APHASIA REHABILITATION

EFFICACY

Parloff (1986) observed, "In an inconsistent and uncertain world, it may be perversely comforting that over the past 80 years doubts, skepticism, and incredulity regarding the efficacy of psychological treatments have been dependably constant and certain. Neither variation in the nature of claimed nor apparently authoritative evidence has diminished critics' regular and expectable expressions of mistrust."

The efficacy of treatment for aphasia has remained controversial. Reviews ranging from Darley (1972), to Wertz (1995), and to Holland et al (1996) suggest that treatment for aphasia is efficacious for patients who meet specific selection criteria. Nevertheless, criticism abounds. A *Medical World News* article (Anonymous, 1969) concluded, "The classic aphasic patient...comes in on a stretcher and isn't talking. When he leaves, he is walking but not talking." A *Lancet* editorial (1977) concluded, "Assessment of the value of aphasia therapy is virtually impossible." More recently, Pedersen et al (1995) reported no differences in recovery between patients who did and did not receive therapy. Van Gijn and Dennis (1998) state, "Systematic review of the existing evidence is urgently needed, and probably new trials as well." The methods and phases of outcomes research in general, the levels of evidence that document the efficacy of treatment, the evidence from group treatment studies, and the results of meta-analyses of treatment research are reviewed here. A few final conclusions on the efficacy of treatment for aphasia are provided.

Nosology in Outcomes Research

Considerable confusion results from the inappropriate use of terminology employed in outcomes research. The terms "outcome," "efficacy," "effectiveness," and "efficiency" are not synonymous, but are often mistakenly substituted for each other. *Outcome* describes a difference between two points in time, as, for example, the difference between pre- and post-treatment performance. Improvement in language function after a course of treatment documents a favorable outcome, but it indicates nothing about the efficacy, effectiveness, or efficiency of the treatment. Failure to control for variables that may influence improvement, for example spontaneous recovery, permits no inference about the effects of treatment.

Efficacy is "the probability of benefit to individuals in a defined population from a medical technology applied for a given medical problem under *ideal* conditions of use" (Office of Technology Assessment, 1978). Three constraints are implied in the definition. First, the statistical inference made in efficacy research applies to a specific population, for example, aphasics, and not to a specific individual. Second, a specific treatment protocol must be administered. Third, efficacy research requires optimally trained clinicians, optimally selected patients, optimally delivered treatment, optimally structured conditions, and optimal measures of change. Thus, an efficacy experiment indicates the *possible* benefits of treatment, not the actual benefits.

Effectiveness, according to the Office of Technology Assessment (1978), is "the probability of benefit

KEY POINT:

- In outcomes research, positive outcome refers to a favorable status, not necessarily as a result of treatment. Efficacy refers to improvement brought about by the ideal treatment under ideal circumstances. Effectiveness refers to similar outcomes under usual circumstances. Efficiency refers to a positive outcome brought about with a minimum of cost and waste.

to individuals in a defined population from a medical technology applied for a given medical problem under *average* conditions of use." Effectiveness research examines clinical outcomes obtained by ordinary clinicians from ordinary patients under the ordinary circumstances of actual clinical practice. Thus, effectiveness experiments should be conducted only after the efficacy of a treatment has been established. Because effectiveness examines the benefit of general clinical practice, it may be assessed for an individual patient or subpopulations.

Efficiency refers to effective production with a minimum of waste, expense, or unnecessary effort, in other words with a high ratio of output to input. Thus, two treatments that have been demonstrated to be efficacious and effective, might be compared to determine which is most efficient, for example, which of the two results in the most improvement with the least expense and effort.

Phases in Clinical Outcome Research

The nosology discussed above is further clarified when we consider the different phases in clinical outcome research. While the number of phases has varied over the years, the five phases shown in **Table 29**, according to Robey and Schultz (1998), are those most often employed in contemporary practice. Only a favorable outcome in an earlier phase warrants advancing to a later phase. The evaluation of efficacy precedes consideration of effectiveness.

As indicated in **Table 29**, the objectives in *Phase I* are to develop the research hypotheses, establish the safety of the treatment, and detect the activity of the treatment. Phase I research provides the genesis and evolution of the null hypothesis that will be tested in Phase III. All components of the experiment are addressed: what behaviors are to be changed? does the change have a positive influence on the lives of patients? who should be treated? what competencies must clinicians possess? how is the outcome to be indexed? what level of change constitutes a successful outcome? how is the treatment best delivered? Safety issues dictate addressing negative side effects. Then, if the treatment does no harm, does it help? Means for accomplishing Phase I objectives are case studies, single-subject experiments, and small-group experiments.

If the outcome of Phase I research warrants testing the efficacy of a treatment, the necessary scientific preparations are undertaken in *Phase II*. Treatment protocols are developed and standardized, outcome

TABLE 29 Five-phase model for outcomes research

Phase	Purpose
▶ I	Develop hypotheses, establish safety, detect activity of the treatment
▶ II	Develop treatment protocol, validate outcome measures, optimize dosage
▶ III	Test the treatment's efficacy under ideal conditions, employ external controls
▶ IV	Test the treatment's effectiveness under typical clinical conditions
▶ V	Continue effectiveness testing, initiate tests of the treatment's efficiency, determine the functional outcomes

TABLE 30 **A level of evidence scale for assessing results from treatment studies**

Level	Examples
A	Meta-analysis that includes two or more randomized, controlled trials
B	A randomized, controlled trial with good internal and external validity
C	A nonrandomized, clinical trial; historical controls; or pre- and post-treatment comparison
Strong consensus	Agreement among 90% of a practice guideline panel
Consensus	Agreement among 75 to 89% of a practice guideline panel
Clinical opinion	A study that does not meet Level C evidence above

Adapted from Birch and Davis Associates, Inc. The State-of-the-Science in Medical Rehabilitation, Volume 1. Falls Church, VA: Birch and Davis Associates, Inc., 1997.

measures are developed and validated, the factors that may affect the treatment's activity are assessed, and the dosage is optimized. Whereas Phase I research was designed to detect activity, Phase II research is designed to establish activity—how much of which treatment for whom? In addition, the critical research hypothesis is refined and finalized, and an explanation for why the treatment works is developed. Again, case studies, single-subject, and small-group experiments are appropriate for Phase II.

Phase III research tests the efficacy of the treatment under ideal conditions. It requires large samples and external controls. The outcome measures developed in Phase II, typically for measuring change in impairment and disability, are used; however, Phase III research may employ quality of life assessments. In addition, Phase III research may be a time of discovery, reaping unexpected as well as expected benefits. Phase III studies require a priori statistical power analysis to determine sample size and to control for a type 2 error. Typically, because sample size must be large, Phase III research is based on a multicenter clinical trial. For drugs, FDA approval may follow two or more positive Phase III trials.

If the efficacy of a treatment is established in Phase III, *Phase IV* research is conducted to determine the effectiveness of the treatment, i.e., the same positive outcome with application of the treatment to typical patients under usual conditions of clinical practice. Again, large samples are required; however, control samples are not used. Effectiveness studies might also contrast the therapeutic effects attributable to different forms of service delivery, the effectiveness attributable to different levels of clinician training, or to variations in the defined population. Essentially, Phase IV is the transition between the end of efficacy research and the beginning of effectiveness and efficiency research.

Phase IV research introduces the treatment to the practicing community. *Phase V* research emphasizes the continuation of effectiveness research and the beginning of efficiency research. Here, the functional outcome of the treatment is explored, for example, the discharge location of the patients treated, their quality of life, and their survival rates. In addition, cost-effectiveness

is explored to determine the financial value of the treatment, as well as the general value of the treatment—the quality control necessary, consumer satisfaction, quality of life, and the value to society.

The five-phase model comprises all forms of clinical outcome research. In summary, Phases I and II yield outcomes, Phase III examines efficacy, Phase IV explores effectiveness, and Phase V looks at effectiveness and efficiency of treatment.

Levels of Evidence

Application of the five-phase outcomes research model permits determination of a specific level of evidence obtained in each phase. A level of evidence indicates the confidence associated with providing a treatment. Several levels of evidence scales exist. Two are elaborated below.

Birch and Davis (1997) have provided the level of evidence scale shown in **Table 30**. It comprises three levels and three consensus-opinion positions. Each level relates to the type and quality of research evidence available. Level A indicates a treatment supported by a meta-analysis that includes two or more randomized, controlled trials and other studies that have good internal and external validity. Level B requires a randomized, controlled trial with good internal and external validity and good generalizability. Level C is supported by a nonrandomized clinical trial (case control or cohort study) in which the experimental and control groups are similar or multivariate analysis has controlled for group differences. Results from studies using historical controls, or a quasi-experimental design, such as pre- and post-treatment comparisons, also constitute Level C evidence. Additional levels of evidence in this model include three levels of consensus of opinion. Strong consensus would be agreement among 90% or more members or reviewers on a practice guideline panel. Consensus is agreement among 75 to 89% of members or reviewers on a practice guideline panel. And, clinical opinion is supported by a study that does not meet the level of evidence in Level C, i.e., historical controls or quasi-experimental design.

A second yardstick for levels of evidence has been provided by the American Academy of Neurology (1994). As shown in **Table 31**, it includes three classes of evidence. Class I is evidence from one or more well-designed, randomized controlled clinical trials. Class II is evidence from one or more randomized clinical studies such as case-control, cohort studies, etc. Class III is evidence from expert opinion, nonrandomized historical controls, or one or more case reports.

A levels of evidence scale can be used to evaluate treatment studies.

TABLE 31 **A level of evidence scale for assessing results from treatment studies**

Level	Examples
▶ Class I	One or more well designed, randomized, controlled clinical trials
▶ Class II	One or more randomized clinical studies, such as case control or cohort studies
▶ Class III	Expert opinion, nonrandomized historical controls, or one or more case reports

American Academy of Neurology. Assessment: Melodic Intonation Therapy. Neurology 1994;44:566–568.

For example, a meta-analysis would constitute the highest level of evidence in the Birch and Davis scale. One or more well-designed, randomized controlled clinical trials would constitute the highest level of evidence in the American Academy of Neurology (AAN) scale. Single-subject experiments or case reports would constitute lower levels of evidence in both scales.

Evidence from Group Treatment Studies of Aphasia

Reviews of aphasia treatment studies by Darley (1972), Holland et al (1996), and Wertz (1995) indicate that research on aphasia treatment has not followed the five-phase outcomes model. Considerable data are available, however, and a level of evidence can be applied to each report. Available data result from three designs: single treatment group, comparison of treatments, and treatment versus no-treatment comparisons.

A single treatment group design typically measures the baseline severity of aphasia in a group of patients, then remeasures severity post-treatment. It represents an outcome study: the difference in performance between pre- and post-treatment. Approximately 10 single treatment group aphasia treatment studies have been conducted. These studies have reported that 50 to 96% of treated patients improve. In general, treated aphasic patients have a good outcome. However, these investigations do not demonstrate that the improvement resulted from the treatment administered rather than from an uncontrolled variable, such as spontaneous recovery. Poeck et al (1989) inferred that treatment in a single treatment group study was efficacious by correcting for the amount of spontaneous recovery "demonstrated in a previous experiment"; the improvement in treated patients exceeded that expected from spontaneous recovery alone. While this attempt to control for spontaneous recovery is creative, it may not be convincing. The single treatment group design can indicate that aphasia improves, but it cannot demonstrate that improvement results from the treatment provided. Single treatment group designs represent Phase I and II studies in the outcomes research model, and they provide Level C evidence in the Birch and Davis scale and Class III evidence in the AAN scale.

A comparison of treatments design involves comparison of specific treatment techniques in matched groups, with administration of a pre- and post-treatment outcome measure. At least five comparison of treatments studies have been conducted with aphasic patients. In all studies, patients in all treatment groups made significant improvement. Only one study (Wertz et al, 1981) demonstrated a significant difference in improvement between groups. In that trial, individually treated patients made significantly more improvement than group-treated patients. Confusion abounds in the interpretation of comparison of treatment studies. Because a no-treatment group is not included, no inference about the efficacy of the treatments is permissible; instead only conclusions about the relative efficacy of one treatment versus another can be drawn. Wertz et al (1981) attempted to infer efficacy in their comparison of treatments design, based on the usual lack of spontaneous recovery after 6 months post-onset. This inference requires the validity of this limit on spontaneous recovery. By the models previously discussed, a comparison of treatments design represents a Phase I or II outcome study, or Phase V (efficiency), if the treatments being compared have had previous documentation of efficacy. The results provide Level C evidence in the Birch and Davis scale or Class II or III evidence, depending on whether patients were randomly assigned to treatment groups, in the AAN scale.

KEY POINTS:

- At least 10 single treatment group aphasia studies have been conducted, with reported improvement in 50 to 96% of patients. These studies do not distinguish spontaneous improvement from treatment effects.
- Of five comparison of treatment studies, only one (Wertz et al, 1981) indicated a specific treatment effect, namely better improvement with individual versus group therapy. These studies do not include a no-treatment group and hence do not establish the efficacy of treatment.

A treatment versus no-treatment design requires that two groups are evaluated with the same outcome measure pre- and post-treatment, but only one group receives the treatment. If the treated group makes significantly more improvement than the no-treatment group, one can infer that the treatment was efficacious, provided that the patients were randomly assigned to the two groups. At least seven comparison of treatment with no-treatment investigations have been conducted with aphasic patients, but only three assigned patients randomly. The four investigations that did not assign patients randomly used self-selected no-treatment groups: patients who elected not to receive treatment, who could not afford treatment, who resided where treatment was not available, or who were placed on a waiting list. These studies are difficult to interpret, because variables that may influence response to treatment are not controlled. Nevertheless, three of the four investigations report positive results: treated patients made significantly more improvement than patients who did not receive treatment. These investigations represent Phase I or II studies in the outcome research model and constitute Level C evidence in the Birch and Davis scale and Class III evidence in the AAN scale.

Two of the three investigations that employed random assignment of patients to treatment and no-treatment groups (Wertz et al, 1986; Katz and Wertz, 1997) reported positive results of treatment. Wertz et al (1986) assigned aphasic patients randomly to treatment by speech pathologists for 8 to 10 hours each week for 12 weeks or no-treatment for 12 weeks. Study patients were aphasic subsequent to a first, single, left hemisphere infarct and ranged between 2 and 24 weeks post-onset. Initial severity and time post-onset were covaried in the outcome analyses. At the end of the treatment trial, treated patients made significantly more improvement than no-treatment patients, implying that treatment for aphasia was efficacious. Katz and Wertz (1997) assigned study patients randomly to three groups: computer language treatment, computer stimulation with no manipulation of language deficits, and no treatment. Study patients were aphasic subsequent to a first, single, left hemisphere infarct and were at least 6 months post-onset. Patients in both treatment groups received 3 hours of treatment each week for 6 months. No-treatment patients were followed untreated for 6 months. At the end of the treatment trial, the computer language treatment group had made significantly more improvement than the computer stimulation and no-treatment groups, and the stimulation and no-treatment groups did not differ significantly in the amount of improvement attained.

Lincoln et al (1984) assigned aphasic patients randomly to treatment and no-treatment groups at 4 weeks post-onset. They prescribed 2 hours of treatment each week for 24 weeks. Study patients had suffered a stroke or strokes, hemorrhagic and/or ischemic; localization, unilateral or bilateral, was not reported. At the end of the 24-week treatment trial, there was no significant difference in improvement between treated and untreated patients. However, less than 30% of the treated patients received the 48 hours of treatment prescribed. The Wertz et al (1986) and Katz and Wertz (1997) investigations represent Phase III, efficacy studies in the outcomes research model, and the evidence provided is Level B in the Birch and Davis scale and Class I in the American Academy of Neurology scale. The investigation of Lincoln et al (1984) is difficult to classify. The design, a randomized, controlled trial, represents a Phase III efficacy study in the outcomes research model; however, the less than opti-

mal conditions (single or multiple strokes, noncompliance with the treatment) is more characteristic of a Phase IV, effectiveness study.

Were aphasia therapy a drug, the treatment would likely meet FDA approval, with two large Phase III studies documenting efficacy. Neurologists should keep this in mind in comparing speech therapy for aphasia with other widely prescribed but less validated treatments.

Meta-Analyses

"A meta-analysis is a mathematical means for synthesizing independent research findings scattered throughout a body of literature" (Robey, 1998). It quantifies the effects (outcomes) of a collection of primary studies and provides the average effect size and its confidence interval. The results estimate the degree to which a null hypothesis, for example, aphasia treatment is not efficacious, is false on the basis of all available evidence. Benchmark criteria are computed for small-sized effects (.20), medium-sized effects (.50), and large-sized effects (.80). While meta-analyses permit the compilation of much larger quantities of data than individual studies, they suffer from the problem of lumping together studies with different patient selections, different specific treatment techniques, and different outcome measures. Three meta-analyses of aphasia treatment studies have been reported. A meta-analysis constitutes the highest level of evidence (Level A) in the Birch and Davis scale. Meta-analysis in not included in the American Academy of Neurology scale.

Whurr et al's (1992) meta-analysis included 45 aphasia treatment studies. The results indicated a medium-sized effect (.59), implying treated aphasic patients "moved from the 50th to the 73rd percentile, a shift of 23 percentage points." Employing the distribution of the normal curve, the authors concluded that "the 23 percentile shift suggested that 73% of the patients who received therapy can expect to improve slightly more than half of a standard deviation as a result of treatment when compared to those aphasic patients not receiving therapy."

Robey has reported two meta-analyses of aphasia treatment studies. The first (Robey, 1994) included 21 studies and indicated that improvement in treated patients was, on average, nearly twice the recovery of untreated patients when treatment began in the acute stage. This exceeded a medium-sized effect (.50). In the chronic stage, treatment exceeded the criterion for a small-sized effect (.20). Robey concluded that treatment for aphasia is effective. In a more recent meta-analysis (Robey, 1998), 55 treatment studies were included. The results confirmed those of the earlier analysis and demonstrated "the utility of aphasia treatments, generally considered, for bringing about desirable clinical outcomes." Specifically, Robey concluded that outcomes for treated patients are superior to those for untreated individuals in all stages of recovery. Outcomes are greatest when treatment is begun in the acute stage of recovery, 2 hours of treatment each week results in greater gains than less intensive treatment, and large gains are achieved by severely aphasic persons when treated by a speech-language pathologist.

Treatment for Aphasia Is Efficacious for Whom?

A contemporary survey of the aphasia treatment literature indicates continued controversy about the efficacy of speech therapy. Advocates for the efficacy of aphasia treatment sometimes substitute emotion for evidence, while skeptics sometimes misinterpret the available empirical evidence. What therefore can be concluded about the efficacy of treatment for aphasia?

KEY POINTS:

- Of seven treatment versus no-treatment studies, only three had randomized selection of groups. Two of the three clearly showed treatment efficacy. The third found no benefit but contained design flaws of inclusion of single and multiple strokes and also lack of compliance with the prescribed treatment regimen. Taken together, these studies provide convincing evidence of efficacy of aphasia therapy.
- Three published meta-analyses of aphasia treatment studies have all confirmed the efficacy of aphasia therapy.

KEY POINT:

- Aphasia therapy appears to benefit most patients. Patients most likely to improve include those with single strokes, treated soon after onset, in good general health, and with at least 3 hours of therapy weekly for a period of months.

First, the results of meta-analyses and well-controlled treatment trials indicate that treatment for aphasia, in general, results in more improvement than no treatment. Certainly, treatment may not benefit all aphasic people, but the collected evidence indicates that it benefits most. Second, treatment appears to help some aphasic people more than others. Selection criteria for the patients most likely to profit from treatment include: 1) aphasia subsequent to a first, single, left hemisphere infarct; 2) six months or less post-onset; and 3) relatively good health with no significant coexisting medical problems. Third, the intensity and duration of treatment appears to influence outcome. While the optimum intensity and duration have not been determined, the literature implies a minimum of 3 hours a week, often for a full 6 months. Patients who receive 48 hours of treatment or less do not appear to benefit much from aphasia therapy. Fourth, there is no evidence to support one type of treatment over another. While aphasia treatment, in general, appears efficacious, how much of which specific treatment for which subgroups of patients remains to be demonstrated.

TREATMENT

The literature on treatment of aphasia, and the variety of treatment techniques in use may give the impression that there are as many types of therapy as there are therapists treating aphasic patients [see, for example, Chapey (1994), Helm-Estabrooks and Albert (1991), and Rosenbek et al (1989)]. Rosenzweig et al (1996) observed, "The exact forms of speech therapy are mainly improvisations supported by some degree of clinical success rather than being generated by theories." A review of the treatment literature by Horner et al (1994) does not, however, support this assumption. Clinicians who treat aphasic patients typically have a theoretical basis for their efforts. In fact, several specific theories for therapy exist. Each is based on a definition of aphasia, relating to a theory of aphasia therapy and to the selection of specific methods employed. Three such theories for therapy are stimulation-facilitation, cognitive neuropsychological and/or psycholinguistic treatment, and functional communication. While other theories for therapy exist (e.g., modality model, processing model, minor hemisphere mediation model), there is considerable overlap among theories. Those elaborated below can be considered representative. It is necessary initially to consider some general treatment principles that are incorporated in most approaches to aphasia treatment.

General Principles

Byng (1995) has provided an answer to the question, "What do clinicians do when they say they do aphasia therapy?" Eight principles are incorporated into most theories and subsequent therapy techniques. The clinician (speech/language pathologist) seeks to:

- Delineate the patient's premorbid uses of language;
- Facilitate the patient's accommodation to the changes in his or her communication skills;
- Investigate the effects of the aphasic deficits on the whole language system;
- Attempt to remediate the aphasic deficits;
- Increase the use of all potential means of communication to support, facilitate, and compensate for the impaired language;
- Enhance the patient's use of his or her remaining language;
- Provide opportunities for the patient to use his or her newly acquired language or communication skills in familiar communication situations;
- Modify the communication

habits of the patient's family or friends to accommodate the aphasia.

These principles make clear that therapy is not confined to only one of the World Health Organization's dimensions—impairment, disability, handicap. While some theoretical approaches to therapy emphasize one dimension, for example, impairment, each theoretical approach seeks generalization of treatment effects to reduce severity in the other dimensions, for example, disability and handicap. Moreover, therapy is not confined to a clinical cubicle. Some theoretical approaches emphasize treating aphasic people in the contexts where they communicate, i.e., home, community, work environment. All theoretical approaches seek generalization of improved communication to functional contexts. Finally, treatment is not directed only at the aphasic person. The behavior of those who communicate with the aphasic person is modified to increase the probability of the aphasic person's communicative success.

Stimulation-Facilitation Therapy

Probably the oldest and most enduring theoretical basis for aphasia therapy is stimulation-facilitation. It was popularized by Schuell and colleagues (1964) and is employed by the majority of aphasia clinicians as a means to reduce language impairment. As summarized by Duffy (1994), the stimulation-facilitation approach considers aphasia to be a multimodality disruption, involving auditory comprehension, reading, oral-expressive language, and writing. Deficits in auditory processing, however, are the primary culprit in aphasic communication. Mending these is the primary focus of treatment. Thus, treatment tasks employ intensive auditory stimulation in a stimulus-response format. Treatment hierarchies, arranged in tasks that are easiest to most difficult for the patient, are employed to ensure that the stimulus is adequate for the aphasic person's level of severity. For example, a hierarchy for treating auditory comprehension deficits might begin with single words in a "point to the _____" format. Repetition of the auditory stimulus follows failure, and the use of cues is employed, for example, "it is used for _____, point to the _____." As auditory comprehension improves, the auditory stimulus is increased in length, for example, "point to the _____ and the _____," and complexity, for example, "before pointing to the _____, show me the _____," in a hierarchical fashion.

The theoretical premise is that an aphasic person has not lost language and, further, that the patient's use of language can be "stimulated" or facilitated" by application of the appropriate therapeutic methods. In addition, even though strengthening or mending auditory processes is the underlying principle, treatment may attack deficits in any language modality: auditory comprehension, reading, oral-expressive language, and writing. Specific examples of treatment tasks for different modalities are found in Rosenbek et al (1989).

Helm-Estabrooks and Albert (1991) discuss a variety of stimulation-facilitation methods for treating different types of aphasia. These include visual action therapy (VAT) for treating global aphasia, voluntary control of involuntary utterances (VICU) for improving oral-expressive language, the Helm elicited program for syntax stimulation (HELPSS) for improving syntax, and treatment of Wernicke's aphasia (TWA) for improving auditory comprehension. An example of the stimulation-facilitation approach, employed in a hierarchical method, is melodic intonation therapy (MIT) (see Appendix A). Repetition of verbal stimuli, using exaggerated rhythm, pitch, and hand gestures, is employed to improve oral-expressive language

KEY POINT:

- Speech therapy techniques incorporate eight general principles. The therapist must: 1) delineate the subject's prior use of language; 2) facilitate accommodation to changes in communication; 3) investigate the specific aphasic deficits; 4) attempt to remediate the language deficits; 5) increase all possible communication modes; 6) enhance use of remaining language skills; 7) provide opportunities to use new language skills; and 8) modify the communication habits of family members and friends. These efforts seek to aid the aphasic patient's deficit, disability, and handicap all together.

KEY POINT:

- The stimulation-facilitation method of treatment involves repetitive drilling in areas of language deficit. Most currently employed speech therapy techniques follow this model.

in Broca's aphasic patients. The program is divided into three levels. In the first two, multisyllabic words and short, high-probability phrases are musically intoned. In the third, longer sentences are intoned, then produced with exaggerated speech prosody, and finally spoken normally.

Again, while the emphasis in stimulation-facilitation treatment is designed to reduce language impairment, the assumption is made that improved performance will carry over and result in reduced disability —improved functional communication—and reduced handicap—improved quality of life. A stimulation-facilitation approach has the advantage of being appropriate for a variety of aphasic impairments and severity. It was the primary treatment method employed in both Veterans Administration cooperative studies on aphasia (Wertz et al, 1981, 1986).

Cognitive Neuropsychological and/or Psycholinguistic Treatment

The cognitive neuropsychological and/or psycholinguistic approach to treatment views aphasia as a modular or relational processing disruption in a model of normal language. More specifically, a psycholinguistic approach designs treatment to restore disrupted language by organizing stimuli to repair the linguistic deficits or impaired module in the psycholinguistic model employed to identify the deficits. A cognitive neuropsychological approach designs treatment to restore or compensate for language deficits or language-related processing deficits in the model of normal cognition. Both methods utilize single-case treatment designs to explore facilitation, reorganization, and/or relearning. Facilitation is a treatment designed to improve access to intact information when the problem is impaired access, while reorganization treatment attempts to reroute processing through intact components in the model to bypass the processing deficit. Relearning requires learning lost information or lost rules.

An example of the cognitive neuropsychological and/or psycholinguistic approach to treatment is "mapping therapy" developed by Saffran and colleagues (1993) for treating agrammatic aphasic patients. The focus is on improving sentence comprehension and sentence productions deficits. The therapeutic hypothesis is that an agrammatic patient has difficulty using syntactic structure to assign thematic roles in sentences—who is doing what to whom. Passive sentences (e.g., "The car was fixed by Tom") present little difficulty, because they are nonreversible (Tom cannot be fixed by the car), and agrammatic patients can use the semantic content to determine who did what to whom. However, in reversible passive sentences (e.g., "The boy was helped by Martha"), there is no semantic guideline to assist comprehension (Martha could be helped by the boy). Saffran et al hypothesized that agrammatic patients have difficulty "mapping" the relationship between the verb and its noun arguments. Their treatment is designed to assist these patients in assigning roles by the nature of the stimuli employed. Thus, treatment is a stimulus-response format that includes color-coding specific items to assist patients in understanding and producing correct thematic relationships—who is doing what to whom.

Cognitive neuropsychological and/or psycholinguistic treatments can be employed to treat deficits in all modalities (auditory comprehension, reading, oral-expressive language, writing), depending on where the patient's performance breaks down in the normal model of language or cognition employed. The treatments attack specific deficits and, therefore, are impairment based. Again, assumptions are made that mending impairments will generalize to reducing disability and handicap.

Functional Communication Treatment

A functional communication approach to aphasia treatment posits that communication reflects the application of pragmatic rules, unconstrained by modality, linguistic, or neurolinguistic considerations. Aphasia is believed to result in ineffective or inefficient language use in natural communication contexts. The rationale for treatment is to facilitate more normal communication by emphasizing pragmatic function over linguistic form and to enhance intermodality flexibility, including establishing strategies for circumventing and/or repairing communication breakdowns. Thus, the focus is on functional communication, rather than on production of specific linguistic content. A variety of functional treatments exist. These include Kagan and Gailey's (1993) training conversational partners for aphasic adults, which emphasizes how nonaphasic people can assist the aphasic person use his or her residual ability to produce maximal communication, Holland's (1991) conversational coaching techniques, which focus on assisting the aphasic person and nonaphasic communication partners to repair communication breakdowns, and Lyon and colleagues' (1997) aphasia partners program, which pairs an aphasic person with a nonaphasic volunteer to reintegrate the aphasic person into his or her community.

A widely used functional communication method is that of Davis and Wilcox (1985), promoting an aphasic's communicative effectiveness (PACE). This approach emphasizes use of the aphasic person's retained communicative strengths rather than attempting to overcome linguistic deficits. While verbal communication is encouraged, PACE promotes the use of other modalities, i.e., gesture, writing, drawing, to compensate for what cannot be said and to circumvent the influence of disrupted language on everyday communication. A typical PACE activity involves clinician and patient communicating information in pictured situations. A stack of drawings is placed, face down, between clinician and patient. Each takes a turn by selecting a drawing and communicating its content to the other. Principles that guide the task are: 1) new information is exchanged, because neither the patient nor the clinician knows what is contained in the pictures; 2) both participants act as both the conveyer and the receiver of information; 3) all modes of communication, viz., speech, gesture, writing, drawing, are considered equally appropriate, because conveying information rather than speaking is the goal; and 4) there is immediate knowledge of results consequent to the success or failure to convey or receive information. Thus, PACE simulates what people do when they communicate, i.e., exchange information (a transaction) or simply "hang out" (an interaction). PACE activities can be expanded to include family members and other aphasic persons. For example, a game might involve conveying a specific war; one member of the group must communicate a specific war (Revolutionary, Civil, World War I, etc.) and then request a song associated with that war.

Because functional communication treatment focuses on context rather than on linguistic content, it is designed to reduce disability and handicap. The goal is to make communication "go" even when the words do not "go" quite right.

Summary

Examples of different theoretical bases for aphasia treatment have been provided along with specific therapeutic analogies. Two caveats are necessary. First, only a few of the theories for therapy have been examined, and only a few examples of treatment tasks for each theory. Second, a clinician typically includes

KEY POINTS:

- Neuropsychological and psycholinguistic treatment methods employ a modular diagram of linguistic or cognitive functions necessary for a communication task; they seek to assist the patient by facilitation of intact functions, reorganization of functions to use preserved modules, and relearning of modules which are deficient.
- Functional communication therapy emphasizes the communication of ideas, without regard for the specific linguistic content or the mode of communication.

Case 8

D.M., a 37-year-old male, awoke "unable to talk and paralyzed on his right side." His local hospital provided a diagnosis of "stroke" with subsequent "right hemiplegia and expressive aphasia." After 1 week, he was discharged home to the care of his mother. After 2 weeks at home, he entered a rehabilitation program in a Veterans Administration Medical Center 150 miles from his home. A neurologic examination at 3 weeks post-onset demonstrated features of a large, left hemisphere infarct in the middle cerebral artery distribution. A CT scan indicated a cortical and subcortical lesion involving the left frontal, temporal, and parietal lobes and extending into the insula. The neurologist concluded that D.M. had suffered "an occlusion of the left middle cerebral artery with right hemiparesis and predominately expressive aphasia."

A speech and language evaluation at 3 weeks post-onset indicated Broca's aphasia with coexisting apraxia of speech and apraxia of phonation. Performance on the Porch Index of Communicative Ability (PICA) was at the 33rd overall percentile. The Boston Diagnostic Aphasia Examination (BDAE) confirmed the presence of Broca's aphasia. Performance on the Communicative Abilities of Daily Living (CADL) indicated a total score of 62. Speech was characterized by whispered, groping articulatory movements; attempts at self-correction; and inconsistency in repeated productions of the same utterance. An ear, nose, and throat evaluation revealed no vocal fold paralysis. D.M. produced vocal fold closure on nonvolitional coughing and clearing his throat, and he produced normal phonation when he laughed. Thus, articulation was considered apraxic, and his inability to phonate volitionally suggested apraxia of phonation.

D.M. received 1 hour of speech therapy, 5 days a week, for 2 months. Initial treatment focused on producing voice and providing functional communication. The early treatment sessions included 30 minutes on reducing the apraxia of phonation and 30 minutes on developing functional communication through gesture, writing, and drawing. If phonation could be obtained, speech treatment would shift to mending D.M.'s apraxic articulatory behavior.

Initial attempts to obtain phonation involved providing a strong push on D.M.'s abdomen while he exhaled slowly. Typically, this results in rapid vocal fold closure with a subsequent, audible grunt. With D.M., the technique resulted in an increased rush of air, no sound production, and a sore stomach. Treatment shifted to placing the clinician's hand on D.M.'s thyroid cartilage and providing firm, manual manipulation while encouraging him to produce an /a/. After three sessions, D.M. was successful in producing an /a/ in 30% of his attempts. After an additional seven sessions, he produced volitional phonation on 90 to 100% of his attempts.

Once phonation was achieved, speech treatment shifted to mending the apraxia of speech. Imitative drill ("I'll say it, and you say it after me.") was initiated on three sets of consonant-vowel-consonant combinations: a set beginning with /m/, e.g., mom moan, mean, etc.; a set beginning with /p/, e.g., pen, pan, pone, etc.; and a set beginning with /t/, e.g., tan, Tom, tone, etc. Accuracy on all sets was approximately 10% pretreatment. Initial treatment focused on /m/ stimuli, and /p/ and /t/ stimuli were followed untreated. After four sessions, /m/ stimuli were 80 to 100% correct, and /p/ and /t/ stimuli remained at pretreatment levels. Subsequent treatment of /p/ stimuli indicated 80 to 100% correct performance after three sessions, and subsequent treatment of /t/ stimuli indicated 80 to 100% correct performance after an additional four sessions. This improvement on sounds, only when treated, might permit inference about the efficacy of the effort.

Simultaneous with the motor speech treatment, functional communication was attacked by encouraging D.M. to use gesture, drawing, and writing to communicate. This included developing a communication board containing printed and pictured stimuli to convey biographical information (name, address, age, occupation, etc.); an orientation list (current date, location, daily schedule, etc.); and basic needs (bathroom, food, drink, blanket, etc.). Use of the communication board was drilled by questions during treatment, for example, "Show me you have to go to the bathroom." In addition, the communication board was explained to other rehabilitation personnel, and all encouraged D.M. to use it. After phonation and some intelligible speech emerged, spoken attempts were added to his functional communication, and a PACE format was included during treatment sessions. This involved encouraging D.M. to use gesture, drawing, and writing to communicate information he was unable to say.

After 2 months of treatment, at 3 months post-onset, D.M.'s PICA overall performance was at the 53rd percentile, a gain of 20 percentile points from pretreatment. His CADL total score was 113, a gain of 51 points from pretreatment. D.M. was encouraged to continue treatment, but he elected to return home. Follow-up evaluation 1 month after discharge, at 4 months post-onset, indicated his PICA overall percentile had dropped by two percentile points, and his CADL total score was unchanged. D.M. indicated that his communicative ability was adequate for his needs, and he had resumed part-time employment. There is minimal evidence to demonstrate that the treatment was efficacious. Throughout the 2 months of treatment, there was a cotherapist, namely, spontaneous recovery. Nevertheless, D.M.'s significant improvement documents a favorable outcome.

a variety of theoretical approaches in the management of an aphasic person to accomplish all of the eight general principles discussed at the beginning of this section. As indicated, some treatments are primarily directed at reducing language impairment, and others are directed at reducing disability and handicap. Those that mend disability and improve functional communication are being employed more often in contemporary clinical practice, because emphasis is being placed by third-party payers on improving functional communication.

PHARMACOTHERAPY OF APHASIA

A recent and potentially exciting area of aphasia research involves the use of drugs to stimulate recovery of language functions. Albert and colleagues (1988) initiated investigations in this domain with a report that the anti-Parkinson drug bromocriptine seemed to improve speech output in patients with transcortical motor aphasia. The authors pointed to the known effects of dopamine agonist drugs in stimulating initiation of movement in disorders such as Parkinson's disease; transcortical motor aphasia shares with Parkinson's disease a difficulty in initiation of motor behavior, in this case speech. The drug has also been utilized in other nonfluent aphasias, though the only randomized trial reported did not document definite benefit.

Preliminary use of stimulant drugs such as methylphenidate has also shown promise in promoting speech output. This area of research is still in its infancy, but there is considerable hope that pharmacotherapy may be an adjunct to behavioral therapies for aphasia. In addition, the effects of stimulant and dopamine agonist drugs raise the concern that sedative and tranquilizing drugs may actually impede language rehabilitation. One study (Goldstein, 1995) found that patients treated with the anticonvulsant drugs phenytoin and carbamazepine, benzodiazepines used for sleep or anxiety, and some antihypertensive drugs seemed to retard stroke rehabilitation. While a prospective study has not been reported, physicians caring for aphasic patients should avoid these drugs if possible.

KEY POINTS:

- The dopamine agonist drug bromocriptine may have a role in increasing speech fluency in transcortical motor aphasia.
- Other drugs such as stimulants may help stroke patients regain language functions faster. Drugs which slow learning should be avoided during rehabilitation.

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NOTES