me to be far too ready to deny their less debilitated lung-cancer patients the benefits of the treatment which is available to them to-day. We have started an investigation in conjunction with the general practitioners who send patients to us, to try to assess how far the palliative treatment given has been worth while. We are anxious to know what quality of life these patients lead before death and how they die.

Surgery offers a real chance of cure to the fortunate few, but does not at present do more than begin to touch the fringe of the problem presented by the thousands of patients dying with lung cancer. Radiotherapy is shouldering the burden of treatment to an increasing degree, and is doing useful work in relieving suffering in a way which is not possible at present by any other means, and is still not sufficiently available to all who need it. Chemotherapy with the nitrogen mustards has so far, in my experience, added nothing to this group of patients to compensate for the general ill effects it has produced, let alone done anything to advance treatment.

Conclusions

The startling rise in the recorded death rate from lung cancer is in large part due to change in numbers and age of the population and to improved diagnosis. It is due in part to a real increase, but we are not yet in a position to say how great that real increase is.

The size and the difficulty of this problem are clear to us all. Although the five-year survival rate for the fortunate few who are operable when they first reach hospital has risen from nothing to between 20 and 30% in 20 years, and while much can be done with *x*-ray treatment to relieve suffering and sometimes prolong life for the less fortunate ones who are, nevertheless, still in fairly good general condition, there are still thousands of these patients each year for whom no real attempt at treatment is being made. Since we all have to die, since some 90,000 people are doing so each year with lung symptoms, and since this number is falling well, we should ask ourselves how far we are performing a useful service by helping to make a public issue of a comparatively small change within that group, which may be in large part due to our own method of recording. We should not be too readily swayed by those who demand that the public be told "the truth" while we are still attempting to sort the facts from the fancies for ourselves, especially since "the truth" when told may not appear to them in at all the same light as it does to us. Anthony Hope once wrote that "telling the truth to people who misunderstand you is generally promoting a falsehood."

The moment the word cancer is mentioned to the public (of which doctors are but a part) emotion is aroused and any calm objective view of the value of evidence presented becomes most difficult. It is important that the medical profession take note of this difficult problem of lung cancer and help to deal with it more efficiently, but it is also important that they do not make matters worse by merely joining in a cry and increasing the alarm. A sensible view of the relationship of smoking to this problem, for instance, should be and, after some wild comments in the lay and medical press, is now being presented to the general public; but no comparable effort seems to be going into an endeavour to persuade the authorities to cleanse the air of our industrial towns. As a profession which speaks so much and so rightly of the need to allay the cancer fear we should beware of putting extravagant accounts of rising cancer death rates and their causes before the public, especially when neither the magnitude of the one nor the degree or responsibility of the other has yet been fully established.

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FACTORS IN THE MORTALITY OF CLOSED HEAD INJURIES

BY

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It has been estimated that in this country every year road accidents account for over 20,000 cases of head injury severe enough to require admission to hospital. Their number and mortality are matters of some concern. Mortality figures for non-missile head injuries vary widely, depending on the selection of cases and on the circumstances in which the particular series is compiled, but most clinics in this country and abroad quote rates of 12–20%, and emphasize that in 60–70% of the fatal cases death occurs within the first twenty-four hours. These early deaths are due frequently to the severity of the brain lesion, but there is a danger of assuming too readily that such is necessarily the case and of failing to recognize those complications for which treatment may be effective. Some early fatalities —and later ones too—are due to intracranial haematomas; and there are other cases in which extracranial factors such as chest complications are responsible.

The remarkable capacity of the brain to recover from or compensate for the effects of injury has also to be remembered. Thus, aphasia and hemiplegia are com-

monly seen in the early days after injury, but they do not frequently cause permanent disability. Patients with very severe lacerations of the brain may survive for days or weeks. Brain-stem lesions carry a high mortality, but even with them recovery is possible: in the present series one patient with clinical evidence of such a lesion remained unconscious for six months following injury, and thereafter made a steady recovery, so that ten months after the accident she was talking and was later able to return home and look after her family.

Such observations make it pertinent to inquire more closely into the immediate causes of death in these cases, even though in the last analysis the head injury may have been responsible, and to seek ways in which the mortality may be reduced. Broadly speaking, the factors responsible may be divided into four groups: (1) the effect of the injury on the brain itself; (2) intracranial complications, particularly haemorrhage, and to a less extent oedema and infection; (3) extracranial factors such as chest complications, fat embolism, and metabolic disorders; and (4) injuries elsewhere in the body and other incidents, as, for example, cardiovascular complications precipitated by the injury.

Many of these factors are interrelated, but in this paper attention is directed especially to three factors which previous experience had indicated were important in the mortality rate of head injuries and for which treatment may be effective—namely, intracranial haematoma, chest complications, and metabolic disorders.

Case Material

Between November, 1948, and February, 1952 (three and a quarter years), 1,000 patients were admitted to the accident service of the Radcliffe Infirmary, Oxford, with closed (non-missile) head injuries. As this hospital is the only one in Oxford to receive such cases and all accident cases are admitted to this service, the opportunity was taken to study an entirely unselected group of head injuries admitted direct from the scene of the accident. These numbered 821. The remaining 179 cases were transferred from other hospitals in the region for further treatment of a major head injury or a suspected complication.

Intracranial Haematoma

Although extradural, subdural, and intracerebral haematomas develop in no more than a small proportion of head injuries (6.2% in this series*), their recognition is important, for they are essentially acute operative problems and also carry a high mortality rate. A review a few years ago showed that when all cases, including those disclosed only at necropsy, are considered the average mortality from extradural haematoma was 50% and from acute subdural haematoma over 60% (Lewin, 1949).

The diagnostic problem was emphasized by James and Turner (1951), who found that in their district more patients died of untreated intracranial haematomas than were coming under their care. Recognition of them is not always easy, but, as a general guide in management, once the conscious level of a patient has begun to improve following the injury, then one should anticipate a steady recovery, even though this recovery may be arrested at any stage for hours, days, or weeks. Any deterioration in the patient's condition, whether it be in the level of his consciousness or in the development of fresh neurological signs, or both, should raise the question of an intracranial clot and lead to exploration if there is any doubt of the diagnosis. A full appreciation of the patient's conscious level and neurological state at the outset of treatment is the foundation for further management.

In practice the indications for exploratory burr-holes are usually clear-cut, and this useful procedure need not be applied indiscriminately. Thus in this series 97 patients were explored; in 56 an intracranial clot was disclosed, and in 8 a subdural hygroma—a proportion of positive to negative exploration of 2 : 1. In a further 9 patients exploration disclosed raised intracranial pressure from the effects of cerebral contusion and oedema, and led to a decompression being performed. Within this same period there were four patients in whom a clot was disclosed at necropsy. These four patients remained in deep coma from the time of injury until death; in two there was a subdural haematoma with cerebral contusion, the patients dying one hour and three hours after injury; in one there was an extradural haematoma on the one side and a subdural haematoma on the other, with cerebral contusion and a fractured base, the patient dying six hours after injury; and in the fourth patient, who died 56 hours after injury with clinical evidence of a brain-stem lesion, there was, in addition to bruising of the pons and mid-brain, a frontal intracerebral clot. It is not the purpose here to discuss whether surgical treatment in these patients could have affected the issue, but to record that they had intracranial clots and to emphasize that it is in this group of patients who remain in coma from the outset that so many of the diagnostic difficulties lie.

Extradural Haematoma

In only half the cases of extradural haematoma is the march of events in any way classical and uncomplicated by other factors. In the others there is, in addition to the extradural clot, severe cerebral contusion or clots in other situations. These patients are usually unconscious from the outset, and this accounts for many of the diagnostic problems. Thus there were 29 patients with extradural clots in this series, of whom 23 recovered and 6 died—a mortality of 20%. Of these 29 cases 12 were complicated by other factors; 5 of these were fatal (one undiagnosed, see above), and at necropsy all showed severe cerebral contusion with a fractured base. These patients are very worrying and may tax all the resources of a neurosurgical unit. Thus three of the seven recovered patients in this group required two or three operations before they finally improved. However, the remaining 17 cases of extradural haematoma were uncomplicated and only one was fatal: the patient stopped breathing as he came into the admission room, and did not recover, although the extradural clot was evacuated immediately under artificial respiration. It should be remarked that 10 of these 17 patients were operated on at a time when their pupils were equal and reacting. A dilated pupil in these circumstances is not so much a sign of extradural haematoma as an indication of impending death.

Delay in operating also results in an increase in the mortality: extradural haematoma is one of the few surgical emergencies in which success or failure is so dependent on the time factor. For example, if there has been a short

TABLE I.—*Intracranial Haematoma in 1,000 Cases of Closed Head Injury (69 Haematomas in 62 Patients, of whom 43 Recovered and 19 Died. Gross Mortality, 30.6%)*

Type	Single Haematoma		With Subdural		With Intracerebral		With Extradural		All Cases		
	R	D	R	D	R	D	R	D	R	D	Total
Extradural ..	21	2	2	3*	0	1	—	—	23	6	29
Acute subdural (<14 days after injury) ..	13	9†	—	—	0	1	2	3	15	13	28
Chronic subdural (>14 days after injury) ..	3	0	—	—	0	0	0	0	3	0	3
Intracerebral ..	4	3*	0	1	—	—	0	1	4	5	9

R = Recovered; D = Died.
* Includes 1 case disclosed at necropsy.
† Includes 2 cases disclosed at necropsy.

*62 patients with haematomas in the total series. Among the direct admissions there were 32, an incidence of 3.7%.

lucid interval of up to four hours, and then a rapid onset of coma with a dilated pupil, exploration should be made at once. The melancholy fact is that the patients in this group, untreated, are usually dead within six hours of the onset of coma and are probably beyond recovery for some time before this.

Another point concerns the nature of the exploration. It is not sufficiently realized that nearly one-third of these clots (in this series 31%) occur in sites other than temporal—frontal, parieto-occipital, or cerebellar—and the teaching that a temporal burr-hole is necessarily the correct exploratory site leads to these clots being missed. The site of scalp bruising and laceration, and the position of a fracture line, are reliable guides to the site of the haematoma, and burr-hole exploration should be made accordingly.

Thus, although there will always be an appreciable mortality in extradural haematoma for the reason that nearly half the cases are complicated by other brain lesions, with timely surgery the mortality from an uncomplicated extradural clot should be low.

Acute Subdural Haematoma

By contrast, acute subdural haematoma, by which is meant a haematoma coming to operation or necropsy within 14 days of injury and large enough to be causing cerebral compression, usually accompanies cerebral contusion and laceration, and is therefore associated with a correspondingly high mortality rate. Of 28 patients in this series with acute subdural haematoma, 15 recovered and 13 died—a mortality rate of 46%. Doubt has been expressed whether surgery has anything to offer during the first 24 hours after injury in these cases, in which deterioration in the patient's condition is so often due more to the cerebral lesion than to the pressure of the clot; but it is difficult to see how in some cases such a differentiation may be made clinically. It is safer to explore when there is evidence of compression and also of deterioration in the patient's condition. The mortality at this time is certainly high: of 12 patients operated on within the first 24 hours, three recovered and nine died. However, three patients did recover, and in retrospect this number might well have been increased by at least two if an early tracheotomy had been recommended in one patient and a further decompression performed in the other—points which illustrate the interdependence of the various factors present in the acute head injury.

Decompressive Operations.—Burr-hole drainage of the clot may be enough if the haematoma is fluid and is very large, but in our experience there have been several cases in which this proved inadequate and the underlying cerebral contusion and oedema has required a decompression. Thus in the 20 patients of this series with acute subdural haematoma in whom at operation no clot other than the subdural was found, a bony decompression was performed in 11, in addition to drainage of the surface haematoma. Six of the 11 patients so treated recovered, and it was estimated that in at least 8 of the 11 patients the underlying brain damage was as much a factor in their clinical picture as the surface clot. There are also some cases of cerebral contusion without gross haematoma in which the bruising and oedema lead to raised intracranial pressure and steady deterioration in the patient's condition. A timely decompression in these cases may prove helpful; of nine such patients in this series in whom a decompression was provided by a bone flap or a subtemporal craniectomy, five recovered.

The high mortality of this severely injured group of patients may have resulted in a too conservative swing in their management, and the fact that some of these patients may be saved by early surgery requires reconsideration.

Acute Traumatic Intracerebral Haematoma

Comparatively little attention has been given to the possibilities of surgery in this condition, but with the help of angiography in suspected cases in the future it should be possible to diagnose these haematomas more often and to attempt treatment. Small intracortical clots and lacerations filled with clot are common enough, but these intracerebral clots are truly subcortical and usually measure 5 cm. or more in diameter. They may occur at all ages and in brains with apparently healthy vascular trees. There were nine such cases in this series. Decompression alone would seem inadequate for their treatment as judged by the fatal issue in the three patients in whom this was done; on the other hand, four of the five patients in whom the clot was evacuated through a cortical incision recovered.

Chest Complications

Intracranial haematomas are uncommon, but, unfortunately, chest complications are all too common, and almost the rule in any case of prolonged coma. They are undoubtedly the cause of many deaths after head injury, and this is not surprising when we remember the ways in which the chest may be involved soon after injury. Thus there may be an associated chest injury with haemothorax and pneumothorax. A serious head and chest injury is a very lethal combination; there were 15 such cases in this series, with six recoveries and nine deaths.

The second way, and by far the commonest, is by the inhalation of vomitus or of blood from a fractured base during the early hours after the injury, when the patient is still comatose. Such a calamity is promoted by the almost invariable rule of transporting these patients on their backs, and by nursing a comatose patient with his head elevated in order to lower his intracranial pressure. This it may do, but the small benefit derived is nothing compared with the fatal risks of such a position. Massive inhalation may result in pulmonary collapse and immediate death, but a treacherous condition may also arise in those patients who do not die immediately. The main air passages may have been cleared by suction or by coughing, but blood and vomitus remain in the small bronchioles. The resulting areas of peripheral pulmonary collapse and oedema leave the patient with a diminished pulmonary reserve, and then a further minor incident may be terminal.

A third complication is pulmonary fat embolism, seen usually in patients with multiple injuries and coming on some hours after the injury. There have been at least eight such cases in this series, and there were probably others which were not detected.

Fourthly, there is the hyperacute pulmonary oedema secondary to brain-stem compression, a condition well known to neurosurgeons and which has recently been produced experimentally by Cameron (1948). Its sudden onset, with the production of quantities of thin blood-stained fluid, may be as dramatic as is its relief if the intracranial pressure can be reduced as by evacuation of a clot.

The lungs, therefore, are particularly susceptible following head injury, and, in those patients who survive these early complications, secondary bronchopneumonia or even lung abscess may develop later, or may follow as a result of prolonged coma.

Effect on the Brain.—The effect of the head injury and the accompanying unconsciousness on the lungs is not, however, the only problem. Anoxia and chest infection have a profound adverse effect on the brain, perhaps especially after recent injury. The brain cannot tolerate even a few minutes' anoxia (Howkins *et al.*, 1946); yet this is a not infrequent risk following head injury, with the upper respiratory tract filled with secretions and the patient cyanosed and breathing rapidly and irregularly. Moreover, it has been recognized for a long time that infection elsewhere in the body adversely affects a neurological lesion: witness the loss of tone in a paraplegic patient who develops a urinary infection, or the access of neurological signs in the child with a cerebellar tumour who develops an incidental infection. In patients with head injuries the same obtains. Onset of chest infection halts recovery and may also result in a deterioration of the patient's mental condition.

Treatment of Chest Complications

To mitigate the effects of these chest complications the basic treatment for the unconscious patient in this series has been nursing, prophylactic penicillin, and oxygen. The first essential was to obtain an unobstructed airway 24 hours a day. The patients were nursed flat from the outset, lying on their side, and, if that was not completely satisfactory, in the semi-prone or "tonsil" position. They were turned from side to side two-hourly. Suction was available at the bedside of all patients in coma so that the nurse could easily clear away secretions in the nose and mouth with a soft rubber catheter. Physiotherapy twice daily to favour full expansion of the lung bases was also given, and if there was difficulty in clearing the upper respiratory tract efficiently there was no hesitation in giving these patients the benefit of full postural drainage with the foot of the bed elevated for short periods. These methods were supplemented where necessary by the use of simple airways, and oxygen was given intermittently or, in the severest cases, continuously.

In these ways it was possible to prevent and treat many of these chest complications. Although it is difficult to present reliable figures, the use of antibiotics has undoubtedly helped to prevent and to lessen the effects of chest infection.

Tracheobronchial Toilet

In some cases in which the obstruction extended down into the trachea and bronchi it was not possible to clear them by the measures described above. In this event the anaesthetist has sucked out the main air passages either by a catheter passed down the trachea directly or through an endotracheal tube which was afterwards removed. These methods proved valuable in the early hours after the accident and were also successfully employed in eight patients at a later phase of their illness. In a further three patients, bronchoscopy was successful in relieving a lobar collapse.

Where it became necessary to intubate a deeply comatose patient soon after admission and the airway was found to be unsatisfactory without the endotracheal tube, it was sometimes left in for some hours. Endotracheal intubation in these circumstances is useful, but experience has shown that it is unwise to continue this method for many hours, particularly if it looks as if the patient may survive the immediate effects of the brain injury and is likely to remain in coma for some days. The tube becomes encrusted with secretions and is less effective; and frequent endotracheal intubation leads to oedema of the pharynx and larynx with ulceration, so that ultimately the respiratory embarrassment is aggravated rather than relieved.

Tracheotomy

It was in such circumstances that tracheotomy was first employed in March, 1949. In this series 13 tracheotomies were performed. In none have we regretted doing it, and in some we wished it had been done earlier. There have been no complications following this operation, even though many of them were performed when the patients were desperately ill. In some instances the operation took place in the patient's room so as to disturb him as little as possible. The figures in regard to ultimate recovery are not impressive, since this resulted in only 3 of the 13 patients; but this emphasizes that operation was considered only in the severest cases, and the benefit of special nursing which was available to all these patients rendered the number of cases in which the question of a tracheotomy arose relatively few. It is perhaps more important to observe that in all cases any chest infection present cleared up once the tracheotomy was performed, and the improvement in the patient's condition was usually immediately apparent; it became easy to clear the trachea adequately, there was no further trouble with the airway, and the patient could now be nursed with the head elevated if desired. It was striking to observe a bilateral bronchopneumonia resolving rapidly after tracheotomy. Another feature noticed was the long survival of some patients with severe brain injuries who had a tracheotomy. Thus it was possible to nurse four patients in coma for periods of eight to twelve weeks and two others for periods of a year without chest complications.

To date, of 18 patients who have had tracheotomy performed, nine improved to such a degree that the tube could be removed; of these, four recovered satisfactorily and two are still improving. In patients from whom the tube was removed the stoma closed quickly without complications.

The experience of these first cases suggests that tracheotomy is a valuable adjunct in the treatment of selected cases, although one would deprecate its use save in the severest head injuries, for simpler measures and good nursing will usually suffice. An early tracheotomy should be seriously considered in patients who have survived the early hours but are likely to remain in coma for some days, and in whom respiratory incidents are beginning to appear. It is useless to wait until the patient is *in extremis* and has already suffered the effects of anoxia. Some of our early cases were operated on too late. Of the last four patients who underwent tracheotomy only one died, and necropsy of that patient showed a normal respiratory tract. Similar experiences of tracheotomy have been reported from America by Echols *et al.* (1950), who advise it in cases in which coma is likely to persist more than 24 hours, and in which other methods of maintaining an adequate airway are inefficient.

Results

Table II summarizes the chest complications present in this series. The total incidence was 6.7%, which includes every case at necropsy in which anything more than mild basal oedema was found. Thus 30 cases were listed as "contributory" in view of the necropsy findings, even though clinically there was no evidence that the lung changes played an important part in causing death. However, the fact that nearly half of all the fatal cases showed significant lung changes illustrates the importance of this extracranial factor.

TABLE II.—*Chest Complications in 1,000 Cases of Closed Head Injury*

Chest Complication	Recovered	Died		Total
		Main Cause of Death	Contributory	
Injury	6	4	5	15
Bronchopneumonia	10	5	11	26
Pulmonary collapse	4	3	5	12
Pulmonary oedema	1	—	4	5
Lung abscess	—	1	—	1
Fat embolism	2	1	5	8
Total	23	14	30	67

Metabolic Disorders

It has been known for a long time that disturbances of fluid and electrolytic balance may arise in comatose patients, but their nature and the extent to which they are present in patients with head injuries who survive the early hours after injury have been little studied. The dangers of dehydration, whether from inability to administer fluids adequately to the unconscious patient, or as a result of over-zealous application of dehydration as a method of treatment in head injuries, have been recognized (Munro, 1938). Cumings (1942) reported some cases of uraemia following head injury; and earlier in this series (Higgins *et al.*, 1951) it was observed that some patients developed a striking disturbance of chloride balance characterized by retention of this ion in the blood without a corresponding excretion in the urine.

It was decided, therefore, to study biochemically a consecutive series of patients who after the first 24 hours following injury remained sufficiently unconscious to require tube feeding, and to follow their progress until recovery of

consciousness or death. It may be mentioned here that our general routine of treatment was to pass a small rubber oesophageal tube or polythene tube through the nose 12 to 24 hours after injury, and to administer fluids, three to four pints (1.7 to 2.3 litres) during the first day, and thereafter increasing the amount as required. All our patients were fed by mouth, and it was not found necessary to resort to intravenous drips for feeding purposes. The details of the treatment and the full biochemical findings are described elsewhere (Higgins *et al.*, in press).

Table III summarizes the results. The high incidence of transient disorders such as glycosuria and changes in the plasma proteins, which in general corrected themselves without specific treatment, were of interest, since they provided

TABLE III.—*Metabolic Disorders in 76 Unconscious Patients*

	No. of Patients
Normal	8
Transient disorders	50
Proteinuria .. 32 of 44 patients examined	
Raised blood urea 22 of 50 ,, ,,	
Low plasma protein 22 of 50 ,, ,,	
Glycosuria .. 13 of 50 ,, ,,	
Major disorders	18
Hyperchloraemia and hypochloruria	9
Hypochloraemia and hyperchloruria	5
Water deprivation	2
Respiratory alkalosis	2
Renal uraemia	3
	76

further evidence of the general systemic disturbance which may follow head injury, although they were of little importance therapeutically. On the other hand, the 21 major metabolic disorders which developed in 18 patients deserve further comment, for, from earlier experience with these disorders and with the results of similar disturbances in cases unassociated with head injury, it would appear that they are not usually correctable spontaneously and are associated with a high mortality. It is believed, therefore, that the successful therapeutic correction of these disorders in 12 of the 18 patients, with recovery of seven patients, played a part in lowering the overall mortality, although it will be appreciated how difficult it is in such a complex setting to ascribe recovery or death to any one single factor. Water deprivation was uncommon in this series owing to the policy of giving fluids early, and in those cases in which therapeutic dehydration was employed the effect of this treatment on the patient was carefully watched.

It is not proposed to discuss here the causes of these metabolic disorders. Some certainly follow coma from whatever cause, but clearly the possibility of a central factor in some cases of head injury has to be borne in mind. Further work requires to be done on these metabolic changes, but the evidence suggests that the patient's fluid and electrolytic requirements are important aspects of his treatment and that the time has come when their assessment, at least in severe head injuries, should be made in conjunction with our biochemical colleagues.

Multiple Injuries

In addition to his head injury a patient may sustain other injuries which may influence considerably not only the prognosis of the case but also its management. Thus among 821 direct admissions there were, excluding those patients with simple contusions and lacerations, 269 cases with injuries in addition to the head injury—an incidence of 32.7%. Of these 269 patients 27 died, and, as indicated in Table VI, it was concluded that in seven cases at least death was due mainly to these other injuries. In 107 patients there were two or more injuries in addition to the head injury. Table IV indicates the principal injuries sustained. Fractures of the face and limbs were the commonest injuries. It was clear that the ready availability of specialist services within the framework of an accident service for the many problems that arose in their management proved an important factor in the successful outcome of many cases.

TABLE IV.—*Other Injuries Sustained by 269 Patients with Head Injuries (All Direct Admissions. Incidence 32.7%)*

Injury	No.
Fractures	332
Face (malar only, 50; nasal bones only, 20; maxillae, 37)	107
Upper limb (clavicle, 40; shoulder, 14; humerus, 6; forearm and wrist, 35; hand, 13)	108
Lower limb (pelvis, 15; femur, 17; knee, 6; leg, 32; foot, 5)	75
Spine (body, 15; processes, 10)	25
Thorax (ribs only)	17
Chest (12 with rib fracture)	13
Abdomen	8
Spinal cord and peripheral nerves, without fracture	8
Eyes	7
Major facial lacerations and soft-tissue injuries	15
	383

One difficulty in the management of the unconscious patient who has multiple injuries is the recognition that other injuries are present, fractures of the spine and pelvis and injuries to the chest and abdomen being easily missed unless systematically looked for; another is to decide which injury should take precedence of treatment and to accept in some cases a compromise between what is ideal for the particular injury and what is practicable in the circumstances. One should not accept unnecessary delay in the treatment of other injuries merely on the grounds that the patient has had a head injury, for this may delay recovery from shock and increase the risks of infection; but it is also important, particularly in the early hours after injury, to avoid, if possible, procedures which may interfere with the essential management of the unconscious patient or mask the developing signs of an intracranial complication.

Results

Table V summarizes the results in this series. The overall mortality was 9%, weighted by the higher mortality of those selected cases transferred from other hospitals with severe head injuries and complications. Of the 821 patients admitted directly from the accident 62 died, a mortality of 7.5%.

TABLE V.—*Mortality in 1,000 Consecutive Cases of Closed Head Injury*

	Cases	Deaths	%
Direct admissions	821	62	7·5
Transferred cases	179	28	15·6
Total ..	1,000	90	9·0

Cause of Death.—The cause of death in the 62 fatal cases among the unselected group has been assessed on the clinical evidence and necropsy findings (Table VI). A

TABLE VI.—*Cause of Death in 62 Direct Admissions*

Cause	No. of Cases
Head (cerebral contusion, 38; intracranial haematoma, 7; cerebral haemorrhage from vascular disease precipitated by minor head injury, 3)	48
Chest complications (pulmonary collapse (inhaled vomitus), 2; bronchopneumonia, 3)	5
Other injuries (chest, 4; ruptured liver, 1; multiple fractures, 2)	7
Other causes (coronary thrombosis, 1; hypertensive cardiac failure, 1)	2
	62

necropsy was performed in all but three cases. Although there were seven deaths due mainly to other injuries, two others from heart disease, and three in which the clinical and necropsy evidence left little doubt that intracranial haemorrhage from cerebrovascular disease had been precipitated by a trivial head injury, these have nevertheless been included in the total number of deaths, for a more complete account is then given of the many problems involved in accident work. Where there has been any doubt about the main cause of death the rule has been to ascribe it to the head injury. The three patients who died mainly from bronchopneumonia were all over 70 years of age. Of the 45 patients who died directly as a result of the head injury, five died within one hour of the accident.

Mortality of Head Injury.—To set alongside these figures one should mention those patients with minor head injuries who were treated in the casualty department and then sent home, and also those who died before reaching hospital. Within the period under review at least as many cases were treated in the casualty department for minor head injuries and sent home as were admitted. With the kind help of the local authorities it has also been found that during this same period 12 patients died of head injury on the roads before they reached hospital. One estimates, therefore, that the mortality of cases of head injury severe enough to be admitted to hospital was certainly well below 10%.

It may be encouraging to note that whereas the gross mortality of the first 500 cases in this series was 11.4%, in the last 500 cases it was 6.6%, and this lower figure has been maintained to date. The series would have to be extended much further, however, before one could conclude that this apparent improvement was due to anything other than extraneous circumstances.

Summary and Conclusions

Attention is drawn to the frequency and mortality of head injuries, and it is suggested that in addition to the immediate effects of the brain injury there are other factors, some of which are preventable or amenable to treatment, which play an important part in the mortality rate. The paper is based on 1,000 consecutive cases of head injury admitted to an accident service in the last three and a quarter years.

Intracranial haematoma developed in 3.7% of an unselected series. The need for urgency in the evacuation of extradural haematoma, and the incidence of these clots in sites other than temporal, are stressed. The value of decompression in some cases of acute subdural haematoma and in certain other cases, and the possibilities of surgery in acute intracerebral haematoma, are discussed.

Chest complications are common in head injuries and account for many deaths. Their causes and treatment are outlined. The use of tracheotomy in selected cases is described.

Evidence is presented to show that metabolic disorders may develop in these patients. It is suggested that their management, and attention to the patient's fluid and electrolytic requirements in the light of the biochemical findings, may help in lowering the mortality rate.

Injuries to other parts occur in over 30% of cases. Their frequency and variety illustrate the advantages of treating head injuries within the framework of an accident service.

The overall mortality of the 1,000 cases was 9%. Among the 821 unselected cases there were 62 deaths, a mortality of 7.5%.

It is concluded that there are several ways by which the mortality rate of head injuries may be reduced. Although further attention to intracranial complications is required, it is believed that as much benefit will be derived from the prevention and treatment of some of the extracranial factors.

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The problem of head injuries remained one of Sir Hugh Cairns's prime interests throughout his career. It was my privilege to have had the benefit of his advice and constant help throughout this study, and I wish to record my debt to him.

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STREPTOMYCIN RESISTANCE IN PATIENTS WITH PULMONARY TUBERCULOSIS PREVIOUSLY TREATED WITH P.A.S. ALONE

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In the treatment of pulmonary tuberculosis with streptomycin alone the emergence of strains of tubercle bacilli resistant to the drug is well known. That the same thing happens when isoniazid is used alone is now established (Medical Research Council, 1952a). But the possibility of resistant strains emerging when cases of pulmonary tuberculosis are treated with sodium or calcium *para*-aminosalicylate (P.A.S.) alone has received much less attention. This is perhaps partly because of the less rapid emergence of resistant organisms when an attempt is made to induce P.A.S. resistance *in vitro* (Steenken and Wolinsky, 1950) and partly because of the greater technical difficulty of the resistance tests. Nevertheless, the emergence of P.A.S.-resistant strains in a large proportion of treated cases has been reported in the literature. Some of these reports are summarized in Table I.

The results suggest that P.A.S.-resistant strains may emerge at a later period of treatment than is usual when streptomycin or isoniazid is used alone, but that, with prolonged treatment, they are isolated from a notable proportion of patients who remain sputum-positive. It is logical to imply that if these patients are later treated with streptomycin and P.A.S. in combination P.A.S. will no longer prevent the emergence of streptomycin-resistant tubercle bacilli. This probability has often been emphasized but is still ignored by many clinicians, partly perhaps because no direct evidence has, so far as we know, been provided. In addition it has been suggested that resistance to P.A.S. is transient and, by impli-

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