REFERRAL: "55-year-old, male, with severe expressive aphasia; please evaluate and treat as indicated."

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Comments by: A. Damien Martin, Ph. D., Speech Pathology, Veterans Administration Hospital, New York, New York

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PATIENT: J. H. BIRTHDATE: 12-16-18 AGE: 55

REFERRAL INFORMATION: This 55-year-old male, patient, was reported to have suffered a left middle cerebral artery thrombosis on 6-1-65 resulting in right facial paralysis, right hemiplegia, and "slurred speech." Neurologic examination at this time reported no cranial nerve involvement except that causing the right facial weakness. The patient was discharged from the VAH on 8-19-65, having had no speech evaluation or therapy with "...full confidence that he could return to the same job (bookkeeper)."

On 11-20-70, J. H. was readmitted because he had developed a tonoclonic seizure disorder. Neurologic findings were essentially unchanged from those of August, 1965, except for the reported seizure patterns. Seizure control medication was initiated and the patient was discharged from the hospital.

On 7-12-73, the patient was admitted due to acute onset of "aphasia." Neurologic findings were essentially as above except for an exacerbation of the right hemiplegia, the expressive speech disturbance, and a dense dysphagia. The diagnosis indicated a second left middle cerebral artery thrombosis. Because of the impenetrable voluntary swallowing disturbance, a feeding gastrostomy was performed on 8-21-73. J. H. was also referred to the Speech Pathology Service (see title) on 8-30-73 for evaluation.

EVALUATION DATA: J. H. has been examined by this Service on a number of occasions since the original referral. The Porch Index of Communicative Ability (PICA) has been used as the

primary measure of overall severity of communicative dysfunction. However, a number of other measurement procedures have also been administered, including the Peabody Picture Vocabulary Test--Part V (DeKenzi and Vignolo, BRAIN, 1962), and a clinical oral-peripheral examination. Results of these procedures are summarized on the attached Speech/Language Data Sheet.

The most obvious finding from the data is the glaring disparity when comparing the patient's verbal performance with his ability to communicate graphically or through gestures. The modality percentile scores on the four PICA examinations illustrate this. The Minnesota Test for Differential Diagnosis of Aphasia (Schuell Test), administered for comparison on 12-20-73, also confirmed the vast performance gap in communicative output.

All tests indicate that the patient understands what is said to him. Token Test (Part V) and PPVT scores certainly confirmed this. Both the PICA and the Schuell Test indicate that J. H.'s reading ability is within normal limits. Input for communication, therefore, certainly seems to have been spared by the two cerebrovascular incidents.

With regard to graphic output, a few deficits were noted upon evaluation following the original referral. On the PICA of 9-12-73, J. H. exhibited a few spelling errors and a consistent pattern of incompleteness when writing sentences. The spelling errors also were present in spontaneous writing of nouns; however, this pattern seemed to disappear when the nouns were written from dictation. Over time (from 9-12-73 to 3-18-74), the spelling errors decreased in frequency, and the incompleteness disappeared by merely asking the patient to be sure to write in complete sentences. The incompleteness was felt (and was confirmed by J. H.) to be a telegraphic compensatory mechanism used by the patient in an attempt to reduce writing time with minimal effect to the information being communicated.

The extremely reduced verbal ability has been pervasive of all tasks requiring oral function. The oral-peripheral examination, performed on 12-20-73, revealed no overt paresis of oral structures, except possibly the right facial weakness which has been present from the outset. This weakness has been felt to be the reason for the intermittent mild-moderate drooling from the right corner of the mouth. All oral, pharyngeal, and/or laryngeal reflex mechanisms have been noted to be normal. Functional problems in swallowing, nonverbal oral activities, and verbalization were felt to be the result more of an extremely dense oral/verbal apraxia with the possibility of a mild overlay of dysarthria involving corticobulbar fibers.

GENERAL IMPRESSIONS AND CLINICAL RECOMMENDATIONS: The standardized tests and clinical examinations indicate that the last cerebrovascular incident rendered the patient speechless in the presence of relatively normal language function. Because of the somewhat static recovery profile exhibited in the four PICA examinations between 9-12-73 and 3-18-74, it seems unlikely that J. H. can expect any reasonable degree of recovery of oral/verbal function. These predictions are reinforced by the medical decision that gastrostomy feeding would be indicated rather than extended therapy to improve the patient's swallowing ability. Therefore, it is the opinion of this clinician that a longitudinal plan of therapy designed to improve the patient's ability to verbalize is unrealistic and unwarranted. This seems even more apparent in light of environmental demands.

J. H. was originally discharged from the VAH to a nursing home because his sister (only living relative) did not feel that she could care for him, primarily due to his feeding difficulties and the spontaneous communication deficit. This situation has, however, been reversed most likely because of the communication therapy that has intervened.

THERAPY PROGRESS REPORT: When J. H. reported to the Speech Pathology Service for evaluation, his primary means of communication was through the graphic modality. He wrote abbreviated notes when his needs made overt communication inevitable. It was through interaction in this way that the clinical staff of this Service realized that he was dissatisfied with this method of communication and that his writing was actually deteriorating through the maladaptive habituation of his compensatory telegraphic writing.

This fact, plus the indications from testing indicating good auditory comprehension and facility of gestural response, led to a decision to make a concerted attempt to shift the patient toward the use of structured gestural output in lieu of writing for communication. Since one of the clinical staff was trained in Amerind Sign, an approach to gestural language which was developed by Dr. Madge Skelly at the VAH in St. Louis, Missouri, as a variant of American Indian Sign Language, a number of sessions were initiated on an outpatient basis to introduce J. H. to Amerind and begin his language metamorphosis.

J. H. responded quickly and positively to this approach. In fact, formal instruction in Amerind was never actually necessary because of the patient's ingenuity and initiative in developing his own arbitrary gestural symbols. Once a consciousness of the power of gestural communication was developed, J. H. rapidly invented a wide range of signs that were easily decoded by those in his environment. The shift in emphasis toward gestural output apparently was a major factor in the decision by J. H.'s sister to have him moved from the nursing home and live with her family.

Active therapeutic involvement with the patient has been terminated; however, long range outpatient followup is being pursued to check the reliability of the predictions made regarding the patient's verbal output. If, upon further testing, significant changes are noted in the patient's ability to verbalize, an active hospital based home therapy program will be initiated with a change in emphasis toward verbal output.

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SPEECH/LANGUAGE DATA

PATIENT: J. H. BIRTHDATE: 12-16-18 AGE: 55

PICA	9-12-73	12-20-73	1-23-74	3-18-74
OVERALL %ile	59	63	70	69
GRAPHIC %ile	98	99	100	98
VERBAL %ile	2	6	5	6
GESTURAL %ile	91	92	99	100

PPVT

RAW SCORE	141
IQ (18 - 5)	131
%ILE (18 - 5)	100

TOKEN TEST

V SCORE	21/22

ORAL EXAM

STRUCTURAL:	NO ABNORMALITIES
FUNCTION :	ALMOST NO VOLUNTARY CONTROL
	(R) MILD FACIAL WEAKNESS
	MILD - MODERATE DROOLING (R)
	TONGUE: STRENGTH O.K.; MOVEMENT SLOW
	REFLEX GAG, COUGH, PALATE - O.K.
	DYSPHAGIA: SEVERE
RESPIRATION:	GOOD
PHONATION :	WET HOARSENESS

COMMENTS BY: A. Damien Martin, Ph. D., Speech Pathologist
VAH, New York

The test results, as well as the samples of behaviour shown on the videotape indicate quite clearly that the patient is suffering from a severe dysarthria coupled with a severe oral facial apraxia. I would agree that, for all practical purposes, the patient is not aphasic.

Before outlining the direction and course of therapy which I would recommend, I would like to review some of the findings on the peripheral speech mechanism evaluation.

First, I found it surprising that there was no evidence of primitive reflexes. In my experience, the patient who has such severe dysarthria generally will show some evidence of primitive function. However, this is all to the good, since no work need be done to inhibit and eventually eliminate those reflexes. I also found it surprising that the gag, palatal, and tongue retrusion reflexes were still intact. Especially since the swallowing reflex is so obviously impaired as evidenced by the patient's drooling and his inability to swallow food.

I would like to see a further evaluation of the laryngeal area since the quality of the obtained phonation gives some indication of vocal fold involvement as well. The similarity between the rather strained cough on demand and the reflexive cough a little later on the tape would seem a further indication of some possible laryngeal involvement.

One of the first steps in therapy planning is the decisions as to the goals of therapy, although these goals may change as one works with a patient. Here my choice of a primary goal would be the rehabilitation of the swallow pattern. As speech pathologists we are often called upon to deal with other than verbal problems. Swallowing is one of these related areas. At the New York Veterans Hospital we frequently find it necessary to work on rehabilitating swallowing patterns which have been disrupted as a result of neurological impairment or surgical intervention. Each patient presents a specific individual pattern of impaired swallowing, therefore, it is impossible to set forth a blueprint for rehabilitation of swallowing. However, the PSM exam and the videotapes in this case do give us a few guidelines as to where one might start.

The intact palatal and tongue retrusion reflexes might be used to initiate and stimulate swallowing. Again, it is difficult to decide on a particular task, and impossible to evaluate its effectiveness if the patient is not directly in front of you. However, one method of utilizing the reflexes would be to place, with a straw, a few drops of cold water on the back of the tongue

or the palate. Resistive exercises designed to strengthen the weakened speech musculature and to control drooling could be used in conjunction with other techniques.

I do not feel speech performance per se would be the immediate or primary goal in this case. However, there would be no reason not to introduce articulatory facilitation techniques at some point in therapy (Schuell et al., 1965, pp 349-352).

DISCUSSION: Jay Rosenbek, Ph. D.
University of Colorado

I've arranged this discussion according to a set of management questions. By so doing I hope to be both specific enough with regard to the patient whose communication difficulty is skeleton for this discussion and general enough with regard to the literature on rehabilitation so that we can have a lively exchange about philosophy and technique. The ordering of these questions implies nothing about their relative importance, nor with some exceptions the order in which they might be asked. The list is representative not exhaustive; descriptive rather than prescriptive.

Question One: What is the patient's speech diagnosis?

Treating a patient prior to diagnosis is improper. Speech pathology is evolving differential treatments for the dysarthrias, dyspraxia and dysphasia. In a very real sense we are arriving at the time (perhaps we have been there for decades) when we can treat labels, bearing in mind, of course, that an individual wears that label.

The differential diagnosis in this case appears to be apraxia of speech and dysarthria presumably of the spastic type. I'll not go into the details of the neurological and speech examination as those have been provided by Dr. Berry, nor will I catalog the additional diagnostic tests that might have been performed as this would take us far afield of our purpose. Rather I will merely underline Dr. Berry's own diagnosis - severe apraxia of oral structures. This diagnosis seems consistent with the test findings. In addition the patient would seem to have mild spastic dysarthria[1] and very, very mild aphasia. My treatment emphasis would be on the apraxia.

[1]During the discussion Dr. Brookshire suggested the proper diagnosis was ataxic dysarthria.

While I have previously defended labeling, the truth is that they are almost exclusively fit for discussion among friends. Apraxia is one label that frequently disrupts communication. Descriptions are a more universal message and so as not to impair communication with a word - apraxia - lets identify the disturbed linguistic dimensions. They are two: articulation and prosody. And what abnormal mechanism accounts for these deficits? Goodglass and Kaplan (1963) provide us with the answer - a disruption of movements most obvious on request and only somewhat improved upon imitation.[2] Our patient's movement disorder appears to be of that type. Having labeled and/or described the patients problems and determined the relative severities of them other management questions are more readily answered.

Question Two: Should the patient be treated?

Yes. As Luria observes in Traumatic Aphasia (1970) "The 'rebirth' of speech can come about only as a result of special retraining." Equally appropriate is Wepman's (1951) contention that such a patient may continue to improve for a considerable length of time post-trauma. Given this specific patient, however, his severity and duration of apraixa, I would consider the management of oral communication to be frankly pragmatic and necessitating completion against a background of family and patient counseling that provides them no false hope about the return of oral expression.

Question Three: What is the patient's prognosis?

Prognosis for this patient as for patients with Wernicke's aphasia resides in the interaction of a list of variables known to us all. Of specific interest in this case is the prognostic significance of the dense oral, nonverbal apraxia. Butfield (1958) notes that severe "mouth apraxia" signals a poor prognosis for return of oral communication. My feeling is that we can apply the same principle to psychomotor (praxic) functioning and speech that we do to lower levels of motor function and speech. Adequate speech is scarcely possible when bulbar involvement disrupts life sustaining processes such as chewing and swallowing. So too can we predict severe limitation of volitional oral communication so long as apraxia invades primary oral function. In this regard Luria (1970) observes that "preservation on the basic levels is most important for the restoration of articulatory movements in severe disturbances of expressive speech." Praxis is the basic level referred to.

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[2]As many of you know that description defines apraxia according to Goodglass and Kaplan. Apraxia, in this patient's case, apraxia of speech and oral nonverbal apraxia, is the term I'm most comfortable with and I'll continue to use it throughout this paper.

The duration of the profound oral, nonverbal apraxia also bodes ill for this patient as the condition had been present for approximately two months at time of test. Vignolo (1964), for example, considers that persisting anarthria including oral apraxia which is still present after two months is a poor prognostic sign. The effect of severity and duration is to make his prognosis for spontaneous recovery poor indeed. I would hasten to add that the prognosis being referred to is for spontaneous recovery of oral communication only. Obviously he functions rather normally in other modalities, so well in fact that he might be employable, assuming his health can be secured.

His prognosis for improvement with therapy is better, presumably, but still not good. In our center we would hold out no hope but would try a variety of therapies specifically tailored to apraxia before abandoning it entirely. Dr. Berry has not yet shared with us his specific therapy paradigms. If he tried to manage oral movements and the patient is still as we see him, then his prognosis for return of oral communication is only fractionally short of hopeless. If the patient has been subjected to the facilitation, motivation, and stimulation therapy sometimes defended for the aphasic patient, his failure to thrive need not darken his prognosis unless its predictable failure severely impaired motivation.

Question Four: Should therapy be initiated at the level of nonverbal movements?[3]

In America, Schuell, Jenkins, and Jimenez-Pabon (1964) advocate working at the level where a patient begins to experience difficulty and in the case of a patient with sensori-motor (apraxic) involvement who cannot imitate verbal gestures such as sounds and syllables, nonverbal gestures must receive therapeutic attention. She recommends in-out and lateral tongue movements with a tongue blade as target among her specific techniques. She also advocates abandoning these drills when the "patient can imitate phonation and move the tongue voluntarily." Luria (1970) begins with nonverbal or as he calls them, "practical movements of the oral apparatus" and builds speech sound gestures on these practical movements. The [p] is his first speech target and he teaches it by first teaching the

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[3]This question fomented warm discussion. Dr. Martin concentrated almost exclusively on the techniques for managing such movements, especially swallowing. Mr. Keith observed that muscle strengthening accomplished at least in part by nonverbal exercise was warranted.

patient a series of blowing tasks.[4] As he makes the transition from blowing to sound production he uses a variety of aids such as pictures of correct production, phonetic placement and the mirror, all of which will be discussed in subsequent sections of this paper. Like Luria, Butfield and Zangwill (1946) teach nonverbal gestures and then associate speech sounds with these nonverbal movements. This was Goldstein's (1948) management philosophy as well and Butfield and Zangwill acknowledge his contribution to their methodology.

At the University of Colorado we employ oral, nonverbal movements in therapy according to two criteria: (1) If we are unable to establish any speech sounds using integral stimulation, phonetic placement or phonetic derivation. (2) If the movement is a phonetic dimension of a sound to be taught. These criteria, at very least, tacitly assume as rapid as is possible movement to speech gestures. This brings us to our next question.

Question Five: What speech stimuli should be introduced first?

Wepman (1951) begins with sounds the patient can imitate. Like Luria (1970), his experience nominates [p], [b] and [m] followed by vowels [i], [eɪ]and [a]. In Wepman's first group, or group of sounds that are likely first candidates for teaching, he also includes [f], [v], [θ] and [ð]. You recognize that some in this first group are among those most often in error in the single-word performance of apraxia patients (Shankweiler and Harris, 1966; Johns and Darley, 1970).

Our experience has been only slightly different from Wepman's. Our single-sound stimulability test given to each apraxic patient prior to therapy usually discovers that one or more of the vowels are easiest. Phonation with an open vocal tract on [a] is the usual starting place and this sound can yield often imperfect but passable [i] and [u] with a bit of lip spreading or rounding. With this patient, attempts to establish vowel contrasts can begin immediately as he approximates some vowels on integral stimulation rather easily.

Bilabial consonants may be next although we have had patients who gained quick control over other, presumably more difficult sounds such as the apico-alveolar fricative [s]. An almost universal finding appears to be that the voiceless are easier than the voiced consonants. Perhaps this seeming difference

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[4]The interested reader can refer to his rather complete discussion of therapy in Traumatic Aphasia. Mouton (1970).

is in large measure artifact, however. Probably more crucial than voicing per se is number of valves employed. Voiced consonants require both laryngeal and labial or lingual valving. Systematic testing might reveal that [h] and the non-English bilabial fricative [θ] would be equally as easy as voiced [a].

The important things seem to be to allow the patient's idiosyncratic performance to govern order and to establish immediate, volitional control over even a limited number of speech sounds early in therapy.

Question Six: Should therapy be drill or general stimulation?

Alajouanine and Lhermitte (1964) have observed that the modality bound disorders such as apraxia need a direct, exercise therapy and that aphasia(s) respond to more general stimulation techniques. Even Wepman (1951) who popularized, if not fathered, motivate, facilitate, stimulate therapy; in his work on recovery from aphasia, emphasized the use of drill in cases of experience disturbance secondary to a "verbal apraxia." He, of course, reminds us that a narrow focus on the speech modality leaves us at best purblind and that drill is inadequate. I think most of us would agree with both his emphasis and his admonition. Schuell, who implicates auditory deficit in all aphasic types, suggests specific drills but emphasizes helping the patient regain auditory control over articulatory events.

These authors then would probably argue that our patient be given a trial period of exercise or drill therapy as opposed to a more general stimulation therapy. Our experience has been that the apraxic patient thrives on drill and will, given good health and a reasonably supporting environment, practice to the limits of his clinician's endurance. If drill promises anything for this patient, that promise will be realized in only a few sessions and care would have to be exercised to prevent his investing a fortune in drill only to receive a paltry, frustrating compensation.

Question Seven: Should practice be massed or space?

This question has infrequently been asked and no experimental and very little ancedotal data are available. In an earlier publication (Rosenbek, Lemme, Ahern, Harris and Wertz, 1973), we advocated massed practice but our rationale was hopelessly inadequate. Apraxic patients like aphasic patients exhibit what Wepman (1972) has characterized as a "shutter" effect. At intervals the patient is capable of profiting from stimulation. Our experience has been that continuous testing to determine the proper rate and number of stimulus presentations for each patient on each day is an essential therapeutic activity. The mere repetition of stimulus presentations may violate the "shutter" principle.

Question Eight: Which of speech pathology's general techniques is appropriate with this patient?

The three prime candidates, or so it seems to me, are integral stimulation, phonetic placement and phonetic derivation. With minimal integral stimulation this patient appears from the tape to be producing approximations of [i], [ou], [ʌ], and perhaps [a] and [mʌ]. These approximations may well be modifiable with phonetic placement procedures. The majority of clinicians recommend manipulating apraxic articulators because of the nature of the disorder (relative preservation of function in all modalities save speaking) explanations of proper points of articulation and pictures showing proper gestures are generally recommended as well.

Probably this patient needs to develop more predictable control of respiration also. Texts are replete with specific phonetic placement drills for helping such a patient learn to coordinate respiration and phonation. Schuell, Jenkins, and Jimenez-Pabon (1964), for example, discusses specific procedures so I'll not repeat them here. In passing, it should be emphasized that attention to respiration is as crucial in apraxia as it is in dysarthria.

Phonetic derivation has a significant place in apraxia therapeutics. By phonetic derivation I mean simply the process of deriving an articulatory gesture from any other intact verbal or nonverbal gesture. For our patient, [I] yields [aI] if he drops his jaw and [a] if he rounds his lips; blowing yields [p] with slight modification. Any front line clinician can generate a plethora of additional examples. The point I would make is that we can manipulate our patient's articulators and the stimuli we introduce in ways that yield discrete articulatory gestures and such manipulations should not be delayed even one session after diagnosis is complete.

Of general interest to many clinicians is the use of recurring utterances as material for derivation. Who among us has not tried to mold such utterances into volitional, purposive communication? I admit to failure in such attempts. I'm not sure of the reasons but a possible explanation is provided by Alajouanine (1956). According to him, one sign that a recurring utterance is breaking up and that a patient is moving toward volitional productions is the patient's ability to inhibit or check the utterance. It may be that the stereotype, even if meaningful, should be inhibited in our therapy. Perhaps we should not allow this patient to respond with his unintelligible grunt and only after he can check it himself should we begin work on articulation.

Serial utterances such as counting on the other hand are useful sources for phonetic derivation. We have molded "want to" from "one, two" and isolated the [w] from [wʌn] and the [aɪ] from [faɪv]. Unfortunately this patient is so profoundly involved that he has no serial utterances so that sources of derivation would have to be previously taught gestures.

Question Nine: What is the importance of the other modalities in a total therapy program for this patient?

He needs functional communication.[5] The graphic modality can give him that ability. Dr. Skelly (1973) in her book on glossectomee rehabilitation has an excellent section on teaching her patients a graphic shorthand to replace inefficient and unnecessary syntactic completeness. Her suggestions are applicable to this and other profoundly involved patients as well. Our experience has been that patients are initially reluctant to adopt such a shorthand both because they want to talk and because if they do write their habit is to do so as they did premorbidly. This reluctance can be overcome with counseling and training.

Analysis of this patient's graphic performance reveals mild deficits. If, and only if, he requested it or his job demanded it, you might teach him to scan for relatively minor errors in his writing and correct them. Because his aphasia is so mild I would anticipate that he could learn compensatory self-correction rather readily. Again, I would emphasize that such efforts should be initiated only if he wants or needs them. Based on this patient's history I would suspect that such work will be unnecessary for economic or life-demand reasons but another compelling reason exists for working extensively on reading as well as writing.

Maintaining reading and writing competence may delay degeneration of language comprehension which can result from such profound impairment of speech. It therefore behooves us to work up a carefully graded set of reading and writing exercises for this patient so that he is required to perform higher level linguistic activities. It is the opinion of Dr. Hans Waengler, a phonetician on our staff, that such activities delay but not preclude the eventual deterioration of overall linguistic functioning. Maintenance of proficiancy at a premorbid level, according to him, can be accomplished only with articulatory gestures. Be that as it may, retarding degeneration of skills is as legitimate a therapeutic aim as returning them to normal.

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[5] Dr. Berry discussed a system of sign language employed with this patient which allowed him to communicate and because he could, to escape living out the rest of his life in a nursing home.

Question Ten: What are the possible uses of intrasystemic facilitation with this patient?

According to Luria, intrasystemic facilitation refers to any procedure that restores function by shifting that function "down to a lower level, i.e., have it carried out at a more primitive automatic level, and thus avoid the effects of disorders involving the voluntary execution of the motor acts "or" to shift the function up by giving it new meaning and transferring its execution to the level of higher cortical processing." According to Luria we utilize intrasystemic facilitation everytime we incorporate a "lower level" behavior such as blowing into an articulatory gesture such as [p]. In other words, some forms of phonetic derivation are example of this form of facilitation.

Another form of intrasystemic facilitation useful in apraxia therapeutics is singing. Rather than going into details let me refer you to two papers, one by Albert, Sparks and Helm (1973), the other by Keith (1973). These authors have outlined possible paradigms for restoring functional communication using singing. Albert et. al. hypothesize that singing facilitates language use in the nondominant hemisphere. Whatever the reason, and it probably has to do with timing of articulatory gestures, some apraxic patients are aided by strongly rhythmical stimuli. The more traditional use of primary, equal and even stress may be beneficial for the same reasons. If singing or rhythmical stimuli are going to be useful with this patient, their palliative effect will be evident within a very few sessions. A good rule is to try rhythm as part of the initial diagnostic workup.

Question Eleven: What are the possible uses of intersystemic facilitation with this patient?

Intersystemic facilitation implies, according to Luria (1970), restoration of function through the utilization of another system or systems. His example serves to clarify the concept. He describes a patient with Parkinsonism whose festinating grasping movements could be slowed and regularized if the patient preceded each squeeze of his hand with an eye blink. As Luria describes it, grasping was now "carried out on the basis of a new functional system." Blinking "was incorporated as the initiating stimulus to the act of grasping."

In apraxia therapeutics this concept of intersystemic facilitation allows us to codify and understand the usefulness or effect of a variety of common methods. Visual input, for example, is almost universally acclaimed as an intersystemic

facilitation for apraxic patients. The mirror guides the patient's tongue as does watching the clinician. Visual sounds are introduced early in therapy and we attempt to help the patient incorporate visual cues into what are normally motor-auditory-tactile articulatory schemata. Dabul and Bollier (1973) in an interesting, unpublished paper advocate eliminating the auditory altogether.

Written stimuli, descriptions of proper points of articulation, and diagrams of articulatory gestures are also useful within the context of intersystemic facilitation. These last techniques, if my experience with chronic, severe apraxic patients is valid, will promise little or nothing for this particular patient; mirror work may.

On the other hand, a form of intersystemic facilitation employing a variety of total body and limb gestures and modeled somewhat after the Luria "blink, squeeze" example has produced satisfactory results in one patient and could be tried with this patient. What we suggest is combining a definite, rhythmical body gesture with sound and syllable productions. For example we might ask this patient to say [a] as he brings one arm and then the other up to his chest. In another easier form of this same facilitation, the patient produces the target utterance while the clinician moves the patient's arm through any of a variety of rhythmic patterns.

The type of limb gesture does not seem crucial. It should, of course, be simple and efficient. Parallel directions for the oral and limb gestures such as simultaneously moving the arm laterally for [i] and toward the midline for [a] are probably advisable as well. The gestures (we use several with each patient) may be chosen from the range of motion exercises completed by the physical therapist. Georgianna Johnson, a physical and speech therapist who taught me this procedure, has success pairing articulatory gestures with alternate raising and lowering of the shoulders. Whatever gestures are used, they should be repeated again and again, they should be forceful without being painful, and the clinician should take an active role in guiding and coordinating these gestures.

This method of intersystemic facilitation has inevitable but far from insurmountable shortcomings. Patients sometimes are reluctant to cooperate. An adequate explanation and knowledge of results usually are sufficient to get the patient's total cooperation. In addition, many speech pathologists lack the necessary training so that they too are reluctant to make a wholehearted investment in the procedure. Consultation with a physical therapist pays the compound divident of reducing the speech pathologist's burden and establishing the basis for cooperative team management. Finally, the procedure does not work with all patients and to date we are far from sanguine in

our predictions about who will thrive and who will not. Probably the method is best for the patient with a true apraxia; probably it is little better than a host of other methods for those patients described by Goldstein (1948) whose expressive deficit reflects a disruption of inner speech rather than apraxia and is therefore truly an aphasia, not an apraxia.

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