A Diagnostic Dilemma

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Introduction. Once one has specialized in the communication disorders of brain-damaged adults and has interacted with a variety of these patients, certain syndromes are reasonably anticipated. Inevitably, patients enter our clinics who defy unambiguous classification and thus remind us of our need to ever expand our repertoire of facts and theories. A description of such a patient is the subject of this paper. Also provided is information gained about the psychiatric syndrome that ultimately determined the direction of care for the patient.

Case History. Mr. C.A.C., a 52 year old, right-handed, Caucasian veteran, was hospitalized at the V.A. Hospital in Denver, Colorado. He had learned English as his only language, had completed the 11th grade and was married with two adult daughters. His occupation had been a television salesman for discount department stores. He has been unemployed for two years, having been fired after an altercation with a fellow employee. Mr. C. sustained a blow to the right side of his head that dislodged several teeth. According to a family member, Mr. C. began to experience memory problems shortly after this episode, with progressive deterioration of mental abilities especially noticeable six months prior to his hospitalization. The family attributed the patient's problems to this event and had filed a personal injury complaint. Litigation was pending. At the time of our contact, he was economically dependent on one of his daughters. A claim of Social Security disability benefits had been filed but no action had yet been taken.

Mr. C. was mildly hypertensive but did not require treatment. There was no history of drug, alcohol or tobacco abuse. Familial history was positive for diabetes mellitus and hypertension. Although the family had no history of dementia, psychiatric disorders or genetically transmitted neurological disease, the patient's sister had recently died as the result of an inoperable brain tumor. A family member reported that her symptoms prior to death included poor memory, difficulty naming things and inability to understand conversation.

Neurological Assessment. The patient's presenting symptoms included anoma, impaired auditory comprehension with confabulation, poor recall and faulty calculation skills. Verbal repetition was intact for even complex phrases. Memory for events, family names and telephone numbers was also intact. The patient's behavior was inconsistent relative to orientation, vigilance, attention and right-left discrimination tasks.

All laboratory analyses were normal, ruling out metabolic, toxic and infectious etiologies. The physical examination and brain scan were also normal. Intriguing but inconclusive findings were obtained from the arteriogram, a C.A.T. scan and an E.E.G. Decreased flow in the left carotid artery with normal intracerebral flow was evidenced on
the arteriogram. The C.A.T. scan revealed enlarged sulci and ventricles suggestive of minimal to moderate cerebral atrophy. The E.E.G. was considered a normal waking record with alpha radiating into the right temporal area somewhat better than into the left temporal. Lacking clear evidence for a vascular or space-occupying lesion, the neurologist diagnosed the patient's condition as presenile dementia of unknown etiology.

**Language Assessment.** When seen for evaluation by a speech pathologist, Mr. C. tended to speak only when addressed, was occasionally echoic, and denied or was unable to describe any problems he encountered when conversing with others. The "Profile of Speech Characteristics" from the Boston Diagnostic Aphasia Examination suggested Wernicke's aphasia, i.e., relatively normal melodic line, phrase length, articulatory agility and grammatical form contrasted with significant paraphasia, word-finding difficulties and impaired auditory comprehension. The "Z-Score Profile" is summarized in Table 1 by averaging the standard deviations for each group of subtests and organizing them according to being on the negative or positive side of the profile. Not well contrasted are the performance differences between oral reading and reading comprehension and between writing to dictation or copying and more propositional writing tasks. Interpretation is difficult, as stimuli differ between subtests and scoring protocols vary; however, where comparison is possible, the patient tended to perform better on oral reading and on writing to dictation.

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| -.4 SD              | +1.0 SD            |
| -.7 SD              | + .9 SD            |

The Porch Index of Communicative Ability (P.I.C.A.) served to clarify these distinctions. Fortuitously, the patient accurately read aloud each stimulus item on subtests V and VII without commensurate comprehension scores. Higher mean scores were obtained on dictation and copying tasks, subtests C, D and E, compared to the propositional naming task, subtest B. Also to be noted on the completed score sheet (Figure 1) are prominent islands of errors within subtests and the presence of the score "2" which may be indicative of an attentional deficit, poor concentration or memory for the task, "noise build-up" or fatigue. An analysis of the results obtained from both test batteries lead to the diagnosis of transcortical sensory aphasia, the features of which are described in Appendix A.
### Porch Index of Communicative Ability

**By Bruce E. Porch, Ph.D.**

**Figure 1**

**SCORE SHEET**

Name: C.A.C.  Case No.: 001  Test No.: 1

Date: 8-10-77  Time: 11:30 to 12:31  Total Time: 61 min.

Test Conditions: STANDARD

Patient Condition: AMBULATORY, NO APPARENT PARESIS OR APH.

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**MEAN SCORE:** 79, 76, 76, 76, 76, 76, 76, 76, 76, 76, 76, 76, 76, 76, 76, 76, 76

**Response Levels:**

- Overall: 10.67
- Gestural: 11.66
- Verbal: 9.80
- Graphic: 9.87

- **Note:** PT READ CARDS ON V / VII ALMOG WITHOUT ERROR

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**Figure 1. Initial PICA Evaluation**
Four and a half months after the initial evaluation, Mr. C. was re-tested with the P.I.C.A. Changes in his behavior were evident (Figure 2) the most notable being the absence of the item score "2", less prominent islands of error, and an increase in perseverative behavior. A comparison of the two P.I.C.A. administrations is reported in Table 2 by modality and overall means. Percentile levels are presented using both left hemisphere norms and extrapolated bilateral norms. The Ranked Response Summary (Figure 3) shows a relatively consistent pattern of deficit which was interpreted as evidence of organic impairment, a point of view that stood in contradiction to the psychiatrist's final diagnosis.

Table 2. Summary of P.I.C.A. Comparative Data

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Psychiatric Assessment. Similar behaviors to those previously described were observed by the psychiatrist in structured interviews with Mr. C. Further, anxiety was apparent when mention was made of his deceased sister and the circumstances of her death. Inconclusive results were obtained when the patient was examined under hypnosis and under the influence of sodium amytal. In the latter condition, it was concluded that the dosage had been insufficient to create a toxic delirium. A preliminary diagnosis of hysterical psychoneurosis taking the form of conversion to his sister's behavior was abandoned when it was learned that the patient's symptoms predated those of his sister.

Retaining the idea of a significant emotional component underlying the patient's behavior, the psychiatrist argued that Mr. C. was unable to cope with the stresses created by his economic situation and the recent death of his sister. Reaction to these pressures coupled with the potential for financial gain by remaining disabled were the factors contributing to the conclusion that Mr. C. manifested Ganser's Syndrome, a hysterical disorder that eclipsed any organic component.

Discussion. Neurology and Speech Pathology Services ceased their interactions with the patient when he was transferred to Psychiatry. Mr. C. did not respond to psychotherapy and was discharged against medical advice when the family became angry with the treatment program.

Having interpreted the patient's behavior from a neuropathologic bias and lacking knowledge about Ganser's Syndrome, my involvement with the case did not end. It seemed important to learn about this psychiatric
Porch Index of Communicative Ability

Score Sheet

Name: C. A. C.  Case No. 001  Test No. 2

Date: 12-22-77  by: C. L.  Time: 5:35  to 10:50  Total Time: 57 min

Test Conditions: Standard

Patient Conditions: Same as previous test

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Response Levels:
- Overall: 16.33
- General: 11.55
- Verbal: 9.15
- Grapho: 9.50

Note: Pt. perseverated frequently on II, III, IX, & IX

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Figure 2. PICA at four-and-one-half months.
Figure 3. PICA at four-and-one-half months.
syndrome so as to avoid future "misdiagnoses." The information gathered about Ganser's Syndrome is summarized in Appendix B.

According to Whitlock (1967), Ganser first described the syndrome in 1897. Since that time controversy has thrived. Areas of debate include 1) whether the syndrome constitutes an hysteria or a psychosis, 2) whether or not it occurs in the absence of organic deficits, 3) the required number of symptoms before the syndrome can be diagnosed, and 4) the differential diagnosis of the syndrome from other disorders that have similar behavioral manifestations. These are reflected in Appendix B.

Of pertinence to this case are the five behavioral features which constitute the syndrome. Vorbeigehen, or approximate answering, was evident in the patient, although the term employed to describe it was paraphasia and the P.I.C.A. scores that reflected it were 5, 6 and 7. Ingraham (1967) and Whitlock (1967) expressed the opinion that this criterion alone was a poor one on which to base the diagnosis of Ganser's Syndrome because it can be found with a variety of organic disorders. Interestingly, Whitlock (1967) attributed to Critchley the statement that Vorbeigehen was the first symptom of sensory or jargon aphasia in the course of organic dementia. Tsai (1973), on the other hand, stated that it was this feature that clinically distinguished his ten cases from other types of patients.

Hallucinatory episodes and hysterical sensory conversion symptoms were never observed with Mr. C. nor did he regain normal mental functions with subsequent amnesia during the period of our contact. The remaining symptom of clouded consciousness was possibly an aspect of his behavior given the occurrence of the P.I.C.A. item score "2," the islands of errors, and the inconsistent responses typifying his performance on orientation tasks, although other explanations are also feasible.

Even being equipped with background information about Ganser's Syndrome did not dispel the diagnostic dilemma created by this case. The question remained about the etiology of Mr. C.'s deficits, especially given the variety of neuropathologies implicated in reported cases of Ganser's Syndrome. If the etiology was organic rather than hysterical, the distinction between a progressive dementia and a more discrete left hemisphere impairment could not yet be determined.

Conclusion. The bases for the disparate or contradictory diagnoses reached with this patient were many. Objective neurological tests failed to provide strong etiological evidence to explain Mr. C.'s behavior. Ignorance of Ganser's Syndrome hampered interdisciplinary cooperation. From a speech pathologist's perspective, had this knowledge been available, a more assertive involvement with the patient's case could have provided at least periodic, objective reassessment to document the progression, stabilization or improvement of language abilities and could have offered a program of family education and/or patient rehabilitation.

Not to be ignored is the influence semantics may have had, for the behavior observed did not appear as disparate as the diagnostic labels applied. Since these labels reflected different assumptions and therefore, different patient management decisions, the impact was not a spurious one. Underlying this influence is the truism that higher cortical functions are complex, thus promoting a multitude of interpretations. The interdisciplinary interest in neurophysiological and neuropsychological processes inevitably creates overlapping areas of professional responsibility. In
acknowledgment of that responsibility, we as speech pathologists can continue to sensitize ourselves to the theories and terminology of other disciplines, as this case presentation hopefully illustrated. Such sensitivity would enhance our ultimate goal – to provide appropriate patient care programs in an interdisciplinary setting.

REFERENCES


APPENDIX A: Transcortical Sensory Aphasia

Etiology:
Vascular insufficiency; exogenous toxin exposure; CVA (in resolution)

Anatomical Findings:
Focal cortical or subcortical lesion of the left posterior temporal lobes
Diffuse cerebral atrophy especially involving the left temporal lobe
Bilateral temporal lobe atrophy
"Watershed" lesion sparing Wernicke's area, Broca's area and the connections between them

Behavioral Description:
Preservation of:
verbal repetition
patient-initiated serial or automatic speech
recitation of memorized material and songs
oral reading
writing to dictation and copying

In the context of significant deficits of:
auditory comprehension for language
reading comprehension
naming skills
With frequent co-existing disturbances of:
attention for acoustic stimuli
memory for spoken language
Spontaneous speech is fluent and syntactically well organized with some paraphasia. A patient may not initiate conversation. Some echolalia may be present.

APPENDIX B: The Ganser Syndrome

Etiology:
Psychogenic (a dispute exists as to whether this syndrome constitutes a form of hysteria as originally described by Ganser or a psychosis); stress related

Anatomical Findings:
No autopsy reports; features of the syndrome have been reported in conjunction with:
sagittal meningioma
exogenous toxin exposure
vascular defect in the left parietal area
neurosyphilis
space occupying lesion of the left hemisphere
syringomyelia with hydrocephalus
closed head trauma

Behavioral Description:
Vorbeigehen - verbal responses that are approximate to appropriate answers;
randomness is a striking feature
Visual and auditory hallucinations
Clouding of consciousness resulting in confusion and disorientation to time and space
Disturbed sensory perception of an hysterical nature
Transitory state with amnesia for the episode

Common Misdiagnoses:
Malingering
Schizophrenia
Organic dementia
Dysphasia, especially nominal aphasia
Hysterical pseudo-dementia

DISCUSSION

Q. Did the patient confabulate, and how disoriented was he?
A. Yes, he did confabulate, especially when he was expected to answer yes-no questions about information contained in paragraphs read aloud to him. Findings relative to orientation were inconsistent. For instance, he was consistently oriented to the fact that he was in a V.A. hospital but when queried as to which floor he responded, "The 6th floor" despite visual cues that would allow him to determine that he was on the first floor. He could find his way to clinics unscourted. In my opinion, many of the things he was asked to do relative to orienting himself reflected his comprehension deficit and expressive problems rather than disorientation.
Q. Was he oriented to time and person?
A. Yes, he was, in my opinion. The psychiatrist stated he was disoriented to person, but the neurologist did not observe this.

Q. Were C.A.T. scans, E.E.G.'s, etc. repeated?
A. No, they were not, although it would certainly be desirable.

Q. He changed from a BL to a left pattern.
A. Yes, you are referring to the higher mean score on Subtest X of the P.I.C.A. compared to the mean score of Subtest VIII on the initial test. This was not true of the second test.

Q. Did you say he was treated as a hysterical patient?
A. Yes.

Q. How was he treated and was it effective?
A. He was treated psychotherapeutically but I do not have details of the specific techniques employed. According to the psychiatrist, the patient did not respond to any attempts at psychotherapy.

Q. Did you treat him?
A. No. I was only able to test him after which he became a patient in psychiatry.

Q. Where is he now?
A. He went to the Mayo Clinic shortly after he left the V.A. All I was able to learn from the family was that Mayo Clinic's diagnosis supported an organic etiology. I do not know what their diagnosis was.

The family has since gone to the Colorado Citizen Advocacy Office and filed a complaint against the V.A.'s treatment of Mr. C.

Q. One of the things this brings out is that there is no single diagnosis. Sometimes there is a medical diagnosis and a speech diagnosis for the same patient. I think it is necessary for us to remind our medical colleagues that we are speaking of the speech diagnosis. We are not disagreeing with them.

To raise the issue of whether you see aphasia in dementia, people such as Bornstein would say, yes, you see aphasia, apraxia and agnosia in dementia. I do not find it too useful to call is aphasia. It is like talking about two different disorders that have different courses.

A. I share your discomfort. In reviewing the reported cases of transcortical sensory aphasia, it seemed that very different patients neurologically were being described. Some cases reflected dementia in which only the speech areas were spared of damage once autopsy was performed. It would appear that in such cases very little information could be available for linguistic processing. Other cases report discrete lesions in the left hemisphere, which would suggest a line or argument similar to Geschwind's for a disconnection syndrome. Certainly these patients would differ prognostically and therapeutically even though the language behavior may be similar.