

# Head Injuries

## A Clinical Lecture

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### **Editor's note:**

*This article is text of a lecture delivered by a surgeon of the Indian Medical Service during the colonial era. The audience probably comprised of general practitioners. The racial tones notwithstanding, the article gives valuable insight into the surgeon's understanding of pathophysiology of head injury and its management in the early twentieth century.*

The subject of my lecture today is a vast one. I will treat but a small portion of the subject, always endeavouring to give you a sound basis to work from in your consideration of these cases, in so far as they may concern you.

You no doubt imagine that anything to do with the diagnosis and treatment of injuries of the head must be very modern, connected with x-rays, ventricular puncture, and surgeons specialized in operations on the head. The impression is false, for as long ago as 3,000 and 2,500 years before Christ, a surgeon living in Egypt fully described their treatment. No doubt accidents occurring during the building of the pyramids provided him with many such cases. In one of these pyramids was found the now famous Edwin Smith Papyrus, which, when translated, brought to light the astonishing fact that treatment and care of the cases was just as thorough then as it is now in our most modern surgical clinic.

In 490 B.C., Hippocrates wrote a book giving full instructions to the surgeon on the treatment of injuries of the skull and brain.

It was not until the beginning of the seventeenth century that surgery recovered sufficiently from the turmoil and religious oppression of the Middle Ages to recommence making strides.

From then until the present time, the names of such great men as Pott, the Hunter Brothers, Cooper, Lister, and more recently Jackson, Horsley, Cushing, Dandy and Martel, are associated with remarkable advances that have been made since those early days. Do not think that these advances

have changed the problems which face us – these remain for all time – each case is a mystery to be solved; it presents its clues which, if followed up correctly, will lead to the solution of that particular mystery. What these advances have done is to give us more ways of attack on the problems which present themselves; sometimes a way of even putting the trouble in order. But even with these ingenious extra helps there is no short cut. We must observe a rigid routine in the examination of these cases, using to the full all powers of observation and deduction before thinking of using any other instruments. Those of us who may be too stiff-necked to follow such a method, invariably fail to produce any useful result.

The surgical anatomy of the skull is intricate, and I do not propose to go into any detail here. Suffice it to say that upon your knowledge of the anatomy of the skull depends the exact diagnosis of the head injury with which you may be confronted.

Much more important is your concept of the elementary physiology of the skull, for upon this depends your understanding and diagnosis of the conditions of concussion, compression, or contusion.

Let us consider the brain, for a moment, as one of those encapsulated organs. This is an unusual description of the brain, but it permits us more easily to envisage some interesting aspects of the functional relationship of the brain to its skeleton, the skull.

A moment's thought will bring us to the conclusion that it is in the matter of rigidity that various organ capsules differ, and that on this character they may be classified into three groups:-

In the first group, the capsule is fully extensible; the kidney and the spleen belong to this group.

The second group, exemplified by the testis, has only partially extensible capsules.

The third group, represented by the brain with its capsule the skull, has a non-extensible covering.

This variation in the rigidity of the capsules in these groups will of necessity modify considerably the mechanics

of the circulation of different organs, under given conditions. There is of course, a primary need for the flow of blood to be continuous, though any tissue, and in the first two groups the extensibility of the capsule allows pulsation and elastic recoil to occur. But in the case of the brain, the mechanism must be different, because the capsule of this organ is unyielding. Therefore, as at each pulsation the brain expands and the skull does not, then room must be made for the pulsation to occur by the expulsion of an equal quantity or volume of the low-pressure intracranial fluids. This is why the veins leaving the skull and the cerebro-spinal fluid in the subarachnoid space of the spinal cord show arterial pulsation.

The mechanism is adequate, but the margin is very small. After violent exercise when the pulsations in the brain are at their widest normal excursion, we are apt to be aware of an unpleasant thudding in the head which indicates that the brain can only just find room for its circulatory excursions. Again, if one has had a slight headache, it is at once aggravated by exertion.

This circulatory peculiarity is fundamental in any consideration of cerebral pathology; it is possible to say that, leaving out destructive lesions, all cerebral symptoms are of circulatory origin.

We will briefly consider how this comes about. Each arterial pulsation is accompanied by an outflow of the low pressure fluids, chiefly in the form of venous blood. For this to occur, the flow in the veins must be absolutely free. Now, since the pressure in the veins is very low, the slightest swelling in the brain, or part of it, will at once cause a collapse of the veins, and a consequent obstruction of a greater or lesser venous territory. The vicious cycle thus commenced is added to by the swelling now occurring from venous congestion, and so on, the disturbance of function becoming progressive. The brain is thus uniquely sensitive to very slight changes in its bulk.

When an organ like the kidney is bruised and swells, it matters very little how soon if ever, it gets back to its normal size. When the brain has been bruised, it must get back to its normal size or its circulation will remain permanently disturbed. A simple bruise, of no ultimate importance to an organ with a yielding capsule, is thus a relatively serious matter with the brain. The great difficulty with which the brain recovers from even simple injuries is one of the most important functional consequences of its rigid encapsulation by the skull.

There is still a traditional reverence for the ancient delusion that fracture of the skull is the lesion of most importance. We have all had cases of head injury under our care. A skiagram of the skull is taken at the first

opportunity. After inspection by the radiologist and by the doctor in charge of the case, a sigh of relief is heaved there is no fracture!- and the less wary of us rock our minds to sleep in that cradle of insecurity until perhaps it is too late to avert the catastrophe. Fracture of the skull is usually an insignificant element of a head injury.

The mechanical significance of the fracture of the skull is simply this – the skull has been distorted until the limit of its elasticity has been passed. It is this distortion, and not the crack in the material of the skull, which is of importance.

Immediate and dramatic effects are not always produced, and because of this and the superstition about the significance of fracture, it is apt to be assumed that the average cranium is on the whole a very satisfactory protective to the brain inside. Since the nature of the so-called minor head injuries has been completely understood, faith in this beneficent fortitude of the skull has been completely shaken. We now know that the skull is only moderately effective in its protective function, and quite considerable degrees of distortion may be caused by only slight external violence. Local violence may cause local bending and permit of localized bruising of the brain underneath.

Under the condition of more severe external force being momentarily applied, a far more serious general distortion may occur always – with or without fracture. This general distortion causes very interesting instantaneous and transient paralysis known as concussion of the brain. There is likely to be produced a widespread bruising of the brain substance that is of great practical importance.

It is of importance to note that, all the evidence would lead to the conclusion that actual distortion of the skull is the immediate cause of most, if not all, of the injuries of the brain. There is no evidence for the belief that injury may be produced by the brain being thrown about inside the undistorted skull as a result of some external violence. No distortion, no brain injury, is probably true.

This liability to distortion would appear to be a racial variant. For instance, the willingness of the Negro to use his head as a battering ram, is well known, and it is said that an experienced American policeman will use his truncheon on the head of a Negro less hopefully than he would use it upon the head of a white man. As far as my experience goes, the head of the average Hindustani comes intermediate between these extremes of the scale. There are many variants in this country – there are many races and many mixtures. It appears fairly clear then, that in some races the resistance is fairly high, and that there is a tendency for the more highly civilized races to be less resistant.

It will be noticed that so far, we have not once concerned

ourselves with the anatomy of the skull, either topographical or developmental. It may or may not be possible to show a difference in the thickness or rigidity of European and Negro skulls, but, when the far more delicate test of function is applied, there seems to be a demonstrable difference. In the one, the protective function is satisfactory; in the other, the protective function has become impaired, and the physiological disadvantages are fully manifest.

Can this present disadvantageous functional relationship of brain and skull be the result of some strong evolutionary tendency? And if it is so, then what is the advantage which compensates for it?

When the modern European's cranium is compared with those of his ancestors, it is found to be remarkably light and thin. It is not improbable therefore that this tendency towards reduction of massiveness is an inherent character of the race and is progressive. It is natural now for you to ask how far such a process could conceivably go. It may even occur to you to ask if a rigid cranium is a necessary structure.

Without any other consideration but that of function, a very definite answer can be given. However, much more of its protective massiveness may be lost, it must always preserve sufficient rigidity to keep its form, because the skull must maintain its functional relationship with the brain, i.e., that of skeleton to the brain.

Since the great war, wherein so many head injuries occurred, many observations have been made on those who survived. Those who suffered loss of a part of their brain skeleton when seen standing, show greater or lesser depressions where the loss of bone has occurred. In the large deficiencies the soft tissues may be found to be one and half inches below the remainder of the surface. Observe the same cases when lying down, and you will see that the concavity has disappeared, the soft tissues having filled the gap. You will find that the larger the opening, the greater the depression while standing. Question one of these men and you will be told that sudden exaggerated movements, such as change of posture, cause severe suffering. You will also be told that changes in atmospheric pressure affects them very much. Now there are others, in whom the breach of continuity of the skull has been closed by some skilful surgeon, these will tell you that they now do not suffer from any symptom, as they used to.

In point of fact, the skull forms the neatest possible solution to the problem of how to support such a large mass of soft tissue. It is an exo-skeleton exploited in a remarkably ingenious way, which is worthy of a moment's consideration.

The other obvious way of supporting a large mass of soft consistency would be to provide it with a stiff connective tissue stroma. Now, were this even possible, it would mean that every fibre would have to be provided with a coat such as is provided for each cerebral vessel the so-called perivascular lymphatic. And that would have to be continued to the very finest of its ramifications. This would at least double the bulk of the whole organ. The presence of this fibrous tissue would enormously add to the complication of intercommunication, which is the essence of brain as it is.

Actually, as the arrangements are, the brain, made up of almost entirely functional elements, is of reasonable size and is kept within bounds by the exo-skeleton, the skull.

After these interesting diversions into the function of the skull in relation to the brain, let us now consider the actual practice of treating head injuries, and apply where possible the principles learnt in our discussion.

The accident happens, the on-lookers or relatives, if there at that time, rush in upon you and clamour for something to be done at once. If you have the fortune not to live close to a road point where accidents are likely to occur, then you will surely be summoned to your hospital to treat the case. Both of which sudden calls will be likely to disturb your calm and upset your judgement. Go quickly to see the cases, but refuse to be rushed into doing anything without first carefully examining them, and then make up your mind as to what the exact condition is with which you are faced.

The patient lies unconscious and pale, arms and legs are inert, eyelids drooping, the eyes unseeing, both pupils dilated and sluggish, the pulse rapid and small. Such is the picture of concussion, a condition of temporary suspension of cerebral functions, following immediately on injury, and lasting a variable time, but with recovery in twenty-four hours. It follows directly on the infliction of that degree of distortion of the skull which would cause a momentary compression of the brain. There is momentary displacement of the cerebro-spinal fluid and blood from the veins, and the condition is due to consequent anaemia of the brain.

Recovery begins from the medullary centers upwards, and is usually initiated by the reflex act of vomiting, the other functions recovering more gradually. There is complete amnesia for the period of unconsciousness.

It is of importance at the stage before us to submit the patient to skilled, methodical, and competent medical care. Hurry nothing and carry out a rigid routine always. Have the man undressed and put to bed. Cut the hair off. Whilst this is being done, employ your time in getting the story of the accident from an eye-witness. The first sight of such a

case is worrying, but more often than not when the case is looked into, it is not so bad as at first appeared. All cases, however, do not recover, which ones will do so it is impossible to say at this stage, and thus the importance of a rigid, careful routine examination is clear.

There is blood on the face, hair and clothes, where is it from? Look for wounds in the ear, the mouth, or on the head, face or neck. Blood may be found in the meatus of one, or more rarely, both ears: look for a torn drum, or a wound in the wall of the meatus. Never syringe out such an ear as you would at once wash into the wound some infection and so set up a meningitis, should there be a tear of the drum with a fracture of the bone behind. Beware of blood being washed into an ear from the outside. Epistaxis may be abundant, but the nose bleeds easily, and it is usually not of great importance; when persistent it may indicate fracture of the anterior fossa. Blood from the mouth might come from a fracture of the anterior fossa, pituitary fossa, or the middle cranial fossa, but you will be well advised to look for a bleeding point along the gums and teeth or on the tongue, before thinking of one the former more serious causes.

Never omit to look carefully at the face, even during the period of concussion. One angle of the mouth may be seen to be drawn up, or one cheek may be sucking in and out with each respiration, indicating involvement of the facial nerve in a fracture of the petrous mass of bone.

A black eye may be caused by a local injury, when it will be of little importance, but again look carefully to distinguish between this local injury and the black eye with proptosis, due to an intra-orbital haemorrhage following fracture of the orbital plate.

Strabismus is a sign which is apt to cause some trouble in interpretation. If there are relatives about enquire from them whether the patient suffered from the condition before the accident, other wise you will, one day, attach a wrong interpretation to this sign. Internal strabismus points to paralysis of the external rectus muscle of the eye, and a fracture of the petrous angle with involvement of the sixth nerve. This is the commonest nerve involved in fracture of the base of the skull. External strabismus is due to an injury to the common motor nerve to the eye as it passes through the foramen in the sphenoid bone.

It will be seen that, even at this early stage, we may, by careful examination, arrive at a fairly accurate diagnosis.

We have not mentioned the temperature, pulse, and respiration rate. These data, of course, should be obtained, and orders given for them to be observed every half hour, and a graph made. Never simply write down the figures

representing the rates; a mass of such figures conveys nothing to us after three or four hours. Make a graph, using coloured ink or pencil, one colour for each set of observations to be recorded. Then, when looked at in a few hours' time, we can at once tell what is happening, and what has happened. The urine must be examined. Note on this chart when urine is voided, when the patient vomited, etc. Thus, we have all the available data collected together.

After twelve, eighteen or twenty-four hours, the signs will be all the more marked. Bleeding will have stopped and you may now find cerebrospinal fluid trickling from one ear. It will not usually be possible to say whether or not there is cerebrospinal fluid coming out from the nose or mouth on account of the presence of the normal secretions.

By now ecchymoses will have commenced to appear. Evert one or other of the lower eyelids, and you will perhaps see a red blotch. This is an infiltration of the subcutaneous tissues of the lower lid with blood which has gravitated down from a basilar haemorrhage.

Just below the tip of the mastoid process, there may be present a bruising, which, if present, is of greatest help. It clearly indicates a severe contusion, or fracture through the base of the skull. I have seen this sign present without any other.

Examine the nape of the neck for the presence of a large, diffuse, and perhaps increasing swelling, which will come from a fracture of the posterior fossa.

The vault of the skull may show any one of a variety of fractures. Examine this region carefully; a haematoma overlying a fracture is particularly deceptive.

At this stage, you may meet with certain phenomena which are very disquieting. The concussion is noticed to have deepened and to have passed into true coma. The pulse is feeble, the respirations interrupted and there is marked cooling of the face, hands and feet – signs of impending death.

Another case may show a pulse of 40 to 50, respirations stertorous, one pupil larger than the other, face and head persistently turned to one side, so that when they are turned to opposite side, they turn back to their original position. There may be localized or generalized convulsions, both tonic and clonic. There are all reactions indicating a particularly a severe lesion and a bad prognosis. They are due to cerebral compression by blood or bone fragments, basal or ventricular haemorrhage, or cerebral contusion.

The normal picture of concussion passes of more or less quickly, and generally within twenty-four hours of its onset.

Sensibility, movements and consciousness return in that order. Concussion having gone, the local signs are displayed, hemiplegia, motor or sensory mixed; regional paralyses, etc. can all be seen now.

Even if there are no such signs, the concussion must disappear completely in order to dispel all fears for the patient. Persistence of any sign or symptom spells danger. After an interval in such a case, coma may set in. The classical lucid interval has passed and the stage of compression has established itself. This coma is accompanied by paralysis, unequal pupils, stertorous breathing, and a slowing pulse rate with a high blood pressure. Early examination of the fundus of the eye, even during the lucid interval, might have given some indication of the impending disaster, by the presence of papillary stasis.

The onset of this coma usually indicates interference with the intracranial circulation, the mechanism of which we have already discussed. The haematoma may be extradural, subdural, or both. From the point of view of opening the skull later on, it is of importance to remember that, although usually the bleeding is from the middle meningeal artery or one of its branches, it may come from pial or from intracerebral veins. The main haematoma always affects one part of the brain more than the rest, and consequently gives rise to symptoms which vary with its situation. In the early stages, the symptoms will be those of cerebral irritation, due to the venous stasis; later, they

	<b>Irritative</b>	<b>Paralytic</b>
Hemispheres	Irritability, Stupor, Reslessness, etc.	Coma
Motor Cortex	Jacksonian fits	Hemiplegia, hemiparesis
Mid-brain	Contracted pupil	Dilated and fixed pupil
Medulla	Vomiting	
(a) Respiratory center	Slow stertorous breathing	Shallow irregular breathing
(b) Cardiac	Slow pulse	Rapid, weak pulse
(c) Vasomotor	Raised blood pressure	Falling blood pressure

are paralytic, due to anaemia of the part caused by capillary compression. As the compression sets in, it is preceded by a wave of irritative signs, due to venous stasis. Later, as the paralytic signs become evident in a succeeding wave, so that you will see in one and the same patient both types of sign. The table before you gives you the main localizing signs, together with those of concussion and compression.

Let us now pass to cerebral contusion. This may be of a major or a minor type.

The patient, having recovered from his concussion, passes almost imperceptibly into a state of stupor. He lies curled up in bed, and at times, he is noisy, resentful of interference, disoriented, restless and irritable. He may even become really violent and have to be restrained. After an interval, he may become rational again, only to relapse into stuporous condition once more, after a longer or shorter period. During the time he is mentally clear, he has complete amnesia for the previous period of stupor. Lumbar puncture

	<b>Concussion</b>	<b>Compression</b>
General condition	Unconscious, slowly regains consciousness	In a classical case: lucid interval, increasing drowsiness and coma
Appearance	Pale, respiration shallow	Flushed; respiration becomes stertorous
Pulse	Increased in rate and feeble	Slow and bounding. Rapid when cardiac center fails
Temperature	Subnormal	Unequal on the two sides
Musculature	Relaxed; reflexes diminished or absent	Varies on the two sides
Sphincters	May be incontinent	Become incontinent
Pupils	Moderately dilated; equal in size, react sluggishly to light	On side of injury: pupil contracts and later dilates. The opposite side follows

will reveal fluid under pressure, and perhaps blood. This condition of alternating lucidity and clouded consciousness may persist for weeks. The patient must be watched with the greatest care, and not allowed out of the care of a competent doctor, no matter how the relatives, or the patient himself, in his lucid interval, may press for permission to leave the hospital.

Minor contusions should be suspected when there is violent headache, giddiness, insomnia, and mental disability. The condition may arise after an injury with or without concussion, or follow as a sequel on major contusion. The headache is very markedly affected by posture, being increased by physical exertion, mental stress, and curiously enough by lying down. The giddiness is similarly affected.

In either form, there may be a raised temperature, without other signs to account for it.

You must also bear in mind that there are late sequelae to head injuries which are very grave, such as meningitis, cerebral abscess, and chronic subdural haematoma.

The treatment of head injuries can be divided into two important steps, early observation and collection of data concerning the case. If there is a compound fracture and a

wound present, it must be excised at once. If this is done by the surgeon in direct charge of the case, then he will decide what he will do. But if you are faced with a case with a wound present, it is your duty to excise the wound at once with all the aseptic precautions possible. Do not first rub the surface with gauze soaked in antiseptic. You will do no good, but only push any infection that may be present deeper into the tissues. Gently dab the edges of the wound with tincture of iodine or alcohol picric acid solution; take a sharp knife and cut away the edge of the wound, making sure that you are cutting through healthy tissue only. Apply firm dressing (dry) when you are sure that there remains no damaged tissue or debris in the wound. If the haemorrhage worries you, apply a bandage, twisted and plaided, around the head at the level of the brow. Into the knot put a pair of forceps and turn them around until the required degree of tightness is obtained. This tourniquet may be left in place for at least twenty minutes if there has been severe bleeding. Do not make any attempt to sew up the wound. More harm has been done by ill-advised attempts to close wounds, which, if they had been left open, would not have given any trouble. Time and again have I seen doctors so keen to sew up a wound, that they do not even take time to carry out the ordinary and elementary step of cleansing their own hands! Cleaning the wound never entered their heads. You can only expect disasters if you do not carry out the elementary rules of surgical cleanliness.

The next step in the treatment, after limitation of, or elimination of, sepsis, is one which directly concerns the surgeon, relief of intra-cranial pressure.

In a case of simple concussion, recovery will invariably be complete in twenty-four hours. It is necessary to warn the patient, and his friends, that a period of at least three weeks' complete rest is essential, and a very gradual return to normal life is equally essentially, if subsequent headaches are to be avoided. I have seen the headache, occurring three months after discharge from hospital, so severe that it was necessary to perform a decompression to obtain any relief. If the return to normal life be gradual, then there is little or no possibility of the occurrence of such complications. Direct surgical treatment is only called for during the first twenty-four hours, by the onset of coma and paralysis after a lucid interval.

In the presence of compression the only treatment is operation, and to be successful, it should be performed in anticipation, i.e., before the major signs of compression are present. Careful observation is necessary to localize the symptoms, for only in those cases where localization has been possible can there be any hope of a complete

recovery. Other cases may recover but will more likely be left with some symptom or other to bear witness that their treatment has been empirical. It is in this matter of observation that you are of the utmost importance. You may be the only doctor to see the case for some hours, and upon your observations the surgeon will depend largely for the early history of the case. Therefore, concentrate upon setting down in writing all you see, at once. The interpretation will become obvious later, when you have collected sufficient data. Keep the pulse and respiration chart with care. Note the type of respiration, and so on. But always make your notes in writing – do not leave it to your memory.

The operation of craniotomy not only allows the removal of haematoma, and ligation of bleeding point, but also offers an exit for further oozing, and relieves the tension in the brain caused by oedema and the contusion. Arterial haemorrhage may be diagnosed in a patient who, after complete or partial recovery from the stage of concussion, sinks into coma or shows paralytic signs. If the site of bleeding can be localized, then operation may be undertaken with some confidence. If, however, this has not been possible for one reason or another, then sub-temporal decompression is the operation of choice, and is a life-saving measure. In cases of gross injury with evidence of bulbar involvement, sub-tentorial decompression should be performed.

The treatment of major and minor contusion consists of rest in bed in a sitting position, and relief of the intracranial disturbances.

The intra-cranial disturbances have their origin in the increased intra-cranial and cerebral tension. In slight cases, this tension may be reduced by giving full doses of magnesium sulphate by mouth, daily. In severe cases, the choice lies between this and intravenous hypertonic saline, with the weight of evidence in favour of the magnesium sulphate in large doses by mouth and per rectum as the better treatment. The dose per rectum is three ounces of magnesium sulphate in six ounces of water, run into the rectum very slowly and retained as long as possible. This, plus the doses by mouth, will cause a copious evacuation of fluid *per anum*, so relieving the body of a fair amount of its fluid content. During this treatment, only enough water will be allowed, to keep the mouth from becoming dry, otherwise you will defeat your ends. A rapid release of pressure may be affected by lumbar puncture. This must be done with greatest care, with the patient in the lateral position, and never sitting up. I have seen lumbar puncture being performed on a case, where the intra-cranial pressure was raised, with the patient sitting up. The fluid was allowed

to flow freely from the needle, and of a sudden the patient collapsed, gasped once or twice and died. The medulla had been forced down into the foramen magnum by the sudden release of pressure, causing immediate death of the unfortunate patient.

When there are persistent symptoms such as delirium, sub-temporal decompression allows a rapid resolution of the cerebral oedema and a means of removing surface clot or of arresting the haemorrhage.

In conclusion, a word with regard to convalescence, a matter of greatest importance in the treatment of head injuries and one too often lost sight of with recovery of the patient from the immediate effects of the injury.

The whole scheme varies greatly with the type of patient, his social standing, his education and financial means, and last but not least with the mentality of his closer relatives. In general, however, some plan along the lines indicated below should be adopted:-

For the first four to ten days nurse the patient in darkened room, in bed. Read to him for short periods, but he should not be allowed to read himself. Only one visitor per day should be permitted, and it must be seen to that the person allowed in is a sensible relative or friend, of some influence with the patient.

If there is good progress under this regime, the next step is to have the patient out of bed in an easy chair for gradually increasing periods each day. The room may be better lighted now but not open to the direct sunlight. Listening to the gramophone or wireless, looking at the pictures of some illustrated paper, or doing a jigsaw puzzle are suitable amusements for this stage.

Along these lines of gradual convalescence, the patient should be quietly led back to normal life. Some will recover rapidly, others more slowly; the speed of convalescence must be carefully gauged by the medical attendant.

Return to work should under no circumstances be allowed before at least four to five weeks have elapsed from the time of return to complete consciousness. When work is resumed, arrangements should be made for the patient to be able to continue this gradual return to normal.

Very frequently, bromides will be found useful and in many cases, essential for long periods; in some cases up to a year or more.

Finally, the patient's reactions to treatment are sole guides to what should and what should not be done in any given case. Only by an assiduous study of the individual and his particular natural history can the practitioner hope to attain success in the treatment of that patient.