(viii) Residual symptoms having at least the severity of those termed traumatic psychasthenia are very common. The condition is of good prognosis up to about two years, but any change which persists longer than this is apt to be permanent. In assessing the degree and importance of residual defect standardized tests are of little value. Symptoms that are reported by a reliable and unbiased witness as occurring but which cannot be detected at interview must be accepted. Many of the minor changes of this condition—such as lack of initiative, retardation, loss of memory for engagements, indecision—which need not transpire at interview, may destroy the usefulness of anybody doing responsible work.

(ix) While the syndrome traumatic psychasthenia may remain stationary, it may, on the other hand, be the starting-point of a progressive deterioration into dementia.

(x) Permanent change is more likely (a) at the extremes of life; (b) when organic disease exists—for example, arteriosclerosis; (c) when there is a constitutional psychopathic tendency. It must be recognized, however, that even in young adults, and even in absence of any of the grosser psychotic sequels, traumatic psychasthenia may persist.

MENTAL SYMPTOMS ASSOCIATED WITH HEAD INJURY

THE NEUROLOGICAL ASPECT*

BY

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In considering the mental disturbances which frequently result from injuries to the head we recognize at once two groups or chains of possible causal circumstances. The first consists of material elements and comprises the organic injuries of the brain and the subsequent reactive changes in the cerebral tissues. The second consists of abstract elements and comprises the emotional disturbances connected either immediately with the accident itself or with the patient's adaptation to his normal life afterwards, and if the question of compensation arises it adds to this group further elements of anxiety and possibly greed. Both the material and the abstract factors react by their different means on a mental constitution and a mental content which are in large measure peculiar for each individual. The thread of the material effects and the patient's mental constitution are the warp and the woof of the weave of mental symptoms, and the emotional effects are printed on; but we who find it difficult enough to make out the pattern may often be in doubt as to what is imprint and what is woven in the texture.

The range of our subject includes cases of many different kinds, and I have grouped them below chiefly according to their physical nature, but I shall have time only for brief discussion of two or three of these groups.

Classification in Groups

The subject as I see it contains the following groups. In many cases the initial mental symptom is unconsciousness, using the word in its popular sense—that is to say, the abolition of all signs of mental activity.

1. Cases of "simple concussion" in which the unconsciousness is usually of brief duration and there is no indication that

the brain has been organically injured.—I have never seen any "mental" symptoms persisting in such cases, with the exception of one quite atypical instance which will be referred to in group 7.

2. More severe cases in which unconsciousness is usually more prolonged and there is conclusive or strong presumptive evidence of organic injury of the brain.—In such cases recovery is occasionally characterized by profound mental disorder lasting many weeks or months, and it is with such instances that we are most concerned to-day.

2a. Rare cases of this kind in which, recovery having reached a certain stage, relapse occurs and the patient sinks into a state of dementia—cases of "late dementia."—I have seen three such cases, and the descriptions of them are fairly common in the literature.

3. Cases in which there has been actual loss or known gross local damage of cerebral tissue which may in itself be responsible for mental changes.—I have in mind cases with open wounds through which cerebral tissue has been extruded, cases of gun-shot wounds and perforating injuries, and cases of depressed fracture in which the damage is local.

4. Cases of intracranial haemorrhage, particularly (a) traumatic subarachnoid haemorrhage, and (b) subdural haematoma.—Here the physical factors appear relatively simple, while on the psychological side the cases differ obviously from those of the earlier groups in that the sequence of mental changes is from normality down to coma instead of from coma upwards.

5. Cases in which fits occur and are accompanied by intellectual deterioration.—The fits are likely to begin long after the injury, and the factors responsible for the mental symptoms are evidently different from those operating in the preceding groups.

6. Cases of so-called "traumatic neurasthenia."—The physical evidences of injury, if there were any, have disappeared (or have been allowed for), but mental symptoms of a nature that we know well have become apparent. This is a difficult and complex group, which we must try to unravel.

7. Cases in which the mental symptoms, persisting after the accident, are those of one of the well-known types of psychosis and probably depend essentially on the patients' antecedent mental constitution.—Cases of manic-depressive psychosis, schizophrenia, and paranoia have all been described following head injuries. I have myself had one case in which a dementia with schizophrenic features followed, in a boy of 16, an injury which caused unconsciousness for about half an hour and no outward signs of organic damage to the brain. It is difficult to know for how much the physical factors count in such instances.

Stages towards Intellectual Recovery

In group 2 the physical concomitants usually leave little doubt that the brain has been organically damaged. There are probably signs of fracture of the skull and possibly cranial nerve disturbances, reflex changes, signs of local weakness of limbs, or blood in the cerebrospinal fluid. The initial stupor may last a number of hours or several days, and the return of mental activities is gradual. In the vast majority of cases the patient passes as "normal" mentally within a few days, and if I describe cases in which recovery takes weeks or months it is because of their great intrinsic interest and because of the light they throw on other cases. The course of events is somewhat as follows.

THE IRRITABLE AND NOISY PHASE

The patient ceases to be entirely passive when moved or touched, and makes grimaces or movements—and soon utters words—expressive of resentment. Within a very short time he may become abusive when attended to or even when spoken to. This phase varies in duration from a few hours to many weeks. It is sometimes accompanied by periods of restlessness and noisiness, and I have had

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several cases in which the patients for weeks were almost continuously noisy and unruly throughout their waking periods. As the patient's abusiveness subsides and it becomes possible to carry on a conversation with him, two features become apparent—confusion and perseveration of words. Thus:

How old are you? Eh?—How old are you? How old am I?—Yes. I don't know.—What is your work? I sell things.—What do you sell? Oh, everything.—What kind of things do you sell? Oh, everything.—Do you sell motor cars or vegetables, or what? Everything.—Do you sell motor cars? No, everything.—Do you sell oranges? Yes, everything.—Have you a shop? No, everything.

A patient who has been noisy for any length of time is likely as he becomes more controlled to remain talkative for a considerable further period. His talk circles round a very limited number of ideas and is characterized by frequent repetition, thus revealing both the perseveration of his ideas and the deficiency of his memory. At the same time elation is likely to make itself apparent as a feature of his mentality, so that he becomes generally pleased with himself and his surroundings, and is what is commonly called "fatuous."

DISORIENTATION AND CONFABULATION

There may follow a more subdued state in which the patient answers rationally but is disorientated in time and place. This again may last many weeks.

One of my patients, a consulting engineer, spent a considerable part of his recovery period (in imagination) in India inspecting a line and locomotives with which he had been concerned several years before, and when questioned he was ready to confabulate about his daily visits to India. Another, confined for months to hospital, used to describe visits to his own home and the very questionable goings-on of his grown-up sons and daughters. These two patients thus showed all the elements of Korsakoff's psychosis. One of them had a good deal of subarachnoid haemorrhage, and I think that, apart from this, confabulation is uncommon; in my experience it does not occur during the more rapid recoveries.

THE RETURN OF MEMORY

During this time memory begins to return. The answer to the question "Have you ever seen me before?" instead of being "No" or "I don't know" becomes "Yes, I think so," and finally "Yes" with confidence, and eventually he can tell exactly when and describe what conversation took place. Memory at first—like developing memory—is patchy. The patient has "clear" or "lucid" periods, and he remembers what takes place during those periods. It may be possible to recognize the periods that he will remember by such signs as an increase in his alertness, more thoughtful answers, and a more respectful attitude towards the doctor; but I must say that I have sometimes been disappointed later to find that the patient remembered nothing of a period in which it seemed to me that all these signs were present. Such periods may at first be of only a minute's duration, and then the patient again answers confusedly, but gradually they become longer.

Memory returns as part of the general recovery, but the patient remains amnesic with regard to a period of variable duration preceding the accident.

THE RETURN OF ACTIVITY

When the talkative phase has subsided, or if it has never been present, the patient's general mental activity,

as evidenced by his behaviour, is in most cases low. He lies idly in bed or sits about, seldom speaks spontaneously, or if he does he is muttering something to himself about years long past; he takes little interest in what is going on around him, and shows no desire to read or to engage in other forms of mental or physical activity. At first he carries out single actions in response to an immediate external stimulus. As he improves the effect of an external stimulus is more prolonged, it produces more and more "affective drive," it carries him further. Part of this improvement may be due to conditioned reactions, but it is apparent where conditioning can hardly be a factor, and the patient performs new activities with less and less urging. At a later stage he takes in stupidly something of the content of the picture on the front page of a newspaper that is placed on his bed. As the days pass he looks at the pictures with more understanding, but it may be a long time before he reads the headings of the paper with intelligence.

When memory returns and he becomes normally orientated, he begins again to talk spontaneously. Simultaneously, spontaneous physical activity has been developing. It may, however, be years before the patient regains the degree of spontaneous mental and physical activity that he had before his accident, or he may never regain them. We shall return to this point in considering "traumatic neurasthenia."

The Emotional State

The patient's emotional state throughout this period of intellectual recovery may go through different phases. At first he is irritable, resentful, abusive. At a subsequent stage he is often elated and childishly happy. This has already been mentioned, and I shall refer here only to some of the possible consequences of it. The patient is so pleased with himself and so anxious to be nice to everyone that he would be easily wheedled into making a bad settlement of whatever claim he might have for damages. One of my patients told me how a "terribly nice gentleman" had visited him at his home in Sussex and explained to him that the accident (in which he had been knocked down by a lorry) was entirely his own fault, and that he would have no claim for damages at all. I do not suggest that any advantage was taken in this instance of the patient's pleasure and simplicity, but the danger is obvious. Elation, when it occurs, usually persists in some degree throughout the whole of the patient's illness. It has a mitigating effect on some other symptoms, and it seems to prevent the occurrence of an anxiety state such as is common in the later stages of recovery. Elation is usually accompanied by a lack of reserve and by some deficiency in the patient's normal respectful behaviour towards the doctor and other strangers with whom he comes into contact. If the patient's emotional setting is not that of elation the alternative is depression, querulousness, anxiety, and outbursts of temper.

Physiological Nature of the Symptoms

It is evident that the lack of spontaneous speech, lack of intellectual and physical activity, amnesia, failure to retain impressions or to appreciate their full significance, represent abolition or severe impairment of function. On the other hand, the early irritability and abusiveness and the later elation and talkativeness are in all probability due to release of function—release of functions represented at a cortical level by the abrogation of the higher cortical functions of reserve and thoughtfulness, or what the Romans called "gravitas." The disorientation in place

and time may also represent some release of function—some uncovering of almost disused functions—again at a cortical level. The circling of the patient's thoughts around a small number of ideas is probably largely due to the failure to receive new impressions at their proper value, or, while the patient is disorientated in time, it may be due to the liberation of a few memories of the period in which he fancies he is living. In either case it would seem to be due to the partial release of certain functions and the failure of others.

Most of these symptoms are probably the result of diffuse rather than local cortical disturbances, but a major importance for the intellectual defects attaches, in my opinion, to impairment of the function of the cortex of the frontal lobes. Elation is also, I believe, the result of a frontal lobe disturbance. I have seen it with localized injuries of the frontal lobe, and in its extreme form it merges with "Witzelsucht," or pathological tendency to joking, which also occurs with frontal lobe lesions.

Prognosis

You will have gathered from the trend of my remarks that even when mental symptoms are prolonged their course is towards recovery, and in the majority of cases recovery is eventually complete. It is seldom more than six months before the patient behaves as a normal individual, though he may still have some residual symptoms. But you will have noticed, too, that I made a special subgroup of certain cases in which after partial recovery the patients lapsed into a state of dementia which persisted. I cannot stay to discuss this group further, but it is noteworthy that the youngest of my three patients was 55 years of age.

Meningeal Haemorrhage

In all the cases we have been discussing the symptoms have begun with stupor and gradually or quickly receded. I must make some mention of two small groups of cases in which it is possible to watch the progress of the symptoms in the opposite direction, from normality down to stupor. I refer to the cases of subarachnoid and subdural haemorrhage resulting from injury. In the former subgroup stupor may take from a few minutes to an hour or two to develop, according to the severity and perhaps the site of the haemorrhage. The symptoms are excitability, restlessness, irritability, unreasonableness, lack of reserve, an activity which is rational but not fully comprehended and which is accompanied by a hypomanic emotional state, disorientation, confusion, then diminishing activity, paraphasia, drowsiness, stupor. More frequently than not the early symptoms are marked by concussion.

In this connexion I would like to tell the story of one of my patients who had a moderate amount of blood in his cerebrospinal fluid. He was knocked unconscious by a blow on the face, and his amnesia begins about five minutes before the blow. At the time the blow was struck he and another man had agreed to have a fight and were on their way to a suitable place; it may be that his emotional state then had an effect on his subsequent behaviour. He was taken "unconscious" (in the popular sense) to hospital. He remained without signs of mental activity while a cut lip was stitched, and then rather quickly he appeared to "come round." He at once became extremely excited, violent, and threatening, and was arrested as drunk by a policeman who had come with him in the ambulance. He has no recollection of any of this, and was un-

doubtedly unconscious throughout. He remained in the police cell for eight to nine hours, and late in the evening was bailed out comatose by his brother. The next morning, still stuporous and irritable and, when roused, abusive, he was produced in court and, still quite unconscious, was actually induced to stand up while he was lectured by the magistrate and "bound over." He subsequently passed through a period of very noisy and unruly behaviour, followed by confusion, disorientation, perseveration, and so on, and it was nearly four weeks before he again became conscious.

With the subdural haematomas the progress towards stupor is much more gradual and there is not the same element of irritability—not the same positive elements in the mental disturbance. The decline can be watched over weeks or months, and the steps are those of a simple dementia: reduction of mental and physical activity, reduction of interests, periods of disorientation, perseveration of ideas, amnesia, confusion, drowsiness—all perhaps variable but increasing with each aggravation—and finally stupor.

Although the course is downwards instead of upwards the actual symptoms in these two subgroups are similar to those of phases in the preceding cases.

Our Ignorance of Essential Pathology

We are almost completely ignorant of the physical and functional changes in the brain which are essentially responsible for these mental symptoms following injuries, and I have not found in the literature any detailed pathological reports of cases of traumatic psychosis. In the great majority of cases the lesions in the brain are widespread, and in the cases of intracranial haemorrhage an extensive area of cortex is affected either by pressure or by direct contact with effused blood, and it is rational to correlate the temporary dementia with depression of activity over such a considerable fraction of the cerebral cortex.

Whatever the disturbance is which affects the brain after injury it is one that in certain cases takes months to recover. Oedema, haemorrhage, local circulatory disturbances, the effect of degenerating tissue elements on the cortical cells, damage to association tracts with subsequent "compensation" for the deficiency, changes in the myelin sheaths, and reactive changes of the interstitial elements in the brain may all exert their influences, and in the presence of so many possible factors it is useless to theorize. The elements of Korsakoff's psychosis which appear are probably due to the toxic effects of degenerating blood and other tissue.

"Traumatic Neurasthenia"

It will perhaps have occurred to you that some of the cases that are dubbed "traumatic neurasthenia" are characterized by symptoms of the very same nature as occur in the final stages of recovery in the cases I have been discussing. Such are slight confusion, inability to think clearly, inability to sustain mental activity, lack of spontaneous conversation and spontaneous physical activity, and lack of reading in a person who has been accustomed to read. These are fairly good indications of a minor degree of that receding dementia which is a feature of the more obvious and prolonged mental disturbances, and such symptoms undoubtedly have a basis in organic cerebral injury. I always distrust the label "traumatic neurasthenia" if the patient's injury has been a serious one. With this psychasthenia the emotional state is variable, but more often leans towards depression. There may be superimposed factors of anxiety arising from the

patient's environmental conditions—including lack of income and the matter of compensation. But in some cases there is another factor which I believe is of greater importance, and that is the difficulty which the patient has, because of his persisting intellectual disturbance, in adapting himself to his normal life, and particularly to his occupation. An example will make clear what I mean.

A butcher, aged 50, had had a serious accident seven months before I first saw him. He said he was unable to attend to his business because whenever he went into his shop he felt "awful," and after a few minutes had a feeling that he simply must get away from it. He complained also of headaches, depression, inability to concentrate, being easily tired, lack of interest, disinclination to meet people; he found it a great effort to write a letter or to make a simple calculation, and his memory was very bad. He had formerly been an active man with a good many interests and many friends, but since the accident had been content to sit about idly, and seemed to have lost all his old interests. One of the reasons why he did not want to meet his former friends was that he had nothing to say to them. There was thus ample evidence of intellectual impairment persisting from the time of the accident. Consider the situation of this man when he goes into his shop. His activities there are very largely habits and reactions conditioned over half a lifetime. But they require memory, judgement, power of rapid calculation, grasp of details—qualities which he no longer possesses in adequate degree. All his strongly ingrained habits and conditioned reactions are frustrated by his persisting mental impairment. Is it any wonder that he feels awful and wants to run away after a few minutes? In the case of an employee the conflict is still greater because of the added feeling of compulsion to do the work.

It is not surprising, then, if true neurasthenic elements are often superimposed on the picture of persisting psychasthenia. In assessing such cases, whether with the neurasthenic element or not, it is important to obtain evidence which will reveal the degree of persisting impairment of the patient's mental activity. Fortunately the prognosis is in general good.

Other Types

The next largest subgroup is formed of those patients in whom a constitutional tendency towards neurosis of the anxiety type pre-exists and the accident serves to precipitate a period of aggravation. They are usually to be distinguished from the last group by their volubility and the multiplicity of their symptoms. Their "headaches" are the typical feeling of pressure on the top of the head or of a tight band round the head, and they are not brought on by stooping and by physical effort as are those of patients who are suffering from organic after-effects of head injuries. Usually the accident has been of a less severe type than in the cases I discussed earlier, and a history of antecedent symptoms of neurosis may be obtained.

Gross hysterical manifestations following injury are, I think, usually the result of pre-existing tendencies, and occur in persons of poor mentality. Minor degrees of hysteria are possibly common, in the sense that a patient may unconsciously take refuge in a continuance of subjective symptoms rather than make the effort to return to the conditions of his normal life or relinquish a claim to compensation.

Then there are those who make the most of their symptoms in an effort to obtain the maximum amount of compensation. We scoff at these, but we must be careful to recognize that the difficulty of obtaining fair compensation gives rise, especially among the working classes, to a great deal of genuine anxiety.

Finally there are cases in which the injury seems to precipitate an attack of depression in a patient whose pre-existing mental constitution was of the cyclothymic or manic-depressive type.

In referring to these latter groups I have stepped on to the verge of a big subject, with which I hope some other speaker will deal—namely, the influence of the patient's individual mental constitution on the symptoms arising in consequence of a head injury.

TREATMENT OF VIRUS DISEASES OF THE SKIN*

BY

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It is a disappointing fact that our knowledge of viruses and the lesions they produce has not added greatly to our therapeutic efficiency in the management of virus diseases of the skin, especially as the most striking examples of lasting immunity occur in certain virus diseases, and few bacterial diseases exist which will bear comparison in this respect with diseases such as measles, mumps, varicella, variola, and zoster. The latter impressive examples are apt to obscure the fact that, in general, no essential differences distinguish the immunity following virus from that of bacterial diseases and it appears to be most probable that viruses are living agents having many points of close resemblance to the pathogenic bacteria. Similar antibodies are developed in virus diseases, and the presence of these antibodies has been demonstrated by the well-known bacteriological tests of complement fixation, agglutination, precipitation, and neutralization. Specific therapy therefore follows the lines of bacteriological procedure, and would chiefly depend upon the use of vaccines and sera.

Unfortunately experimental and clinical evidence indicates that in many virus infections specific antibodies are practically ineffective when the virus has once reached a susceptible cell. No doubt there are exceptions to this rule, and in 1935 Laidlaw, Smith, Andrewes, and Dunkin demonstrated that an anti-influenza serum had considerable potency therapeutically, even when given to mice two or three days after experimental infection with influenza virus. As a rule, however, unless specific antibodies reach the cell before the invading virus, infection is very likely to ensue, and the use of hyperimmune serum or that of convalescent patients is almost limited to prophylaxis. Similarly the induction of active immunity by living or dead vaccine may be very effective in the prevention of infection but of little or no value when infection has occurred.

The various means of producing active immunity in virus diseases are:

1. Vaccination with living virus:

- (a) Human strains—for example, herpes.
- (b) Modified by animal passage (vaccinia).
- (c) Attenuated by time or temperature (rabies).
- (d) Neutralized with immune serum (distemper).

* Read in the Section of Dermatology at the Annual Meeting of the British Medical Association, Belfast, 1937.