TRAUMATIC SENSORY APHASIA.

BY V. ZACHARY COPE, LONDON.

INJURIES to the head may give rise to various derangements of memory and speech. Following concussion, there may ensue a general mental dullness, accompanied by slowness and uncertainty of speech and impaired memory; commonly this does not last for more than a few days, and can be attributed to the general effect of concussion on the higher cerebral centres. More definite and serious is the impairment of speech consequent on injury to the specialized centres concerned in that function.

Many cases have been recorded in which motor aphasia has followed a traumatic lesion in the region of Broca's convolution, and it is recognized that the treatment of such cases is operative; but very little attention has been paid to those cases in which the aphasia is of the sensory variety, and due to injury to the region of the auditory and visual word-centres. It may be that the paralysis which is likely to accompany cases of motor aphasia calls attention to the condition sooner and demonstrates more urgently the need for intervention, or more likely the sensory lesion occurs much less commonly than the motor variety; but the fact remains that very few cases of traumatic sensory aphasia have been recorded. In the literature accessible to me I have only been able to collect five cases, and any opinions put forward here are based on these and on my own case recorded below.

The clinical history of a case of traumatic sensory aphasia may be one of two types. Nearly always, concussion, either slight or severe, follows immediately after the injury. Unconsciousness may last for anything from a quarter of an hour up to eight days.

In the first type of case, the aphasia is noticed as the patient recovers from the unconscious state; in the second variety there is a latent period between the recovery from unconsciousness and the observance of speech defect. In the case recorded by Dr. Kinneir Wilson, the patient left the hospital apparently well the day after the injury, but two days later returned in a half-dazed condition and unable to express his thoughts. In one case, no mention is made of unconsciousness, but it is stated that the speech defect came on gradually.

The further course of a case, if left without operative treatment, is variable. Considerable improvement occurs within a few weeks, and in a few months the patient may be put down as "recovered," but some defect of memory or speech commonly remains, and in one of the recorded cases hardly any improvement took place for the two years the patient was under treatment.

The prognosis is bad as regards the complete recovery of the function of speech if the case is treated on expectant lines. Recovery ought to be more complete in children, since in them the opposite side of the cerebrum would more readily compensate for the injured side.

Attention must be drawn to the fact that epileptiform fits occurred some time after the injury in two out of the three cases treated expectantly.

In one instance death resulted from the status epilepticus some years after the injury.

The condition may be caused by direct violence applied over the left parieto-occipital region, or by contre-coup injury from a blow on the right side of the cranium. In one case it was due to a penetrating wound over the region of the superior temporal gyrus on the left side. In Heiligtag's case there was accompanying fracture of the skull, but no record of depression of bone.

Since there is no record of depressed fracture in any recorded case the actual lesion causing the condition must have been either hæmorrhage leading to pressure over the sensory centre for speech, or laceration, cortical or subcortical, of that area of the brain.

It has been contended by Phelps that pial hæmorrhage by itself is not sufficient to cause speech defect. He asserts that if any hæmorrhage localized in some part of the speech zone proves to be of cortical origin, it must still be regarded as a concomitant rather than a causative factor in production of the speech defect. Whilst admitting that in most cases of pial hæmorrhage there is some superficial cortical contusion or laceration, it appears more likely that traumatic sensory aphasia is, as a general rule, caused mainly, if not solely, by the pressure of blood-clot over the cortical sensory speech centres; this view receives support from the six cases under discussion. In the two patients operated upon, the removal of the clot caused immediate cure of the condition; in a third (Heiligtag), a pulse-rate of 60 and a headache persisting after eight days of unconsciousness suggest increased intracranial pressure. Dr. Kinneir Wilson's patient showed a marked latent period between the recovery from the concussion and the development of the aphasia; this is in keeping with the occurrence of hæmorrhage, and the latent period suggests a comparison with that which occurs in middle meningeal hæmorrhage. In the other two cases also, the account given appears to indicate an interval before the symptoms of aphasia developed. On *à priori* grounds it appears more likely that hæmorrhage rather than cortical laceration would give rise to sensory aphasia unaccompanied by other lesions, for a cerebral contusion extensive and severe enough to put the sensory speech-centres out of working order would be likely to injure the kinæsthetic area or the hemianopic cortical centre, while it is comparatively easy to imagine a single ruptured pial vessel causing an accumulation of blood that would give rise to considerable direct pressure over the speech centres; such hæmorrhage would tend to stop naturally when the pressure had reached a certain limit. The continuous pressure would in due time cause permanent changes of the underlying cortex.

Treatment.—The line of treatment to be adopted follows from the view which is taken as to the cause of the condition. If the cortical or subcortical laceration is the main etiological factor, expectant treatment is indicated. On the other hand, if pressure of the blood-clot is the main cause, it seems clear that the skull should be trephined, the dura opened and the clot removed. The trephine opening should be made about two inches above the left external auditory meatus, and more bone may be removed in a backward direction if necessary. The subdural region should be explored carefully by a flat dural separator, and there should be no hesitation in incising the subcortical region

for clot if none be found superficially. To illustrate this line of treatment I here relate the case which came under my observation and treatment.

AUTHOR'S CASE OF TRAUMATIC SENSORY APHASIA, TREATED SUCCESSFULLY BY TREPHINING AND REMOVAL OF CLOT.

Ellen B., aged 14, was brought to the Bolingbroke Hospital about eight o'clock on the evening of August 14, 1913, with the history that she had fallen down a flight of six steps and struck the back part and left side of her head against the step or

FIG. 381.—Spontaneous attempt at writing.

ground. The accident had occurred three hours previously; during the intervening time she had been semi-conscious and had vomited frequently. Up to the time of the accident she had been a lively and intelligent girl in full possession of her faculties.

When admitted, the patient's temperature was normal, pulse 76, respirations 16 per minute. She lay with eyes closed, but when approached opened them and said, "Don't hurt me, daddy," and repeated the word "daddy" frequently during the initial examination. She had no recollection of the accident, and with difficulty told of her doings that day. At the same time she continually relapsed into a more drowsy condition; her eyeballs kept rolling upwards.

There was an abrasion and a very small scalp-wound behind and above the left ear. There were no signs of fracture of the skull-base, and no paralysis of cranial or peripheral nerves. The pupils were of normal size and reacted to light; the knee-jerks were elicited and the plantar-reflex was normal.

FIG. 382.—Spontaneous attempt.

FIG. 383.—Attempt at copying capital letters.

Her condition did not improve; she remained in an irritable state, curled up on her right side in bed, and resented any interference. Vomiting occurred several times. She spoke no word spontaneously, but apparently heard what was said to her. At first her condition was taken to be that of simple concussion and cerebral irritation.

Gradually, however, her pulse became slower, till it was about 50 per minute, whilst

her mental condition remained anomalous, in that she seemed fully conscious yet did not utter a word. On August 19, when I saw her first, the condition was much as described above. There was no paralysis, but I could not elicit the knee-jerks; there was some photophobia; the pupils were equal, slightly dilated, and reacted to light normally. Ophthalmoscopic examination showed congestion of the retinal vessels and blurring of the edges of the disc.

On the next day (Aug. 20) the aphasic condition was more fully investigated by my house-surgeon (Mr. O. R. L. Wilson), to whom I am indebted for these notes.

The only word she could be got to utter spontaneously was the nick-name of her sister, and this name, "Doodles," she repeated throughout the examination. She gave plenty of indication that she understood at least some of the words spoken to her, but

how are you.
hasn't any sense

FIG. 384.—Copying small letters.

could not answer any question intelligibly, a jumbled mutter being all that she could attempt. She was unable to repeat words spoken to her.

She did not understand written questions or commands, though it was clear she saw them. She was able to write spontaneously, but her writing was disconnected nonsense. She copied capital letters correctly, but came to grief when attempting to copy ordinary handwriting.

She was unable to write from dictation, nor could she name objects seen. Reading aloud from printed or written matter was not in her power, and she was unable even to pick out objects of which the name was given to her by speaking.

She readily understood pantomimic gestures, and imitated them. She appeared to appreciate the use of some but not all objects shown to her.

From the above examination it was evident that there was some serious lesion in the higher centres for understanding both printed and spoken words, but especially affecting the visual higher centres.

On August 21, operation was undertaken. A horse-shoe-shaped flap was made with its base above the left ear. The trephine was placed so that the centre-pin was two inches, or a little more, above the external auditory meatus. No clot was found outside the dura, which was tense, bulging, and non-pulsatile. When the dura was incised a small amount of dark blood was found underneath. By carefully searching in every direction under the dura by means of a flat metal instrument, a considerable amount of old black blood-clot was discovered in a direction towards the occipital lobe. This clot escaped under considerable tension—it leapt out—and altogether several drachms came away. Some more bone was removed in a backward direction and careful search made for further extra- or intradural hæmorrhage, but no more clot was found. The cerebral cortex was also explored by needle and knife, but no subcortical hæmorrhage was found. The dura was sewn up with thread and the skin flap replaced and sutured with silk-worm gut without drainage. The pulse at the end of the

NELLIE
NELLIE
NELLIE
NEL
anint
ansint

FIG. 386.—Copying.

operation was 100; at the beginning it had been 50 to the minute.

Recovery of speech after the operation was exceedingly quick. The next day

BOLINSBROKE.

BOLINSBROKE

BOLINS

BOLINSBROKE

FIG. 385.—Copying.

the patient asked for some dinner, and in general showed a marvellous improvement. Within a week, practically all her disabilities were removed, and instead of the stolid, non-comprehending countenance, was the intelligent, mobile face, readily smiling, and appreciating every remark made to her.

So far as I can ascertain, this is the first case in Great Britain to be trephined for traumatic sensory aphasia.

PREVIOUSLY RECORDED CASES.

Case 1.—(BEEVOR.) *Vide Lancet*, 1893, 1, 726.

A man, aged 50, fell fourteen feet, and became unconscious for a quarter of an hour. Next day he was drowsy and could not talk, there was no definite palsy. Spontaneous speech was imperfect, and though he understood spoken commands, he talked nonsense. He wrote spontaneously, but could not understand written commands. He could neither repeat words spoken to him, nor pick out objects named, nor write from dictation. He could not copy printed characters, nor was he able to name objects shown to him, though he could recognize their use.

The lesion was diagnosed as probably concussion or hæmorrhage over the visual word-centre.

Recovery is stated to have occurred within three weeks, but memory remained impaired, especially that of names of people and things, moreover a fit occurred during recovery, and another somewhat later than the time the case was reported.

There had been a scalp wound two inches above the external auditory meatus, and Beevor at the time said, "If therefore it had been necessary to operate, I think it would have been advisable to trephine at the scar over the region of the first temporo-sphenoidal convolution, and if nothing were found, to work backwards and upwards."

Case 2.—(MACVICAR.) *Scott. Med. and Surg. Jour.*, 1898, iii, 407.

A painter, aged 26, on December 27, 1897, struck his head against the edge of a dresser whilst under the influence of drink. There was an injury over the right parietal eminence. For two or three days the symptoms were those of ordinary concussion, but on December 30 his wife noticed that his speech was much altered. He could point out anything named, but failed to name most things at sight, though on a wrong name being suggested he repudiated it. He understood all that was said, but nothing written. He could copy print and could write his own name, though he could not name the letters after he had written it. He was blind to figures, completely letter-blind, but not absolutely word-blind.

The patient was given 7-gr. doses of potassium iodide and gradually recovered. The visual word-centre took fourteen days to recover as regards words, and nineteen for numerals. Four months after the accident the patient could write a short letter correctly and read fluently.

MacVicar stated, "It is probable that the nature of the lesion was a hæmorrhage, either single, or multiple minute hæmorrhages. The disturbance of the brain as a whole, through the medium of the cerebro-spinal fluid, was evidenced by the severe symptoms of concussion, the short unconsciousness with some delirium, the persistent vomiting, headache, and slowed pulse, from which he took some days to recover."

Dr. MacVicar has kindly informed me that the patient, several years after the relation of his case, came under treatment in hospital in a condition of status epilepticus. The fits continued till death, which supervened about twelve hours after admission. A post-mortem was granted. Softening, cortical and sub-cortical, was present in the areas mentioned in the case.

Case 3.—(KINNEIR WILSON.) *Brain*, xxix, 416.

Patient (a man) fell from a cart and injured the left side of his head. He remained over-night in military hospital in a more or less unconscious state, but left next day apparently well. Two days later, he returned in a half-dazed condition, quite unable to express his thoughts.

His condition when under observation was as follows: no hemianopia, slight weakness of right arm, sensation unimpaired.

Speech: excellent articulation and phonation.

Little word-deafness. Great word-blindness.

Cannot repeat a single syllable to order. Can sing tunes well, frequently with words. Can whistle accurately. Cannot write or read. Can copy written and printed language.

Cannot read what he has copied. Can draw well.

Cannot name objects, but indicates their use perfectly. No apraxia or mind-blindness. Pours out a torrent of words and nonsense in conversation.

Dr. Kinneir Wilson has kindly informed me that the patient's condition remained *in statu quo* for the two years during which he was under observation.

Case 4.—(HEILIGTAG.) *Deut. Wochensch.* 1910, xxxvi, 2147.

A man, aged 25, on November 4, 1909, was struck on the head by a stick. He had a wound over the region between the left parietal and occipital bones. Also a fracture. Another wound on the right side in a corresponding position.

Patient unconscious; vomiting occurred, and there was slight fever for a few days. Pulse 110, but came down to 60. Remained unconscious for eight days. Complained of headache after wound had healed. Could not read, but could write and speak. This condition was diagnosed as subcortical alexia, and operation was intended, but Dr. Heiligttag writes in March, 1914, that the patient refused operation, and that the clinical symptoms of the case are about the same as at the time of publication.

Case 5.—(BALL.) *Dublin Jour. of Med. Science*, 1888, Sept., 243.

Patient, aged 26. Ten days before admission was struck on the head by a penknife in the left temporal region. Gradually found difficulty in using correct words. The external wound healed. He was able to write his name correctly and to copy writing, but was unable to write from dictation or from sentences which he originated himself. Could not read. Word-deafness partial. No paralysis.

Five days after admission operation was performed. Wound of the squamous portion of temporal. It was found that the knife had penetrated the dura. Dark-coloured blood extruded. Clot removed. Drainage.

Much better the same evening. Worse next morning, but on removal of drainage tube he improved.

The recorder of the case commented, "In this case, I believe the knife penetrated the superior temporo-sphenoidal convolution, traversed the fissure of Sylvius, and probably injured Broca's convolution; and that his symptoms were due to a blood-clot in the fissure of Sylvius which was breaking down."

It is quite clear that injury or pressure over the superior temporo-sphenoidal convolution would have accounted for all the symptoms of this case.

CONCLUSIONS.

1. Traumatic sensory aphasia is often, if not generally, due to subdural hæmorrhage pressing on the cortical sensory speech centres.
2. The aphasia affects the visual more than the auditory speech-centre.
3. If treated expectantly, recovery is generally incomplete, and there is a risk of epileptiform convulsions developing later.
4. Treatment by trephining and removal of the clot is the best course.