

Subclinical Aphasia Following Closed Head Injury:
A Response to Sarno, Buonaguro and Levita

Claude M.J. Braun
Université du Québec à Montréal and Concordia University, Montréal, Canada

Jacinthe M.C. Baribeau
Concordia University, Montréal, Canada

Though many authors have reported the occurrence of isolated symptoms of aphasia or other language deficits in subgroups of closed head injury (CHI) patients, frank aphasia was reported to occur in only 2% of 750 cases (Heilman, Safran and Geschwind, 1971) and 14% of 50 cases (Levin, Grossman and Kelly, 1976) in the two perhaps best controlled studies noted in the literature. Research at the New York University Medical Rehabilitation Center (Sarno and colleagues) has indicated, however, that 32% of CHI patients manifested frank aphasia (Sarno, 1980) and that all CHI patients manifested a subclinical aphasia syndrome (Sarno, Buonaguro and Levita, 1986).

Several aphasiologists have argued that language impairment in CHI should be interpreted as a reflection of underlying cognitive disorganization (Gillis and Dixon, 1982; Hagen, 1981; Malkmus and Burditt, 1981). In a review article, Holland (1982) clearly took the stand that language disorders following CHI are not aphasia, but are secondary to cognitive and memory impairments, and objected to Sarno's term "subclinical aphasia" for CHI patients as "inappropriate labelling." Recent new results by Sarno and colleagues and absence of an empirical refutation by any detractors justifies this presentation.

Sarno and colleagues recently published in the June issue of Archives of Physical Medicine and Rehabilitation (1986), test results which further support their argument for a subclinical aphasia syndrome in CHI. Their sample of 125 CHI patients achieved scores on a visual naming test, a word fluency test and the Token Test within the range of aphasia. In studies by Brooks and Aughton, 1979; Brooks, Bond, Jones and Risvi, 1980; performance on the Token Test by CHI patients was not as impaired as Sarno et al. reported.

The only report suggesting percentages of post-traumatic aphasia as high as those reported by Sarno and colleagues consists of a study quoted by Sarno (Bricolo, Turazzi and Feriotti, 1980). Included in this study were 135 severely damaged closed and penetrating brain injured inpatients. Nonverbal psychological functions were not assessed, precluding the differentiation between generalized intellectual dysfunction and aphasia.

The study by Sarno and colleagues was based on a group of CHI patients whose mean duration of coma was not reported (the reader is informed that median duration of coma exceeded 24 hours). Language tests were administered at a mean of 45 weeks (median = 26 weeks) after trauma. The authors report no neurological or neuropsychological data on the subjects, relying on language tests and biographic data to define the groups, to the exclusion of other clinical criteria.

This latter aspect of the study raises serious questions about the authors' suggestion that CHI invariably results in clinical or subclinical aphasia. Several reports in the literature have indicated that language disturbances recover relatively more, and more quickly, than other neuropsychological functions (Alajouanine, Castaigne, Lhermitte, Escourolle, Ribaucourt, 1957; Conkey, 1938; Ruesch, 1944). Furthermore, it is useful to have some clear

nonverbal clinical measure of the severity of the injury to enhance any interpretation of traumatic language disturbance.

The purpose of the present study was to clarify the issue of objective performance deficits on several similar language tests in a clinically well-defined group of severely damaged CHI patients tested with a wide ranging battery of neuropsychological tests long after the period of presumed spontaneous recovery.

METHOD

Subjects. Forty-one cases (39 males, 2 females) of severe CHI admitted to an experimental university-based perceptual-cognitive computer remediation program were tested. All cases had sustained motor vehicle accidents except two who had fallen. Table 1 provides further description of the group. Patients with a history of pretraumatic alcoholism (N = 7) or with current alcoholism (N = 2) and/or drug abuse (N = 10 pre-trauma, 3 post-trauma) were not excluded from this study because the point of the study was to demonstrate relative absence of an aphasia syndrome following CHI. No subjects had pre-traumatic histories of neurological or psychiatric consultation. In the Sarno et al. (1986) sample 3% of CHI cases had audiometric deficits. These cases were not excluded from the study, nor was the audiometric deficit defined. In the present study, 38% of the sample manifested audiometric deficits defined as hearing loss >25 dB SPL-ANSI at 750, 1000, 1500, 2000, 3000, 4000 Hz and >30 dB at 500 and 8000 Hz (these cut-offs represent 2 SDs below the norm in our laboratory's acoustic conditions). None of our subjects had a hearing loss in the frequency range of speech (600-2000 Hz).

Table 1. Subject characteristics.

	Mean	SD	Median	Range
Age	28.66	7.84	27	18-51
Education	11.05	2.63	11	5-19
Duration of coma (days)	76.54	141.26	35	7-912
Interval since trauma (months)	70.78	54.53	55	12-240

Note. Coma was defined as being terminated if a patient manifested all 3 of the following criteria: 1) opening eyes spontaneously, 2) producing a comprehensible verbal response, 3) obeying a command motorically (Teasdale and Jennett, 1976). Though duration of coma varies considerably, brain damage was confirmed in all cases by means of radiological and/or neuropsychological evaluation.

Procedures. Patients were tested by Ph.D. and M.A. psychology students using the recommended standardized procedures. Because a normative interpretation of the tests will be used in subsequent sections, references for the norms and administration procedures of each test are provided in Table 2. A score on a test was judged deficient if lower than 2 SDs below the normal mean.

Table 2. Norms and purposes of tests administered. Asterisks mark tests of verbal functions.

<u>Tests related to frontal lobe function</u>	<u>Percentage deficient</u>
Design Fluency (Jones-Gotman and Milner, 1977)	42%
Finger Tapping Right Hand	38%
Finger Tapping Left Hand (Bornstein, 1985)	49%
Dynamometer Right Hand	20%
Dynamometer Left Hand (Bornstein, 1985)	15%
Wisconsin Card Sorting Test (Heaton, 1981) (perseverative errors)	10%
*Benton C.O.W.A.T. (Benton, Hamsher, Varney and Spreen, 1983)	47%
Kolb Sequential Praxis (limbs) (Kolb and Milner, 1981)	23%
<u>Tests related to temporal lobe function</u>	
*Russell Adaptation of WMS (Semantic Memory) (Russell, 1975)	50%
Ekman Test of Discrimination of Facial Emotions (Ethier, 1985)	26%
*Verbal Contextual Test of Emotion Discrimination (Ethier, 1985)	63%
Russell Adaptation of WMS (Designs) Russell, 1975)	54%
<u>Tests related to parietal lobe function</u>	
Benton Test of Right-Left Discrimination (Benton, 1969)	13%
Tactual Performance Test (Right Hand)	20%
Tactual Performance Test (Left Hand) (Heaton, Grant and Matthews, 1986)	28%
Two-Point Aesthesiometry (Right Hand)	29%
Two-Point Aesthesiometry (Left Hand) (Semmes, Weinstein, Ghent and Teuber, 1960)	25%
Semmes Test of Personal Orientation (Semmes, Weinstein, Ghent and Teuber, 1963)	10%
SDSS (Right Body Side)	10%
SDSS (Left Body Side) (Antofani and Smith, 1979)	36%
<u>Tests of attention</u>	
D-2 Test of Attention (Brickenkamp, 1962)	50%
Simple Reaction Time (Both Hands Combined) (Braun, Ethier and Baribeau, 1987)	85%
<u>Pure tests of verbal function</u>	
Vocabulary (WAIS-R) (Wechsler, 1981)	10%
Token Test (Short Form) (Spellacy and Spreen, 1969)	2%

RESULTS

Tests of verbal functions used in our laboratory indicated that verbal delayed semantic memory (30 minutes), verbal fluency and comprehension of emotionally laden phrases demonstrated important deficits, in decreasing order. There were few deficits in vocabulary (from the WAIS) or on the Token Test. On the whole, tests of verbal functions were less affected by CHI than tests of attention and of memory, in that order (Table 3).

Table 3. Pearson product-moment correlations and levels of statistical significance.

	Education	Coma	Interval	Token	Vocabulary	Cowat	Deficits
Age	-.006 NS	.184 NS	.244 NS	-.173 NS	.207 NS	-.029 NS	.421 **
Education		.049 NS	-.076 NS	.382 **	.445 **	.076 NS	-.064 NS
Duration of coma			.075 NS	-.069 NS	-.017 NS	.016 NS	.420 **
Interval since trauma				-.565 ***	-.135 NS	-.176 NS	.436 **
Token test					.222 NS	.244 NS	-.340 *
Vocabulary test						.016 NS	-.060 NS
Cowat test							-.575 ***

Note. *** = $p < .001$, ** = $p < .01$, * = $p < .05$, NS = non significant.

Duration of coma, as consistently reported in the literature, was not related at all to the aphasia screening test (Token Test), to vocabulary performance, or to verbal fluency. Interestingly, the longer the interval between trauma and testing, the less these subjects were deficient on these verbal tests, particularly with regards to the Token Test, where the trend is statistically significant. On the other hand, the total number of neuropsychological deficits was significantly positively related to duration of coma and to the interval between trauma and testing.

DISCUSSION

The discrepancy between these results and those of Sarno and colleagues could be partially explained by the much longer interval between trauma and testing in the present study. A difference in selection of subjects cannot be ruled out either, considering that the NYU Medical Rehabilitation Center has a Speech Pathology service whereas the present rehabilitation program does not. On the other hand, all subjects of the present study were referred by rehabilitation centers with speech therapy services, except for two cases. Finally, the cases evaluated here were also most likely more severely damaged as indicated by the marked difference in audiometric results, and probably, duration of coma.

The results nevertheless very clearly support the claims of authors who have objected to Sarno's (1980, 1986) suggestion of a universal subclinical aphasic syndrome following CHI. The present sample was more deficient on attention and memory tasks than on verbal tasks and was more frequently deficient on nonaphasic verbal tasks than on an aphasia screening task. The very low percentage of frank aphasia (2%) following CHI reported by Heilman, Safran and Geshwind (1971) was corroborated exactly in the present study.

It is not being argued that, with emergence from coma, some closed head injury patients may not manifest an aphasic-like phenomenon. The results presented here suggest that verbal, and particularly so-called aphasic

deficits as measured in this study, tend to recover following CHI. The data also suggest that most other neuropsychological sequelae manifest the inverse trend. Whereas verbal performances were not found to correlate with duration of coma, the summed deficits of a comprehensive neuropsychological test battery did correlate significantly with duration of coma. These results support previous research except for that of Sarno and colleagues (Groher, 1977; Morsier, 1973).

These findings demonstrate that not much of a case for "subclinical aphasia" in CHI can be made, that verbal deficits following CHI can most logically be explained as a reflection of underlying cognitive dysfunction, and that some verbal deficits observed following CHI have a good prognosis for recovery relative to other neuropsychological functions. These conclusions need to be supported by more extensive evaluation of language and other functions in closed head injury.

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DISCUSSION

- Q: You appear to have placed a lot of emphasis on the factor of chronicity. If a language disturbance is not chronic then it cannot be aphasia?
- A: Aphasia evolves but it is aphasia. When a patient comes out of coma following CHI he or she has a lot of problems that look like aphasia but which may not be aphasia. It would be important to test CHI patients at various phases, particularly following the confusional stage, to see how these language deficits evolve, using the proper aphasia batteries to see if there are clear aphasia profiles or not.
- Q: A big problem with the 1980 and 1986 papers by Sarno and colleagues was the absence of neurological control. Basically these series comprised very heterogenous samples of people with heterogenous deficits. Would you comment on the neurological data regarding your group?
- A: Pioneers in the investigation of coma in CHI, people like Jennett and Teasdale, contributed something very important. Even in the recent literature, coma duration is considered one of the most valid indices in CHI. Coma duration has been demonstrated to relate to the ensemble of neuropsychological deficits in CHI patients. It also has the advantage of being relatively easily obtainable in the absence of appropriate neurological imaging data. I would rate coma duration, post-traumatic amnesia duration, neuropsychological test results, and magnetic resonance imaging as good indicators of persistent damage to the central nervous system. In my experience, CT scanning becomes a weak indicator of brain damage with extended post onset time and yields an inordinate proportion of false negative judgments.
- Q: Have you done anything to exclude gross hemorrhages, subcortical involvement, or cortical problems such as lacerations?
- A: No. The patients were not selected beyond the criteria of CHI and chronicity (minimum 1 year post-onset time). I personally don't find localizing information in CHI to be very useful in the context of my research questions. Most of the damage, as has been stressed by Harvey Levin, is subcortical, is very diffuse and extends to the brain stem, the corpus callosum and other long fiber tracts. In addition, there can be coup and contre-coup effects, hemorrhages and lacerations. These are very difficult to tease out reliably.
- C: These considerations may be important in considering the differences between Sarno and colleague's results and your results. Without this information it is not clear which reports might be more representative of the general situation in CHI.
- Q: What one person defines as a 6-day coma can be defined as a 21-day coma by another. Do you feel that more precise definitions are required if we are to use coma duration for purposes of comparison?

A: Yes, but we need more than definitions, we need to measure coma duration using scales. The most commonly used one in CHI is the Glasgow Coma Scale (Jennett and Teasdale). We tried to adjust the hospital records to meet the Glasgow Scale model by making verifications with the treating physicians by telephone. But we find that the patient's account, the family's account and the physician's account of coma duration can be at great odds.

Q: We have interpreted lifting of coma to occur when any one of the three responses specified by Jennet and Teasdale (eye opening, verbal response to command, motor response) occurs. You have defined it as requiring presence of all three. It would seem that you are talking about the duration of obtundation rather than coma.

A: Our definition is probably closer to the lay person's definition of coma than the neurologist's definition. Perhaps the next speaker, a neurologist, could shed some light on this issue.

C: The definition used here for coma does not correspond to the definition most commonly used in neurology following Plum and Posner's classic work on the subject (Stupor and Coma). According to their conception, coma is absolute loss of any response. Coma as defined here includes states that would be termed lethargy or stupor by neurologists.

Q: I think that your data shows that frank aphasia is not as common following CHI as other disorders. What is your perspective on other communicative disorders in that population?

A: What has impressed me is the wide range of disorders of affective deficits in CHI patients' communication. One of the test results reported in Table 2 concerns a "verbal-contextual" task which is a verbal-semantic analog of the Ekman test, wherein a brief sentence contextually laden by one of the six primary emotions described by Ekman is to be matched with one of six verbal labels of these emotions, namely joy, fear, surprise, anger, disgust, sadness. Our patients manifested tremendous difficulty in accomplishing this task. They also had great difficulties with the Ekman test consisting of identification of emotions of faces. I feel that we should explore CHI patient's production and reception of prosody. Finally, I feel that disorders of speech output should be further investigated in light of the severe handicaps that CHI patients manifest in this respect. In particular, my inclination would be to investigate dysfunction at the various tube and valve levels of the phonatory apparatus and to estimate the differential involvement of motor cortex, basal ganglia, cerebellum, and brain stem respiratory and other motor circuits.