PSYCHIATRIC ASPECTS OF HEAD INJURY.

By E. GUTTMANN, M.D.Munich, L.R.C.P.E.

In the past head injuries have been of comparatively little concern to the psychiatrist. As judged from statistics of admission to mental hospitals they are a small problem. Mapother (1937), for example, who took a very broad view of the causal connection found in L.C.C. mental hospitals—3 per thousand in observation wards, 4 per thousand in psychiatric O.P. depts. in general hospitals, 7 per thousand of the admissions attributable to head injuries. The increasing interest that psychiatrists take in head injuries at present is mainly due to the increasing amount of extramural work that they are being called on to do. In general hospital and outpatient practice they are bound to encounter a considerable number of problems connected with head injuries, both in the acute and in the later stages. The psychiatrists and neuro-psychiatrists in the Services have probably had this experience first, but when adequate provision of psychiatric service becomes available in the general hospitals, many cases will come into the orbit of psychiatrists which hitherto have been “disposed of” without much regard for their mental aspect.

PATHOLOGY.

The morbid anatomy of cerebral injury in general is not the subject of this review. The reader can be referred to A. Meyer’s chapter in this volume, and to the “General Pathological Considerations” by G. B. Hassin in Brock’s recent book (1940) on Injuries to the Brain and Spinal Cord.

CLINICAL CLASSIFICATION.

The standard Nomenclature of Disease has the general heading “Psychosis due to trauma (traumatic psychosis)” with the following sub-headings:

- Traumatic delirium.
- Post-traumatic personality disorder.
- Post-traumatic mental deterioration.
- Other types (to be specified).

Considering this inadequate, Bowman and Blau (1939) propose the following classification:

A. Primarily traumatic mental disorders (mental disorders due primarily to head trauma):

1. Acute.
   - Concussion syndrome.
   - Traumatic coma.

...
II. Subacute.

Traumatic delirium.
Traumatic Korsakoff.
Other types (to be specified), e.g. stupor, apathetic syndrome, twilight state, mixed.

III. Chronic.

Post-traumatic personality disorders:
(a) Of adults.
(b) Of children.

Post-traumatic mental defect conditions:
(a) Special (to be specified), e.g. memory, aphasia, alexia, amnesia.
(b) Generalized.

Mixed type.

Punch drunk.
With traumatic convulsion disorder.
Other types (to be specified).

B. Secondary mental disorders (mental disorders associated, precipitated, or complicated with trauma, but not due primarily to head trauma):

I. Psychoneurosis (type to be specified) with head trauma (e.g. conversion hysteria, anxiety state, terror neurosis, malingering, etc.).

II. Psychosis (to be specified) with head trauma (e.g. general paresis, psychosis due to alcohol, psychosis with cerebral arterio-sclerosis).

III. Mental deficiency (amentia) with head trauma.

Davidoff and Hoch (1940) classify the mental states caused by head trauma as follows:

(1) The acute stage.
(2) The subacute or Korsakoff type.
(3) The chronic state.
(4) The mixed type.

1 and 2 are also called post-concussion syndrome. The chronic states are divided into:

A. Post-traumatic mental deterioration or defect where organic factors predominate, and where the previous personality plays a relatively lesser role in the symptomatology.

B. Post-traumatic personality disorder. The previous personality is a relatively greater factor in determining the course.

C. The combined type.

D. Traumatic neurosis.

The personality disorders (b) are divided into the milder form—vaso-motor changes, lack of concentration, fatigue, emotional instability, unreliability and irritability—and the severe form with delusions and hallucinations, as well as definite antisocial and asocial behaviour.
The Brain Injuries Committee of the Medical Research Council (1941) has issued a glossary of psychological terms commonly used in cases of head injury. Desirable as it seems to have a uniform terminology, the definitions proposed in several instances differ so much from the common usage of the terms employed that it must be feared they will not find general recognition. They will be quoted at the appropriate points in the following chapter. Symonds (1942) has given some useful explanations and comments.

**Symptomatology.**

The psychiatric symptoms which develop after head injury are well known and can be found in text-books. Numerous articles published just before or at the beginning of the war reviewed them for the practitioner.

Symonds has given the most comprehensive and up-to-date description in Brock's monograph (1940). The Brain Injuries Committee of the M.R.C. made a valuable contribution by publishing definitions of the most commonly used terms, thus attempting to combat the well-known difficulties due to the use in psychiatry of an ill-defined nomenclature.

According to this glossary, "Concussion is a state of unconsciousness or impaired consciousness, however fleeting, suddenly produced by mechanical force applied to the skull, and usually followed by retrograde amnesia." This official definition, if accepted, will do away with the confusion arising from the fact that some used the word concussion for the mechanical act, instead of, or in addition to, the psychological effect. However, retrograde amnesia is not common in milder cases, and moreover it is difficult to assess owing to the circumstances of the accident. One would prefer "retrospective amnesia," and it might be suspected that it is this that was actually meant. In the glossary a state of absolute unconsciousness is called coma, and this is characterized by the absence of all psychologically understandable responses to stimuli or inner needs. In the absence of psychological response, the intensity of the disturbance (often called the depth of the coma) should be characterized by the presence or absence of neurological phenomena, such as swallowing, pupillary and corneal reflexes, plantar responses, etc (Glossary). Semi-coma describes a state in which psychologically understandable responses are elicited only by painful or other disagreeable stimuli. If one wants to be precise, the emphasis has to be on the "psychologically understandable." Movements of limbs after pinprick need not have psychological meaning.

On recovering from unconsciousness the patient passes through a state of clouded consciousness (unless he wakes up suddenly). This state of clouded consciousness, unfortunately, is called confusion in the glossary.*

The glossary distinguishes three degrees of clouded consciousness: "Severe" describes a state in which the patient, though for the most part inaccessible, will occasionally show adequate response to simple commands forcibly given. "Moderate" signifies a state in which the patient, though out of touch with his surroundings, can be got to give relevant answers to simple questions. "Mild"

* This usage leads to formulations like the following: Prolonged confusion is common after severe closed head injury. . . . Associated injuries may lead to confusion in diagnosis (Ritchie Russell, 1942).
finally is a state in which the patient, though presenting the characteristic features of confusion (i.e. clouded consciousness) to some degree, is capable of coherent conversation and appropriate behaviour. The general characteristics of clouded consciousness are "impaired capacity to think clearly and with the customary rapidity, and to perceive, respond to, and remember current stimuli; there is also disorientation."

The glossary defines two other expressions which apply to the acute stage, viz. delirium and stupor. Traumatic delirium is a state of much disturbed consciousness with motor restlessness, transient hallucinations, disorientation, and perhaps delusions. The restlessness of the unconscious (comatose) state with its extra-pyramidal hyperkinesis and pseudospontaneous movements must be distinguished from the activities of the delirious patient who acts in keeping with his dreamlike mental experiences (Curran and Guttmann, 1943). Traumatic stupor, according to the glossary, is a state in which the patient, though not unconscious, exhibits little or no spontaneous activity. As in general psychiatric usage, stupor means absence or reduction of spontaneous activity not due to clouded consciousness (Curran and Guttmann, 1943), there seems to be little reason to describe the state in question otherwise than as aspontaneity or lack of initiative.

"Traumatic automatism," according to the glossary, is a state in which the patient, though capable of responding normally to his immediate environment, subsequently has amnesia for the period in question. In this state there is always some degree of confusion with disorientation. In other words, automatism is used to describe the mildest form of clouded consciousness. This usage was introduced by Symonds, who describes six stages of recovery from concussion, with automatism as the last. His description of the six stages may conveniently be given here:

- **Stage 1.** Complete paralysis of cerebral functions, which may extend for a moment to the vital centres.
- **Stage 2.** Coma. Flaccid paralysis, abolition of reflex function. Pulse and respiration have returned.
- **Stage 3.** Stupor. Reflex functions return; one observes movements of simple purposeful type such as withdrawal of a limb from a painful stimulus. The patient may respond to forcible command, but is otherwise unconscious of his surroundings.
- **Stage 4.** Restless confusion; the patient is resistive and often violent. Voluntary movements and speech return, but without control or direction.
- **Stage 5.** Mental confusion. The patient's behaviour is quieter and more controlled, his speech more coherent.
- **Stage 6.** Automatism. The patient responds naturally to simple questions and performs accustomed actions in an orderly and effective way. He is still dazed and imperfectly aware of his surroundings; he repeats the same question. He has no memory of this phase in retrospect.

Symonds (in Brock, 1940) assumes that this same sequence of events is to be observed in a case in which the process of recovery occupies a period of days, and also in a case in which recovery is complete within a few minutes. All these stages are covered by the retrospective (post-traumatic) amnesia, and, in Symonds' view, it is only the duration of these phases which varies with
the severity of the "general contusional" effect. It is the logical outcome of this view to employ the duration of the amnesia as a measure of the severity of this effect. This, in fact, was proposed originally by Ritchie Russell (1938) in his classical paper on the exclusively practical consideration that it is easier and more reliable to assess amnesia than a disturbance of consciousness, regarded as equivalent to the memory gap. Although this theoretical assumption is debatable (the amnesia covers the Korsakoff stage of recovery, during which the patient may be quite lucid), the method of using the length of the post-traumatic amnesia as a measure of the severity of the injury has found wide practical application (Ritchie Russell, Symonds, H. Cairns, E. Guttmann, etc., etc.). It is practically the only one that lends itself to statistical comparison. It would be desirable to reach agreement about the classes to be used; most writers divide their cases into five groups: Post-traumatic amnesia up to five minutes (o); up to one hour (A); up to one day (B); up to one week (c); and longer (p); but other divisions are employed, and even the same writer is not always consistent in the use of these classes. There is agreement that "severity" in such comparisons refers to the general concussive effect only; the surgical severity of each case must be judged independently (laceration, compound fractures, cranial nerve injuries); and localized cerebral injury needs separate assessment. The general concussive effect of high-velocity impact on a small area of the skull is very small (for example, in gunshot wounds), and it is therefore not paradoxical that these cases would not appear to be "severe" if measured in terms of post-traumatic amnesia. The amnesia is a satisfactory measure of the "concussion," and therefore of practically all cases of blunt injury. But this method is a simplification for practical purposes; to be exact, each case of head injury should be assessed as cranial, local cerebral, and general concussive injury, to which may be added the psychological trauma and the social-economic significance.

For clinical psychiatric purposes, E. Guttmann (1942) suggests a simple classification. He suggests calling "mild" those cases which wake up without a transitional period of clouded consciousness, and "severe" those which pass through an amnesic-confabulatory (Korsakoff) stage. When the patient gradually wakes up, going through a stage of clouded consciousness only, he speaks of moderate severity. This classification is useful only for patients observed at the time, not in retrospect, and it does not assume that all cases necessarily pass through the same stages.

Little has been published on the psychology and psycho-pathology of the post-traumatic memory disturbances. Symonds (in Brock, 1940) has called attention to the shrinkage of the retrograde amnesia, and E. Guttmann (1942) has pointed out that this observation is important in regard to the theory of this puzzling phenomenon. Disturbance of recall at the time of assessment is one of the factors to account for an apparent gap in the past memory. It would seem preferable not to call amnesia the failure to recall recent events (such as observed in Korsakoff states, traumatic or otherwise), and to speak about retrograde amnesia only after the function of recall has settled down again. Post-traumatic amnesia only can be assessed in retrospect. Delirious episodes may or may not occur during the recovery from severe
head injury. Guttmann and Winterstein (1938) have pointed out that they are never observed after knock-outs in boxers. Curran and Guttmann (1943) attribute them to intercurrent infection, excessive loss of blood, respiratory or circulatory embarrassment, and the possible significance of a constitutional element or chronic intoxication is mentioned. Povitskaya (1942) points out that cerebral trauma sustained during alcoholic intoxication leads to greater restlessness and resistiveness, and this, as well as a history of chronic alcoholism, is clearly correlated statistically with a higher incidence of delirium, resembling delirium tremens. All mental symptoms are more highly coloured in alcoholic subjects; in non-alcoholics delirium is rare. Nightmares, horror, are almost a rule after concussion, irrespective of its seriousness.

Eichler (1939), on the other hand, described cases of Korsakoff psychosis with manic affect and grandiose ideas, combined with disturbance of pupillary reactions and absent tendon reflexes in which there were no predisposing factors such as alcoholism, or syphilis, and no demonstrable constitutional element.

T. C. Graves (1938) drew the attention to certain groups of cases of head injury followed by mental symptoms in which treatment of chronic infective processes (in the teeth, nasal sinuses) were followed by considerable improvement of mental symptoms.

The neurological symptoms and signs cannot be dealt with here, but a few borderline items may be quoted.

M. Scott (1940) demonstrated three cases of stupor following head injury due to a subdural collection of clear fluid—so-called subdural hygroma. After evacuation of the fluid the patients recovered. This type of lesion should be suspected in every case of head injury in which the normal tendency to recovery is not shown after proper measures for the relief of raised intracranial pressure and oedema. The treatment is opening by trephine or burr.

Bay (1939) pointed out the importance of olfactory examination, as anosmia is not infrequently the only objective sign of intracranial damage after head injury. Leigh (1943) encountered 72 cases with impaired sense of smell among 1,000 consecutive cases of head injury. This sign may follow violence to any part of the head, but frontal and occipital injuries are the most common. A head injury which causes defects of smell is generally a severe one.

E. Stier (1938) found loss of libido in 33 cases (? out of how many) of head injury of varying severity: in half of the cases—all over 40 years of age—there was also loss of potency. But Fleck (1939) pointed out that these disturbances of sexual functions occur only in cases of cerebral contusion, not in cases of simple concussion.

Witzelsucht does not occur in all frontal injuries (Bostrom, 1940). It has localizing diagnostic value only if it occurs within the framework of a changed personality. The latter usually consists in extraordinary levity with regard to all the problems of life, and excessive indifference towards the patient's own defect. The patient is incapable of emotional orientation. The lack of spontaneity as observed after frontal injuries is often difficult to distinguish from psychically conditioned inhibition.

Headache is probably the most important symptom after head injury. Its
omnipresence is generally taken for granted. However, E. Guttmann (1940b) who made a systematic study of its occurrence, found that less than 50 per cent. of his patients—a consecutive series of head injuries admitted to an accident ward—complained of headache when waking up from unconsciousness. Only 20 per cent. had headache when leaving hospital. Cases with short amnesia were more liable to complain of headache than those with long amnesia, and the age of the patients did not make much difference in the frequency. When followed up three and six months after the accident, only 20 per cent. of the patients complained of headache, mild or severe, and this figure included all headaches occurring in the follow-up period, some of which were probably due to other causes.

Rowbotham (1942) examined 500 cases suffering from the effects of injury to the head and found headache in 50 per cent. of them, but these cases were probably a selection of bad risks.

Dereux (1939) described the post-concussional syndrome as the "subjective syndrome." From varying authors he gathered figures about its frequency, varying between 95 and 50 per cent.

The syndrome of headache, giddiness and nervous instability (minor contusion syndrome) has, according to Symonds (in Brock, 1940), doubtless an organic basis. The symptoms constituting it are in the main symptoms of instability rather than loss of function, and they may occur, not only in the course of recovery from severe injury, but also as the more immediate effect of slight injury without disturbance of consciousness.

The onset of any or all of these symptoms may be delayed. After brief loss of consciousness, for example, the sufferer may appear to have completely recovered in a few hours, yet in the course of a few days may begin to complain of headache, giddiness, or undue fatigue of body and mind. Occasionally it may be a week or more after the accident before the patient seeks advice on account of such symptoms, and he may have continued at his work during this time. The same may be true in the case of a man who has not lost consciousness at all, but has sustained a bruise or laceration of his scalp. This latent interval has often been attributed unjustly to the effects of a compensation neurosis, but it is in fact observed quite commonly in cases where no such possibility exists.

Many of the patients who suffer from headache and giddiness complain also of symptoms which Symonds groups under the heading "Nervous instability." Most frequent is an undue liability to fatigue, both mental and physical. There may also be intolerance of noise and light (apart from the aggravation of headache by these causes); insomnia; anxiety; and depression. The patient's spontaneous complaint is usually that of nervousness. Doubtless these symptoms are partly, often largely, due to psychogenic causes. They cannot, however, always be ascribed to the memory of the accident, for that is often blotted out by the retrograde amnesia; nor, as Ritchie Russell (1932) has pointed out, are they by any means confined to compensation cases. They are most common in persons with a family and personal history containing evidence of neurotic predisposition, and are also dependent upon the degree of vexation and distress occasioned by the circumstances arising out of the
accident. They are more apt to occur in elderly and arteriosclerotic than in young and healthy persons.

The patients who exhibit these symptoms of minor post-traumatic mental disorder may present no measurable evidence of organic damage. Formal tests of memory, power of calculation, or reaction-time may show no defect. It is difficult, however (according to Symonds), for anyone who has studied a large number of these cases to escape the conclusion that the organic factor is present.

Experience of the more severe effects of brain injury shows that the highest levels of cerebral function are the most vulnerable and the last to recover. Close observation of the symptoms which are described under the term "nervous instability" has convinced Symonds that they are in part due to a disturbance of function at these highest levels, as a physical effect of the trauma. As a result the sufferer is not himself. His best qualities are in abeyance, revealing the second-best. He is slightly less of a man, and more of a child. Hence there is a tendency for his reactions to inner need or external stress to be inadequate or neurotic. The greater the stress, as in the case of a workman with dependants, and without reserves of capital, the greater the liability to neurosis.

E. Guttmann (1943a) has also found the importance of psychological and social factors in the causation of late symptoms. Observation in young soldiers with no apparent predisposition brings out one point not mentioned by Symonds, i.e. the precipitation of a neurosis by an injury in a person who had been under psychological stress before.

In Schaller's view (1939), the post-traumatic concussion state produces reversible changes of brain function which, in severe cases, may become irreversible, with demonstrable pathological change. The post-traumatic psycho-neurotic state is characterized by fear, suggestion and wishful thinking; it is due to the precipitation of psychic complexes, following a period of meditation, in patients presenting inadequate personality traits and subjected to adverse mental influences. In comparing 100 cases of each group, he finds (among other differences) headache in 77 of the encephalopathics, as against 97 in the neurotics. Of the former, 30 per cent. went back to work in spite of symptoms, of the latter only 6. He gives the differential diagnosis of the major symptoms in the following table (slightly modified):

<table>
<thead>
<tr>
<th><strong>Encephalopathy</strong></th>
<th><strong>Neurosis</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Wishes to work.</td>
<td>1. Does not wish to work.</td>
</tr>
<tr>
<td>2. Euphoric, aggressive, periods of explosive irritability.</td>
<td>2. Depressed, emotional, complaining.</td>
</tr>
<tr>
<td>3. Amnesia of injury; memory and concentration difficult.</td>
<td>3. Mentally alert.</td>
</tr>
<tr>
<td>4. Changes from original personality make-up.</td>
<td>4. Aggravation of inherent personality defects.</td>
</tr>
<tr>
<td>5. Often severe injury, followed by unconsciousness.</td>
<td>5. Frequently slight injury.</td>
</tr>
<tr>
<td>6. No hysterical symptoms and signs.</td>
<td>6. Hysterical symptoms and signs.</td>
</tr>
</tbody>
</table>
Encephalopathy.

7. No exaggeration in statement and behaviour.
8. Tendency to improvement.
10. Constant and precise symptomatology.
11. Headache frequently absent.
12. Dizziness, vertigo.
13. Intolerance to heat and alcohol.

Neurosis.

7. Exaggeration.
8. Tendency to aggravation.
10. Multiplicity, changeability and indefiniteness of symptom.
11. Headache rarely absent.
12. Dizziness, giddiness.
13. No such intolerance.

Lewis and Jones (1941) have shown how frequent headache and dizziness are in effort syndrome, and Lewis (1942) found mild headache, dizziness and fatigue equally often in neuroses of mixed type as in post-concussional neuroses. Only severe headache was more frequent in the latter.

According to Symonds (in Brock, 1940), total disability due to permanent mental incapacity of the organic type is a rare consequence of generalized brain injury. But though total disability is rare, residual symptoms of mental disorder belonging to the organic reaction type are by no means uncommon. In cases of moderate or severe injury these symptoms may be very slow to recover, so that it may be a year or more after the accident before the patient has regained his normal mental state. In some cases recovery is never complete. In the intellectual sphere there may be slight but permanent impairment of memory, especially for recent events, impaired judgment, and insight may be imperfect. The patient may consider himself normal, though it is apparent to his intimates that he has never been the same man since his accident. An increased liability to fatigue over mental tasks is an almost constant feature of the syndrome, as also an increased susceptibility to the effects of alcohol. The tolerance for alcohol is one of the most delicate tests of the degree of recovery.

These defects of mental function are of relatively small consequence in a manual labourer, but they may so reduce the capacity of the intellectual worker that he is permanently unfit to return to his business or profession.

There is no constant relationship between the degree of residual mental defect and the severity of the generalized brain injury as measured by the duration, either of the period of unconsciousness, or the post-traumatic amnesia. There is a general correspondence, but with striking exceptions. Apart from irritability, changes of mood and general disposition are rare in adults, though previous personality traits may be exaggerated. In children, on the other hand, changes of behaviour may be a striking symptom of cerebral injury (Blau, Tramer, Northfield). This aspect is dealt with in the chapter on Child Psychology.

The diagnosis of intellectual deterioration following head injury is only one aspect of the diagnosis or of the measurement of dementia in general. M. B. Brody (1942) has recently published several important contributions to this problem, and he has worked out an abbreviated technique for measuring deterioration by means of a "discrepancy score," which follows the lines of the Babcock Test. He uses the 1916 Stanford Binet Vocabulary Test applied
with his own short technique (Brody, 1941). Secondly he uses tests of opposites, general information, substitution, sentence repetition, and uses the discrepancy between the second set of tests and the vocabulary score as a measure of deterioration. His test is simple and short in application and scoring, and it requires no elaborate apparatus. It has not been tried out yet in cases of head injury, but it seems promising for this purpose. The same is true of the Shipley Hartford Scale for measuring intellectual impairment. Another technique worth considering is the application of the Wechsler Intelligence Test. The author himself is very reserved in his expectations as regards this use of his test. He considers the task of measuring mental deterioration quantitatively as far from easy:

"While we cannot as yet use mental tests for obtaining the person's deterioration quotient or even express his loss of ability in terms of such and such per cent., we can use tests to determine relative degrees of deterioration in a rough way. Various studies have proved that individuals with mental impairment do much better on certain tests than they do on others, and it is possible to compare various disease entities with regard to degrees of impairment in individuals suffering from them." The author finds that the following tests out of his battery hold up with age: Information, comprehension, assembly, picture completion (vocabulary). Tests which do not hold up with age are: Digits forward and backward, arithmetical reasoning, digit symbol test, block design and similarities. His table of comparison between various clinical groups shows that both organic brain diseases and psycho-neuroses score higher on verbal than on performance tests. His table 31 (page 149) also shows little difference between neurosis and organic brain disease. In other words, one cannot expect much help in the differential diagnosis of the later stages of head injury from Wechsler's battery of tests.

Nadel (1938) examined a comparatively small number of cases with organic disease of the left or of both frontal lobes, and compared them with a similar number of cases of organic disease located elsewhere in the nervous system. He used a battery of tests, including Koh's Block Designs, a colour-sorting test, Rorschach and others, and analysed the results with regard to the quality of the behaviour rather than numerically. The frontal cases lost the abilities which the author calls abstract behaviour, i.e. the ability to devise a course of action and to vary the approach to a given problem. But when the procedure was altered so as to require no choice of behaviour, they would succeed with the problem. Such a behaviour is called "restricted" or "concrete."

Steinberg (1941) studied in detail 37 cases of head injury in a mental hospital. He found deterioration—as determined by the Babcock method—with the same frequency in cases with and without fracture of the skull. Factors of prognostic significance with regard to deterioration were found to be the patient’s age at the time of injury, the occurrence of convulsions and of paranoid reactions. Most of his cases showed a progressive deterioration, in particular those with a prolonged psychotic syndrome following head injury.

Brussel (1942) studied the difference in response of the neurotic and the organic post-concussion case to the Rorschach test. He described the following signs as characteristic of the neurotic:
1. The number of responses is not more than 25.
2. The number of human movement responses is not more than 1.
5. Shading shock occurs.
6. Refusal or rejection of more than one card.
7. More than 50 per cent. pure formal response.
8. The percentage of animal response is greater than 50.
9. More than one formal colour response.

These neurotic signs are contrasted to those described by Piotrkowski as characteristic of the organic case. In addition to the usual method he uses a graphic method which has not been described yet in detail. The significant performance of the organic patient in this modification of the test is:

1. Attempt to include details irrelevant to the concept, losing relation to the whole.
2. Totally indiscriminate use of the blot to include every detail.
3. Intense effort toward precise reproduction.
4. Automatic colour matching.
5. Absence of addition pertinent to the concept.

The performance of the neurotic is characterized by the omission of details without disturbance of the integrity of the whole, and by the inability to make supplementary additions.

Using both these methods the author reached nearly 100 per cent. agreement with the clinical diagnosis.

Halstead (1939, 1940) studied cases of local (operative) cerebral injury by, means of an elaborate sorting test.

A tray with 62 articles of everyday use was shown to the patients and the range of the patients' interest and familiarity with the objects assessed. The patient was then asked to place together those objects which seemed to him to belong together. His immediate recall of the objects used was tested next, and finally his capacity for abstracting was assessed by asking him why he placed the objects together in the manner he did, and whether he could think of another way of grouping them. There were also some objects added to or taken away from his groups in order to find out whether he was able to reach the level of abstraction.

Normal persons and patients with lesions outside the frontal lobes recalled a greater number of the test objects which had been grouped than of those which had not been grouped. This effect of grouping was absent in frontal lesions. Frontal cases also showed paucity of categories, the tendency to produce groups of one object, and a marked resistance in shifting to alternate ways of grouping. The disturbance of abstraction thus indicated was present even in cases in which the post-operative social adjustment was good, and in which the formal intelligence of the patient was unaltered.

Examination of disability following a head injury should be based on the end-results of the injury, and not on the initial symptoms and signs (Kessler, 1942). The latter are to be utilized only when final examination fails to disclose evidence of pathology. In the absence of mental changes, severe head
trauma may be finally assessed 6–12 months after the initial injury. The important point to emphasize is that at the same time the symptoms of headache and dizziness are being checked, and the severity of the resulting disability in the human economy is being determined by reliable accurate tests of increased intracranial pressure. If vestibular disturbance prevents a man from walking, but allows him to sit without difficulty, regardless of the spinal fluid findings, an estimate of 50 per cent. should be made. If there is involvement of the optic disc, the disability thus caused is added to the 50 per cent., but the total is not to exceed 100 per cent.

Another method of estimating permanent disability is one in which subjective symptoms and objective signs are grouped according to their severity:

1. Cases with non-localizing symptoms.
2. Cases with localizing symptoms.
3. Cases with epileptic manifestations.
4. Latent cases.
5. Cases with late aggravation.

For the first group a rating between 5 and 20 per cent. is suggested. In the serious cases of the remaining groups, 20–50 per cent. are recommended, whilst those of an extreme character are rated at 100 per cent.

In the case of traumatic neurosis, it becomes incumbent upon the physician to evaluate the man’s permanent incapacity to work. A man who is lame in both legs may be perfectly able to work, while a neurotic may be completely incapacitated. Any system of evaluation is therefore necessarily arbitrary. Kessler’s own method is to allow a compensation for a period of one year; during that interval the patient is observed in a curative workshop, and given psychotherapy. Final awards vary from 33–100 per cent.

“Punch drunk” or traumatic encephalopathy is the result of repeated injury to the skull, as most commonly seen in professional pugilists. Apart from the neurological signs (slurred speech, disturbance of equilibrium, extrapyramidal disturbances) it is characterized by intellectual impairment, which has to be diagnosed from pre-existing mental defect. Will (1939) and Jokl (1941) have published case-histories and discussed problems of prevention.

**Precipitated Psychoses.**

Psychoses not “organic” in type following head injury are of considerable interest to psychiatrists; in particular, schizophrenic reactions after physical trauma touch on the fundamental problem of aetiology in psychiatry. Generally it can be said that research and publications are still at the stage of assessing the frequency and sorting out the available material.

According to A. J. Lewis (1942), post-traumatic psychoses have an incidence of 19 per hundred thousand of the male population. The rate rises from 10 in the 20–29 age-group, to 16 in the 30–39 group, 20 in the 40–49 group, and 25 in the 50–59, 60–69 groups. This increased incidence as age advances cannot be accounted for by an increase in accidents sustained, but must be construed as another instance of how the ageing process is itself, with its reduction of functions and loss of resilience, a very prominent cause of these traumatic
psychoses. In short it is more an involutional or presenile disorder here than a traumatic one, and the age distribution is very similar to that of presenile and other degenerative organic psychoses.

Malzberg (1937) reported that in New York State the percentage of first admissions of persons with traumatic psychoses rose from 2.1 per million of total population in 1910 to 9.5 in 1935.

Tennent (1937) studied 44 cases of psychoses following head injury admitted to the Maudsley Hospital from 1923 to 1936. All showed the typical organic syndrome in the beginning. In five cases, when the confusion decreased, hallucinatory experiences and bizarre or paranoid delusions became prominent—in other words they developed schizophrenic psychoses. Four out of these were reserved, exclusive and definitely schizoid before their injury. Two had had hallucinatory experiences before. None of the cases improved. In five others the organic reaction developed into a depressive illness. In three of them there was a history of treatment for a similar depressive illness some years previously. Two others developed a manic reaction—proof of a manic-depressive psychosis. Of the rest of the cases four recovered, two became permanent inmates of mental hospitals, and one died.

Three cases occurred in children under 16 years of age; one was a depressive who had had a manic phase before. Two showed behavior disorder and demoralization, one permanent, so that he had to be admitted to an institution as a moral defective. The other improved, but remained a problem.

The remainder were acute traumatic psychoses which recovered, but a third of the patients showed residual symptoms which interfered with their health and their ability to earn their living.

Vyner and Swim (1941) found 67 cases of post-traumatic psychoses in a resident population of 8,796 patients at a mental hospital, i.e. 0.76 per cent. An inference as to the rarity of this group is obviously not permissible, since the duration of treatment of traumatic cases as compared with, say, chronic schizophrenics was not taken into account. The cases were divided into traumatic delirium (15 cases), post-traumatic personality disorder (27 cases) and post-traumatic mental deterioration (25 cases). Twenty-six of the cases showed focal neurological signs; 18 had epileptic fits. There were 30 alcoholics among the group, and 20 patients showed signs of cerebral arteriosclerosis at the time of the injury. The pre-morbid personality was described as schizoid—exclusive, unfriendly and shy—in 18 patients, and as psychopathic in another 4. Fracture of the skull had no relation to the type of severity of the mental disturbance that followed, but patients with fracture were more prone than others to develop convulsions. Ten cases had air encephalograms done, but in 4 the ventricles were not filled (? by the lumbar route: no attempt at verification by cisternal injection or ventriculograms seems to have been made). Six cases showed moderate generalized cerebral atrophy; there was little difference in the extent of atrophy in the comparatively recent cases and those of long duration. From this it would seem (to the authors) that the cerebral atrophy is not progressive, but reaches its highest within one or two years following the injury. However, the number of cases seems too small to allow such a generalization. The same is true of the authors' statement that the
presence or the duration of unconsciousness following head injury is not related to the severity of the mental reaction.

Hoch and Davidoff (1939) studied 250 cases of post-traumatic psychoses, about half of which were complicated by alcoholism, cerebral arteriosclerosis, senility, neurosyphilis, mental deficiency, etc. The prognosis was found worse in these-complicated cases, whereas fracture of the skull was of no prognostic value. The most interesting findings of the author refer to the effect of the pre-psychotic personality type on the outcome, as given in the following table:

<table>
<thead>
<tr>
<th>Personality Type</th>
<th>Introvert</th>
<th>Extrovert</th>
</tr>
</thead>
<tbody>
<tr>
<td>Recovered</td>
<td>3</td>
<td>24</td>
</tr>
<tr>
<td>Much improved</td>
<td>3</td>
<td>50</td>
</tr>
<tr>
<td>Improved</td>
<td>11</td>
<td>41</td>
</tr>
<tr>
<td>Unimproved</td>
<td>47 (73%)</td>
<td>72 (38%)</td>
</tr>
</tbody>
</table>

With regard to the more specific problem of schizophrenia, Brill quoted Kraepelin as saying that in a very large number of cases schizophrenia is entirely independent of external conditions of life; it is the outgrowth of a constitution and cannot be produced by external causes. The first World War has amply demonstrated that the most severe hardships, the most exhaustive psychic traumas as well as the cerebral injuries have not produced any increase in the number of schizophrenics. In reply to a question about the possible precipitation of schizophrenia by trauma, Dr. Brill stated that "a patient who reacts schizophrenically is constitutionally a schizophrenic." Those who have such a constitution can get schizophrenic episodes for one reason or another—it may be due to age, stress, or accident. If a person was well past the age of 40, i.e. past the dangerous period of schizophrenia, one might say that the accident acted as a precipitating cause. But the author would still maintain that neither the insurance company nor the Government are responsible for supporting the patient for the rest of his existence.

Bostroem (1940) stated very authoritatively that psychoses of endogenous type after head injury develop only if there exists an endogenous predisposition.

Comparing the incidence of schizophrenia in the average population with that in a group of 1,554 head injuries Feuchtwanger and Mayer-Gross (1938) arrived at the conclusion that the incidence of schizophrenic "processes" among the latter was only slightly (0.55 per cent.) higher. Including, however, schizophrenic reactions and defects, the figure was more than three times as high as in the average population. Twenty-three case-histories are given in detail. In five cases paranoid psychoses were closely connected with manifestations of traumatic epilepsy; in four other cases traumatic epilepsy also existed, but the onset of the schizophrenic psychosis followed an improvement of the epileptic symptoms. In the largest group a schizophrenic process was observed without any epileptic signs.

Shapiro (1941) found that in 21 out of 2,000 cases of schizophrenia a close relation between trauma and the onset of psychic symptoms was shown. In ten cases there was no injury to the brain, but the mental symptoms developed in a close time relation to a severe head injury. All the patients in this group had a positive family history, and were introverted personalities. Thus in these
cases the trauma may have precipitated latent tendencies. The trauma acts simply as a precipitating factor. The remaining eleven cases showed evidence of cerebral damage (confusion, dizziness, defect in memory and orientation, persistent headache with irritability). In seven cases in this group the pre-psychotic personality was well integrated, and only in two cases was a positive family history present. In these cases the trauma did more than precipitate a psychotic reaction. In producing pathological changes in the brain, the injury would seem to have contributed to the formation of the clinical picture.

Morsier (1939) published three cases of schizophrenic psychosis following head injuries of greater or lesser severity. He expressed his opinion that, in the present state of our knowledge, the causal connection must be admitted, if there was no heredity, and if transitional symptoms existed between the accident and the manifestation of schizophrenic symptoms. As symptoms of transition (Brückensymptome) he would accept not only epileptic fits but also signs of traumatic encephalopathy, such as headache, vertigo, disturbed memory.

Keller and Miller (1940) reported the interesting case of a woman admitted in a manic state. She had been unhappily married to an alcoholic. When she intended to leave her husband he struck her, knocked her down and beat her head against the ground. She escaped, and she does not seem to have been unconscious at all. A fortnight later she developed severe headache, and about four weeks after the incident her behaviour changed; she began to talk loudly, became demanding, irritable and careless of her appearance, and went out with strangers at night. Admitted in a state of manic excitement with restlessness, flight of ideas, etc., nothing was found on examination to point to organic cerebral damage. But on the eleventh day of treatment she developed a Jacksonian attack, followed by a series of further convulsions within the next 24 hours, ending fatally. The post-mortem showed three linear fractures of the skull, extensive thrombosis of meningeal veins, a large area of necrosis in the right cerebral hemisphere, swelling of the brain, and widespread petechiae.

Jequier and Bovet (1939) reviewed the problem of the precipitation of G.P.I. by cranial trauma. They are inclined to recognize the traumatic character of the disease if (1) the patient was free from symptoms at the time of the accident, (2) the accident was concussion or fracture of the skull (not an emotional shock), (3) symptoms existed continuously between the accident and the diagnosis of G.P.I., (4) the development was rapid, and (5) the incubation period short (often indicated by the patient's low age).

E. Guttmann (1943c) studied the effect of suicidal head injuries on depressed patients. He saw two patients with endogenous and one with reactive depression recover mentally as a result of a severe injury to the frontal lobes in two, to the left temporal lobe in one case. One case of psychopathic depression with a laceration of the posterior part of the left temporal lobe showed no change. One recurrent endogenous depressive was not changed by a mild concussion. He compared his own cases with two cases of injury to the basal part of the frontal lobes, also suicidal, resulting in a marked change of personality, and he reached the conclusion that in his case the recovery was not due to the localization of the injury but to the cerebral damage in general, and to psychological factors, such as the fact of being ill, change of environment, etc.
Baonville and collaborators (1939) published a case of an excitable psychopath who, in a suicidal attempt, shot himself through both frontal lobes. Apathy, loss of initiative and disturbance of attention were the main features in the clinical picture after surgical recovery; egocentricity, suggestibility and puerile euphoria are also mentioned. There was some disturbance of memory and concentration. The patient died subsequently in an epileptic fit, and the post-mortem showed destruction of areas 8, 9 and 76.

POST-TRAUMATIC EPILEPSY.

P. B. Ascroft (1941) investigated the fate of men who sustained gunshot wounds of the head in the last war, with special reference to the development of epilepsy. He examined 540 case-histories of patients who had survived long enough to be re-examined four years after they were first boarded for head injury by the Ministry of Pensions. Only 317 cases were satisfactorily complete. Out of these, 107, or 34 per cent., had fits. Fits were twice as frequent when the dura was penetrated. 45 per cent. of such cases were affected. When the wounds were septic, the incidence of epilepsy was much higher. In about a quarter of the cases epilepsy starts in the first month after the injury, in half of the cases within the first six months. In a third of the cases in which the course of the illness was known, the fits had ceased some years before the patient was last examined.

H. G. Garland (1942) studied a mixed group of epileptics. He found a history of head injury in 6 per cent.; the age distribution of incidence suggested that most of the cases seen were of the idiopathic type.

Feiling (1942) reviewed some literature on the incidence of traumatic epilepsy following blast injury, but did not arrive at any definite conclusions as to what factor or factors really determine the origin of epilepsy.

Harrower-Erickson (1940) studied the Rorschach responses of patients with focal epilepsy who had been treated by local brain excision. Though in most of her cases no connection with a previous head injury was established, her findings may be applicable to cases of post-traumatic epilepsy. Her most important finding was the rarity of the occurrence of the "restricted" picture, found in other organic lesions. Though most of her patients showed some deviation from the norm, they did not exhibit such characteristics that the term "epileptic personality" could be attributed to them.

Elridge (in Brock, 1940) in 362 head injuries (peacetime hospital admissions) found 17, or just under 2 per cent., cases which developed seizures shortly after the injury. In 43 cases of late epilepsy (from another source) the onset of the fits was on the average over five years after the original injury.

Troeger (1938), comparing the families of traumatic epileptics with those of cases of idiopathic epilepsy, found no evidence for the assumption that heredity plays a part in the origin of post-traumatic epilepsy. In some of his cases endogenous factors might have been of some importance, but this is impression rather than scientific evidence.

Wilson Gill (1941) published three cases of narcolepsy in which he assumed a trauma to the skull to be the responsible factor. Some cases of narcolepsy are known to be due to a localized mild encephalitis; trauma to the skull may be
expected to cause similar effects, if damage is localized in the region of the hypothalamus and the third ventricle.

**Prognosis.**

Symonds and Ritchie Russell (1943) studied the prognosis of head injury in Service cases. Out of 242 consecutive cases 91 per cent. returned to duty, 9 per cent. were invalided. A follow-up showed that a further 11 per cent. were invalided later. Out of 718 cases which were not admitted immediately after the injury but at various intervals afterwards, the prognosis was much worse; this was not unexpected since the cases were largely selected for admission because their progress had been unsatisfactory elsewhere. Their relatively bad prognosis did not depend on more severe injury, as judged by the duration of post-traumatic amnesia. There was a positive correlation between the bad prognosis and a higher incidence of predisposition to mental disorder. In selected cases of flying personnel the prognosis for return to duty was four times as good as in all the other cases of the series. It may be assumed that the main reason for the better prognosis was that the air crews were a highly selected group in respect of absence of predisposition to mental disorder.

E. Guttmann (1943a) studied the prognosis in civilian head injuries. Three hundred consecutive admissions to an accident service in a mixed urban and rural district were the material of his investigations. Four per cent. of the cases died in hospital; two became invalids through cerebral damage. Eighty per cent. of the cases were treated in hospital for fourteen days or less. The average working time lost by wage earners was roughly eight weeks. In mild cases (i.e. those with a post-traumatic amnesia of one hour or less) the average loss of working time was four to five weeks, in severe cases (post-traumatic amnesia one to seven days) about nine weeks, and in the severest cases fourteen weeks. In patients who stayed off work for an unusually long time, excluding the cases of cerebral damage, it could be shown that social and psychological factors rather than the physical effects of the accident were responsible. The same author had shown in an earlier investigation that only roughly 20 per cent. of the cases had any subjective symptoms three months after the accident or later.

Ritchie Russell (1942) followed up a consecutive series of 200 admissions of head injury, and he found that 87 per cent. of them returned to full work within six months of the injury. His figures showed very clearly that the duration of the disability bore little relation to the presence of a fracture. Counting all symptoms, including slight ones such as an occasional headache, he found the duration of symptoms under 2 months in 40 per cent., 2 to 6 months in 5 per cent., 6 to 18 months in 15 per cent., and over 18 months in 40 per cent. of the cases.

The largest clinical and statistical analysis on the prognosis of cranio-cerebral injury was published by Cedermark (1942). He was able to follow up over 1,700 cases. 146 of these died after their discharge from hospital, whereas the expectation compared with the average population in Sweden would have been 102 only, yet in only a few cases had the subsequent death any connection with the injury. The author is inclined to think that the increased
mortality was due to arteriosclerotic changes. The death-rate became normal about eight to ten years after the injury. In 7 per cent. of the cases the earning capacity was reduced by 10 per cent. or more. The figures for insured and un-insured patients did not differ. There was a positive correlation of 0.84 between the occurrence of objective symptoms and of symptoms of neurasthenic and psychogenic kind. Late symptoms were found in about a third of those followed up, in about half of the fracture cases and in a quarter of the cases without fracture. These figures did not vary at three, five and ten years after the injury for adults; among younger patients the figure tended to fall over a longer period of observation.

In only four cases was there reason to suspect a serious change of personality; in no case was a chronic psychosis found. Five patients had episodic psychoses and severe psychogenic reactions during the later course of recovery. In three of these cases, however, the connection with head injury was questionable.

Epileptic fits were found in 1 to 2 per cent. of the cases observed over ten years. In a number of cases, however, the traumatic character of the epilepsy was doubtful.

The writer's material did not allow of any definite conclusion as regards the effect of long bed treatment on the final outcome, but his figures for the duration of in-patient treatment needed are of interest. The median for in-patient treatment was 7.6 days, with the lower and upper quartiles at 3.7 and 16.5 days respectively. The median for fracture cases was 14.2 days. The working time lost for the total material was up to 1.7 weeks for one quarter of the cases, 3.7 weeks for half of them and 2.4 months for three-quarters. The author gives also figures for the time his patients spent in bed, in hospital and at home together. This combined period was up to one week for a quarter of the cases, two weeks for half of them, and one month for three-quarters. Finally, he determined the time when the patients considered themselves recovered, i.e. the point when the after-effects disappeared. For the whole material the lower quartile was 1.4 weeks, the median 3.5 weeks, and the upper quartile 2.5 months.

TREATMENT.

Surgical treatment in the strict sense of the word is not the subject of this review. As regards the problem of dehydration, it can fairly be said that most neurosurgeons in this country employ it only in selected cases, i.e. in those in which manometric tests have shown an increased intracranial pressure. Lambert Rogers has recently put the case for dehydration, but, as Ritchie Russell has pointed out, his evidence is not convincing, since it refers only to selected cases.

Cairns (1942b) has emphasized that there is no need to keep a patient in bed for any rigidly determined length of time after he has recovered consciousness. An investigation by With lends support to his view. This author reviewed 394 cases treated with individualized confinement to bed according to their symptoms. The time of bed treatment was:

<table>
<thead>
<tr>
<th>Duration</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Under 1 week</td>
<td>in 65%</td>
</tr>
<tr>
<td>1 to 2 weeks</td>
<td>&quot; 24%</td>
</tr>
<tr>
<td>2 to 3 &quot;</td>
<td>&quot; 8%</td>
</tr>
<tr>
<td>3 to 4 &quot;</td>
<td>&quot; 2%</td>
</tr>
<tr>
<td>Over 4 weeks</td>
<td>&quot; 1%</td>
</tr>
</tbody>
</table>
The results of a follow-up study 5–15 years afterwards were most encouraging. Work was resumed within one month of discharge in 66 per cent. and within one to three months in 18 per cent. Less than 1 per cent. were permanently disabled.

The rehabilitation—i.e. the non-surgical treatment of head injuries—was discussed at a meeting of the Royal Society of Medicine (1942). Jefferson, opening the discussion, formulated his view: “We must beware of the danger of coming to regard all who have had a head injury as neurotic, though it would be not far wrong to regard all of them, as I remember that we do, as potential neurotics.”

According to Jefferson, two stages should be considered in rehabilitation. The first covers the period during which the patient is in hospital, confined to bed or ambulant. He warns against over-emphasizing the value of physiotherapy. Rehabilitation in a diversional form should be instituted early, whilst the patient is confined to bed. It will take the form of reading, or being read to, jigsaw, crossword or other puzzles, drawing, needlework, basket-making, sewing, knitting and rug-making. The patient convalescing from head injury should be encouraged to use his mind, and take an active part in his treatment. This first stage will last about six weeks. The second stage is that of hardening. Military cases, at this stage, should be taken over by their respective service. Civilian cases should have gymnastics and physical training, and work in the garden, or in the carpenters’ and engineers’ shops; walks, organized games and visits to the town, for the purpose of finding out how the patient reacts, will play a part. It is an essential of rehabilitation that it should be planned by a time-table each day. H. Cairns (1942b) pointed out the importance of the time factor for deciding when a patient should be considered fit for return to work or duty. He drew up a tentative time-table grading the severity of the injury according to the post-traumatic amnesia. As a working rule he suggested that the shortest time in which ability to carry out full work may be expected to return is as follows:

<table>
<thead>
<tr>
<th>Post-traumatic amnesia</th>
<th>4-6 weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td>5 minutes–1 hour</td>
<td></td>
</tr>
<tr>
<td>1–24 hours</td>
<td>6–8</td>
</tr>
<tr>
<td>1–7 days</td>
<td>2–4 months</td>
</tr>
<tr>
<td>over 7 days</td>
<td>4–8</td>
</tr>
</tbody>
</table>

Rehabilitation, according to him, begins when the patient commences to talk, and to respond properly. He rarely finds a reason why the patient should not be allowed to get up a few days after he has recovered consciousness. If he gets a headache he can be put to bed, given some aspirin, and he can get up again the next day. Inhibitions, however, rarely necessary, for headache is not a conspicuous feature of this stage of recovery. From the day of getting up, graduated physical and mental exercises may begin. The importance of psychological factors is stressed, and no attempts at rehabilitation are considered likely to be successful unless the patient’s anxieties and fears are assuaged, and unless he is helped through the phases of depression and other disturbances of feeling that so often beset him during recovery from head injury.

Russell Brain (1942) found that the incidence of neurosis is much higher after
head injuries due to industrial accidents than to those following road accidents, and he showed that in industrial accidents the high incidence of neurosis is associated with the less severe injuries. In both groups the incidence of neurosis is highest in the unskilled labourers. Men of any occupation are specifically more likely to develop traumatic neurosis if they are injured in an industrial accident, even though their organic injury is likely to be less serious. The remedies he suggests are: (1) The rehabilitation in all cases of slight injury is primarily in the hands of the general practitioner or casualty officer who first sees the patient. These "front line workers" need to learn the right psychological handling of the injured man, and to avoid suggesting serious injury when none exists. They should also learn to avoid the hospitalization of really slight cases. (2) All big centres need a unit of doctors trained to deal with head injuries, both slight and severe. (3) Occupational therapy should merge into therapeutic occupation, i.e. organized light work. (4) The social responsibility for the injury should be more widely recognized, and the emphasis changed from compensation to rehabilitation. He finally suggested alterations in the Workmen's Compensation Act to that effect:

In another paper on rehabilitation after head injury, Cairns (1942) described some causes of failure. Return to work too early is one of the common ones. A physical softness on return to the outside world is another cause of breakdown, particularly in the Services. The more serious causes of failure include loss of intellectual capacity, epilepsy, and disabilities resulting from gross brain damage. In this group of cases the patient will not be able to return to his previous occupation, but, with adequate facilities for vocational training, much can be done to make these patients into useful members of society, and, at the same time, to regain for them their self-respect.

A. J. Lewis (1942) considers the treatment to be more preventive than actual. He regards the essentials of preventive treatment as (1) To decide early what plan to adopt, and, as far as possible, to adhere to it. (2) To let the patient know, as soon as may be, that he will, or will not, have such and such residual disability, and that he need fear only so much incapacity, or none at all eventually. (3) To see that misguided relatives or friends do not tell him a highly coloured story of the accident, but that it is explained to him soberly and with due allowance for his amnesia and other symptoms. (4) Not to prolong the period of rest and inactivity, but to institute early some mild work or interests, not more exacting than his state warrants, and gradually to increase both the opportunity for activities and the incentive, taking care on the other hand to avoid overtaxing him to the point where frustration and "catastrophe reaction" could lead to exaggerated concern over his disability. (5) To help him in any financial, legal or domestic embarrassments to which the accident has conduced; a skilled social worker is here most valuable. (6) To do everything possible to bring the phase of special examinations to an end, except in so far as they are necessary for assessing progress or deciding on special methods of treatment or disposal.

Munro (1938) considers only 30 per cent. of his cases as operative. In the non-operative cases he divides convalescence into three stages. The first begins when the intracranial pressure has been re-established to normal level. During
this period the damaged tissues should heal, and this can be accomplished by keeping the patient flat on his back in bed for two weeks from the time when his intracranial pressure has been demonstrated to be back at normal.

The second period begins at the end of the second week of convalescence. It is divided into two parts, the first in the hospital, and the second at home. The length of time involved depends on the activity, the intelligence and cooperation of the patient. Five to six weeks are usually required. During this period the patient should get out of bed and gradually increase his activity. He should also demonstrate to himself and to his family that he can move about without symptoms. Before leaving the hospital, the patient and his friends and relatives are seen, and are told that his injury is healed, what kind of injury it was, and what the permanent after-effects are likely to be. The patient is reassured against his common fears, and is given a schedule of how to live for the next four weeks.

The third period extends from the end of the first month after discharge to the time when the patient returns to work. The psychological management is directed to the purpose of getting the patient himself to wish to go back to his own activities.

In a recently published book on the after-effects of brain injury Goldstein (1942) includes a long chapter on the treatment of aphasia and kindred disorder. It contains a detailed description of the approach to the re-education of patients with focal disturbances which will be most valuable to all who have to deal with such cases. The author stresses the general principles and the individual approach. A thorough psychological analysis of the preserved faculties and of the patient's ability to use them form the basis for the training which should enable the patient to regain his functions, though using new techniques. The author also describes his methods for improving the working and earning capacity of the patient with cerebral injury, vocational guidance and other ways of social readjustment.

REFERENCES.

BARCROCK, H. (1941), J. Psychol., 11, 261.
BARBIERI TALMIER, G. (1939), Schizophrenia sup., 7, 3.
BECK E. (1939), Nervenartz., 12, 142.
Idem (1939), D. Z. Neurochirurg., 169, 284.
BOSTROEM, A. (1940), Münch. med. Wschr., 87, 985.
Idem (1941), Abstr. War Medicine, 1, 224.
[1944.

BY E. GUTTMANN, M.D.

Idem (1942a), War Medicine, 2, 772.
Idem and Ritchie Russell, W. (1941), Brain, 64, 93.
Evans, Jameson (1940), Brit. J. Ophth., 24, 614.
Faulk, F. (1939), Arch. Z. Psychiat., 107, 72.
Fox, T. H. (1940), Mil. Surgeon, 86, 478.
From, J. (1930), Lyons Med., 169, 628.
Gill, A. W. (1941), Lancet, i, 474.
Graves, T. C. (1938), J. Ment. Sci., 84, 552.
Guttmann, E. (1942), paper read before the Oxford Medical Society.
Idem (1943b), Lancet, i, 10.
Idem and Winterstein, C. E. (1938), ibid., 84, 347.
Idem (1939-40), Amer. J. Psychiat., 96, 1263.
Harrower-Ericsson, A. (1946), ibid., 42, 1681.
Kroll, E. (1941), Medical Aspects of Boxing. Pretoria.
Karst, K. H. (1938), J. Belg. de Neurol., 35, 598.
Leigh, A. D. (1943), Lancet, i, 38.
Idem and Jones, M. (1940), Lancet, i, 813.
Malzberg (1937), Psychiat. Quart., 11, 445.
PSYCHIATRIC ASPECTS OF HEAD INJURY.

MEYER, J. E. (1940), Arch. f. Psychiat., 113, 368.
MOORE, TH. V. (1938), Consciousness and the Nervous System. Catholic University of America.
MYERS, C. S. (1940), Shell Shock in France. Cambridge.
NADEL, A. B. (1938), Behavior following Cerebral Lesions. New York.
Raven, J. L. (1942), Lancet, i, 115.
REHWAHL, E. (1940), Med. Klinik, 36, 867.
ROOSSEN, R. (1941), Nervenarzt., 103, 274.
Idem (1941), ibid., 93, 389.
SCOTT, M. (1940), ibid., 44, 444.
Idem (1940), ibid., 43, 279.
Idem (1940), ibid., 43, 591.
STEINBERG, D. L. (1941), Elgin State Hospital Papers, 1, 145.
VAN VALKENBURG, C. T. (1940), Lancet, i, 1003.
VERJAAL, A. (1939), Z. Neurol., 186, 211.
WILLIAMS, D., and Denny BROWN, D. (1941), Brain, 64, 223.
WILLIAMS, D., and Denny BROWN, D. (1941), Brain, 64, 223.
Wolffson, L. N. (1938), Psychiat. Quart., 12, 137.
ZILLIG, G. (1941), Nervenarzt., 14, 145.