Cathy, A Case Study: Dysphasic, Functional, Mixed?

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A speech pathologist in an acute care hospital is asked to assess a variety of individuals with communication disorders. Brain injured patients exhibit a variety of behaviors which may or may not be dysphasic in nature. The site of a lesion may be difficult to determine, especially if the patient is suspected of sustaining brain damage as a result of a metabolic disorder. The patient may have central language deficits, but other behaviors can be present that indicate a variety of cognitive dysfunctions. Premorbid personality also contributes to the behaviors observed after injury.

The purpose of this paper is to present a case study where dysphasic-like symptoms were observed. Additionally, some unusual behavior was noted that could have resulted from diffuse brain damage, a psychogenic disorder or a combination of the above. The question arises, "is it possible for a patient to exhibit both dysphasic and functional involvement?" If the etiology cannot be clearly defined, should the speech pathologist take this patient into a therapy program?

Case History

Cathy is a 31 year old female who had been admitted to the hospital emergency room eleven times in the year preceding the most recent admission. Her usual diagnoses were overdose of alcohol, and/or insulin, secondary to diabetes. She had been diagnosed as diabetic five years ago.

A study of her background revealed an abused childhood and depressing adult life. She completed the 11th grade, had been married twice and had four children. None of the children were living with her at the time of the injury. She had intermittantly been under psychiatric treatment at the state mental hospital and had spent six months in prison for forging checks. In recent years her occupations included bartender, dancer and prostitute.

Ten months prior to this hospitalization, a psychiatric consultation was requested. According to the psychiatrist she appeared depressed over the death of a close friend but there was no evidence of thought disorder, psychosis or organic brain syndrome. Intelligence was rated as average and memory for short and long term events was intact. She exhibited a passive, dependent personality.

On October 8, 1976, according to the medical chart, she was admitted to the emergency room in a comatose state. There was no evidence of head trauma and her pupils reacted to light. Lab studies were all within normal limits except for a slightly lowered glucose count of 63 (normal range is from 70 to 120). A Computerized Axial Tomography (CAT Scan) showed questionable slight ventricle enlargement. The electroencephalogram (EEG) was compatible with metabolic encephalopathy. An echoencephalogram showed no shift of midline structures. Her electrocardiogram demonstrated sinus tachycardia and ST-T wave form abnormalities. A cut on her upper lip suggested that she may have had some seizure activity during the night. Considerable amounts of rigidity resembling the cogwheel rigidity seen in parkinsonism was noted. The diagnosis on her chart was encephalopathy secondary to hypoglycemia.

Two weeks after admission, Cathy was transferred to the rehabilitation unit. At that time a communication evaluation was requested. In the clinic she did not respond to any visual, auditory or tactile stimuli. She was unable to hold an object in her hand and usually stared straight ahead unless forced to look at an object or establish eye contact. On the ward the only spontaneous behavior was eating, which she did with her hands, swallowing only when her mouth could not contain another piece of food.

A neurologist observed that her behavior resembled akinetic mutism. Akinetic mutism is an alert-looking immobility that characterizes certain subacute or chronic states of altered consciousness in which sleep-wake cycles have returned, but evidence for mental activity remains almost entirely absent (Plum and Posner, 1972).

It was decided that a trial period of therapy should be initiated. The short term goal was to provide controlled intense stimuli in order to encourage Cathy to respond to her external environment. The following are a summary of behaviors observed at critical periods of change during therapy.

Number of weeks following onset	Behaviors Observed
2 weeks	Initiated therapy, daily for 2 or 3-1/2 hour sessions.
3 weeks	Able to demonstrate function of common objects intermittently. Perseverations noted.
4 weeks	Able to imitate her name and names of common objects. Speech is whispered (has been heard to clear her throat and cough with phonation). Continues to perseverate unless cued.
6 weeks	Identification of objects is inconsistent. Attention span has increased. Will follow a few simple commands. First spontaneous response: "Go home". Still not voicing.
7-1/2 weeks	Able to read printed words but unable to match them to the correct object. Graphic responses are unintelligible but differentiated.
8 weeks	Speech is now voiced. Output is characterized by perseverations, jargon and neologisms. Continues to require cueing for correct responses.
9 weeks	Is becoming more restless and hostile. Is able to say a few intelligible sentences but syntax is incomplete and responses are delayed.
11 weeks	Is speaking spontaneously when she needs something. Prefers to use one word rather than a sentence. Still has confusion with yes and no responses and substitutes neologisms intermittently.

Ten weeks after therapy was initiated, Cathy was discharged from the hospital in the custody of her guardian. Because of her growing hostility toward the staff it was decided to set up a home therapy program to be administered by her guardian. This person had attended many of the therapy sessions and appeared to have a good grasp of the problem and the remediation program. She was seen in the speech clinic every two weeks and suggestions were made

regarding the home program.

Cathy has continued to improve in the speech and language areas but still has deficits in both long and short term memory. Cognition is still not up to her age level and prognosis for total recovery is poor. She is presently going through a vocational rehabilitation program, with the goal being placement in a sheltered workship environment.

Discussion

Hypoglycemia in persons with diabetes usually results from the use of insulin or an oral hypoglycemic agent. It is one of the most common and serious causes of metabolic coma, exhibiting almost limitless combinations of clinical signs and symptoms. Pathologically, hypoglycemia directs its main damage at the cerebral hemispheres producing laminar or pseudolaminar necrosis in fatal cases but largely spares the brainstem (Plum and Posner, 1972). "Among the permanent brain disturbances resulting from hypoglycemia are mental deterioration, schizophrenia, affective disorders, hemiparesis, aphasia, choreiform movements, Parkinsonism, epilepsy and narcolepsy" (Plum and Posner, 1972).

In view of her background, many staff members suspected a functional involvement that would account for her unusual behavior. A physician administered drugs in an attempt to break down her inhibitions and elicit voiced speech. These attempts did not alter her behavior.

It is still not clear as to why she did not initially voice her verbal responses even though she was heard to cough and clear her throat. While rare, this clinician has observed head trauma patients and severe aphasic patients with an etiology of cerebral vascular accident, who attempted speech without voicing. This whispered behavior usually lasted only a few days. An inability to vocalize may be an indication of diffuse brain damage.

Cathy's progress followed an orderly recovery pattern that is usually observed in stroke patients with language deficits, even though there was a great deal of behavior that is not usually seen in dysphasia. Many of her communication deficits were characteristic of central language involvement.

If functional involvement is suspected along with central language process impairments, should we as clinicians decline to treat these patients? Metabolic coma is one of the more serious types of coma and can cause severe deficits. As in all types of brain damage, early intervention is critical to recovery. It is hoped that this paper will encourage more research directed toward the patient whose deficits cannot clearly be defined.

Summary of Questions and Comments Following the Presentation

The group appeared divided as to whether therapy was of benefit to this patient. Some felt she would have recovered spontaneously. Others suggested that the supportive interaction in therapy was a positive factor.

It was suggested that some of her behaviors were similar to patients who had overdosed on large amounts of Darvon.

Attempts to do a CAT Scan and EEG were unsuccessful later in her hospital stay because of her inability to cooperate.

To my knowledge, a psychiatric consultation was not requested.

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