

Closed head trauma and aphasia¹

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SUMMARY A prospective study has been done on the relationship between closed head trauma and aphasia. The most frequent type of aphasia seen after closed head injury is an anomic aphasia. This aphasia is often associated with other defects of higher cortical function. The second most common type of aphasia is a Wernicke's aphasia. Other types of aphasia were not seen in this study. The areas of the head which when injured produce aphasia are the right orbitofrontal region and the left temporoparietal region. The prognosis for recovery appeared highly variable.

Brain (1965) records that Valerius Maximus (30 A.D.) described a learned man of Athens who lost his memory for words after being struck on the head with a stone. Although the association of language disorder with head trauma was known even to the ancients, there are even now few detailed descriptions of aphasia after closed head injuries. Goldstein (1942), Schiller (1947), Russell and Espir (1961), and Walker (1968) have written about injuries that resulted in aphasia and other defects of higher cortical function, but these injuries were, for the most part, penetrating wounds. There have been cases of closed head injury and aphasia reported in the literature by Lichtheim (1885), Russell (1932), and Perlo and Schwab (1969), but these have been single case reports and in most the aphasia was reported merely as an accessory finding. Goldstein (1948) studied a woman who had suffered head trauma and had anomic aphasia, but despite the history of trauma, he attributed the defect to arteriosclerosis. Glaser and Shafer (1932) studied 255 patients with head trauma from non-penetrating wounds. Sixteen of these cases developed aphasia. One of the 16 had a 'complete motor aphasia'. Ten patients had a 'partial motor aphasia', four had a 'mixed sensory and motor aphasia', and one had a 'pure sensory aphasia'. The authors did not mention how they examined their patients for aphasia or on what basis they were classified. Included in this series of patients were cases with depressed fractures,

abscesses, and intracerebral haematomas. Although these authors studied the relationship between the location of the injury and other signs and symptoms, they did not do this for their cases of aphasia.

The purpose of this study, therefore, was to do a prospective investigation on the relationship between closed head trauma and aphasia.

METHODS

During a 10 month period adult patients admitted to the Boston City Hospital with a diagnosis of head trauma were screened for aphasia and other defects of higher cortical function. Patients admitted with the diagnosis of head trauma were usually admitted because of unconsciousness or history of previous unconsciousness, the presence of a skull fracture or evidence of extensive soft tissue injury, neurological signs, or evidence of severe injury to other parts of the body. The aphasia screening examination, which was not done until the patient was alert and cooperative, consisted of evaluation of spontaneous speech, comprehension of spoken and written language, repetition, naming, ability to write, calculation, right-left orientation, and finger recognition. If a patient showed any defect on this screening test he was given a more detailed aphasia examination as well as a thorough neurological examination. The period of unconsciousness as well as the severity and area of head injury were noted. All other pertinent clinical and laboratory data were recorded and patients were followed until their aphasia cleared or they were lost to follow-up. Patients were excluded from this series if there was no clear history or evidence for head trauma. They were excluded if they did not have aphasia before a seizure or it did not persist at least a week after a seizure. Patients with subdural haematomas were included only if the aphasia persisted after operation, and there was evidence that the cerebrum was not damaged by the removal. Patients with depressed

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fractures, intracerebral abscesses, and cases in which there was operative intervention with manipulation of the cerebrum were also excluded from our series.

We classified our aphasics according to the Benson and Geschwind (1971) classification. Anomic aphasia was defined as a fluent aphasia in which the patient demonstrates verbal paraphasias and circumlocutions, has normal comprehension and repetition, and has abnormal naming for all kinds of material especially to confrontation. Wernicke's aphasia was defined as a fluent aphasia with paraphasia, poor comprehension for spoken and written language, and poor repetition. Broca's aphasia was defined as non-fluent aphasia with good comprehension, which may improve slightly in repetition, series speech, and singing. Global aphasia was defined as a non-fluent aphasia with poor comprehension, poor repetition, and poor naming. Conduction aphasia was defined as a fluent aphasia with literal paraphasia, good comprehension, but very poor repetition, and impaired naming. We defined the syndrome of the isolated speech area as a fluent aphasia with poor comprehension, but with excellent repetition and echolalia.

The location and severity of the major area injured in 100 cases of head trauma requiring admission, but without aphasia, were examined in order to compare the areas injured in this non-aphasic group to the areas injured in the aphasia group.

RESULTS

During the 10 months this study took place, approximately 750 patients were admitted to the Boston City Hospital with a diagnosis of closed head trauma. Of this group there were 15 cases of aphasia after closed head trauma. Two cases were excluded, and 13 met our criteria for inclusion. One excluded case was a patient who had an anomic aphasia while lethargic, but in whom when he became alert no aphasia could be detected. The second patient excluded had a persistent anomic aphasia but had had his anterior temporal lobe needled by the neurosurgeons before he was examined for aphasia.

The mean age of the aphasic group was 57, and the mean age in the control group with closed head injury but without aphasia was 43. In the group of 13 aphasic patients there were six known alcoholics. Previous studies have shown that of the entire patient population of this hospital approximately 20% are known alcoholics, while among patients with head injuries in this institution the figure is much higher.

Seven of the 13 patients with aphasia had evidence of right orbitofrontal trauma, four had evidence of trauma in the left temporoparietal region, and two had no external evidence of trauma (Table 1). In the nonaphasic, or control group, 27 of the 100 cases with closed head trauma had evidence of trauma to the right orbitofrontal region, 29 to the left orbitofrontal region, nine to the right temporoparietal

TABLE 1
AREA OF HEAD INJURED IN 13 APHASIC PATIENTS

	<i>Orbitofrontal</i>	<i>Temporoparietal</i>	<i>Occipital</i>	<i>Other*</i>
Right	7	0	0	2
Left	0	4	0	

*No evidence of injury.

region, nine to the left temporoparietal region, six to the right occipital region, six to the left occipital region, and 14 either had no evidence of injury or were injured in the midline, on the nose, or on the mandible (Table 2).

TABLE 2
AREA OF HEAD INJURED IN 100 NON-APHASIC PATIENTS WITH HEAD TRAUMA

	<i>Orbitofrontal</i>	<i>Temporoparietal</i>	<i>Occipital</i>	<i>Other*</i>
Right	27	9	6	14
Left	29	9	6	

*No evidence of injury, or injury on mandible or nose.

We will now give three illustrative cases.

CASE 1

(BCH no. 2109370) Four days before admission this 35 year old alcoholic female had been beaten over the head by thieves. After this episode the patient's landlady noticed that the patient was 'talking funny', but did not become concerned until the day of admission when the patient had a major motor seizure and was admitted to Boston City Hospital.

Three days after her admission, the patient was transferred to the Neurological Unit. General physical examination revealed hepatomegaly. There was no external evidence of head trauma. On neurological examination she was alert and cooperative. She is right handed and a high school graduate. She was oriented to place, person, and year. She did not know the month or day. Her memory for recent events was poor and she could recall only one out of three objects in three minutes. On aphasia examination her spontaneous speech was fluent, but contained circumlocutions and empty phrases, while comprehension and the ability to repeat verbal material appeared normal. Her major difficulty was in naming objects. She could, however, tell what these objects were used for and she could select the correct name if given a choice. The patient could not read aloud, or show comprehension of written language, whether tested by commands, yes-no and multiple-choice questions, pointing or skin galvanometry. She could not recognize words, syllables, or letters visually or tactually. She had difficulty in recognizing words that were spelled to her but could spell when given words. Her writing, both spontaneous and to command, was excellent. She wrote well to dictation and could copy, but she could not

read what she had written. Although the patient could match colours well she could not name them correctly even when the answers were given in the form of a multiple-choice question. When asked to name colours from memory she did well. The patient confused her right and left, could not name her fingers, even when given the name in a multiple-choice question, and could not move the finger the examiner requested her to move or even point to the correct finger. Her calculations were poor and she did not correct with multiple-choice questions. Her drawings of cubes, clocks, and flowers were normal and the patient never demonstrated a hemianopsia.

The patient had a lumbar puncture which showed xanthochromic fluid but was under normal pressure and contained no cells. Her EEG, brainscan, and left carotid arteriogram were all normal.

She was discharged one month after her admission and at the time of her discharge her findings were essentially unchanged.

The patient was seen in clinic eight months after discharge. Her anomic aphasia persisted, and although she could read occasional letters and words she remained dyslexic. Her writing remained normal and she no longer showed right-left confusion, finger agnosia, and acalculia. The remainder of her examination was unchanged.

CASE 2

(BCH no. 2011891) An 80 year old right-handed woman had been in good health except for decreased vision which resulted from advanced glaucoma. On the way to church she tripped, fell, injured her head, and was knocked unconscious. When admitted to Boston City Hospital, however, she was noted to be lethargic and aphasic.

A consultant from the Neurological Unit examined her on the following day and noted that the patient had a large contusion over the left parietal area. Although the patient appeared alert her intellectual function could not adequately be tested because of severe aphasia. Her speech was fluent and she would not even stop speaking when the examiner asked her to stop. Her speech was markedly paraphasic and contained many neologisms. She showed no evidence that she could comprehend written or spoken language. She would not repeat, name, do series speech or write. On examination of the cranial nerves there was a questionable decrease of blink on the right side but no consistent field defect could be obtained. The patient moved the right side slightly less than the left and she had a right-sided extensor plantar response. Her cerebrospinal fluid contained 35,000 erythrocytes/cu. mm and skull radiographs showed a left temporo-parietal linear non-displaced fracture.

The patient regained her strength on her right side, but when discharged on the 32nd day she remained severely aphasic.

CASE 3

(BCH no. 2109684) A 56 year old right handed male alcoholic was beaten on the head during a mugging.

He was unresponsive on admission, but shortly after admission he awoke, was noted to be aphasic, and was transferred to the Neurological Unit.

On admission to the Unit it was noted that the patient had a contusion over the right orbit and a right conjunctival haemorrhage. On neurological examination it was noted that the patient was alert and fully oriented. While he could not remember the events which surrounded his injury, he could recall three objects in three minutes and seemed to have a good recent memory. The remainder of his intellectual functions were unremarkable. On aphasia testing the patient's spontaneous speech was fluent, but contained frequent circumlocutions and empty phrases. His comprehension of spoken language was excellent as was his repetition. He exhibited a major difficulty in naming. While he could not name objects he saw or touched he could tell what they were used for, and could select the correct name if given in a multiple-choice fashion. The patient showed little evidence that he comprehended the written material. His writing, both to dictation and spontaneously, was also extremely poor. The patient did not demonstrate acalculia, finger agnosia, or right-left confusion. Other than decreased pin prick sensation in a stocking-like distribution and decreased ankle jerks the remainder of the neurological examination was normal.

The patient's lumbar puncture, skull radiographs, and EEG were all normal.

He remained in the hospital for 22 days and during this time his clinical picture remained unchanged. Five months after his discharge he was seen in a local tavern by a physician from the Neurological Unit. The patient recognized him, came over to the physician and asked, 'Do you have a . . . You know what I mean. A thing to buy a drink with?'

DISCUSSION

There were only two types of aphasia seen after closed head injury. Nine patients had a classical anomic aphasia and four had a Wernicke's aphasia (Table 3). No other type of aphasia was seen.

We found three additional cases of anomic aphasia secondary to closed head trauma but since these patients had their trauma before the time this study was started they were not included.

Weinstein and Kahn (1952) and Weinstein and Keller (1963) described a naming defect seen in

TABLE 3
TYPE OF APHASIA

Type of aphasia	Aphasic patients (no.)	Aphasic patients (%)
Wernicke's aphasia (sensory aphasia, receptive aphasia)	4	30.7
Anomic aphasia (amnestic, nominal)	9	69.3
Total	13	100

tion, in addition to a selective vulnerability of the right orbitofrontal region as compared with the left.

Courville (1942) and others have noted that the dorsolateral surface of the temporal lobe was a frequent site of contusion, whereas other regions of the speech area, including the frontal and parietal opercula, were rarely contused. In our series, a parietal lobe contusion was seen in one case at post-mortem examination and in another case a contusion of the dorsolateral temporal lobe was seen during the removal of a subdural haematoma. Several other cases had arteriography which suggested swelling of the temporal lobe. Most of our cases have had either bloody or xanthochromic spinal fluid. Although the pathology of all these cases of post-traumatic aphasia is not known, it appears that contusion of the dorsolateral surface of the temporal lobe and temporoparietal junction is the most likely aetiology for these aphasias.

It is not known for certain why trauma in the same area of the head produces an anomic aphasia in some patients, a Wernicke's aphasia in others, and in some it produces no aphasia. There very well may be a difference in the severity of the trauma or a small difference in the area injured. We were, however, unable to measure these differences in our material.

Although we had some difficulties with long term follow-up, a problem peculiar to our institution and patient population, in our cases recovery varied greatly and we could find no constant relationships that would help the physician to prognosticate.

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