SOME OBSERVATIONS ON MASKED EPILEPSY AND THE SIMULATION OF TRAUMATIC EPILEPSY BY CEREBRAL TUMOUR, WITH SPECIAL REFERENCE TO HEAD INJURIES WITHOUT FRACTURE*

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In an analysis of 500 cases of head injury without fracture, epilepsy was discovered for the first time after the accident in 17 of them. But, in each case, cerebral tumour was either proved to be present or else strongly suggested clinically. So far as it was possible to judge by the history of the accident in the various cases, the injuries seemed to be of a trivial nature. The investigation, therefore, resolved itself into an attempt to ascertain, if possible, the relationship between head injuries without fracture and epilepsy in cases of cerebral tumour. The cases fell under the following clinical classification: (1) Those in which epilepsy was the predominating feature, and referred to here as traumatic epilepsy simulated by cerebral tumour; (2) those in which epilepsy was more or less obscured by the subjective symptoms common to traumatic neurosis, and referred to here as masked epilepsy.

I. TRAUMATIC EPILEPSY SIMULATED BY CEREBRAL TUMOUR

There was some evidence to indicate that a protective mechanism exists between a tumour and the brain, which may be either permanently or temporarily damaged after a head injury without fracture. The persistence of epilepsy on the one hand and its gradual subsidence on the other after a head injury were taken as evidence of the progress occurring in the traumatic breach. Illustrations representing each type will be given.

Epilepsy Persisting.—A timber porter, age 45, when standing, was struck in the right postparietal region by a 12-lb. wooden plank which fell flat from a distance of 10 feet. He was momentarily dazed, but after an hour continued his work. The following morning he had, for the first time, an epileptic fit and complained of headache. The fits increased in frequency, and he was sent to hospital, a week after the accident, as a case of traumatic epilepsy. On admission there were double papilloedema (1·5 D), right pyramidal signs without much weakness, and slight motor aphasia. The cerebrospinal fluid contained 0·3 per cent. protein, and globulin in excess. During the first fortnight the fits (right Jacksonian) increased in frequency and at the end of

that time he became totally hemiplegic and aphasic. Mr. Dickson Wright exposed a
growth situated in Broca’s area which at autopsy was found to be a carcinoma
surrounded by haematoma. Previous to the accident the patient was apparently in
good health.

Epilepsy Subsising.—A labourer, age 24, was kicked on the forehead when playing
football. He struggled to his feet and continued the game. The following day he had,
for the first time, an epileptic fit. For 10 years he attended a London hospital for
epilepsy and was relieved by luminal. He had been free from attacks for two years
when, at the age of 36, he fell down a manhole, not in a fit (according to an eye-
wit ness). He got to the surface almost at once and continued his work. The following
day he had a seizure, and thereafter several attacks each day for a month when he
was sent as a case of traumatic epilepsy by a firm of solicitors. He was admitted to
hospital. According to the history the fits had often begun in his left hand, immediately
after which he became unconscious. The fits observed in hospital were generalized.
A physical examination disclosed double papilledema (R. 8D; L. 4D); left hemi-
plegia (weak left face, left extensor response, without apparent paralysis). The
cerebrospinal fluid contained nothing pathological. A cystic glioma attached to the
plia over the right motor arm area was exposed and 10 c.cm. of yellowish fluid were
removed by Mr. Wakeley. Dr. Carnegie Dickson reported a section of the growth as
an astrocytoma.

II. MASKED EPILEPSY

Epilepsy was masked in 12 cases. Among the circumstances, apart
from the subjective symptoms common to trauma, which tended to obscure
the recognition of corticomotor and corticosensory fits were: (a) Complete
amnesia of the somatic experience; (b) difficulty in describing a cortical
experience, especially when it was sensory; and (c) the sudden onset of
hemiplegia.

(a) Complete Amnesia of the Somatic Experience.—Unlike experimental
epilepsy, the epileptic phenomenon observed clinically is, as a rule, composed of
two elements, viz. psychological and somatic. The psychological element
in some of my cases was so prominent a feature of the attack as to obscure
completely all recollection of the somatic element. In spite of a careful
search for evidence of cortical experiences in those who suffered from periodic
attacks of disturbances of consciousness, the difficulties in detecting epilepsy
were greater in the case of non-convulsive fits. Such terms employed by the
patients as ‘collapse,’ ‘stroke,’ ‘faints’ and ‘giddiness’ sometimes led to the
disclosure of epilepsy. If the case about to be described had been sensory
instead of motor, it would have been impossible to interpret the significance
of the term ‘giddiness.’

A gardener, age 24, was sent for recurring attacks of giddiness which followed a
blow on his forehead caused by the swinging of an iron gate. Giddiness, which
occurred in the morning, had continued for a period of one month. He gave a history
of two severe attacks of headache unaccompanied by vomiting. His appearance was
heavy but otherwise healthy. Although intelligent in answering other questions, he
was unable to provide any further information on the nature of the giddiness than:‘I just come over giddy.’ He had no idea of its duration but thought the ‘turn’
lasted a ‘short time.’ A physical examination revealed no signs of organic disease.
The optic discs were normal. When the examination was finished he was asked to
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dress, but instead of doing so, he remained lying on the couch, and there occurred a typical left Jacksonian fit which began in the arm and spread to the face and leg simultaneously. The left plantar was extensor and remained so for about five minutes. When, after he had dressed, he was asked to relate what had happened, it was impossible to elicit anything more from him than, 'I just came over giddy.' No recollection of having been convulsed was retained. A week later he was admitted to hospital. A physical examination revealed nothing abnormal. Renal efficiency tests were normal and the Wassermann reaction was negative. The cerebrospinal fluid contained 0.06 per cent. protein, and globulins were present. There was no increase in cells. After two months' rest in bed the symptoms subsided. He has been under observation for three years and the left plantar reflex has been extensor for the last three months.

(b) Difficulty in Describing Corticosensory Experiences.—In four cases the somatic experience was but vaguely retained and could only be symbolized

in a language the clinical interpretation of which presented difficulties. Thus, in addition to prolonged observation, a grim and laborious coaxing was necessary in leading the patients to symbolize their sensory experiences in language which admitted of clinical interpretation. In others the somatic element was recalled in a clean-cut and well-defined description.

A tailor, age 52, was sent because he was 'nervy and giddy' after striking his head against a mantelpiece. An examination revealed kinesthetic signs in the left hand (compass test 4 cm.). No other evidence of organic disease could be detected. There was no papilledema. He was persistent in his refusal to admit having difficulty in using his left hand, and he denied having headache. During the same week he was admitted to hospital and in a few days' time said he had recovered and wanted to return home. After a lengthy interrogation relating to his left side, he admitted this much, that his left hand sometimes went 'twiddley,' and sometimes, 'it goes funny all down that side.' After being persistently urged he ultimately symbolized the sensations.
in his head, not as headache, but scornfully by the description: 'Them ain't headache, them is hell'; and thereafter persistently denied that he suffered from headache. The cerebrospinal fluid showed 0.1 per cent. total protein, and an excess of globulin; no other pathological changes were noted. The following week Mr. Wakeley exposed the meningioma shown in fig. 1.

A tram driver, age 60, as the result of a mechanical defect in the magnetic brake control of the tram he was driving, collided with a stationary tram in front. He was thrown backwards against the window at the rear of the driving cabin, his head striking the glass without breaking the latter. He felt dazed but did not lose consciousness, and when the necessary brake adjustments were completed he was able to continue driving. The following day he began to lose confidence in himself because of giddiness. A week after the accident headache and pain behind the eyes were noticed for the first time and he was obliged to stop work. A month after the accident he was sent to hospital, where a physical examination disclosed no signs of organic disease. He appeared to be mentally retarded and was admitted for observation. The cerebrospinal fluid showed three cells, 0.06 per cent. total protein, and excess of globulin. During his first week in hospital he was observed to be drowsy on some occasions. A month after his admission he complained of unpleasant smells in the ward which lasted from 15 minutes to an hour. He was unable to give any information as to their nature. Three months after his admission papilloedema appeared in the right eye and within a fortnight the left eye was similarly affected. He complained
of 'numb feelings,' but, although he said they were present on one side only, he differed from time to time as to which side was affected. Papilledema increased to 5D. Mr. Tanner performed a left subtemporal decompression; the brain was found to be under great tension.

The following case illustrates the unmasking of an uncinate fit, perhaps by the added impressions consequent upon new experiences.

In brief, a woman, age 56, was stunned by a toss from her horse. She finished the day's hunt. The following day she felt 'headachy, nervy, and a sort of burny feeling in my nose.' Not having quite recovered her former health after six months, she made Africa her destination for a complete change. While she was there she 'kept on smelling a sort of smell like burning natives,' and since her return to England this sensation has periodically recurred. She was vague about its duration and frequency. Sometimes she said 'I have numbness in my hand or leg.' When asked which side she replied 'What difference does it make, it lasts such a short time.' But when she referred to the numbness, she invariably looked at the palm of her left hand, which she stroked with her left thumb. A diagnosis of tumour has not yet been substantiated in this case.

A school teacher, age 45, was admitted to hospital after slipping on the floor and striking the back of her head. On examination no physical signs were detected. She had headache and sickness in the morning. When questioned as to her sensory experiences she disclosed the fact that periodically there was a sudden appearance of 'white nuns or groups of nurses dressed in white' to the left of her bed. When further questioned she said that if she believed in ghosts she would think they were ghosts. The patient was not decompressed but the brain at autopsy was found to contain the tumour with hemorrhage as shown in fig. 2.

(c) The Sudden Onset of Hemiplegia.—Difficulty was sometimes experienced in discovering the sequence of events centring round an accident. Mental phenomena following in the wake of trauma in general were sometimes of such a nature as to produce a degree of introspection. Thus, symptoms hitherto indefinite or disregarded by the patient may have become a prominent source of worry. Preexisting disease was, therefore, sometimes in a double sense discovered by accident, and the aetiological relationship between the head injury and the epilepsy was for a time left in doubt.

The sudden onset of hemiplegia with unconsciousness or a state of coma lasting hours or days masked the existence of epilepsy in six cases of occipital tumour, and in one case of a cyst in Broca's area. In all these cases there was an excess of protein in the cerebrospinal fluid beyond the amount usually found in thrombotic lesions. With or without loss of consciousness the somatic expression of an occipital fit, in some of my cases, was characterized by repeated attacks of blindness lasting from seconds to minutes. But the psychological reaction preceded and succeeded the somatic process.

A woman, age 64, fell on the pavement, as a result of which her left forehead was bruised and her left eye discoloured. She was unable to relate what had happened to her when she returned home a hundred yards away. The following morning she was in a comatose state and when she recovered she complained that she had lost her sight for a short time. The same week she was seen by a local ophthalmologist who diagnosed detachment of the left retina and lens. A fortnight after the accident she appeared to
have recovered, but she complained of periodic attacks of loss of sight and her relations noticed that she was at times so drowsy that she could not be roused. One evening, two months after the accident, she 'collapsed' in her home. This was followed in the morning by headache and vomiting which continued on and off for three days. On her admission to hospital there were left homonymous hemianopia and loss of postural sense on the left side. No other organic signs were discovered. The retina and lens appeared to be normal. The cerebrospinal fluid showed 0.1 per cent. protein, and slight excess of globulin, but no other abnormal changes. She frequently complained of 'blackness before my eyes when I feel awful.' Headache was not once complained of and she denied its existence. A fortnight after her admission she was

![Fig. 3.](image_url)

A bricklayer, age 55 years, was discovered unconscious on a scaffold 20 feet above the ground. An accident was inferred, but details were entirely lacking. He was taken to an infirmary nearby, where he recovered in three days and was sent home. A fortnight later he went into the country to recuperate and remained there for three weeks. When getting off the train at Paddington on his return home he 'collapsed' and thereafter was regarded as having lost his memory. Three months later he was sent to hospital as a case of 'loss of memory following an accident.' An examination revealed sensory aphasia. It was difficult to obtain his interest, because with one hand covering his forehead he kept repeating 'Oh dear.' There were no pyramidal signs. The sensory system could not be tested. When the light of an ophthalmoscope torch was placed at a good range to his left, his gaze invariably moved at once towards the light; but this never occurred when the light was placed
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...to his right. It was only when the light moved from the right inwards as far as to reach the nasal field that his gaze was directed to it. He was admitted as an in-patient. The cerebrospinal fluid contained 0·2 per cent. protein, and globulin in excess; all other tests were negative. A fortnight after his admission Mr. Tanner performed a left occipital decompression, and exposed a large tumour which extended forwards into the sensory area. A section of the growth showed carcinoma.

Of the remaining cases, the following presented the most interesting feature. A right-homonymous-hemianopic and partially sensory-hemiplegic patient, with a history of several hunting accidents and what had been diagnosed as two attacks of cerebral thrombosis, presented twice during a period of observation the following fit: his eyes rolled up and slightly to the right in tonus, then with head slightly flexed forwards the lids went into momentary clonus. The clinical picture somewhat resembled the reaction of the upper lids to the removal of a bit of grit or smoke blowing into the eyes, but there was in addition a total lack of conscious expression in his face for over one minute. The convulsive part of the attack, on both occasions, lasted about five seconds. The patient had no recollection of its occurrence, but, although uncommunicative on the subject, he was able to recall that 'everything went black.' When the nurse was asked if she noticed what had happened she replied: 'Oh yes, he is always getting neuralgia in his eyes like that.' Thereafter it was possible with the help of members of his family, who regarded this sign as a 'disagreeable habit,' to trace the existence of these attacks for a period of five years at least. Mr. Tanner and Mr. Dickson Wright exposed but did not remove a large left occipital tumour.

A male, age 38 years, was admitted to hospital with complete right hemiplegia and motor aphasia. 'Yes' and 'No' were the only words he could utter, not always relevantly. There was no papilledema. Pyramidal signs were present on the right side and there was marked loss of sense of position when he attempted to touch any part of his paralysed side with his left index finger. Previous to his admission he had remained in bed in a London hospital for two months, where, according to the notes supplied, his case was diagnosed as one of cerebral thrombosis. The cerebrospinal fluid was found to contain 0·06 per cent. protein, excess of globulin, and 12 lymphocytes. After his admission his wife supplied the information that seven years previously he met with an accident on his bicycle, as a result of which he remained in a local hospital for three weeks with concussion. Shortly after the accident he would at times lose the power of speech for a few seconds, and she added that his present state began with an epileptic fit which 'left him paralysed on the right side, and speechless.' In view of the excess of cells in the cerebrospinal fluid an abscess was suspected and Mr. Colledge decompressed over Broca's area. A cyst, from which 2 oz. of fluid were removed, was there found attached to the pia. There was no abscess.

CRANIOCEREBRAL REACTION TO VIOLENCE

A short reference to the history of the reaction of the skull to violence is perhaps worthy of note. Hippocrates observes: 'For what is pressed inwards is broken off from another bone which continues in its natural state, and the "esphlasis" must necessarily be accompanied by a fissure.' Celsus says: 'Sometimes, though rarely, it happens that the bone remains sound and entire, whilst in consequence of a blow some vein ruptured in the membranes of the brain discharges some blood internally, which, stagnating there, excites some violent pains and in some produces blindness.' This, by the way, is the first recorded reference to subdural hæmatoma. He further states: 'If the fissure is not manifest, ink is to be laid upon the bone and
afterwards abraided by a rugine, in which case the fissure, if there be any, will retain part of the black substance.' James, in presenting the views of his time, remarks: 'It appears from geometry that among figures of equal perimeters the circle comprehends the greatest space. But the figure of the cranium is nearly spherical and therefore if it be pressed inwards its capacity must be diminished. Whether, therefore, the convex figure of the cranium be changed by intropression without fracture, or the fractured bone be forced from its place, the effect will be the same, which is compression of the brain.' The observations of Wilfred Trotter mark a new era in the advance of knowledge of head injuries. Trotter regards the skull as an elastic structure capable of being distorted in part or as a whole as the result of a blow on the head. He emphasizes the fact that the absence of fracture is not evidence that the skull has not been considerably distorted and the brain seriously damaged.

**PERSONAL EXPERIMENTS**

The elasticity of fresh skulls representing eight decades was tested as follows. The sawn rim of the vault of the skull to be treated was fixed on a block of lead 8 in. in diameter and 4 in. thick. A glass tube 36 in. long and 3/4 in. in diameter was, by means of an iron stand fitted with adjustable brackets and clamps, firmly held vertically over the vault and just free from touching it. A magnet connected to a galvanic battery was fixed inside and at the upper end of the tube (fig. 4). Balls of hard steel, of various weights ranging from 14 gr. to 1 gr., were at different times fixed to the magnet and released from it by means of a switch on the battery. The balls after impact on the skull rebounded inside the tube and the first or maximum of the particular series of rebounds was measured by means of an adjustable steel furl on the tube. Balls of all the weights used rebounded. The average maximum rebound of a 7 gr. ball was 12 in. However, in view of the curvature of the skull producing deflections of the ball against the inside of the tube, and of other difficulties in providing constants, the test was satisfactory only in so far as the balls always rebounded. The rebound was less from the skulls of the first decade than from adult skulls. When the balls were allowed to fall on the plastic lead base they remained where they fell without rebound.

Elasticity is that property by virtue of which an object recovers its original shape and dimensions when the straining force is withdrawn. The recovery from the strain on the skull, produced by the impact of the ball, is implied in the rebound of the ball and is explained as follows. When the ball comes into contact with the skull, a mutual action, consisting of a pair of equal and opposite forces, takes place between the two bodies. Of these one acts on the ball, the motion of which is changed by the skull; the other acts on the skull which moves against its inherent resistance or stress. At the instant the two come into contact (a) a sound is heard due to oscillations;
and (b) the skull is strained, and begins to sink, and continues to sink through a space through which resistance of the skull is overcome by the ball. Thus, certain changes of figure and dimensions are produced in the skull. Because of its great hardness, deformity of the ball itself is negligible. The resistance of the skull to deformation increases from the instant the ball falls on it; it begins by being zero and increases until the velocity of the ball is wholly destroyed. For an instant the ball and the skull are at rest. Then the skull returns to its original form and by so doing tosses the ball from its surface.
The total height through which the ball falls is therefore 36 in. plus the amount of sinking of the skull at the point of impact, that is the space through which resistance is overcome. The amount to which the skull is capable of sinking within the limits of its elasticity is not known. It is probably very considerable.

**CONCLUSIONS**

In head injuries the skull is by no means fixed. Among other joints the craniovertebral joint permits the head to be pushed out of the way as a first defence against the full force of the wounding load on the skull itself. Yet even the smallest load (a ball of hard steel of the lightest weight) which acts on the skull produces an alteration of dimension or form.

An object falling on the head falls in a straight line; then, if the injury is non-penetrating, the skull itself becomes the wounding body of the brain. However, instead of being a moving body going to the brain in a straight line, the circumference of the skull is constrained to injure the brain in the circumference of a circle. In the cases referred to, the tumours were situated in the cortex in parts remote from the head injury.

Into what shape then is the skull thrown when it is deformed by a head injury? An answer to this question resolves itself into a geometrical conception of the shape assumed by a spherical body which is elastic and which when strained by a blow begins to sink, and continues to sink through a space through which its resistance is overcome. Sinking of the skull will tend to diminish the intracranial capacity and so to crush the brain as a whole.

It is, therefore, suggested that when a cerebral tumour also occupies the brain-containing space, a head injury will tend to crush the intracranial contents beyond the amount due to the sinking of the skull alone acting on a normally contained cavity. Thus, in cases of cortical tumour, it would appear reasonable to regard a head injury, *however slightly it acts on the skull*, as being a possible mechanical factor capable of precipitating the onset of epilepsy.

**REFERENCES**

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Some Observations on Masked Epilepsy and the Simulation of Traumatic Epilepsy by Cerebral Tumour, with Special Reference to Head Injuries without Fracture

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