Depressed skull fracture: data, treatment, and follow-up in 225 consecutive cases

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SUMMARY Data are given of a consecutive series of 225 cases with depressed skull fracture, admitted within 48 hours after the accident. The series is considered to represent the full range of this type of injury in a densely populated area in the Netherlands. The predominance of young male patients with a compound fracture is stressed. The data are compared with other series. The results of long-term follow-up are presented and particular attention is paid to the rate of early and late epilepsy and persisting neurological deficit. The significance of complications like intracranial haematoma and venous sinus involvement is confirmed. Treatment is discussed. In 66% of cases, most of which were compound, some even severely contaminated, the indriven bone fragments were completely or partially replaced with excellent cosmetic result and low infection rate.

Series of civilian cases with head injury and depressed skull fracture were described by Stöwsand and Geile, (1966); Miller and Jennett (1968); Cabraal and Abeyesuriya (1969). Miller and Jennett paid particular attention to the complications in this type of head injury. Nevertheless, information about incidence, site and severity of injury, cause, age, sex, dural tears, complications, etc., is still rather scarce. Probably this lack of attention is due to the fact that the majority of these injuries recover rapidly and completely, provided that treatment is appropriate. It seemed worthwhile to review our cases and to compare our data with those of others. We considered the Glasgow series (400 cases: Miller and Jennett, 1968) the most suitable for this purpose.

Opinions on the immediate handling of these patients diverge, especially if the fracture is compound. Many important questions arise, such as: should indriven fragments be elevated or not, removed or replaced; antibiotics be given, etc. The answer to these questions is not unanimous and may even vary between surgeons in one department. This encouraged a long-term follow-up study of our cases with revaluation of the results of the therapy used.

MATERIAL

PATIENTS This report is based on a series of 225 consecutive cases of depressed skull fracture admitted to the neurosurgical department in Rotterdam between June 1960 and May 1970. All cases were admitted within 48 hours after the incident. Fewer than 10 cases, admitted more than two days after the accident and a few cases with puncture wounds through orbit or nose, were excluded. Gunshot wounds were not encountered. In all cases one or more fragments were depressed by at least the thickness of the skull.

Our service covers a population of 1·3 million people of which 90% live within 20 miles of the hospital. It is estimated that at least 95% of all depressed skull fractures in this area are referred to our department. Therefore this series seems to represent the full range of depressed fractures of the skull in a densely populated area. It is therefore comparable with the Glasgow or Oxford series (Miller and Jennett, 1968).

The estimated incidence of 10 cases of depressed skull fracture per million inhabitants per year in 1960 had risen to 20 in 1970.

FOLLOW-UP Twenty-five patients died in consequence of the injury, usually as a result of a severe brain lesion due to the acceleration component of the injury (Table 1). Of the 200 remaining cases four died later due to causes unrelated to the injury. In the follow-up clinic 185 patients were seen. From one to 10 years
TABLE 1
ANALYSIS OF 225 CONSECUTIVE CASES WITH DEPRESSED
SKULL FRACTURE ADMITTED IN ROTTERDAM BETWEEN
1960 AND 1970

<table>
<thead>
<tr>
<th></th>
<th>No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total number of patients</td>
<td>225</td>
<td></td>
</tr>
<tr>
<td>Death as a result of the injury</td>
<td>25</td>
<td></td>
</tr>
<tr>
<td>Left hospital alive</td>
<td>200</td>
<td></td>
</tr>
<tr>
<td>Subsequent death due to other causes</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Available for follow-up</td>
<td>196</td>
<td></td>
</tr>
<tr>
<td>Attended follow-up clinic</td>
<td>185</td>
<td>94</td>
</tr>
<tr>
<td>Information obtained by letter</td>
<td>6</td>
<td>3</td>
</tr>
<tr>
<td>Patients not retraced</td>
<td>5</td>
<td>3</td>
</tr>
</tbody>
</table>

GLASGOW 1968 400 cases    ROTTERDAM 1970 225 cases

FIG. 1. Age distribution of depressed skull fractures in Glasgow and Rotterdam.

The incidence of road traffic accidents in Rotterdam (51%) is higher than in Glasgow (37%), due to the greater number of vehicle accidents in our densely populated area. This difference is compensated for by a lower number of assaults and sports-injuries (Table 2). To the miscellaneous group belong six children who were hit by a swing.

AGE AND SEX The distribution of age does not differ significantly from the distribution in the

Glasgow series (Fig. 1), nor from the figures given by Stöwsand and Geile (1966). The preponderance of young patients is striking.

In our series, 86% of the cases were male (Fig. 2) (Miller and Jennett: 84%; Stöwsand and Geile: 83%; Cabraai and Abeysuriya: 89%). Even in the group less than 16 years of age more than twice as many males as females were involved.

CAUSE OF INJURY The incidence of road traffic accidents in Rotterdam (51%) is higher than in

Table 2

| CAUSES OF DEPRESSED SKULL FRACTURE IN GLASGOW AND ROTTERDAM SERIES (AS %) |
|-----------------------------|-----------------------------|
|                              | Glasgow (%) | Rotterdam (%) |
| Road traffic                | 37.5          | 51            |
| pedestrian                 | 20.0          | 18            |
| vehicle                     | 14.25         | 30            |
| unknown                     | 3.25          | 3             |
| Industrial                  | 15.25         | 19            |
| Assault                     | 14.5          | 3             |
| Sport                       | 9.25          | 1             |
| Miscellaneous domestic (falls, etc.) | 23.5 | 26            |
|                             | 100.0         | 100           |

after injury (85% after two years or more, 60% after four years or more) all patients were interviewed and examined by the author. Particular attention was paid to the presence of late epilepsy and major neurological deficit like dysphasia, hemiparesis, loss of vision, or visual field defects, etc. However, cosmetic results and less striking complaints like headache, dizziness, etc., were also noted. Radiographs of the skull were taken of most patients. Repeated electroencephalography (EEG) was performed only in about 20% of cases, particularly in those where doubt existed as to the presence of epileptic fits. In these cases heteroanamnestic information was also obtained from family members. In five cases information was obtained by letter, while six patients were abroad and could not be traced.
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On the whole, there is—as might be expected—more similarity between the Rotterdam and Glasgow series than with the Ceylon series of Cabraai and Abeysuriya (1969) in which 50% were caused by assaults and 5% by falling coconuts.

TYPE AND LOCALIZATION OF FRACTURE, LACERATION OF DURA MATER In this series 86% of cases were compound (Glasgow 84%) (Fig. 3). The similarity with the Glasgow series is even more striking when the involvement of the dura mater in the compound cases is considered (Fig. 4). In the relatively small number of closed injuries the findings also corresponded (Fig. 5).

Table 3 shows the distribution over the various bones of the skull. In about one out of every five cases, more than one convexity bone was affected by the fracture line. These cases were classified according to the region where the impression was predominantly situated. In seven cases three or more bones were involved. These cases were attended by a high mortality, as were the cases with occipital localization. The frontal and parietal region were particularly often involved. This distribution is in agreement with the findings of Stöwsand and Geile (1966) and Cabraai and Abeysuriya (1969).

EPILEPSY Jennett (1962, 1969a,b) distinguishes early epilepsy (occurring within the first week after the accident) from late epilepsy (occurring more than seven days after the accident).
According to this author early epilepsy justifies separate consideration because it differs from epilepsy occurring later: it is less likely to persist, it is more likely to take the form of focal motor attacks, and temporal lobe seizures do not occur.

There was a much lower frequency of early epileptic fits in the Rotterdam than in the Glasgow series (Table 4), perhaps due to the retrospective character of our study. Possibly not all fits were noted in the case histories or on the temperature chart. Of the 10 cases observed, five showed localized focal motor attacks and five generalized seizures.

The rate of late epilepsy in Rotterdam (7.1%) was also lower than in Glasgow (9.5%) (Table 4). This is not surprising, as there were fewer patients with a post-traumatic amnesia (PTA) of more than 24 hours in Rotterdam (14.2%) than in Glasgow (23.8%) (Table 5). According to Jennett (1962, 1970) late epilepsy is likely to occur more often in cases with a PTA of more than 24 hours. Of the 166 patients with a PTA less than 24 hours, nine developed late epilepsy; of the 32 patients with a PTA of more than 24 hours, seven developed epilepsy (Table 6). These figures confirm Jennett’s statement about the relationship between length of PTA and late epilepsy. Of the 10 patients with early epilepsy three adult patients developed late epilepsy. Of the 16 patients with late epilepsy eight had generalized attacks, two focal motor attacks, and six temporal lobe epilepsy.

**TABLE 4**

<table>
<thead>
<tr>
<th></th>
<th>Glasgow (%)</th>
<th>Rotterdam (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early epilepsy</td>
<td>9.2</td>
<td>4.4</td>
</tr>
<tr>
<td>Late epilepsy</td>
<td>9.5</td>
<td>7.1</td>
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**TABLE 5**

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<th></th>
<th>Glasgow (400) (%)</th>
<th>Rotterdam (225) (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PTA less than 1 hr</td>
<td>53.5</td>
<td>62.2</td>
</tr>
<tr>
<td>PTA between 1 and 24 hr</td>
<td>22.7</td>
<td>23.6</td>
</tr>
<tr>
<td>PTA more than 24 hr</td>
<td>23.8</td>
<td>14.2</td>
</tr>
</tbody>
</table>

**TABLE 6**

<table>
<thead>
<tr>
<th></th>
<th>Incidence of late epilepsy</th>
</tr>
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<tbody>
<tr>
<td>PTA less than 24 hours</td>
<td>9/166 (5.4%)</td>
</tr>
<tr>
<td>PTA more than 24 hours</td>
<td>7/32 (21.9%)</td>
</tr>
</tbody>
</table>

NEUROLOGICAL DEFICIT Stöwsand and Geile (1966) compared severity of brain trauma in a series of 114 patients with depressed skull fracture with two other groups, one with linear skull fracture, the other one without skull fracture. The group with depressed skull fracture was characterized by a wider range of neurological syndromes—that is, cases without any cerebral symptoms on one hand and, on the other, some with severe cerebral deficit. Severe neurological signs were particularly observed with parietal and temporal depressed fractures.
We can confirm that severe neurological deficit lasting more than six months (like hemiparesis, dysphasia, visual field defects) is particularly observed in cases with temporal, parietal, or frontobasal localization of the depressed fracture (Table 7).

**COMPlications** Miller and Jennett (1968) stressed that serious sequelae are particularly observed in case of complications like intracranial haematoma, involvement of dural venous sinuses by the depressed fracture, and infection. The presence of one or more of these complications in patients with depressed fracture significantly increased the mortality and the incidence of prolonged neurological deficit and late post-traumatic epilepsy.

We diagnosed less intracranial haematomata than Miller and Jennett (Table 8), possibly because some cases were missed. Post-mortem examination was performed in less than half of our fatal cases. We encountered four epidural and five intracerebral haematomata. Of the latter, two were combined with an acute subdural haematoma. Seven showed a temporal or parietal localization, whereas two were localized in the frontal area. All cases were compound. Three patients died, five showed prolonged severe neurological deficit and only one patient recovered completely. Of the 27 patients with venous sinus involvement five died and six showed prolonged neurological deficit.

These findings confirm Miller and Jennett's (1968) statement that intracranial haematoma and venous sinus involvement represent a substantial additional risk to the patient. The third complication, infection, is dealt with under Treatment.

**TREATMENT**

**COMpound injuries** Recent experience with a large number of cases of penetrating missile wounds of the brain in the Vietnam war has confirmed the value of the time-honoured principles of complete surgical debridement, consisting of radical debridement of scalp and periosteum, removal of all pulped brain tissue from the missile track, removal of all indriven bone fragments, and watertight closure of the dura mater (Hagan, 1971; Hammon, 1971).

The treatment of penetrating head injuries and depressed skull fracture in civilian cases has shown a gradual change in the past decades. Classical treatment in compound cases consists of elevation and subsequent total removal of all bone fragments at the fracture site with a second operation for cranioplasty at a later date. This manner of treatment is still the method of choice for many neurosurgeons (Rowbotham, 1964; Meirowsky, 1965; Cabraai and Abeyesuriya, 1969).

Macewen (1888) was probably the first advocate of replacement of bone fragments. Coleman (1942) replaced fragments only if they were clean and if the dura mater was intact, but Leyerl (1957) also replaced fragments in cases of torn dura mater, provided tight closure within 24 hours could be performed.

According to Lewin (1966) removal of all depressed bone fragments, which often contain dirt and hair, is the routine procedure. He replaces the bony fragments only when the wound is essentially clean, the bone fragments are not obviously contaminated, the dura mater is intact, and provided it has been possible to operate within the first few hours. According to Lewin it is only in a few instances that the conditions are sufficiently favourable to justify
this procedure, which is aimed to avoid a large bony defect.

Kriss, Taren, and Kahn (1969), however, report a long-term follow-up in 79 patients with compound skull fractures treated by immediate replacement of the original bone fragments. Only two instances of infection after the procedure were reported and in 75 there was an excellent cosmetic result. They prefer this treatment to classical debridement with or without a second operation to install a prosthesis.

Our way of handling patients with compound depressed fractures has gradually changed in the last 10 years. Initially we performed the classical procedure. Since 1962 we have replaced bone fragments in an increasing number of patients; initially only in clean, but latterly also in contaminated cases. The dura mater was closed if possible, but sometimes watertight closure could not be achieved; the defect was then covered with Gelfoam.

In 60 of our 225 cases we refrained from elevation of the bone fragments for various reasons of which concomitant severe brain damage and shallow depression were the most important (Table 9). Of the 165 patients who were operated upon completely replaced in 82, partially replaced in 27, and removed in 56 cases (Table 10). Compound fractures were present in 152 patients and 13 had closed fractures; 110 patients were operated upon within six hours and 30 between six and 24 hours after the accident. In 13 the operation was delayed up to three days after the accident and in 12 up to one week. Infection occurred in five of the 109 cases with replacement of the bone fragments and in six of the 56 cases in which the fragments were removed (Table 10). There was no correlation between the infection rate and the time of operation (eight of the 10 infected cases were operated within 24 hours after the injury). Neither could any definite relation be established between infection rate and operative procedure; the number of infected cases in the group with replacement of fragments is even less than the number in the much smaller group in which fragments were removed. It should be kept in mind, however, that both groups are not similar: in severely contaminated cases the fragments were preferably removed, especially if the operation were performed more than 24 hours after the accident. Nevertheless, our experience shows that even in compound and contaminated cases excellent cosmetic results can be obtained by replacing the fragments without the development of infection. These findings are in agreement with those of Kriss et al. (1969), and of Jennett and Miller (1971). Various antibiotics were sometimes given systemically, sometimes locally, sometimes not at all, depending upon the opinion of the neurosurgeon in charge. We could not discern any significant difference between the infection rate in these sub-groups. Even in dirty compound cases the fragments were often replaced and no antibiotics given, without subsequent development of infection. However, in the three compound cases where the patient sustained his injury while being in contaminated water, severe meningitis developed despite early antibiotic treatment.

The various types of infection observed in our patients are shown in Table 11. In the four cases with osteomyelitis the infection cleared up after

<table>
<thead>
<tr>
<th>TABLE 9</th>
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<tr>
<td>REASONS FOR NOT OPERATING ON 60 OF THE 225 CASES</td>
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</table>

<table>
<thead>
<tr>
<th>Reason</th>
<th>No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severe brain damage</td>
<td>22</td>
</tr>
<tr>
<td>Shallow depression</td>
<td>31</td>
</tr>
<tr>
<td>Left hospital against advice</td>
<td>3</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>4</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>60</strong></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>TABLE 10</th>
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<tbody>
<tr>
<td>INFECTION RATE FOR DIFFERENT TYPES OF OPERATION (IN 165 OPERATED CASES)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Type of operation</th>
<th>Number of cases infected</th>
</tr>
</thead>
<tbody>
<tr>
<td>Replacement of all fragments</td>
<td>2/82</td>
</tr>
<tr>
<td>Partial replacement of fragments</td>
<td>3/27</td>
</tr>
<tr>
<td>Removal of fragments</td>
<td>6/56</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>TABLE 11</th>
</tr>
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<tbody>
<tr>
<td>TYPES OF POSTOPERATIVE INFECTION</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Infection</th>
<th>No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Meningitis</td>
<td>2</td>
</tr>
<tr>
<td>Meningitis + osteomyelitis</td>
<td>1</td>
</tr>
<tr>
<td>Osteomyelitis</td>
<td>4</td>
</tr>
<tr>
<td>Subdural empyema</td>
<td>1</td>
</tr>
<tr>
<td>Wound infection</td>
<td>3</td>
</tr>
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</table>
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secondary removal of the fragments. Only one of the infected patients, with a severe brain-stem contusion and a subdural empyema developing subsequent to the removal of an acute subdural haematoma, died.

CLOSED INJURIES The problem of treatment in closed depressed skull fracture is less complex than in compound cases. The chances of developing infection are negligible, except in the case of frontobasal fractures involving the paranasal sinuses (most of these, however, are compound).

It is, nevertheless, often difficult to decide whether the elevation of indriven fragments is necessary or not. According to Rowbotham (1964), they are better left undisturbed when they are not causing obvious cerebral symptoms or signs. In his opinion the decision to operate will moreover be influenced by the age of the patient and his type of work, but specific indications for operation exist when a patient is unconscious and thought to be suffering from cerebral compression, when there are signs of underlying brain damage, such as hemiplegia or aphasia, or when a fragment of bone is thought to have pierced the dura mater. Therefore fragments depressed for more than half an inch, those lying end on, or those obviously spiculated should be elevated. A depressed fracture overlying the sagittal or lateral sinus is better left undisturbed to avoid a torrential haemorrhage. Lewin (1966) and Kessel (1969) mention the same indications. This means that the general policy is to advise elevation, although they admit that a depressed fracture alone rarely causes cerebral compression and that epilepsy remains uninfluenced whether the depressed bone is elevated or not. They too prefer a conservative policy in elderly patients.

Of our 32 patients with closed injuries, 13 were operated on and 19 left undisturbed. Initially we handled patients according to the criteria mentioned above, but in recent years more cases were left alone, even when the indentation was more than the thickness of the skull, provided that the patient was conscious and did not show major neurological deficit. Depressed skull fractures overlying major venous sinuses were always left undisturbed unless signs of intracranial haematoma were developing. Our follow-up results in this small group of patients warrant continuation of this policy, as no adverse effects were observed. Follow-up radiographs in these unoperated cases disclosed that in most patients the indentation persisted, even after 10 years, although the sharp edges of indriven fragments usually were rounded off. In a few children less than 10 years of age with plate-like or shallow indentations the deformation gradually disappeared, and after a few years it could not be detected, either by radiography or by palpation.

DISCUSSION

The rapid increase in the number of head injuries in the last decade results in an increasing annual incidence of depressed skull fracture. This series of 225 consecutive cases with this type of injury comprises at least 95% of all cases with depressed skull fracture, occurring during the last decade in a densely populated area of 1.3 million people in and around Rotterdam.

Comparison with the Glasgow series of Miller and Jennett (1971) reveals a remarkable similarity in many of the data obtained: there is the same preponderance of young patients, with striking predominance of male cases and of compound injuries. In both series the dura mater is torn in more than half of the cases. Complications like venous sinus involvement and post-operative infection occur in a similar proportion. There is, however, a wide divergence in death-rate due to the injury between Rotterdam (11%) and Glasgow (3%). This difference is most probably due to the fact that in the Glasgow series most cases are referred to the neurosurgical unit from a large number of hospitals in a widely scattered area. In Rotterdam, on the other hand, all cases with head injuries are referred straight to our hospital. It may be assumed that only a small number of patients with rapidly fatal serious brain damage reach the Glasgow neurosurgical department, whereas most of the corresponding Rotterdam cases are admitted when they are still alive. This supposition is supported by the fact that 16 of our 25 fatal cases died within 18 hours after the injury. The great number of vehicle accidents in the Rotterdam area may be another factor. The larger percentage of patients with a post-traumatic amnesia of more than 24 hours in the Glasgow series may be explained in a similar way: severe cases admitted to hospitals in a widely scattered area and surviving more than 24 hours are more likely to be transferred to a neurosurgical unit. This results in a relative
increase in more severe cases in such a neuro-surgical unit.

Our data confirm Jennett’s (1962, 1969a,b) opinion about the different characteristics of early epilepsy (occurring within the first week after the injury) and late epilepsy (occurring more than seven days after the injury). In our series also late epilepsy was significantly more frequent when the period of post-traumatic amnesia exceeded 24 hours. The number of patients with epilepsy in this series is too small to warrant conclusions about Jennett’s statements regarding the relation between late epilepsy, dural penetration and early epilepsy.

Most patients with a depressed skull fracture recover completely, provided treatment is appropriate. Classical opinions regarding treatment of compound cases were based on war-time experience of missile injuries, and total removal of all indriven and fractured bone fragments was advocated. In the last decade an increasing number of neurosurgeons became convinced that civilian, non-missile and war-time missile cases are not similar. They replaced bone fragments in an increasing number of civilian cases. Excellent cosmetic results without significant adverse effects were reported by Kriss et al. (1969), and more recently by Jennett and Miller (1971). Our follow-up results in 165 operated cases are in agreement with those of Kriss et al. They have led to our present opinion about the handling of these patients, which can be summarized as follows:

Replacement of all or part of the bone fragments is justified in clean or contaminated compound injuries, provided the dura mater can be sutured or the dural defect can be covered with Gelfoam. The operation should be performed preferably within 24 hours after the accident. Meticulous debridement of scalp, periosteum, dura mater, and sometimes brain tissue, is vital. In some cases the most contaminated bone fragments can be removed without adverse cosmetic effect. If, in rare cases, local infection and osteomyelitis develop the infected bone fragments should be removed; likewise in cases who have established infection upon admission. With this way of handling the number of second operations for cranioplasty has declined sharply. The value of ‘prophylactic’ antibiotic treatment in these cases is not yet established and needs further consideration. The value of elevating indriven bone fragments in closed cases is open to doubt. The number of closed cases is, unfortunately, too small to warrant more definite conclusions.

The author is indebted to Professor S. A. de Lange and Dr. R. C. Kruyt for their kind permission to include their cases in this study. He thanks Dr. G. Blaauw for his helpful comment and valuable criticism of the draft paper.

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*J Neurol Neurosurg Psychiatry* 1972 35: 395-402
doi: 10.1136/jnnp.35.3.395

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