Remarks ON THE TREATMENT OF ACUTE HEAD INJURIES*

BY

GEOFFREY JEFFERSON, M.S.LOND., F.R.C.S.,
HONORARY NEUROLOGICAL SURGEON, MANCHESTER ROYAL INFIRMARY;
HONORARY SURGEON, SALFORD ROYAL HOSPITAL, AND THE
NATIONAL HOSPITAL, QUEEN SQUARE

It is not only because head injuries are such common occurrences, but also because skilful treatment of the right kind, and at the correct time, may make so much difference to the result that they are a fruitful subject for discussion on such an occasion as this. Few subjects can boast a more voluminous literature, reflecting the mutations in theory which time has brought and the alterations in practice which logic demanded. Nor is opinion as yet well stabilized on the meaning of certain of the cerebral states observable as the result of cranio-cerebral trauma, and in consequence there are divergencies of opinion as to the nature of the treatment which is most beneficial. At the present time the treatment of head injuries is chiefly non-operative, because experience has shown that the worst cases are not saved by routine decompression, and because it is generally believed that we have in hypertonic solutions a method of lowering pressure which, if it cannot replace operation in all cases, is useful enough in the majority. I believe that the current fashion for hypertonic solutions as a cure-all is wrong, because it is indiscriminately applied: that they have their value when properly applied I shall not deny, but the problem of head injury is not solved by their use, and the time has come for revision.

When a patient with a head injury is brought into hospital to-day it is admitted that he may have a middle meningeal haemorrhage; but then this is something of a rarity, and it is hoped that if he happens to have one it will make its presence known by the classical train of signs. Otherwise it is generally assumed that the patient will not be operated upon, that if he has had maximal brain laceration he will die and nothing will save him (this is true), and that if he has a submaximal injury he will no doubt get better (which may be). In the meanwhile hypertonic treatment will be given, and this panacea will cure him if he is curable. It is my contention that this line of action tends to produce a feeling of unwarranted satisfaction, a feeling that all that could possibly be done is being done. We shall do a better service to surgery and to the patient if we regard every head injury as a potential subject for operation, if we suspect our patients of having not only epidural haemorrhages, but the much more insidious and common subdural haematomas, and thus reduce by a little more the unavoidable death rate in these serious cases. In order to get a proper sense of proportion we shall do well to turn to the available clinical material, and to subject it to scrutiny to determine so far as we can in what respects the cases differ from one another, in which our treatment is adequate, and in what particulars we may improve it.

CLINICAL MATERIAL

An analysis has been made of a consecutive series of cases at the Manchester Royal Infirmary and Salford Royal Hospital (a five-year period at the one, six years at the other). The total number of cases of head injury admitted amounts to 1,004, and of these 230 patients died (23 per cent. fatality). Various mortality rates have been arrived

* Made in opening a discussion in the Section of Surgery at the Annual Meeting of the British Medical Association, Dublin, 1933.

at by other observers, from over 60 per cent. downwards, and if the rate appears to be falling to-day it is chiefly because the slighter cases are admitted more often than formerly, and it is these minor cases which cause an apparent improvement in result.

Much more important than the gross rate is an analysis of the rapidity of death. In 152 cases the time of death after admission to hospital was stated sufficiently accurately, and the death rate per day is shown in the accompanying chart (Fig. 1).

Ninety-one patients, 60 per cent. of total eventual deaths, died within twenty-four hours, and the next highest number before the end of forty-eight hours, meaning that overwhelming damage occurs in a high proportion of the cases. In three cases, for example, tears of the cavernous and superior longitudinal sinuses were found at necropsy, with the skull full of blood. Some four patients were believed to have local haemorrhages, but died whilst active

FIG. 1.—Acute head injuries (152 cases): Death rate per day.

preparations were on foot to relieve them—that is, within an hour or so of admission. This primary mortality seems to me to be fixed and unavoidable, but the case is otherwise with the late deaths—those from the second or third day onwards. Some of these are comprised by fatalities in aged persons who die of pneumonia or exhaustion, but there is a sufficient number of young people in this category to raise the question as to why, having survived the initial danger period, they have eventually succumbed. Given a severe enough trauma it is not surprising that death will speedily occur, but it is

interesting to ponder the various factors which permit survival for longer periods with death as the outcome. Meningitis accounts for some 8 per cent. of deaths, with a special incidence in childhood. It will be seen shortly that unsuspected subdural haematomas have been found among them more often than they were suspected, and evidently we shall have to focus attention in future on the recognition of this cause of death from compression. But all the late deaths are not due to gross compression, and we can only assume damage to centres and to synaptic and tract connexions by haemorrhage, secondary oedema, and necrosis, in which histamine may play a part. In thinking of such centres and pathways we must bear in mind that the subthalamic nuclei of the vegetative nervous system are as vulnerable as, and perhaps even more delicate than, those in the medulla oblongata. We know very little of those metabolic disorders which lead to death, for besides those centres which are immediately necessary to life there are others without whose aid life cannot be long sustained. The thermal crises which proclaim severe injury to the tubular region are inescapable evidences of damage; minor injuries of the same type and with different results no doubt occur. Arising out of this question of damage to different centres is the variability of the placement of the injury in different people. Polar contusion is well known, lacerations of the under surfaces of the frontal lobes and of the

temporal poles commonplaces of observation ; indications of these local injuries are recognizable clinically. It is sometimes assumed that the great variations in behaviour during recovery arise from temperamental peculiarities in the individual. This presupposes that patients sustain a stereotyped injury, the result varying with the make-up of the person injured ; and up to a point this may be true. But it is much more probable that the differences commonly remarked are due to variations in the situation of the chief damage, and I am convinced that evidences of lesions of particular regions are much more frequently to be observed than has been generally believed. Symonds has done notable service in drawing attention to the possibility of recognizing residual focal damage long afterwards, and there is a rich field awaiting more intensive study during the acute phases.

CLINICAL TYPES

When all is said and done the outstanding consideration in any case of head injury is the problem of traumatic stupor: how has it arisen, what does it mean, and how shall it be dealt with? How much of it is due to direct contusion, how much to secondary oedema? These are the mute questions put to us by every case, and on the answer depends the individual treatment. So much is the depth of unconsciousness the one and essential primary clinical point in head injuries that it can be said that immediate anxiety is almost at an end when consciousness is regained. Patients arrive at hospital in all stages of post-traumatic stupor, and they will be found to fall conveniently into three groups.

Group A

Group A includes those who are deeply unconscious or comatose, with widely dilated pupils and sluggish corneal reflexes, cyanosis, stertorous respiration, and flaccid limbs. These patients die very rapidly and nothing can be done to save them.

GROUP A.—Comatose on Admission

Sex	Age	Pulse Rate	Survival in Hours
M	58	140	8
M	64	82	2
F	43	63	12
F	61	104	7
M	59	76	6½
M	7	130	½
M	23	60	12

Group B

Group B, in which the patients are already recovering consciousness when they reach the hospital, is the largest. The patients have no recollection of the accident (retrograde amnesia) ; they are shocked, vomit once or several times, are dazed and restless, but can be induced to answer questions, though some persistence may be required to obtain answers. Often enough these patients do not again relapse into unconsciousness, and though they may be drowsy and restless they can usually be roused periodically for a sufficiently thorough general and neurological examination. When they follow this course they do not give rise to any great anxiety ; they may be irritable, but once they have answered questions the surgeon feels that he knows where he is with them, and will be able to intervene appropriately if there is any falling-off in their condition. It is important, however, to observe that fractures of the skull may be present

in this type of case and, what is more important, laceration of the brain ; it is from this class that the best examples of extra- and sub-dural haematoma are recruited. The cases which follow are random examples of the common uncomplicated cases of the general type just described, the only noteworthy point being the variability from case to case of the pulse rate.

GROUP B.1.—Dazed, but not Unconscious on Admission :
Recoveries

Sex	Age	Pulse Rate	Unconscious	Fracture	Home: Days
M	50	60	Not	No	14
M	47	66	Not	Yes	18
M	27	100	Not	Yes	14
M	53	112	Not	Yes	16
F	44	56	Not	No	10
M	23	60	5 minutes	Yes	21

Not all of these patients remain in such good condition, and some again relapse into unconsciousness or into a stupor from which it is difficult to rouse them. Unless careful observations have been made on admission they then present very difficult problems, for although there is always the strong suspicion of massive haemorrhage in such cases, it is not always present, and some recover again without intervention: the crucial point is the depth of secondary unconsciousness and the presence or absence of localizing signs. If the stupor is relatively light and no limb weaknesses are present a clot can be assumed to be absent, but if there is the slightest doubt it is very much wiser to explore, a brief and conservative intervention being sufficient to establish the presence or absence of intracranial bleeding.

GROUP B.2.—Primarily Unconscious ; Recovery ; again Unconscious

Sex	Age	Pulse	Consciousness	Fracture	Recovery : Days
M	63	104	Unconscious Conscious Unconscious 12 hours	No	17
M	46	74	Semi-conscious Lapsed into complete unconsciousness for two days	Yes	28
F	24	80	Dazed Became unconscious for 36 hours	Yes	14

It is possible that small clots were present in the three cases outlined above, but if so they were not of sufficient size to be localizable clinically.

Group C

The third group (C) presents the greatest difficulty in diagnosis and treatment. This consists of patients admitted in a state of stupor or semi-stupor and who remain non-co-operative and unresponsive for many hours or days. Although they do not reply to questions their general condition is not as a rule bad ; they are well oxygenated, their pupils are normal, and while it is the common rule to speak of them as unconscious they move about in bed, and much information can be gathered if the observer will take the trouble to sit by them for a time. Facial asymmetries may be observed by testing the corneal reflexes, and the movements of the arms watched in the effort to remove an annoying finger from the cornea. The abdominal, plantar, and tendon reflexes furnish corroborative evidence of a unilateral lesion. In a number of these patients no lateralizing signs are dis-

coverable, and after a few days of anxiety they gradually become more conscious and slowly recover. Generally the hospital records of these patients merely state laconically that the "patient is unconscious," often a lazy way of saying that no trouble has been taken to

GROUP C.—Unconscious on Admission: Recoveries

Sex	Age	Pulse Rate	Unconscious	Fracture	Home: Days
M	21	88	2 hours	0	10
F	20	64	3½ hours	+	14
M	33	88	2 hours	+	21
M	21	90	4 hours	0	10
M	18	56	4½ hours	0	5
M	36	80	2½ hours	0	23
M	21	80	24 hours	0	22
F	36	80	24 hours	0	23
F	37	94	2 days	+	15
M	21	80	2 days	0	21
F	21	84	3 days	0	21
F	20	88	3 days	+	21
M	40	120	6 days	+	28
F	23	76	35 days	+	42
M	56	90	28 days	0	42
F	20	70	19 days	+	21
M	21	70	28 days	0	35
M	33	90	10 days	+	21

find out his exact state. The students' teachers are somewhat to blame for this, because the fact of non-cooperation on the patient's part is regarded as a preventive of any useful examination. Periuncutory examination of reflexes ought to be replaced by careful notes on limb movements during the restless turnings of these patients and a clear-headed search for evidences of damage to cerebral centres.

THE CAUSE OF TRAUMATIC STUPOR

Reference has already been made to variations in the depth of stupor, and attention called in particular to the fact that the patient is rarely so deeply unconscious that important clinical observations cannot be made. Following the teaching of Trotter in this country, we assume that concussion is due to vascular arrest, that it is an affair in its purest form of moments only, and that recovery is perfect. This doctrine has been widely received because it is the only understandable explanation of a dysfunction which passes off quickly and leaves few traces behind. That it may not be a complete explanation is very possible, for we know that trauma may temporarily paralyse nervous tissue (for example, the "physiological paralysis" of peripheral nerves) without our being any the wiser as to the exact processes involved. This much, however, is certain, that the longer any paralysis lasts the more certain are we that anatomical damage has occurred, a fact of great importance when applied to the brain mass. Patients remaining unconscious for any length of time may safely be assumed to have suffered neural damage, and there is no doubt that even those who recover consciousness in a few moments may still have had contusions and even lacerations of the brain, and from such injury may spring a subdural haemorrhage large enough to cause death unless the surgeon intervenes.

It is very commonly assumed that prolonged unconsciousness is the result of raised intracranial pressure. This seems to be a logical assumption, since Nature from time to time

performs a human experiment for us by producing a severe local compression in the shape of a middle meningeal haemorrhage. Indeed, the sequence of stupor, recovery, and secondary stupor implies the production of unconsciousness by clot. But is it the clot by reason of its size or the clot by reason of its position which leads to stupor? It so happens that most massive clots are anteriorly placed, and it is undoubtedly the frontal lobe which suffers most. Evidence of a tangential kind can be drawn from experience with intracranial tumours, and we know, for example, that pressure alone does not cause stupor except in its last stages, and with rises of tension far in advance of anything seen in head injuries. The intracranial pressure in cerebellar tumours, for instance, exceeds considerably any so far recorded rise in trauma, and yet these patients are never ordinarily unconscious, whilst we have learned that the only cases which approximate to head injuries in their behaviour are neoplastic lesions of the frontal and temporal lobes. Nor do the demeanour, the lack of cyanosis, nor the actual cerebrospinal fluid pressure readings in traumatic cases bear out the teaching that stupor is due to pressure. Rapidity of compression does not explain why stupor should result from a focal clot, and we know that manometric readings may be normal even when a clot is present, which should signify that the haematoma has a local rather than a general influence. But this discussion on clot pressure is taking me away from my main theme—namely, that stupor in the average case is contusional, and that even when oedema occurs it is very rarely alone the cause of protracted unconsciousness. I believe that it is most often due to contusion of the anterior portions of the hemispheres, and we know that these regions are particularly prone to damage by reason of the anatomical configuration of the fossae and the cranio-cerebral relationships there. Further, the restlessness, the irritability, the anger, indeed the "sham rage" of some patients, recall the animal experiments of Bard, and of Fulton and Ingraham, who produced it by lesions of the under-surfaces of the frontal lobes. The following is a clear-cut example of frontal contusion.

CASE I

A very powerfully built young man, aged 20, fell 80 ft. whilst rock climbing. He remained stuporous with intervals of wild excitement for many days, and finally became quite uncontrollable. In the second and third weeks he was constantly out of bed, tried to get out of the window, and threw the nurses about when they interfered with him. Male nurses had to be called in. He was in hospital five weeks, and finally made an excellent recovery.

Incontinence is another symptom found, in particular in affections of the frontal lobes (sometimes of the temporal); it is a really rare occurrence with tumours elsewhere than in these areas. Incontinence is the rule in the severer head injuries, and I believe that it means the same thing in head injuries as in tumour cases. In some patients residual signs of a severe frontal contusion may be discovered as soon as consciousness is regained: the following cases will illustrate my point.

CASE II

A youth, aged 20, was thrown from a motor cycle ten days before. Remained stuporous for four days, but is now talking well, though his mental processes are very much disordered. He thinks he is at the works in the electrical laboratory, where he has been carrying out researches. He is very interested in the taking of temperatures by the nurses in the ward, and told his brother that he did not know how they conducted their experiments as the instruments had no external connexions. He talks very well, though disorientated in time and place. He is still quite incontinent,

and given a urinal does not know how to use it and does not try. This is a pure frontal-lobe picture. Patient made a complete recovery; he was incontinent five days after return to consciousness.

CASE III

A girl, aged 14, was knocked off a cycle by a motor car. She remained unconscious nine days and then gradually recovered her faculties. Usual retrograde amnesia. She remained incontinent for five days after recovery of consciousness, and was incontinent at night for twenty-one days in all. Finally made a complete recovery.

The only other localizing sign of frontal contusion which is undebatable is anosmia, of which I have seen five cases following injury. It is often permanent, and is a very distressing sequel. No doubt the lack of concentration, enfeeblement of memory, and temperamental changes which can follow severe head injuries have the same pathological explanation.

Time will not permit me to describe further the signs of local contusion in other regions of the brain: evidences of parietal and of occipital contusion, with its visual defects, are presented sufficiently commonly. I must, however, refer to one point of the very greatest importance—left-sided frontal or temporal contusion with aphasia. An injury in this situation interferes, of course, with speech, and at times a patient will be encountered who is recovering from his stupor, who looks at the examiner as if he understood what was said, and who then turns away without replying. Failure to recognize this as an important localizing sign has cost more than one patient his life, for I well remember how a left-sided subdural haematoma was overlooked through non-realization as significant of this absence of speech in one who looked as if he could speak. I strongly suspect that the patients in Group C who remained stuporous for so many days had some degree of aphasia, and that the lack of speech or understanding of speech was mistaken for semi-consciousness.

BASIC STATE AND EPIPHENOMENA

These considerations are of great importance to us when we turn to the treatment of head injuries in general. This brings us up against the question of hypertonic salines and their place in the therapeutics of cerebral trauma. I believe that their use is incorrect if we expect them to return a patient to consciousness, for this they invariably fail to do during the first stages of injury. The patient is not unconscious because his brain is oedematous, but because it has been damaged generally, and probably locally as well, by a severe blow. This generalized traumatized state producing stupor of a duration dependent on the severity of the injury is the "basic state" in head injury, and if it is not too severe and if no new element is added to it the patient will eventually recover, no matter how he is treated, although maybe with some lingering evidence of damage to some area in excess of another. Alterations in this state, and particularly alterations in the normal recovery from it, may be brought about by the production of local compression by a haematoma.

In order to separate these happenings from the ordinary effects of trauma I prefer to call them the epiphenomena. We shall see shortly that subdural bleeding is an event for which we must be ever on the watch, and particularly must we be careful not to do anything which may induce it. Intravenous hypertonic salines not only raise the blood pressure but, by shrinking the brain, increase the space into which haemorrhage may occur. During the first day or so hypertonics are best withheld, and used only when there is definite evidence for their administra-

tion. This means a manometric reading on the cerebro-spinal fluid by lumbar puncture. When should this be done? Not during the first twenty-four hours. Readings at any time are valueless if the patient is restless, for they are then fictitiously high, and the plain fact is that most of the patients in the present series have not needed to have it done at all. This practice is entirely at variance with that of whole-hearted dehydrators, who drain off all the obtainable cerebro-spinal fluid immediately and again daily, meanwhile restricting fluid intake and giving hypertonic solutions. I see no point in this. The only time when dehydration is of great service is during secondary oedema, and that means from the second or third day onwards. The danger is that we can fall into the trap of believing that these methods are necessary for all cases indiscriminately from the time of admission to hospital, and that we become interested in applying them more and more whole-heartedly without safeguards. I do not wish to condemn hypertonics entirely, I ask only for their intelligent use; and I say the same of lumbar puncture.

THE EPIPHENOMENA

Massive Haemorrhages

The classical picture of middle meningeal haemorrhage is well known, and there have been in the present series fifteen examples of epidural clot, of which few have been of the orthodox type. I have referred already to the uneasiness that one feels about the late deaths and the fear that some of them may have been due to unrecognized compressions by massive haematomas and not to the laceration and secondary oedema that was the presumed cause of death. Unfortunately the fatal cases in the present series have not come frequently to post-mortem. Owing to the workings of the Coroners Act necropsies are no longer carried out as a routine in fatal injuries, and are much harder to obtain than was formerly the case. I believe that this has a bad influence on the handling of head injuries, for the overlooking of a middle meningeal haemorrhage is no longer revealed, and a salutary lesson is lost. A false sense of the complete adequacy of our treatment is only too easily engendered. Recourse has had to be made to the records of the Manchester Royal Infirmary over a long period to obtain 100 consecutive post-mortem examinations. Among them were fourteen examples of unrecognized clot.

Middle Meningeal Haemorrhages

Sex	Age	Length of Survival	Fracture Present	Clot
F	1½	2 days	Yes	Huge
M	42	7 hours	No	Large
M	12	3 days	Yes	Large
F	60	3½ hours	Yes	Enormous
M	46	65 minutes	Yes	Extensive

The period of survival in such a retrospective survey as this is of the first importance; the last case, for instance, could scarcely have been operated upon in time, however quickly aware the surgeon was of the needs of the case. The extreme youth of the first patient no doubt disarmed suspicion, for middle meningeal haemorrhage is rare in childhood. This record demonstrates the frequency with which fracture is present in this type of bleeding, as compared with those next to be considered, and also the rapidity with which death can occur from an arterial haematoma.

Subdural Haematomas

Sex	Age	Length of Survival	Fracture Present	Clot
M	38	4 hours	No	Extensive
F	60	16 days	No	Enormous
M	65	2 days	Yes	Large
M	30	8½ hours	Yes	Very large
M	35	7 days	No	Large
M	45	6 days	No	Bilateral
M	21	Hours	No	Extensive
M	60	1 day	Yes	Bilateral
M	35	10 minutes	Yes	Huge

The last patient, of course, could not have been saved by operation, and we have no right to assume that all of these patients would have recovered had they been operated upon. Cases in which there was extensive brain laceration have been excluded, and in most of those set out above it is specifically stated that the brain was not seriously damaged except by indentation with the clot. The absence of fracture in five out of nine subdural haematomas is important when comparing them with the epidural haematomas, where coexisting fracture was the rule. Another important point is the very rapid production of subdural bleeding. The literature contains many references to chronic subdural haemorrhage and insufficient to cases of this type. The cases in this post-mortem record have occurred over a period of twenty years, and were under the care of different surgeons; they may be taken as an average series. Considering the great number of cases admitted the overlooking of these examples no doubt represents a small margin of error, but if they disturb our equanimity it is right and proper that they should do so. Nor are these figures isolated commentaries of past (and present) diagnostic difficulties. Vance, in his series of 512 necropsies, found sixty-one epidural and 132 subdural haemorrhages, but in the latter series he has not made sufficiently clear the extent of coexisting lacerations which would have made recovery improbable, even had the clots been removed. McCreery and Berry, in 520 cases of proved skull fracture, found fourteen cases of unrecognized epidural and fifty-one of unrecognized subdural bleeding. They mention three patients in this latter group in particular, where repeated lumbar punctures were done, with deaths of all within twelve hours. These writers, and others as well, have taken signs of fracture as indications of the severity of the injury, feeling that only those with proved fractures were worthy of serious analysis. The incorrectness of this attitude will be generally recognized from the frequency with which subdural haematomas occur without fracture.

The impression that is forced upon one by reflection on these cases is that, though the syndrome of middle meningeal haemorrhage is well known, mistakes are still made at times, whilst subdural haemorrhage is a common cause of death, and still more often escapes diagnosis and correct treatment. If there is this danger of overlooking subdural haemorrhage, are we on the right lines in our treatment of head injuries? Ought we not to be much more suspicious and much more ready to operate than has been the common practice in recent years? I believe that we are through with the period of operating for cerebral contusion and oedema (save in the late case with papilloedema), but that we have not yet entered as fully as we shall on the new but still limited stage of looking for subdural clots.

Clot Compression

How are cases of clot compression to be recognized? The most important service that we can perform is to

advertise their occurrence and make surgeons suspicious of their presence. On extradural haemorrhage I have little to say. A direct temporal contusion is apt to produce injury to the meningeal vessels, and I have come to be very suspicious of a temporal haematoma. Indeed, if there is the least sign of contralateral limb weakness, and especially if there is a Hutchinsonian pupil, the indication for exploration is absolute. But no single sign can be taken alone. With the subdural haemorrhages the case is still more difficult. Laceration of the brain is common in all head injuries, and I believe that we should often be startled if we could but see how great the damage is, even in patients who recover. Any laceration must be accompanied by a certain amount of bleeding, which varies from a sub-pial stain to a clot of varying dimensions. If by chance a large artery or vein is ruptured the resulting bleeding may cause death within a few hours. In other cases a slowly increasing haematoma is created, which can finally become stabilized to form a large tumour, to be discovered weeks or months later. My own observations lead me to think that these clots have arisen from injuries to the temporal or frontal lobes low down. Trotter, in his chronic cases, stated that the haemorrhages were high and had come from parasagittal veins. The fact is that it is very difficult to discover the exact origin of the bleeding, for some of the clots are so large that they cover great areas of the hemispheres. So much is this so that there is little risk of missing a haematoma if a drill-hole is made just below the temporal crest.

A patient with a subdural haemorrhage may not be unconscious, though he will become so eventually if unrelieved. The latent period of subdural clot compression is longer than in extradural haemorrhage, and may, as has been said, be of almost any length. The acute cases will be mistaken for those of extradural clots, but as long as the dura is opened when an extradural clot is not found no harm will come of this. The following example is that of a subacute haematoma.

CASE V

A man, aged 37, was admitted to hospital in semi-stupor. He had suffered a mild injury to his head in a rough-and-tumble twelve days before but had never been unconscious. As the days passed he became more and more drowsy, and finally no longer answered when spoken to. He could, however, still be roused, when he looked round confusedly, but would not speak. Some slight weakness of the right hand was made out when he resisted attempts at examination. Eye closure to the corneal reflex was also weaker on the right, and the resulting grimace uneven. Pulse rate 60. This was a clear case of total aphasia, and a large subdural clot was found on the left side.

Five other cases of similar type have been operated on, and, as in extradural haemorrhage, the result depends on the extent of concomitant cerebral damage, the perfect cases being those in which there is but negligible damage to the brain proper. In summary, the acute cases look like those with extradural clots; the more chronic are those in which a patient at the end of a day or so begins to get worse, to become drowsy and difficult, and to present some evidence of lateralization of the lesion. The need for bilateral exploration requires to be mentioned, but I feel sure that the clots are more often uni- than bi-lateral.

THE PULSE RATE IN HEAD INJURIES

What use can be made of the pulse rate as a sign of clot compression? I believe that it is a reliable guide provided that the rest of the picture fits in with it, but that by itself it is not an indication for operation. The great variability in the rate will have been observed in the cases outlined above, nor can any hard-and-fast rule be laid down

concerning it. A number of patients who are going to make uninterrupted recoveries have very slow pulse rates; indeed, it is quite impossible to tell from the initial pulse rate alone what the prognosis of the case is going to be. It is true that a very slow pulse rate is a danger signal, but it can very well be a false alarm.

A.—Pulse rate on admission of a series of fatal cases: 64, 88, 96, 80, 60, 56, 140, 54, 88, 50.

B.—Pulse rate on admission of a series of recoveries: 88, 72, 76, 58, 140, 80, 72, 64, 68, 50, 76.

An interesting point is the fact that a very slow pulse rate may develop after the first few days and remain during the period of recovery. In children, in particular, the initial rate is fast, whilst in adults primary shock, a condition seen in purity only in uncomplicated head injuries, will equally cause a rapid pulse. On the whole, slow pulse rates are seen during the first few hours more commonly in the fatal cases than in others. There is, however, a very interesting phenomenon to which I wish to call attention, and that is secondary bradycardia setting in towards the end of the first week and continuing during the second. This has been a not uncommon finding in the present series, and is due to reactionary oedema. Often enough the patients during this phase have been entirely conscious, and apart from the bradycardia their condition has given rise to no alarm. Sometimes they have had very severe headache, but not necessarily so; sometimes no more than a feeling of fullness or oppression in the head has been experienced. We have often kept a patient back in hospital on account of his slow pulse when he has felt well and pressed for his release, and then, no untoward signs having developed, he has finally been allowed to go. It might be assumed that in all such cases with secondary bradycardia the cerebro-spinal fluid pressures would be elevated, but in three separate cases with pulse rates of 56, 48, 54, the cerebro-spinal fluid pressure registered 11, 10, 11 cm. respectively—that is, entirely normal readings. The accompanying chart (Fig. 2)

FIG. 2.—Time in half-days.

illustrates secondary bradycardia continuing over several days in a patient making a perfect clinical recovery, and this chart could be duplicated many times from our material.

In conclusion, I believe that the classification of head injuries on a basis of stupor is a more correct method than that which categorizes the cases according to evidences of fracture. This alteration I have made tentatively to-day. Signs of local contusion must be carefully looked for and recorded, whilst every effort is made to distinguish the epiphenomena, the alterations in behaviour and clinical state induced by the laying down of another condition—local compression by subdural or epidural haematoma on the "basic state" of general contusion.

I must express my indebtedness to Mr. G. F. Rowbotham and Mr. R. J. C. Maxwell for the trouble they have taken in helping in the search of hospital records.

ULCERATIVE COLITIS

A SURVEY OF NINETY-FIVE CASES

BY

T. L. HARDY, M.A., M.D., F.R.C.P.

PHYSICIAN, GENERAL HOSPITAL, BIRMINGHAM

AND

ERNEST BULMER, M.D., M.R.C.P.Ed.,

M.R.C.P.LOND.

ASSISTANT PHYSICIAN, GENERAL HOSPITAL, BIRMINGHAM

The disease known as ulcerative colitis, first clearly differentiated from other forms of colon ulceration by Hale-White in 1888, is a severe inflammatory disorder of the colon, in whole or in part. Clinically it is characterized by rectal discharges of blood, mucus, and pus, and is accompanied by constitutional disturbances such as fever, secondary anaemia, loss of weight, and prostration in varying degrees. A prolonged course, with marked tendency to relapse, completes this brief definition of a disease which, despite much endeavour, remains obscure in its aetiology, uncertain in its course, and fickle in its response to treatment.

Though perhaps of infrequent occurrence in the practice of the individual, the condition is common enough in the medical wards of our hospitals, and constitutes one of the outstanding problems of present-day practice. The often insidious nature of the onset, the slow progression with the late appearance of toxic symptoms, the apparent clinical recovery in certain cases with relapse sooner or later, encourage a false sense of security and obscure the potential dangers of this malady. It is useful in such a disease to avail oneself of the records of a large general hospital over a number of years, and thus obtain a composite picture beyond the experience of the individual. Such a survey has been attempted and the results embodied in this paper, particular attention being paid to the life-history of the disorder. So far as we are aware a series of figures comparable to our own has not appeared within recent years in the medical literature of this country, with the exception of a valuable and exhaustive paper by Hern.¹

No attempt has been made in the present communication to contribute to the vexed question of aetiology, but it may be said that our records give no support to the view, now generally discarded, that ulcerative colitis is a phase of bacillary dysentery. On the other hand, the alleged specificity of Bagen's diplo-streptococcus has yet to be proved, and his special serum is still in the early days of its trial in this country. We are not in a position to make any observations on these two points from our case records. On reviewing the literature it would appear that, apart from the introduction of methods of precision such as the sigmoidoscope and more recently the opaque enema in diagnosis, little has been added to our knowledge of this disease during the present century, so that the earlier contributions to the subject by Hale-White,² Allchin,³ and Hawkins,⁴ among others, require little or no modification with the passage of time.

SCOPE OF INQUIRY

The material on which this paper is based has been derived in the main from a systematic search of the records of the General Hospital, Birmingham, for the period January, 1920, to October, 1932. Apart from a few patients who came under our observation in private practice, the series is a consecutive one, and represents the experience of a general hospital of some 500 beds. During the period under review 104 patients were admitted to hospital with ulcerative colitis, and as a result of inquiry, either by personal interview or by report