The Acute Head Injury: a Multidisciplinary Problem*

A. Earl Walker, M.D.**

Introduction

Your invitation to be a guest speaker at the 1967 meeting of the Japan Neurosurgical Society brought back vivid memories of my visit to your Society seven years ago. In view of those pleasant occasions, I am delighted to be with you to discuss the most serious problem of our modern society, namely traffic accidents.

I do not intend to describe the techniques of the repair of cranial wounds nor the care of intracranial lesions but rather, I would call your attention to the impact of complicating injuries of other parts of the body which may tip the delicate balance between life and death.

The problems of the head-injured patient today are more complicated than those of a few decades ago (Table I). This increasing complexity of the trauma victim stems from the increasing pace of our civilization, manifested by our people driving themselves and their cars faster and faster and using more drugs and more alcoholic beverages. These factors predispose to traffic accidents, the major cause of serious head injuries. The added violence not only aggravates the brain damage but compounds the problem by inflicting multiple injuries (Table 2). The location of the associated injuries vary depending upon a number of factors; particularly

Table 1. Average Number of Injuries per Traffic Accident (Brisbane Series)5)

<table>
<thead>
<tr>
<th>Year</th>
<th>Average Number</th>
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<tbody>
<tr>
<td>1935–39</td>
<td>4.6</td>
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<tr>
<td>1940–44</td>
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<td>6.3</td>
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<tr>
<td>1960–63</td>
<td>6.9</td>
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important is whether the victim was a pedestrian or an occupant of a vehicle (Table 3). Pedestrians are likely to have associated fractures of the extremities and occupants of a car, chest and/or face lesions. With a second wound complicating almost half of the head injuries resulting from traffic accidents, the question is pertinent as to its effect on the course of the craniocerebral lesion. Does a fractured leg with some attendant shock, attenuate or aggravate the effect of a closed head injury? It has been stated that intracranial bleeding is less likely to occur in the presence of shock, but the evidence for this statement is largely conjectural. Although the problem has been recognized, little experimental work has been carried out on the subject. However, even if the compounded effects are poorly understood, there is no doubt that multiple injuries greatly complicate the practical treatment of the patient requiring the cooperative efforts of specialists in respiratory physiology, in the treatment of fractures, in the repair of abdominal injuries, in the treatment of shock, in electrolyte balance and in the treatment of wounds of the paranasal sinuses.

1. Respiratory Complications

There is general agreement that in trauma of all types, an adequate respiratory exchange is essential for the proper repair of tissues. This is particularly true in head injuries for even mild degrees of cerebral hypoxia will cause severe alterations in vital functions both directly and as the result of secondary vascular changes. For this reason, the primary consideration in the treatment of any head injured patient is the establishment of an adequate airway to ensure sufficient respiratory exchange to oxygenate the blood. This may require aspirating debris, blood or

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<th>Road Accidents N=4954</th>
<th>Other Accidents N=6125</th>
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<tr>
<td>Single Injury</td>
<td>65% (3230)</td>
<td>82.5% (5045)</td>
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<tr>
<td>Multiple Injury</td>
<td>35% (1724)</td>
<td>17.5% (1080)</td>
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Table 2. Incidence of Multiple Injuries in Accidents of Various Types

<table>
<thead>
<tr>
<th></th>
<th>Heidelberg N=4954</th>
<th>Birmingham N=500 (fatal)</th>
<th>Canada N=566</th>
<th>Brisbane N=1,000</th>
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<tr>
<td>Head Injury</td>
<td>71%</td>
<td>72%</td>
<td>57%</td>
<td>67%</td>
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<tr>
<td>Lower Limbs</td>
<td>30</td>
<td>29</td>
<td>21</td>
<td>41</td>
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<tr>
<td>Upper Limbs</td>
<td>17</td>
<td>—</td>
<td>15</td>
<td>—</td>
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<tr>
<td>Chest Injury</td>
<td>10</td>
<td>42</td>
<td>20</td>
<td>20</td>
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<tr>
<td>Abdominal and Pelvic Injury</td>
<td>8</td>
<td>45</td>
<td>16</td>
<td>18</td>
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<tr>
<td>Spinal Injury</td>
<td>3</td>
<td>—</td>
<td>3</td>
<td>6</td>
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Table 3. Sites of Multiple Injuries
vomitus from the throat and upper trachea. Some neurosurgeons perform a tracheostomy routinely in comatose patients with head injuries. The arguments for such a procedure are cogent, namely that the amount of dead space is decreased, the trachea can be readily cleaned and the airway may not pass through traumatized or lacerated tissues such as the lip, mouth or jaw. Undoubtedly such a course is wise in selected cases. However, as a routine we prefer to insert an intratracheal tube which may be left in place for a day or so. The introduction of the tube is usually quite easy in these patients. Through it, the secretions may be sucked from the trachea. If vomitus or blood has been aspirated, 50 cc of normal saline or 1.3% NaHCO₃ may be instilled into the trachea and the “boiling” concoction immediately suctioned as a toilet of the tracheobronchial tree. Hydrocortisone (100 mgm) may be added to the fluid. This technique has a place in the treatment when aspiration of vomitus is known but as a routine procedure it adds a risk of bronchial spasm and pulmonary atelectasis. Once a clear airway has been established, it is simple to attach the tube to a respirator to assist or to maintain respiration. Moreover, if a surgical operation is contemplated, the anesthesiologist is working in a much more satisfactory field than if a tracheostomy is carried out. The intratracheal tube may be removed in a few hours or even after a day or more, whereas, a tracheostomy tube must be left in place for days or weeks. Moreover, the patient with a tracheostomy requires constant nursing care which, in many hospitals at the present time, is difficult. This is particularly important in older individuals. In a series of head injuries we reported some years ago, individuals over 40 years of age with tracheostomy had a mortality rate of 70%; in patients below 40 the mortality was halved¹⁰). An intratracheal tube will give most of the advantages of a tracheostomy without the necessity of an operation. If after 24 or 48 hours the patient does not seem to be recovering and the respiratory problems continue, a tracheostomy may be carried out under much more ideal conditions than when the patient is first seen.

A second major respiratory consideration relates to trauma of the chest. Almost 50% of patients with head injuries resulting from traffic accidents have, as a second lesion, some damage to the rib cage. These injuries may not be severe or they may be so extensive that they impede respiratory efforts. For this reason, a very careful examination of the chest is essential in any head injured patient. The presence of pneumothorax or hemothorax, if suspected, should be verified radiologically or by needle tap and the air or blood removed so as to allow adequate pulmonary expansion.

Chest injuries impair gas exchange, increase the respiratory effort and raise the intrathoracic pressure. Gross hemopneumothorax requires transfusions and suction drainage of the chest. The patient with a flail chest may require controlled respiration using an intermittent positive pressure ventilator for several weeks. Atelectasis tends to lower the oxygen tension of the arterial blood due to the decreased ventilation-perfusion ratio in the lung. The fall in O₂ tension stimulates chemoreceptors and respiration is increased. The net result is a respiratory
alkalosis and a degree of hypoxia. In the head injured patient with dilated cerebral vessels requiring more blood, the pulmonary alterations compound the disturbance. However, the administration of O₂ usually compensates for the shunts.

It is essential to minimize the intrapulmonary pressure so that adequate venous return may occur from the brain and so that the mediastinal structures are not displaced causing cardiac and hence secondary vascular embarrassment.

Lacerations of the chest wall require careful care; a sucking wound must be closed and air or blood within the pleural sac removed by a tube left in for constant suction. Fractured ribs, if painful, are probably best immobilized by strapping. This does, however, limit the respiratory excursion and if the patient does not have too much pain such strapping may not be necessary. In all such cases the administration of oxygen and assisted respiration should be considered. The administration of oxygen routinely is probably advisable, but in the less severely injured case, it is not necessary provided the respiratory exchange is adequate.

A constant question arises in the care of the head injured patient with chest injury as to his or her position. In general, a semiprone or lateral position is desirable (Fig. 1), but as the patient stabilizes, a supine position is much more convenient for the attendants and if nasal secretions are not a problem, probably easier for the patient. An oscillating side to side mattress has been devised so that these patients can be shifted from one side to the other every minute or two, thus

![Fig. 1. Sketch to illustrate the lateral decubitus position which allows an adequate respiratory exchange and prevents aspiration of secretions (after Jamieson).](image)
increasing the pulmonary ventilation and tending to eliminate dependent or hypo-
static congestion which often occurs in the individual who lies upon his back fo
some period of time.

2. Cardiovascular Complication

The presence of multiple wounds is apt to produce impairment of the cardio-
vascular system by reason of hypovolemic shock. Consider that the tissues about
a fractured femur may hold more than 2,000 cc of blood and that intra- or retro-
peritoneal bleeding may be greater than a liter! Thus, without any external bleed-
ing there may be more than a 50% decrease in circulating blood with resultant
shock. The inadequate perfusion of vital organs may be due to deterioration of
cardiac function, inadequate blood volume or enlargement of the vascular space.
Although in trauma, hypovolemic shock is the rule and replacement by whole blood
is indicated as an emergency measure, other factors may be contributory. Precise
identification of the hemodynamic abnormality is necessary for the application of
symptomatic measures, such as the relief of lowered arterial pressure by vasocon-
strictors may aggravate the basic condition. The pulse rate and systolic blood
pressure have proved to be poor guides to the blood flow. A better indicator of
the intravascular volume is the central venous pressure or the venous pressure just
before the blood enters the right auricle. An adequate blood volume exists if this
tension is between 5–15 cm. of water, lower pressures are usually indicative of a
decreased blood volume or cardiac deficit and rarely a peripheral vascular
hypotonia. Guided by the central venous pressure, the blood volume should be
restored. If a volume in excess of the normal amount of circulating blood fails to
bring up the central venous pressure, the blood volume should be determined to
confirm the circulatory state of either persistent blood loss or peripheral vascular
hypotonia. If the shock persists in spite of a high central venous pressure, a cardiac
deficit is probable. For this isoproterenal increases both the rate and force of
cardiac contraction and at the same time decreases peripheral resistance. Given in
a saline solution, 0.1–0.2 mgm per hour of isoproterenal is usually adequate to
restore blood pressure, cardiac output and renal function. If multiple transfusions
are ineffective, peripheral pooling, a possible cause, may be combatted by low
molecular dextran; vasoconstrictors may be of value. In such conditions, the blood
lactic acid values may indicate the severity of tissue hypoxia. A persistently
elevated content may be helped by the use of a hyperbaric chamber.

3. Abdominal Complications

Although the abdomen is not so frequently injured as some other parts of the
body, if shock is present and unaccountable, the abdomen should be suspect even
if no external evidence of injury is apparent. In the initial hours after an intra-
abdominal laceration, signs of peritoneal irritation may be absent. This is particu-
larly true if the patient is restless or comatose. Accordingly, the abdominal contents should be explored by catheter, gastric tube and quadrantic paracentesis. The catheter will confirm the integrity of the urinary bladder and examination of the urine should reassure the physician that the patient does not have diabetes or nephritis, either of which would require specific therapeutic measures. The catheter is left in place so that the urinary output may be measured. Evacuation of the stomach contents by a gastric tube prevents aspiration of regurgitated vomitus. The tube should be left in place and suction applied. The insertion of a needle (15-16 gauge) in each quadrant to aspirate any peritoneal debris, such as hemorrhage, urine or intestinal fluid is a reliable technique to test for the presence of a ruptured viscus. The punctures may be repeated if shock continues in spite of blood replacement for the peritoneal extravasation may be delayed by an initial spasm of the arteries or gut.

4. Metabolic Disturbances

Damage to cerebral centers may interfere with normal metabolic processes through a number of mechanisms. Hypothalamic pituitary pathways are intimately related to water regulation, and vegetative hypothalamic centers modify temperature, respiratory rate, carbohydrate metabolism, electrolyte balance and osmolarity of the blood. Relatively minor injuries to these areas may temporarily arrest function and cause severe derangements lasting several days. Neurosurgeons are well acquainted with the rather striking arrest of function which may occur from very minimal insults to many parts of the brain. The insertion of a needle into the prefrontal area for an injection of an anesthetic agent or other substance may relieve pain within a few seconds before an injection can be made. The mere passage of a needle into the ventrolateral nucleus of the thalamus, at times, arrests tremor and relieves rigidity for some period of time. Relatively small lesions of the pituitary gland may produce temporary complete cessation of hypophyseal function. These are but a few examples of the very striking effects produced by minimal cerebral trauma. Similar spectacular alterations may occur as the result of trauma to the head.

Because water constitutes approximately 55% of the body weight, its regulation is particularly important. This is accentuated by the fact that water exchange is so rapid that even a relatively isolated portion of the body will receive water introduced into the general circulation within a matter of seconds. Not only the relationship of extracellular and intercellular fluid is closely related to the electrolytes, particularly to sodium and to a lesser extent to potassium. Sodium, which is mainly present in the extracellular fluid, exchanges rapidly except for the fraction (1%) within bone. Thus, about 70% of the body sodium may be exchanged or shifted within hours. Its normal cell concentration ranges between 136 and 144 milliequivalents per liter. On the other hand, potassium is mainly located intracellularly where it reaches a concentration of 146 milliequivalents per liter,
although its serum concentration is only 3 to 5.5 milliequivalents per liter. The concentration of these salts in the serum with the contributions by glucose and urea determine the serum osmolarity which normally ranges between 285 and 298 milliosmols per liter. Shifts in the total body water and serum sodium will change the osmolarity since in general, the glucose and blood urea nitrogen fractions remain fairly constant. It is apparent that the kidney holds the key to the concentration of these electrolytes and to the osmolarity of the blood. Cerebral influences modifying the kidney activity are mediated by the antidiuretic hormone (ADH) the most striking derangement of which produces diabetes insipidus in which the kidneys pass low concentration urine in great amounts. In some states, however, the electrolytes passed by the kidneys may be varied so that sodium is retained and potassium is excreted.

With these basic concepts in mind, one may consider the clinical patterns of electrolyte disturbance encountered in head injured patients. Alterations are, of course, not seen in all cases of head injury; in fact, even in severe head injuries associated with periods of unconsciousness for some days, the electrolytes may remain balanced. Moreover, there is no pattern of electrolyte disturbance which correlates with a lesion of a particular part of the brain. It is true, that wounds of the central or frontal region are a little more likely to give rise to such disturbances; perhaps they are more commonly associated with injury of the hypothalamic centers.

The most common response to injury is a hypo-osmolarity which occurs in the first few days with a return to normal limits. It is assumed that the hypothalamic centers stimulate both anterior and posterior pituitary glands, to secrete ACTH and ADH and that the ADH effect continues in spite of water loading. During this time there is a positive fluid and sodium balance while urine serum osmolarity ratios are consistently higher than 1. This commonly follows surgical trauma. The sodium retention usually begins within the first 48 hours, continues for 3 to 5 days and is then followed by a naturesis. Water retention occurs about the same time but may last a few days longer. Although the total body sodium is increased, the water retention is sufficient that sodium serum concentrations decrease producing a sodium paradox. This phenomenon has been termed "the inappropriate secretion of antidiuretic hormone".

The above hyponatremia is the usual posttraumatic state but the reverse may occur, namely a hypernatremia. The progressive increase in serum sodium concentration may be associated with a negative fluid balance or the sodium balance may remain constant while a net fluid loss occurs. This condition is thought in most cases to be due to a water deficit alone or to excessive loss of fluid by bowel or kidney such as occurs in diabetes insipidus, or through the use of dehydrating agents. Rarely it may be due to the administration of abnormally high amounts of sodium, particularly if the renal function is impaired. In most cases it seems likely that a combination of excessive renal water loss plus inadequate water replacement with or without an excessive sodium intake is responsible. Since the symptoma-
tology of hypo- and hypernatremia may be similar, it is sometimes difficult to distinguish these two conditions (Table 4). For this reason, blood examinations of the electrolytes are important. Occasionally electrolyte retention causing hypernatremia may occur where water depletion has not been present. These cases are probably related to a disturbed thirst center and neurogenic impairment of the glomerular and tubular salt mechanisms.

Another type of disturbance seen following head injuries relates to a low serum sodium concentration, a negative sodium balance and moderate fluid retention. In these cases, sodium is initially retained and then sharply the balance becomes negative, while the potassium balance reverses from negative to positive. These cases of “cerebral salt wasting” are assumed to be due to inappropriate secretion of ADH with resulting expansion of the body fluid compartments causing suppression of aldosterone and naturesis. The enlargement of the extracellular volume presumably stimulates the postcommissural organ (a volume receptor) to inhibit the secretion of aldosterone from the adrenal cortex and thereby promote renal conservation of potassium and sodium loss through tubular and glomerular mechanisms. Occasionally in the absence of dilutional effects which would presumably eliminate the possibility of expansion of the fluid compartment with suppression of aldosterone, sodium continues to be secreted in excessive amounts; in these cases, careful studies of blood and urine levels with measurement of the sodium excreted in the urine develop during the administration of salt for a period of time may be effective.

One must keep in mind that the common use of corticosteroids in the treatment of head injuries may further modify the secretion of the electrolytes, particularly sodium and give rise to changes in the electrolyte balance.

This discussion of the cerebral or neurogenic electrolyte disturbances emphasizes the necessity for the cooperation of the internist in the care of the head-injured person. Neither the general surgeon nor the neurosurgeon usually has the time nor desire to analyse and follow these patients. Fortunately, in most post-traumatic states, the electrolyte shift is minimal and the dilutional hypotonicity corrects itself within a day or so. In practice, if the kidney function is normal, a urinary output

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<th>Table 4. Signs and Symptoms of Water Imbalance Relative to Solutes</th>
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<tr>
<td>Serum solute &gt;285 mOsm./L.</td>
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<tr>
<td>Lethargic, Ataxia, Twitching, Convulsions</td>
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<tr>
<td>Elevated, Oliguria, Concentrated</td>
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<tr>
<td>Slowing</td>
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<tr>
<td>Serum Volume &lt;270 mOsm./L.</td>
</tr>
<tr>
<td>Confusion or coma</td>
</tr>
<tr>
<td>Weakness, Twitching, Cramps and convulsions</td>
</tr>
<tr>
<td>Normal or elevated</td>
</tr>
<tr>
<td>Polyuria, Dilute</td>
</tr>
<tr>
<td>Slowing</td>
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between 1500 and 2000 cc per day probably means that the electrolytes are in balance. However, if the patient is not progressing satisfactorily, a careful analysis of the electrolytes may give a clue as to the cause of the unsatisfactory convalescence.

It must be remembered that if the kidneys are functioning normally, they will regulate the water and electrolyte balance unless extreme stresses are applied. Some years ago, one American school of thought advocated extreme dehydration for head-injured patients39, and another excessive hydration50; both enthusiastically maintained the superiority of his technique over all others!

### 5. Wounds of the Accessory Cranial Sinuses

#### A. Orbitofrontal Wounds

One of the most frequent wounds encountered in traffic accidents is that of the forehead. Inflicted by striking the dashboard, the wound may involve to a greater or lesser extent the contents of the orbit, the temporal fossa and even the inferior margins of the orbit. Pointed or sharp objects may penetrate the orbital tissues through the orbital roof into the frontal lobe leaving a small wound of entrance.

Special considerations in this type of wound relate to the risk of infection carried in by the foreign body or from a septic sinus, the likelihood of a cerebrospinal leak and its disastrous meningitic sequelae and on the favorable side, the probability of restoring the victim to a normal life if the surgical outcome is successful, since the lesion is usually confined to the frontal pole of the brain. The surgeon aims in this type of wound to achieve not only an adequate debridement but to preserve vision and maintain a good cosmetic result. To gain these goals, it is necessary to have the cooperation of neurosurgeons, ophthalmologists, and otolaryngologists. The primary consideration is usually not the treatment of the cranial portion of the wound, which may be delayed until the orbit and sinuses have been repaired (Fig. 2).

The decision regarding the type of treatment of the orbital lesion must rest with the ophthalmologist. However, a very determined attempt should be made to preserve the eyeball even if there is no vision, or, at least, to delay enucleation until the tissue reaction has entirely subsided. However, massive wounds of the orbit with avulsion of the glove are sometimes encountered. In these cases, the extensive loss of tissue requires some type of graft to cover the exposed temporal or frontal cortex. Perhaps the most satisfactory repair is a fascial graft or split thickness skin graft to the wound after complete debridement of the tissue. A piece of fascia lata or skin is sutured in place to cover the dural defect. This may be in turn covered with a molded piece of tantalum or plastic. By this means, a spinal fluid leak is controlled as well as a potential cerebral herniation. In a few weeks, the graft may be removed and the underlying brain and granulation tissue prepared.
Fig. 2. (Case No. 122 03 49). Roentgenograms of the head of a 19 years old boy who sustained multiple buckshot wounds of the orbit. Although he became stuporous, no intracranial bleeding occurred. His right globe was perforated and the left pupil dilated and unresponsive to right. Four days after the injury, the orbits were explored and the globes repaired. Some vision returned in the left eye.

by compresses for a split-thickness graft. Subsequent reconstructive surgery may be necessary to produce a good cosmetic effect and to prepare the orbit for a prosthesis.

Less extensive and puncture wounds of the orbit are more commonly encountered, often with comminuted fractures of the orbital roof involving the frontal sinuses and perhaps with displacement of the lower rim of the orbit. Although these wounds may have small entrance lacerations, if the agent penetrating the orbit and brain is likely to be infected, such as the tine of a pitchfork or a knife blade, it is advisable to expose the dural laceration and brain tract in order to debride the wound. If the frontal sinuses are implicated, the debridement is particularly indicated. Such orbital penetrating injuries, according to the report of Bard et al\textsuperscript{1)}, have approximately a 25% mortality.

The debridement of compound fractures of the orbit and sinuses is best carried out through a bone flap, a technique usually ill-advised in the repair of cranio-cerebral wounds. However, in the case of penetrating orbital wounds, a bone flap is essential to afford ready access to the frontal lobe as well as to the orbit and sinuses. These advantages outweigh the slight risk of infection of the bone flap. Small frontal lacerations may be debrided and closed in layers before the coronal incision is made for the osteoplastic flap. If there are extensive lacerations in the frontal region, the incision may utilize one of these debrided lacerations. When the bone flap has been reflected, the dura mater is separated from the inner table of the floor of the anterior fossa so that the dura and brain may be debrided. The
fragmented walls of the sinus should be removed, if possible, leaving the supraciliary ridge to preserve a good cosmetic effect. If the sinus walls are not extensively comminuted, only the posterior wall may be removed. From the sinus, it is essential to remove all blood and debris, the mucous membrane lining the sinus walls, and to plug the nasofrontal ostria with bone wax. Tears in the dura mater on the orbital surface of the frontal lobe, which may extend to the cribriform plate, are likely to be a source of cerebrospinal fluid rhinorrhea and should be repaired by dural grafts. Comminuted fragments of the orbital roof are excised and a tantalum plate may be placed over a bony defect to prevent later herniation of the frontal lobe (Fig. 3).

![Fig. 3. (Case No. 123 94 05). Roentgenograms of the skull of a 30 years old man with a lacerated frontal wound overlying a comminuted fracture through which oozed brain tissue. The ipsilateral pupil was dilated and the contralateral limbs paretic. The wound was debrided, and the sinuses entered. The torn dura mater was not completely closed and a rhinorrhea had to be repaired by a fascia lata graft one week later.](image)

B. *Mastoid Wounds*

Severe lacerations of the mastoid region are not common because of its massive structure and the overlying muscles in this area. Fractures, however, extending into the mastoid region are occasionally associated with overlying lacerations. It is therefore important to explore the middle fossa and suture the dura mater that has been damaged in such wounds in the process of debridement of a postrior temporal fracture, otherwise a small encephalocele may develop and give rise to a cerebrospinal fluid rhinorrhea or otorrhea. Usually such meningoceles can be readily treated by turning down a small bone flap just above the ear, locating the site of the rhinorrhea, packing the bony dehiscence with a muscle and repairing the dural defect with a graft.

Fractures in this area frequently involve the facial nerve as well as the structures of the inner ear. If the facial nerve is paralysed, it should be explored through
a mastoidectomy wound and decompressed. This may be done at the time of the primary debridement, if the patient's condition is satisfactory and an operator competent in the use of an operating microscope is available. The results of early decompression, or if the nerve is completely severed, of suture or grafting are excellent.

6. Complicating Injuries of the Cervical Spine

Although in most series of head injuries, cervical spine involvement is reported in approximately 10% of cases, it would seem on theoretical grounds that neck injuries probably occur more commonly and are often unrecognized. Certainly, most active traumatology services will have at all times, at least one patient being treated by both orthopedic and neurosurgeons for a neck injury. The primary consideration of course, in the case of a cervical spine injury is the avoidance of further damage to the cord by manipulation of the neck. When a patient is admitted to the emergency room with the possibility of a neck injury as suggested by the position of the head, asymmetry of the tendon reflexes or a paresis of an extremity, the head should be immobilized by sand bags until it can be put in extension using a canvas halter beneath the chin and occiput. By this means, one may apply 20–30 pounds traction to maintain the head in proper position. With the head so immobilized, AP and lateral roentgenograms of the spine may be made. Some difficulty is often encountered to obtain adequate lateral views of the lower cervical spine because of obstruction by the shoulders. Without moving the head or neck, the patient's shoulders may be pulled down manually or by adhesive tapes as the x-ray film is being exposed. If this is not satisfactory, oblique views may help.

The abnormalities in the roentgenograms of the spine suggesting fracture are well known; (1) displacement of the vertebrae, (2) linear discontinuities of the vertebral bodies of their appendages, (3) irregularities in the position of the spinous processes, (4) the presence of a retropharyngeal mass (hematoma). If any of these signs are present the patient should be treated as if a cervical spinal fracture existed. The problem of getting at the mouth to eliminate secretions of a comatose or stuporous patient in halter traction is difficult. Under such circumstances either a transnasal intratracheal intubation must be carried out or a tracheostomy performed. If surgery is not required for the repair of cranial wounds, the latter may be the most effective procedure since these patients will usually be maintained in a dorsal decubitus position. However, such a procedure may not be necessary initially if the patient's cranial wound does not seem to be serious and his state of consciousness is improving. A nasal intubation for a few hours may be quite satisfactory to provide an adequate toilet of the trachea.

The definitive treatment of the fractured cervical spine associated with head injury will depend upon the severity of the head injury and the presence of intracranial complications such as hemorrhage. In most cases, it is advisable to wait
for stabilization of the intracranial and neck injuries before proceeding with radical therapy. An exception to this rule should be made if there is evidence of progression of neurological dysfunction below the cervical cord or if there is a complete block on lumbar puncture. In most cases, however, after the initial x-rays have been made, Crutchfield tongs are substituted for a halter to maintain the neck in proper alignment. With undisplaced fractures of the neck, this may be followed by subsequent immobilization for some weeks by means of a Minerva jacket or cervical collar. On the other hand, fracture dislocations tend to recur and are probably best handled by an open operation. In general, we would prefer an anterior fusion shock or pulmonary emboli. The amount of circulating blood which may be necessary if a fracture dislocation exists which cannot be readily reduced by extension.

7. **Limb Fractures**

Fractures of the extremities complicate the treatment of the head-injured patient not only by their presence and the positions necessary for the alignment of fragments, but also by the altered physiology due to blood loss about the fracture, shock or pulmonary emboli. The amount of circulating blood which may be extravasated to the tissues about a fracture, particularly of such large bones as the

![Fig. 4](image-url) (Case No. 121 51 19). Roentgenograms of a 71 years old man who fell down a flight of stairs sustaining a bilateral depressed frontal skull fracture passing through the sinus, a pneumocephalus, and a supracondylar fracture. The patient was treated expectantly and the femoral fracture reduced by an open operation one week after the accident.

— 19 —
femur, is quite sufficient to cause shock. The effects of a markedly lowered blood pressure upon an already damaged brain may be quite devastating. In addition, there is some evidence to suggest that pulmonary fat emboli are much more common following fractures of the long bone than were formerly thought, although in uncomplicated cases most of these emboli will be absorbed without difficulty. However, with a complicating head injury, the pulmonary emboli may embarrass the oxygen exchange (Fig. 4).

Summary

A review of the changing clinical picture of the head-injured patient brings out the multidisciplinary nature of the problems. Rarely is the neurosurgeon competent to handle alone the multiple conditions that result from high speed traffic accidents. However, with team cooperation and a nicety of judgment in assigning priorities to the various injuries, the complex situation may be met efficiently and effectively.

References

9) "Road Accidents" Series Chirurgica, Documenta Geigy, 1962, 5:5: