; unless of course, one also believes that a change in the attitudes will induce changes in metabolism! With this, it is clear that West *et at,* do not follow their own theory of stuttering in recommending a programme of therapy.

From the standpoint of their therapy, then, what deductions can be made about the theory of stuttering? In the treatment of both child and adult stutterers what they seem to emphasize on is to remove tension and anxiety; create opportunities for more pleasant speech experiences. The authors assert that if the anxiety in an adult stutterer is controlled, he can be considered to have been treated successfully. Granting that such measures would result in fluency, what becomes then of a theory of stuttering? It amounts to saying that stuttering is caused by tension and anxiety. In other words, the presence of tension and anxiety is the cause of stuttering and the elimination of them is the therapy for it. This would be the inevitable conclusion on the causation of stuttering if we follow the therapy advocated by West *et at* Since West *et at,* do not come to such an obvious conclusion the contradiction is not resolved. If anxiety is treated, adjustment is effected, personality is changed and if all these result in fluency, what happens then to atavistic heredity? To brain injury? To metabolic fault? If stuttering can be removed without altering physiological conditions, then those physiological conditions cannot be the cause of stuttering. It was John Stuart Mill, the 19th century British empirical philosopher who said that if B appears when A is introduced and B disappears when A is removed then probably C is not the cause of B! So in summary it can be stated that the 'theory' of stuttering advocated by West *et at,* does not predict the therapy they prescribe and the therapy they advocate for does not predict the theory they advance. This is in addition to the fact that the 'theory' of stuttering is totally devoid of evidences and the therapy is very inadequately formulated. The criticisms levelled against Van Riper's therapy will hold good here also.

Conclusions: A critical review of the 'theory' and therapy of stuttering advanced by West and his associates warrants the following conclusions. West *et ah,* have failed in providing evidences in favour of an organic approach to stuttering. The authors make statements for which not only there is absolutely no evidence but also the ones that cannot even be accepted as hypotheses for scientific investigation. More then anything the total system of notions is entirely speculatory and several of these fly in the face of contrary evidences. The therapy the authors advocate is unrelated to the 'theory' they hold. The very fact that they advocate a psychological therapy for stuttering negates an organic basis of stuttering, apart from providing a glaring contradiction in their total approach.

REFERENCES

Brutten, E. J. and Shoemaker, D. J. (1966) *The Modification of Stuttering.* Engle-Wood Cliffs: Prentice-Hall.

Dunlap, K. (1932) *Habits, their Making and Unmaking.* New York: Liveright.

Eysenck, H. J. (1957) *Dynamics of Anxiety and Hysteria.* London: Routledge, Kegan Paul.

Eysenck, H. J. (1964) *Experiments in Motivation.* London: Pergamon Press.

Franks, C. M. (1956) Conditioning and personality; a study of normal and neurotic subjects. *F. abnorm. Soc. Psychol.,* 52,143-150.

Golub, A. J. (1952) The heart rates of stutterers and non-stutterers in relation to frequency of stuttering during a series of oral readings. Unpublished Ph.D. thesis, Univ. of Iowa. Quoted from Johnson and Moeller (Eds.) *Speech Handicapped School Children.* New York: Harper and Row.

Harrison, H. W. (1946) A study of the speech of sixty institutionalized epileptics. M.A. thesis. Louisiana State Univ. *Speech Monographs,* 1947 (Abstract).

Hegde, M. N. (1969) The significance of spontaneous recovery of stutterers to stuttering as a classically learned maladaptive habit. To be published.

Heltman, H. J. (1940) Contradictory evidences in handedness and stuttering, F. *Speech Dis.,* 5, 327-332.

Sheehan, J. G. and Martyn, M. M. (1966) Spontaneous recovery from stuttering, F. *Speech Hearing Res.,* 9, 121-135.

Hurlock, E. (1964) *Child Development.* New York: McGraw-Hill.

Jacobson, E. (1938) *Progressive Relaxation.* Chicago: Chicago Univ. Press.

Johnson, W. (1967) Stuttering. In Johnson, W. and Moeller, D. (Eds.) *Speech Handicapped School Children.* New York: Harper and Row.

Jones, M. C. (1924) A laboratory study of fear: The case of Peter. *Pedagog Sem,* 31, 308-315.

Kendrick, D. C. (1960) The theory of 'conditioned inhibition' as an explanation of negative practice effects: an experimental analysis. In Eysenck, H. J. (Ed.) *Behaviour Therapy and the Neurosis.* Oxford: Pergamon Press.

Kopp, G. A. (1934) Metabolic studies of stutterers. *Speech Monograps,* 1,1.

Lehner, G. P. J. (1954) Negative practice as a Psychotherapeutic technique. *J. Gen. Psychol.* 51, 69-B 2.

London, P. (1964) *The Modes and Morals of Psychotherapy.* New York: Holt, Rinehart and Winston.

Palmer, M. F., and Gillett, A. M. (1938) Sex differences in the cardiac rhythms of stutterers. F. *Speech Dis.,* 3, 3-12.

Rathna, N. (1069) Behaviour Therapy and Speech Pathology. *Proceedings of the Seminar on Behaviour Therapy.*

Ritzman, C. H. (1943) A cardiovascular and metabolic study of stutterers and nonstutterers. *F. Speech Dis.,* 8,161-182.

Strother, C. R. and Kreigman, L. S. (1943) Diadochokinesis in stutterers and nonstutterers. *F. Speech Dis.,* 8, 323-335.

Strother, C. R., and Kreigman, L. S. (1944) Rhythmokinesis in stutterers and nonstutterers. *F. Speech Dis.,* 9, 239-244.

Van Riper, C. (1958) Experiments in stuttering therapy. In J. Eisenson (Ed.) *Stuttering: A Symposium.* New York: Harper and Row.

Van Riper, C. (1963 a) *Speech Correction:* Engle-Wood Cliffs: Prentice-Hall.

Van Riper, C. (1963 b) Symptomatic therapy for stuttering. In Travis, L. E. (Ed.) *Handbook of Speech Pathology.* London: Peter Owen.

West, R. W. (1929) A neurological test for stutterers, F. *New. Psychopath.* 10,114.

West, R.W., and Ansberry, M. (1968) *The Rehabilitation of Speech.* New York: Harper and Row.