

## 12. Anomia, Dysfluency, and Chronic Alcoholism: Prognostic Considerations

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Sometimes in daily clinical experiences, clinicians are faced with patients for whom it is difficult to make a prognosis. Most published investigations of aphasia treatment present results obtained from subjects who sustained single left cerebrovascular accidents (CVAs) and had few or no complicating conditions. However, many patients are more complex than this, leaving their prognosis unsure. When such patients present, it seems worthwhile to study them carefully and, if possible, to share the outcome of their treatment with other professionals. To this end, this single-subject treatment study is submitted.

The patient, B., presented with symptoms of moderate anomic aphasia and dysfluency described as stuttering, coincident with a left frontal subdural hematoma 11 years previously. He also had a 15-year history of heavy alcohol abuse with frequent hospitalizations for concomitant seizures. B. had been "dry" for a year, but had recently suffered a relapse and entered an alcohol rehabilitation program. The presence of adult-onset dysfluency and the history of chronic, heavy alcohol abuse raised questions about B.'s prognosis.

The prognostic implications of adult-onset dysfluency were examined first. Two kinds of adult-onset dysfluency cases have been reported in the literature. One kind, which has been called *neurogenic dysfluency*, appears to be a motor execution problem that exists as a distinct, conspicuous behavior apart from other neurogenic communication disorders, although it may occur in conjunction with them (Arend, Handzel, & Weiss, 1962; Canter, 1971; Helm, Butler, & Benson, 1978; Helm-Estabrooks, 1986; Mazzucchi, Moretti, Carpeggiani, Parma, & Painsi, 1981; Quinn & Andrews, 1977; Rosenbek, Messert, Collins, & Wertz, 1978; Rosenfield, 1972; Rosenfield, Miller, & Feltovitch, 1980). Some of these patients improved with treatment programs designed to manipulate rate of speech, whereas other

patients showed no improvement with treatment (Helm, Butler, & Canter, 1980; Helm-Estabrooks, 1986; Rosenbek et al., 1978).

The second reported kind of adult-onset dysfluency has its onset at or near the same time as other neurological symptoms, but appears to be a psychogenic reaction to them (Brookshire, 1989; Baumgartner & Duffy, 1986, as cited in Duffy, 1989; Roth, Aronson, & Davis, 1989). Patients with this kind of dysfluency often improve with treatment (Brookshire, 1989; Duffy, 1989), so they may have a better prognosis than patients with "neurogenic" dysfluency. Therefore, if B.'s dysfluency was "psychogenic" in origin it might be more likely to respond to treatment than if it was "neurogenic" in origin. However, the differential diagnosis of the dysfluency as "neurogenic" or "psychogenic" often rests on the patient's response to treatment. Before treatment was initiated, therefore, the prognosis for improvement of the patient's fluency was considered guarded.

The potential impact of B.'s heavy alcohol abuse on his prognosis was also unclear. Although he was not drinking at the time of the evaluation, B. had drunk heavily for 15 years and had been hospitalized frequently for associated seizures. There are few data available regarding whether or how a history of heavy alcohol abuse will affect a patient's response to language treatment. Rada, Porch, Dillingham, Kellner, and Porch (1977) evaluated alcoholics 1 week after detoxification and found them to be impaired on measures of word fluency, on the *Token Test*, and on graphic subtests from the *Porch Index of Communicative Ability* compared to a control group. This suggests that chronic alcoholics who become aphasic might have a poorer prognosis than aphasic patients who are not alcoholic, since alcoholism alone apparently caused some language impairment that lasted beyond the period of detoxification. Hageman, Lux, and Rucci-Zimmer (1986) stated that although chronic alcoholism is known to cause deficits in memory, reasoning, problem solving, and perceptual-motor skills, the interaction between prestroke alcohol abuse and post-stroke aphasia rehabilitation is unknown and requires investigation. Brookshire (1989) reported improvement of "psychogenic" dysfluency in a patient who had been a chronic alcoholic but who had stopped excessive drinking 5 years prior to treatment. Few other treatment studies mention alcoholism among their subjects. Since alcoholism is a condition that clinicians are likely to encounter fairly frequently, it seems important to begin to document its effect, if any, on a patient's response to treatment.

In summary, the patient was 11 years poststroke and had never received language rehabilitation. He had adult-onset dysfluency coincident with the stroke, and he had a history of alcohol abuse. All of these factors could have potentially negative prognostic implications. Nevertheless, heeding Darley's (1978) advice that all aphasic patients deserve at least a trial period of therapy, the patient was enrolled for treatment.

## SUBJECT

B. was a 50-year-old right-handed male with a high school education. He was 11 years post-onset of a left frontal subdural hematoma that resulted in aphasia and what he and his physician called "stuttering." B. denied any speech or language problems, including stuttering, prior to the CVA and denied any family history of such problems. B.'s sister corroborated these claims. B. never received speech-language pathology services at the time of his stroke or in the following 11 years. He was referred for evaluation by a new physician. Medical history was significant for hypertension and for the alcohol abuse.

The profile of scores on the *Boston Diagnostic Aphasia Examination* (Goodglass & Kaplan, 1983) was compatible with a diagnosis of moderate anomic aphasia. Expressive speech was characterized by normal melodic line, phrase length, and articulatory agility, but included anomic blocks; sound, part-word, word, and phrase repetitions; revisions; and occasional semantic and phonemic paraphasias. The dysfluencies were sometimes accompanied by tremor of the lower lip, but no other secondary stuttering characteristics were evident. There was no dysarthria, oral apraxia, apraxia of speech, or limb apraxia. Reading and writing were moderately impaired. The ability to tap a rhythm and to draw were intact.

No consistency or adaptation effects occurred on repeated readings of the Grandfather Passage, consistent with other reports of neurogenic dysfluency. Treatment techniques that typically result in changes in developmental stuttering, including choral reading, relaxation training, identification and modification of dysfluencies, and delayed auditory feedback were ineffective, as was pacing, which is sometimes effective in cases of neurogenic stuttering.

Dysfluencies occurred as often on functor as on content words. They occurred less frequently during repetition, automatic speech, and oral reading tasks than during confrontation naming, responsive naming, and conversational speech tasks. During these latter tasks, the dysfluencies appeared to be associated with failures of word retrieval and sometimes seemed to be produced as "filler" to maintain B.'s conversational turn while he tried to retrieve the word (for example, "Actually I liked /p/ I /lai/ I /lai/ I /lai/ I /lai/ I /lai/ I /lai/ I liked the post office better," or "And then when I when I when I when I /s:/ . . . when I went in the service . . ."). Canter (1971) described a similar behavior that he called *dysnomic stuttering*, in which speech is sometimes marked by repetition of the previous word or phrase as the person fails to retrieve the desired word. Thus it was hypothesized that B.'s dysfluency was related to his dysnomia, and that a treatment program designed to improve word retrieval ability should result in a decrease of dysfluency. However, efforts to attack the word retrieval problem directly by teaching the patient to circumlocute

were hampered by B.'s inability to inhibit the word and part-word repetitions and revisions that were produced when he could not retrieve a word.

## METHOD

Since it appeared that B.'s dysfluencies might be serving to "fill" the time he needed to retrieve a word, it seemed that a strategy of forcing him to remain silent for a period of time before he was allowed to respond might give him the time he needed to retrieve and produce the word accurately, and would reduce or eliminate the dysfluencies. This "delay" approach was reported by Marshall (1976) to be the most effective retrieval behavior spontaneously used by aphasic adults. He reported that his subjects used filled pauses, unfilled pauses, or some stalling tactic to gain adequate time to produce the word. The goal for B. was to replace the filled pauses and stalling tactics (dysfluencies) with unfilled or silent pauses.

A treatment program was designed that used systematically decreasing periods of enforced delay before allowing the subject to respond. To assess the efficacy of this program, a within-subject multiple-baseline-across-behaviors design was used. The behaviors targeted were confrontation naming, description of action pictures, and conversational speech. Baselines on all behaviors were taken before treatment was initiated on the first target behavior; they continued to be taken on untreated behaviors while the first behavior was being treated. When the criterion on a target behavior was reached (10% or fewer dysfluencies on two consecutive probes), treatment of that behavior was discontinued, but maintenance probes continued to be taken after treatment of subsequent behaviors was initiated.

Treatment sessions were structured so that initially a 5-second delay was imposed before allowing a response, then a 3-second delay, then no enforced delay. These delays indicated only the first moment when B. was permitted to respond, not a moment when he was forced to respond. He could, and did, remain silent for longer intervals if he had not retrieved the word. When no delay was enforced, he was instructed to use a delay whenever necessary before responding. B. had to achieve fluent productions on 90% of the words for two consecutive trials at one delay level before progressing to the next.

The subject's responses were audiorecorded and transcribed verbatim. Half of the tapes for each target behavior were randomly chosen for transcription and scoring by a second listener. Point-to-point interjudge reliability in identifying dysfluencies was 96%.

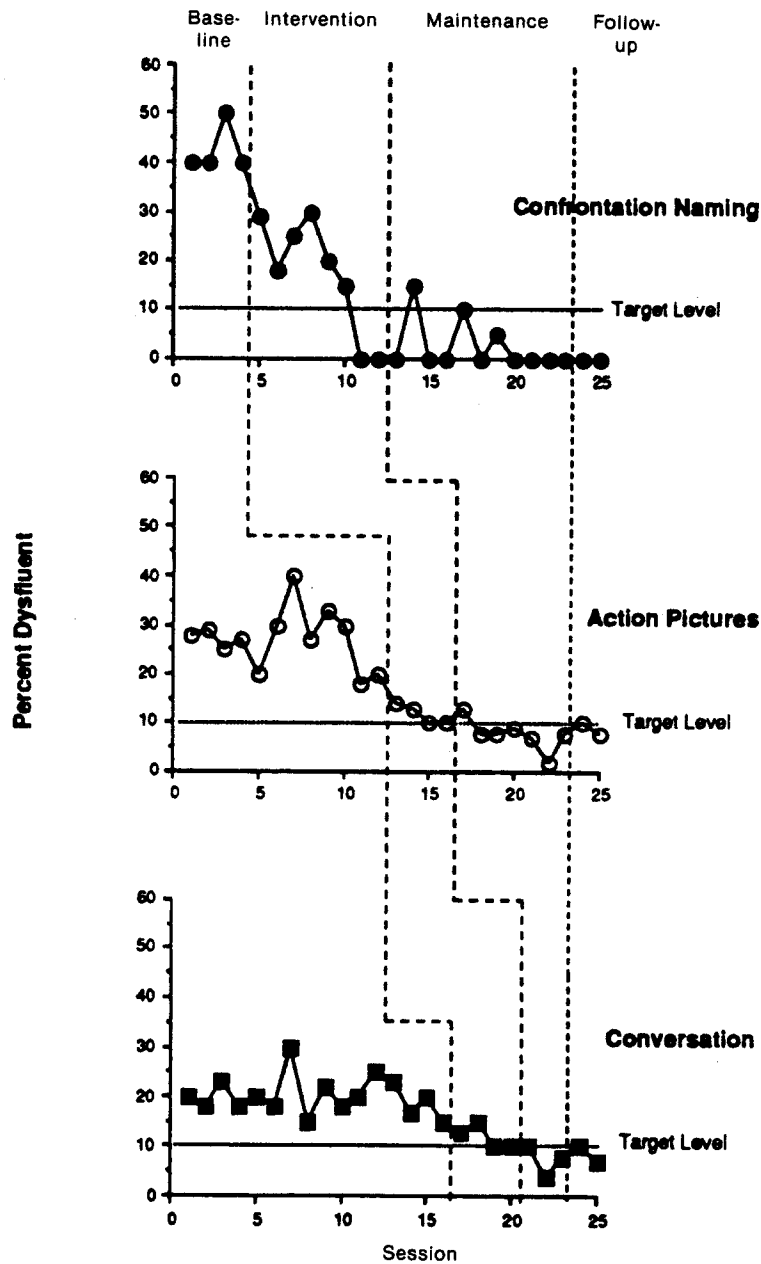
## RESULTS

B.'s response to the treatment program can be seen in Figure 12.1. Dysfluencies decreased following intervention on confrontation naming and the criterion was achieved at session 12. Except for the probe taken at session 14, dysfluencies remained at or below the target level for the remaining maintenance probes. A similar decrease in dysfluencies during picture description can be seen following intervention on this behavior, and, except for the probe taken at session 17, the dysfluencies remained below the target level during maintenance probes. Dysfluencies during conversational speech also decreased below baseline measures after intervention and remained at or below the target level during maintenance probes.

## DISCUSSION

Was B.'s dysfluency a motor speech problem or a reaction to his anomia? Brookshire (1989) suggested that although a positive response to behavioral treatment is not definitive regarding the organic or nonorganic nature of a behavior, such a response "substantially increases the likelihood that the behavior is, to an important extent, psychogenic" (p. 11). As more is learned about the complex interaction between neurochemistry, emotion, and behavior, it is becoming more and more difficult to say that a behavior is psychogenic, with the implication that it has no organic basis. The use of *neurogenic* and *psychogenic* as dichotomous terms needs examination. In any case, judging by the improvement that was achieved following a treatment program designed to replace filled pauses with silent pauses, it seems that B.'s dysfluency was, in large part, a reaction to the anomia, or what Canter called *dysnomic stuttering*. The results of this study show that dysnomic stuttering responds positively to treatment. Thus, the presence of such dysfluency need not be considered a negative prognostic indicator.

Similarly, despite B.'s history of heavy alcohol abuse, including a recent relapse, he did benefit from treatment. One of the limitations of single-subject research designs is the restricted ability to generalize the results to the population at large. It should be noted that B. had voluntarily entered an alcohol rehabilitation program prior to beginning work on his speech and language, and this may very well have had a positive effect on his motivation and attendance that might not have been present if he were not enrolled in the rehabilitation program or, indeed, if he were still drinking. Despite these limitations of generalization, it seems important to begin to



**Figure 12.1.** B.'s response to the treatment program with dysfluencies shown as a percentage of the total words he produced during each targeted behavior. The last two sessions, sessions 24 and 25, were follow-up sessions. Data from session 24 were collected 1 month after treatment was discontinued, and data from session 25 were collected 2 months after treatment was discontinued.

document whether patients with histories of chronic alcoholism respond to treatment. This is especially true given the lack of available information regarding the effects of alcohol abuse on prognosis, the potential that it may negatively affect treatment, and the prevalence of this disease in our society. In this case, a 15-year history of alcoholism did not preclude a positive treatment outcome. It is hoped that other clinicians will begin to study and report on patients with histories of alcoholism, so that more informed prognostic statements can be made.

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