Bullet injuries of the brain

H Alan Crockard FRCS
Senior Neurosurgical Registrar, Royal Victoria Hospital, Belfast

Summary

Experience gained with a wide variety of missile injuries of the brain is presented. Clinical signs and intracranial pressure (ICP) studied in the early post-injury period have been correlated with survival and treatment. Stress is laid on fluid requirements and the importance of controlled ventilation in the management of the labile clinical condition of such patients. Coughing and struggling caused extrusion of blood and brain from the wound, and this was reduced considerably with endotracheal intubation and mechanical ventilation. Post-operatively high ICP could be controlled in potential survivors with continued ventilation.

Development of treatment

The study and treatment of bullet wounds have been the subject of a considerable amount of medical thought and writing since the introduction of gunpowder to Europe in the 14th century. It was soon realized that the sequelae of a sabre wound and an injury by musket ball could be very different, the former healing by first intention, the latter often suppurating and producing unexpected complications due to an unpredictable route through tissue. To the layman the explanation was obvious—saltpetre, a constituent of gunpowder, was the devil’s soil and devils might ride upon the missile. Medical opinion held that there was a specific poison in such wounds and so employed cautery to reduce its consequences.

Progress in the treatment of this type of trauma has been slow, not because of lack of material but for two other reasons. Firstly, the lessons learnt in one theatre of war have been quickly forgotten and secondly, the differing vantage points of those who have studied missile trauma have resulted in a confusion between the primary wound and its secondary complications. As far as head wounds were concerned, their treatment was limited by the extremely poor prognosis regardless of therapy. The general 19th century opinion is summed up in the words of George James Guthrie, the British surgeon-in-chief in the Peninsular War and at the Battle of Waterloo: ‘Injuries of the head affecting the brain are difficult of distinction, treacherous in their course and, for the most part, fatal in their results.

In 1536 Paré, lacking boiling oil at the height of a battle, was forced to use a balm, rose oil, and discovered that this produced superior healing and considerably less suffering in his patients than cauterization. Two centuries later Larrey, Napoleon’s surgeon-in-chief, provided another significant advance, conceiving the flying ambulance (ambulance volante) to evacuate the injured rapidly from the front line to hospital. It was his idea to bring treatment as close as possible in time...
and place to the wounded. Both these men were active in the battlefield and emphasized the need for early surgery and wound drainage. Hunter, on the other hand, was well removed from the scene of combat and drew his conclusions from injured men who had survived the primary insult. This type of controversy can be traced throughout the history of the surgery of trauma, some advocating immediate intervention, others stressing the importance of delay.

In those times the treatment was on occasion more severe than the original trauma. Venesection was routine in the field hospital in the same way as intravenous infusions are today, and was especially popular for head injuries to reduce fever and ‘phlegmonous inflammation of the wound’. Paré cites the case of a man with a gunshot wound of the head and ‘strong constitution’ who, in the 3 days after injury, lost 5 pints by therapeutic bleeding, had a craniotomy, a wound infection, and possibly septicaemia, and survived—‘He was certainly cured thanks to God, without whose benediction these treatments would have been useless’. Small wonder!

John Hunter was commissioned as a staff surgeon to the British troops sent to capture Belle Isle in 1762. What impressed him was the lack of disability in French captives who had sustained limb wounds 5 days before and who had had no medical attention. This and the fact that he was not close to battle himself made him conclude that a conservative approach to gunshot wounds was the best method. In his book *A Treatise on the Blood, Inflammation and Gunshot Wounds* he devotes only 5 lines to wounds of the head, but his approach to such injuries is amplified in his account of a young man accidentally shot with a ramrod, which produced a side-to-side injury from the right parietal to the left temporal region. Forty-eight hours later Hunter trepanned ‘on the hind part of his head and some pieces of bone were extracted from the brain’, but despite this the patient died. In this case, as with all Hunter’s work, however, it was his perception of relevant data which was so valuable. He correlated altered level of consciousness with variations in pulse and respiration and remarked on the pupils’ reactivity to light. At autopsy he noted: ‘Inflammation had begun, for the unifying medium was deposited’.

This problem of inevitable infection and inflammation preoccupied medical thought. Was it a specific poison? Was it a natural method of healing? Was it a contagion spread by the air? John Atkins in his book *The Navy Surgeon* stressed that craniotomy should be performed in the hold of a ship ‘in a close place by candlelight’ for air was the ‘enemy of the brain’.

Generally, however, the results were so bad that no brain surgery was performed in the 19th century wars. With the findings of Lister and Pasteur it was possible to consider surgery. Stevenson, reporting on the Boer War, was able to encourage surgery for head wounds: ‘By the free use of antiseptics that surgical infective disease was reduced to a degree never before seen in any war’. During the First World War the problem of infection was well known and a chief cause of death in neurosurgical cases, but Cushing demonstrated that sepsis could be considerably reduced by the removal of infected bone fragments and clot with extensive, early, and definitive surgery.

The other mainstay of treatment, evacuation to skilled medical attention, was progressing. Jefferson and Cushing and later Cairns organized highly sophisticated teams for dealing early with head injuries, but it was not until the development of the helicopter that evacuation time was dramatically reduced. In the Boer War 2 days elapsed
before cranial surgery could be undertaken; by World War II many casualties had specialist attention within 24 hours, but in Korea most had neurosurgical care within 8 hours of injury and in Vietnam similar injuries were treated within 2 hours. Here then is the crux of modern treatment for penetrating brain injuries—rapid evacuation to a specialist centre.

**Missile wounding**

The early guns were of unpredictable firepower. Gelnhausen in his account of the Thirty Years War describes an attempt to execute a peasant by shooting. The ball bounced off his head 'like from a steel mountain' and he succumbed only when his head was cleaved open with sword. With the improvement in munitions by the 19th century the wounding properties of missiles were more carefully studied. The 'wind of the shot' or 'projectile air' was considered to be the cause of injury and Pirogoff, the Russian surgeon, attributed a soldier's death to a bullet that passed only close to the victim. It was supposed that the bullet forced air in front of it into the wound. In the Paris street fighting in 1848 Hugier noted that rifle bullets produced much larger wounds than he expected.

It was Kocher in 1874 and Horsley in 1894 who developed the concept of the 'hydrodynamic effect' of bullet wounding. They showed that a bullet could pass through an empty can producing neat entrance and exit holes, but if the can was filled with water it would burst apart shortly after the passage

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**FIG. 1** High-velocity missile injury caused by an Armalite bullet. Note expanding cavity from entrance point and complete disappearance of temporal lobe. Reproduced by permission from the British Journal of Surgery.
of the bullet owing to the transfer of energy from the bullet to the contents of the container. This will also occur in a vacuum, thus excluding 'projectile air' as a major wounding factor. This same effect has been shown in the experimental animal's head20 using high-speed X-ray techniques. A temporary cavity forms21 which may be 30 times the diameter of the missile, and the intracranial pressure (ICP) exceeds 1,000 lb/in² (80 kg/cm²)22. As this explosive cavity is produced by the bullet's energy and as this varies with the square of the velocity \( E = \frac{1}{2}mv^2 \) it is not difficult to appreciate how much more devastating is a modern rifle bullet with a velocity in excess of 3,500 ft/sec (1,060 m/sec) compared with that of a revolver (600-800 ft/sec (183-243 m/sec) (Figs. 1 and 2).

One of the first modern experimental studies of bullet wounds of the brain was carried out by Horsley18. He measured blood pressure, respiration, heart rate, and ICP in a dog following a low-velocity bullet wound. After injury there was a respiratory standstill, usually transient, followed later by a rise in blood pressure and ICP. It was Horsley’s thesis that the cause of death was ‘medullary anaemia’ due to raised ICP. He also advocated artificial respiration to overcome the transient apnoea. Gerber and Moody23 have elaborated on this work and have shown a fall in cerebral blood flow in fatal cases.

Apart from autopsy reports, there have been few basic measurements in patients with bullet injuries of the brain owing to the nature of the injury and the locations where wounding is likely to take place. So any clinical observations or measurements made soon after the injury might be useful in the understanding of its pathophysiology and thus pro-
provide a sound basis for treatment.

**Pathophysiology of raised ICP**

Following head injury there is a variable degree of cerebral oedema and, as this increase in brain bulk is contained within a closed cavity, ICP will rise. Some accommodation can take place by the expulsion or absorption of cerebrospinal fluid (CSF), but with the exhaustion of this facility further increases in volume will produce disproportionately large rises in pressure. In addition to gaseous inadequacies, airway obstruction or ataxic respirations will cause a dramatic increase in the central venous pressure which is relayed directly to the contents. Endotracheal intubation, muscle relaxation, and controlled hypocapnic ventilation have been used in an effort to reduce intracranial volume and ICP and improve injured cell metabolism, with encouraging results.

**Materials and methods**

The hospital at which this work was carried out is situated in an area in Northern Ireland where some of the most violent disturbances have occurred. There has frequently been gunfire close to and occasionally inside the hospital grounds. Table I shows the evacuation time of the first 84 patients with penetrating injuries of the brain treated by us during the period 1970–73; one-third arrived within 15 minutes and three-quarters were admitted within 90 minutes of wounding. This rapidity of evacuation was due to the location of the hospital and the efficiency of the ambulance services. This had two major effects: firstly, it meant that many moribund patients were admitted (42% of those who died did so within 6 hours of injury) and secondly, it allowed us to treat and study the injured earlier than had hitherto been possible.

On admission a brief neurological examination was conducted during resuscitation. At least one intravenous fluid line was erected

<table>
<thead>
<tr>
<th>Table I</th>
<th>Interval between injury and admission</th>
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<tbody>
<tr>
<td>Minutes</td>
<td>Numbers</td>
</tr>
<tr>
<td>0-15</td>
<td>28</td>
</tr>
<tr>
<td>16-30</td>
<td>35</td>
</tr>
<tr>
<td>31-45</td>
<td>8</td>
</tr>
<tr>
<td>46-60</td>
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<tr>
<td>61-120</td>
<td>3</td>
</tr>
<tr>
<td>121-180</td>
<td>2</td>
</tr>
<tr>
<td>&gt; 180</td>
<td>3</td>
</tr>
<tr>
<td>Unknown</td>
<td>9</td>
</tr>
<tr>
<td>Total</td>
<td>84</td>
</tr>
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and most patients were mechanically ventilated after endotracheal intubation and muscle relaxation. An ICP transducer was inserted through the wound to lie between bone and dura. In some cases the wound was too small to permit the passage of the transducer and these were studied only postoperatively.

**Clinical condition**

The patient with a severe head injury due to a road traffic accident is immediately unconscious, with small pupils, irregular respiration, and extensor spasms. Blood loss is usually slight and, in the absence of respiratory complications, the condition may be unaltered for
some time. By contrast, the labile condition of the person with a bullet wound of the brain constantly impressed us. Even with severe injuries consciousness was not necessarily lost; the patient was often irritable, struggling, coughing, or vomiting, and with each exertion blood and brain extruded from the wound. It was not uncommon to examine and question such a patient within a few minutes of injury only to see him lapse into coma and die some hours later.

The initial clinical observations were correlated with survival (Table II). One of the most valuable signs was level of consciousness—only 11.5% of those admitted conscious and fully co-operative died, but any clouding of consciousness was associated with a three-fold increase in mortality, and coma was invariably fatal. Pupillary size and response to light were noted. Normally reacting pupils were associated with least mortality, but this factor was not as critical as level of consciousness, for one-third of these patients eventually died. Another feature which we observed was the absence of pin-point pupils, a common finding in deceleration head injuries.

Blood pressure on admission was also recorded, and it was obvious that any deviation from 'normal' (90–150 mm Hg systolic) was a bad sign. Those with high blood pressure also had high ICP and almost invariably died. Sixteen patients admitted with large craniofacial wounds were bleeding profusely from dural sinuses or cerebral or facial arteries and were extremely shocked. Despite massive transfusion many succumbed in the resuscitation room, but 5 requiring up to 10 litres intravenously within an hour of admission survived after surgery. In 3 of the 16 the common carotid artery was ligated during resuscitation to reduce the blood loss; one survived.

**Intracranial pressure**

Of the 93 patients reported by us, 28, 20 had ICP studies, 11 of them within an hour of injury. There was a wide spectrum of initial pressure patterns which could be roughly divided into 3 main groups.

<table>
<thead>
<tr>
<th>Clinical sign</th>
<th>Mortality (%)</th>
</tr>
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<tbody>
<tr>
<td>Alert</td>
<td>11.5</td>
</tr>
<tr>
<td>Drowsy</td>
<td>33.3</td>
</tr>
<tr>
<td>Reacts to pain</td>
<td>79.1</td>
</tr>
<tr>
<td>Coma</td>
<td>100.0</td>
</tr>
<tr>
<td>Pupils</td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>34.0</td>
</tr>
<tr>
<td>Unequal</td>
<td>61.9</td>
</tr>
<tr>
<td>Dilated</td>
<td>96.0</td>
</tr>
<tr>
<td>Blood pressure</td>
<td></td>
</tr>
<tr>
<td>Normotensive</td>
<td>41.0</td>
</tr>
<tr>
<td>Hypotensive</td>
<td>72.0</td>
</tr>
<tr>
<td>Hypertensive</td>
<td>93.0</td>
</tr>
</tbody>
</table>

**High ICP** The patients with high ICP (60–100 mm Hg) had small cranial wounds with little blood loss; all were unconscious and had dilated pupils and a raised blood pressure. Of the 4 studied, 1 survived, the others dying within the first 2 days after injury. In those who had an operation the missile trace was explored and, apart from severe generalized oedema, little else was found.
FIG. 3 ICP and mean blood pressure (MBP) records of a patient with side-to-side low-velocity injury (see text). Time scale in hours post-injury.

FIG. 4 Initial ICP and MBP records of patient admitted 15 minutes after high-velocity frontal injury which caused extensive tissue loss and bleeding (see text). Time scale in minutes post-injury.
Postoperatively there was no change in ICP.

A typical record is shown in Figure 3. This patient was admitted, 30 minutes after a side-to-side low-velocity injury, in coma with dilated pupils and a blood pressure of 160/100 mm Hg. After endotracheal intubation and institution of controlled ventilation there was a reduction in ICP and mean blood pressure. This improvement was temporary, however, and within 5 hours the pressures slowly rose with "plateau waves" appearing between 12 and 18 hours after injury, until at 28 hours there was a sudden terminal drop in pressure, a typical failure of the Cushing response. At no time did the patient's condition improve sufficiently to justify surgery, and at autopsy the missile track was seen to traverse the midbrain structures (Fig. 2).

Low ICP initially The 6 patients in this group had all been injured by high-velocity missiles (Armalite, 0.303 in, and SLR 7.62 mm bullets). Most had large wounds with considerable loss of skin, bone, and brain tissue and were bleeding profusely. Two were moribund on admission, 2 died within 24 hours, but the other 2 survived; both of these had sagittal sinus lacerations successfully repaired with a fascial patch. In all cases both ICP and blood pressure were low when observation was begun, but after sufficient transfusion to raise the blood pressure there was a stepwise increase in ICP (Fig. 4). In most cases the increase in systemic pressure was followed by increased bleeding, and again ICP would closely follow the downward trend in blood pressure, a characteristic of loss of autoregulation. Superimposed on this problem of circulating blood volume was massive cerebral oedema, so that of the 4 patients in this group who had operations, only 2 survived the ensuing rapid brain swelling, which produced an extremely high ICP. This is shown in Figure 4, in which ICP reaches 100 mm Hg within 80 minutes of injury.

‘Normal’ ICP initially A few patients presented with normal or slightly increased ICP and did not appear to be severely injured on admission. Blood loss was not a problem. Coughing or struggling produced rapid increases in ICP which subsided in part only. Such a patient with a tangential high-velocity occipital wound (Fig. 5) was admitted drowsy and irritable 15 minutes after injury. An ICP transducer was inserted between bone and dura. He began to struggle and had to be restrained, when he suddenly deteriorated; the pupils, which had been normal, became fixed and dilated and ICP, normal on admission, rose to 100 mm Hg (Fig. 6). An endotracheal tube was inserted

**FIG. 5** Skull X-ray of patient with tangential, high-velocity occipital injury. Note bone fragments deep in cerebral tissue and fracture lines radiating from the point of impact.
FIG. 6 ICP record of patient in Fig. 5 (see text).

...and controlled ventilation begun after muscle relaxation, with some reduction in ICP. At emergency operation a small tear in the lateral sinus and a large haematoma were found. Postoperatively (Fig. 7) his ICP was 50 mm Hg and he was ventilated for 6 days until the pressure was reduced. He survived and was discharged from hospital with a monoparesis and hemianopia.

**Postoperative ICP studies**

After operation there was usually a rise in ICP, even in patients with little actual brain damage, such as that produced by a low-velocity tangential injury. This secondary rise in pressure occurred 6–20 hours after injury and lasted up to 12 days, the degree and extent correlating well with the amount of brain damage noted at operation. It occurred whether or not the patient was hyperventilated postoperatively (Fig. 8).

ICP monitoring was also useful in alerting

FIG. 7 Postoperative radiograph of patient in Fig. 5 showing the extensive craniotomy required for adequate haemostasis, debridement, and dural repair. An ICP transducer (round shadow) was inserted through a separate burrhole to monitor the postoperative course.

FIG. 8 ICP record of patient with tangential temporal bullet injury showing mild secondary rise in ICP which was seen in most cases. In more severe injuries ICP could rise to very high levels and remain high for up to 12 days.
the clinician to postoperative haematoma formation. In 2 cases, both hyperventilated, a rapid rise in ICP shortly after operation was the first sign of this complication. In one it preceded pupillary dilatation by several hours; in the second case re-exploration was performed before pupillary changes had occurred.

The length of postoperative ventilation was determined by clinical assessment and from the response of the ICP to treatment. There was no unnecessary prolongation of vegetative life, but in those patients who showed a potential for survival treatment was continued for as long as ICP was controlled by this means. Figure 9 shows the ICP and mean blood pressure of a patient with a side-to-side high-velocity injury. Postoperatively the pressures gradually decreased over the 7 days of treatment. The clinical condition was assessed after reversal of muscle relaxants at 48-hour intervals, and on the first 2 occasions ICP rose to very high levels. The second time the patient rapidly developed extensor spasms and ataxic breathing while the ICP rose rapidly and exceeded the mean blood pressure, thus abolishing perfusion. Complete medullary collapse was prevented by re-institution of muscle relaxation and controlled ventilation. A period of 110 hours' hyperventilation was required before the vicious circle of pressure rises and increasing cell anoxia was broken and ICP remained low when the patient resumed spontaneous respiration.

**FIG. 9** Postoperative ICP and MBP records showing effect of 110 hours' hyperventilation (see text).

**Discussion**

The lability of the clinical condition in the early post-injury period was a feature which constantly impressed us. Impressive, too, was the sensitivity of the intracranial contents to coughing, struggling, or even the slightest airway obstruction, and no one working in the resuscitation room needed convincing of the efficacy of immediate intubation and controlled ventilation. Prognosis following bullet injury was necessarily guarded, but certain
Factors such as the missile velocity and the area of brain involved gave some indication of outcome. Overall, 85% of patients with side-to-side injuries died, whether injured by low- or high-velocity missiles, but obviously the faster bullets were associated with higher mortality. It was not our experience, however, that all such injuries were ‘inevitably fatal’. Of the 11 patients whose ICP monitoring was started within an hour of injury, 9 were wounded by high-velocity missiles and 3 survived, and among the total number treated by our department there was a significant number of survivors from such an injury. An injury produced by a tangential impact had the best prognosis, and while it produced extensive fracturing, the patients were often conscious or drowsy on admission. Blood loss was a major problem, but if this could be controlled the outlook for such patients was considerably improved. The initial level of consciousness was a most sensitive indicator, and any clouding of consciousness or pupil irregularity was associated with a greatly increased mortality.

I monitored ICP for two reasons: firstly, to correlate the results with clinical and operative findings in an effort to understand the underlying pathophysiology; and secondly, to assess objectively the effects of various forms of therapy. There are valid theoretical objections to pressure measurements obtained from the open skull, which allows the contents to decompress and reduce ICP. Nevertheless, I felt that the pressure patterns provided useful information which outweighed the theoretical disadvantages. Patients with large, gaping wounds may have had artificially low pressure readings, but there is little doubt that rises in pressure could be meaningfully interpreted.

Cope, reviewing the experiences of the Second World War, concluded that ‘one of the outstanding lessons learned in the war was that the head injured could be moved without any decline in condition’. He reported Jefferson’s stable observations of vital functions before and after a 3-hour road journey. This has certainly not been the experience in Belfast considering, for example, those admitted bleeding profusely, with low ICP and blood pressure and requiring 10 litres of fluid during resuscitation. Manifestly they would not have survived a slower evacuation. Small and Turner qualified Cope’s statement and postulated that, in war, a neurosurgical team with a mortality of 10% was too far from the battlefield. The Royal Victoria Hospital has been at the other extreme positionally and has had a mortality of 56%.

With the availability of helicopter services rapid evacuation to a specialist unit is desirable. For patients injured in outlying areas and admitted to the local hospital it was our policy to recommend intubation and controlled ventilation before and during transportation. Obviously a medical practitioner, preferably an anaesthetist, was required to accompany the patient, and although this taxed overstretched manpower there was no doubt among those dealing with such injuries that the policy provided the best conditions for the injured. Considering the irrevocable rises in ICP caused by coughing and struggling (Fig. 6) it may be that the quality of survival was improved with this approach. The risks of delayed specialist care and spontaneous respiration have been demonstrated by Raimondi and Samuelson, for 37 out of 89 patients transferred to their hospital deteriorated during transit and 21 died. The main causes of death were cerebral haematoma and respiratory distress, and only 2 patients were intubated before transfer. Very few significant haematomas have been seen in the Belfast series, and in the case quoted (p. 119) there was airway obstruction. It is my belief that controlled ventilation
prevents haematoma formation by lowering central venous pressure and bleeding into the damaged area.

The details of surgical technique have been reported elsewhere, but basically the aim was to arrest haemorrhage and remove all necrotic debris, clot, and bone fragments. The dural defect was always repaired with temporal fascia or fascia lata and a skin closure, effected by scalp flap rotation where necessary. Postoperative sepsis was not a major problem and no abscesses have been detected so far.

All patients except those with very minor injuries had a period of postoperative ventilation, the length depending on the severity of the cerebral oedema. No attempt was made to prolong vegetative life—rather was it our aim to improve the quality of potential survivors. The decision to discontinue hyperventilation was made on the basis of clinical judgement, ICP, and CSF lactate studies. Patients requiring only short periods of ventilation to reduce ICP had the best chance of survival.

The mortality and morbidity from head trauma of all kinds is depressingly high and the incidence steadily increasing. Particularly tragic are the results of missile trauma, which have been all too frequent in the present circumstances. It is hoped that the medical experience gained will benefit other similarly injured people. The value of rapid evacuation, adequate resuscitation, and an adequate airway has been demonstrated in cases of open wounds of the head, and encouraging results have been obtained in severe closed head injuries. It would seem that ICP monitoring, which has a minor place in the management of cerebral tumours and vascular disease, is an important tool in the management of severe head injuries, in assessing the effect of therapy, and in the early detection of intracranial complications.

I am grateful for the active support of a great many people, including the nursing and medical staff of the accident and emergency, neurosurgical, and respiratory and intensive care units and the consultants, D S Gordon Mch FRCS, G A Gleadhill MD FRCS, A R Taylor PhD FRCS, and R G Gray MD FFARCS who allowed me to study their cases. Historical references were supplied by Miss E Allen, Curator of the Hunterian Museum of the Royal College of Surgeons of England, and Maj. Gen. W MacLennan and Mr Davies of the Royal Army Medical College. The work was begun during the tenure of a Royal Victoria Hospital Research Fellowship.

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