SURGICAL MANAGEMENT OF DEPRESSED CRANIAL FRACTURES

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RECOMMENDATIONS

(see *Methodology*)

Indications

- Patients with open (compound) cranial fractures depressed greater than the thickness of the cranium should undergo operative intervention to prevent infection.
- Patients with open (compound) depressed cranial fractures may be treated nonoperatively if there is no clinical or radiographic evidence of dural penetration, significant intracranial hematoma, depression greater than 1 cm, frontal sinus involvement, gross cosmetic deformity, wound infection, pneumocephalus, or gross wound contamination.
- Nonoperative management of closed (simple) depressed cranial fractures is a treatment option.

Timing

- Early operation is recommended to reduce the incidence of infection.

Methods

- Elevation and debridement is recommended as the surgical method of choice.
- Primary bone fragment replacement is a surgical option in the absence of wound infection at the time of surgery.
- All management strategies for open (compound) depressed fractures should include antibiotics.

KEY WORDS: Antibiotic prophylaxis, Burr hole, Cranial fracture, Craniotomy, Depressed cranial fracture, Depressed skull fracture, Head injury, Skull fracture, Surgical technique, Traumatic brain injury

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OVERVIEW

The presence of a cranial fracture has consistently been shown to be associated with a higher incidence of intracranial lesions, neurological deficit, and poorer outcome (4, 8, 12, 14). Indeed, Chan et al. (4) found cranial fracture to be the only independent significant risk factor in predicting intracranial hematomas in a cohort of 1178 adolescents. Macpherson et al. (12) found that 71% of 850 patients with a cranial fracture had an intracranial lesion (i.e., contusion or hematoma), compared with only 46% of 533 patients without a cranial fracture. Hung et al. (8) determined that patients with both loss of consciousness and cranial fracture were at significantly greater risk of developing a "surgically significant intracranial hematoma" than those with one or neither condition. Servadei et al. (14) showed the importance of cranial fracture in predicting the presence of intracranial lesions, even in minor head injuries (Glasgow Coma Scale score 14 or 15). These studies underscore the importance of cranial fractures as indicators of clinically significant injuries, as well as the importance of computed tomographic (CT) scans in evaluation of all patients with known or clinically suspected cranial fractures.

Depressed cranial fractures may complicate up to 6% of head injuries in some series (7), and account for significant morbidity and mortality. Compound fractures account for up to 90% of these injuries (3, 6, 17), and are associated with an infection rate of 1.9 to 10.6% (9, 13, 16, 17), an average neurological morbidity of approximately 11% (6), an incidence of late epilepsy of up to 15% (10), and a

mortality rate of 1.4 to 19% (3, 5–7, 17). By convention, compound depressed cranial fractures are treated surgically, with debridement and elevation, primarily to attempt to decrease the incidence of infection. Closed ("simple") depressed cranial fractures undergo operative repair if the extent of depression is greater than the full thickness of the adjacent calvarium, with the theoretical benefits of better cosmesis, a diminution in late-onset posttraumatic epilepsy, and a reduction in the incidence of persistent neurological deficit. There is, however, very little literature to support these management strategies, despite their widespread, and theoretically sound, practice. There is Class III literature that addresses the efficacy of surgical management of these injuries, and it argues against automatic surgical treatment of all compound fractures (7).

Most of the literature reviewed focuses predominantly on infectious complications, seizures, surgical technique (e.g., bone fragment replacement versus removal), or the predictive power of cranial fracture for the presence of other intracranial pathology. Several large studies of patients with cranial fracture shed light on the breadth of issues associated with such lesions and are discussed below, under Scientific Foundation. However, some of these studies were conducted before the CT-scan era, and thus, although important for our understanding of the injury itself, are not included for critical analysis.

PROCESS

A MEDLINE computer search using the following key words: "skull" and "fracture" and "depressed" between 1975 and 2001 was performed. A total of 224 documents were found. The search was narrowed to include the key words: "surgery" or "operation" or "elevation". A total of 122 articles were found, 5 of which met the criteria for critical analysis. In addition, the reference lists of all articles were reviewed, and additional articles were selected for background information. The results of this analysis were incorporated into the review presented here. Papers primarily addressing the following topics were not included: patients with associated medical illnesses, sinus fractures, cranial base fractures, isolated orbital or facial fractures, and pre-CT era reports. In general, papers with the following characteristics were also excluded: case series with less than 10 patients evaluated by CT scan and with incomplete outcome data (mortality or Glasgow outcome score), case reports, operative series with operations occurring longer than 14 days from injury. Several articles with case series of less than 10 patients were examined and reviewed because of the limited number of patient series evaluating the acute surgical management of depressed cranial fractures in the CT era. Selected articles were evaluated for design, prognostic significance, therapeutic efficacy, and overall outcome. In addition, several articles were reviewed for the purposes of historical perspective.

SCIENTIFIC FOUNDATION

Closed, linear cranial fractures are considered nonoperative lesions unless associated with surgical intracranial masses. Controversy surrounds appropriate management of depressed cranial fractures. Compound depressed cranial fractures are depressed fractures with an overlying scalp laceration in continuity with the fracture site and with galeal disruption, and have conventionally been treated with debridement and surgical elevation (3, 6, 9, 14). Simple depressed cranial fractures have no galeal disruption and are traditionally managed with surgical elevation only if the extent of depression equals or exceeds the thickness of adjacent, intact bone, or if there is an associated intracranial hematoma with mass effect that requires evacuation.

The rationale for aggressive treatment of depressed cranial fractures stems from their association with infection and late epilepsy. Cosmetic deformity also plays a role in surgical decision making. Such complications, and their potential sequelae, are well documented. In a series of 359 patients with compound cranial fractures, Jennett and Miller (9) documented a 10.6% incidence of infection, which was associated with a significantly higher incidence of persistent neurological deficit, late epilepsy (defined as seizures longer than 1 wk from injury), and death. Operative debridement reduced the incidence of infection to 4.6% in their series. Operative delay greater than 48 hours from injury dramatically increased the incidence to 36.5%. There was no difference in infection rate between surgical cohorts who had bone fragments replaced versus removed—results supported by a series of 225 patients with depressed cranial fracture reported by Braakman (3), and a treatment strategy reported as early as Macewan in 1888 (9). In a separate report of 1000 patients with nonmissile depressed cranial fractures, Jennett et al. (10) documented a 15% incidence of late epilepsy, which was significantly associated with posttraumatic amnesia longer than 24 hours, torn dura, the presence of focal neurological signs, and the presence of early epilepsy (i.e., within 1 wk of injury). In the closed-fracture patients in this series, there was no difference in incidence of epilepsy between the elevated and nonelevated cases. Additionally, there was a higher incidence of late epilepsy in patients with elevated compound fractures. The authors explain this finding by documenting a higher incidence of those factors independently associated with late epilepsy, such as dural tearing and long posttraumatic amnesia, in the elevated-fracture patient cohort. These series were reported before the CT era, however, they offer us a clear picture of both the range of complications associated with nonmissile depressed cranial fractures and the controversies surrounding management strategies.

The primary question facing the neurosurgeon regarding depressed cranial fracture is whether to operate. Heary et al. (7) reported a group of patients with compound depressed cranial fractures in which nonsurgical therapy was used for a subgroup of 26 patients without clinical or radiographic evidence of dural violation or significant underlying brain injury. They concluded that patients with open (compound) depressed cranial fractures may be treated nonoperatively if there is no clinical or radiographic evidence of dural penetration, significant intracranial hematoma, depression greater

than 1 cm, frontal sinus involvement, gross deformity, wound infection, pneumocephalus, or gross wound contamination. No infectious complications occurred. Similarly, van den Heever and van der Merwe (16) reported an equally low incidence of infection in a group of nonoperatively treated patients that included 139 compound depressed fractures. Surgical indications in their series included clinical characteristics of the wound. CT scans were not routinely used unless a neurological deficit was present on admission.

Although these studies are retrospective and nonrandomized, and, thus, subject to inherent biases, they clearly demonstrate that at least a select group of patients with compound depressed cranial fractures will do well without surgery.

Another challenge to traditional thinking that has surfaced in the literature involves the proper surgical management of compound depressed cranial fractures with respect to the bone fragments. Conventional treatment involves operative debridement, elevation of the fracture, removal of bone fragments, and delayed cranioplasty. However, this subjects the patient to a second operation (i.e., cranioplasty), with its attendant risks and complications. Kriss et al. (11), Jennett and Miller (9), and Braakman (3) showed that infectious complications are not increased by primary bone fragment replacement. Wylen et al. (17) retrospectively reviewed a series of 32 patients who underwent elevation and repair of a compound depressed cranial fracture with primary replacement of bone fragments within 72 hours of injury. Patients treated longer than 72 hours after injury and patients who presented with existing infection were excluded from the study. There were no infectious complications. Blankenship et al. (2) also demonstrated a 0% infection rate in 31 children with compound depressed cranial fractures treated with primary bone fragment replacement, regardless of the degree of contamination of the wound at the time of surgery. Thirty patients in this series were treated within 16 hours of injury. Likewise, Adeloye and Shokunbi (1) report the success of immediate bone replacement, without infectious sequelae, in 12 patients with compound depressed fractures, 11 of whom were treated within 10 hours of injury. Four patients in their series were treated with free-fragment removal secondary to the greater severity of parenchymal injury, suggesting benefit from the decompression that bone removal would provide. Despite the retrospective, uncontrolled, nonrandomized design of these observational studies, they clearly demonstrate the feasibility of immediate bone fragment replacement without a corresponding increase in infectious sequelae, thus, obviating the need for a second surgical procedure.

SUMMARY

The majority of studies are case series. No controlled, prospective clinical trials of treatment using surgical versus nonsurgical management have been published. The majority of data support debridement and elevation of grossly contaminated compound depressed cranial fractures as soon as possible after injury. However, several retrospective studies demonstrate successful nonoperative management of some patients with less-severe compound depressed cranial fractures on the basis of CT and clinical criteria. In the absence of gross wound infection at the time of presentation, immediate replacement of bone fragments seems not to increase the incidence of infection if surgery is performed expeditiously, and this replacement eliminates the need for subsequent cranioplasty and its attendant risks and complications. No controlled data exist to support the timing of surgery or the use of one technique over another.

KEY ISSUES FOR FUTURE INVESTIGATION

To improve the strength of recommendations above the option level, well-controlled trials of surgical technique are warranted, and should examine issues of bone fragment replacement versus removal, dural laceration repair, etc., and their respective relationship to outcome variables, such as incidence of infection, incidence of epilepsy, need for reoperation, surgical complications, and, most importantly, neurological and neuropsychological outcomes.

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TABLE 1. Surgical management of depressed cranial fractures[a]

Authors	No. of patients	Class	Inclusion GCS	Treatment	Outcome	Description	Conclusion
Adeloye and Shokunbi (1)	16	III	GCS 15	Bone fragment replacement versus removal	Infection rate; GOS; timing not specified	Retrospective series of 16 patients treated either with immediate bone fragment replacement (n = 12) or free fragment removal (n = 4) to compare infection rate and outcome between groups.	●No incidence of infectious complication in either group, supporting the safety of bone fragment replacement. ●Selection bias and small number of patients preclude comparison of outcome. **No. of patients / GR (%) / Infection (%)** Replacement: 12 / 100 / 0 No replacement: 4 / 100 / 0
Blankenship et al. (2)	31	III	Unknown	Bone fragment replacement	Infection or nonunion, average 26.5 mo	Retrospective review of 31 children aged 20 mo to 17 yr surgically treated with primary bone fragment replacement for compound depressed cranial fracture to examine the incidence of infectious complications. Thirty of 31 patients were treated within 16 h of injury.	●No instances of infectious complications and no need for subsequent cranioplasty, regardless of degree of wound contamination. **No. of patients / Infection (%)** Replacement: 31 / 0
Braakman (3)	225	N/A	N/A	Bone fragment replacement versus removal	Infection rate; incidence of epilepsy	Retrospective review of 225 patients with depressed cranial fracture to examine epidemiology, assess outcome with respect to incidence of epilepsy and persistent neurological deficit, and to assess the effect of primary bone fragment replacement on infection rate.	●4.4% incidence of early epilepsy. ●7.1% incidence of late epilepsy. ●Primary replacement of bone fragments did not alter infection rate. **No. of patients / Infection (%)** Replacement: 82 / 2.4 Partial replacement: 27 / 11.1 Removal: 56 / 10.7
Heary et al. (7)	54	III	Excellent, good, fair, poor	Surgery and nonsurgical	Mean GOS, 9.5 mo	Retrospective review of 54 prospectively treated patients with compound depressed cranial fractures comparing patients treated surgically versus nonsurgically based on a standardized treatment protocol involving clinical and CT criteria.	●No difference in outcome between patients treated surgically versus nonsurgically with compound depressed cranial fractures that have no clinical or radiographic evidence of dural penetration, significant intracranial hematoma, depression >1 cm, frontal sinus involvement, gross deformity, wound infection, or gross wound contamination. ●No infectious complications occurred in either group with standardized treatment protocol including prophylactic antibiosis. ●Patients with surgical lesions had significantly increased incidence of SAH and cerebral contusion ($P < 0.01$ and $P < 0.05$, respectively). ●Mortality 5.6% of those analyzed, 19% total. **No. of patients / Excellent (%) / Good (%) / Fair (%) / Poor (%) / D (%)** Surgery: 28 / 64.3 / 21.4 / 10.7 / 0 / 3.6 Nonsurgical: 26 / 80.8 / 3.8 / 7.7 / 0 / 7.7
Jennett and Miller (9)	359	N/A	N/A	Early versus delayed; bone fragment replacement versus removal; dura closed versus open	Infection rate, CNS signs, late epilepsy, mortality	Retrospective review of 359 patients with compound, depressed cranial fractures to examine the causes and consequences of infection and to assess the effect of surgical timing and methods on infection rate.	●10.6% overall infection rate. ●Infection was significantly associated with increased mortality, prolonged CNS signs, late epilepsy. ●Incidence of infection was significantly greater in patients with >48 h delay between injury and operation. ●No difference in infection rate between the group whose dura was closed and the group whose dura was left open. ●Primary bone fragment replacement did not significantly affect postoperative infection rate or incidence of late epilepsy.
Jennett et al. (10)	1000	N/A	N/A	Surgery versus no surgery	Incidence of early and late epilepsy	Retrospective review of 1000 patients with nonmissile depressed cranial fractures to study risk factors associated with posttraumatic epilepsy.	●10% incidence of early epilepsy (1 wk). ●15% incidence of late epilepsy (>1 wk). ●Increased risk of late epilepsy with posttraumatic amnesia >24 h, torn dura, focal neurological signs, occurrence of early epilepsy. ●Lower risk of early/late epilepsy with occipital fractures.

TABLE 1. Continued

Authors	No. of patients	Class	Inclusion GCS	Treatment	Outcome	Description	Conclusion
Mendelow et al. (13)	176	N/A	N/A	Prophylactic antibiotics versus no antibiotics	Infection rate	Retrospective series of 176 patients with compound, depressed cranial fractures to determine factors that predispose to infection and the effect of prophylactic antibiotics on infection rate.	•6.3% overall infection rate. •Prophylactic use of ampicillin and a sulphonamide was associated with a significantly lower infection rate (1.9%) than with any other combination (15.6%) or no antibiotics at all (10.5%). •Dural penetration, primary versus secondary closure, sinus involvement, surgery versus no surgery, bone replacement versus removal did not significantly affect infection rate.
Steinbok et al. (15)	111	III	Duration of coma	Surgery and nonsurgical	Incidence of seizures, neurological dysfunction, cosmesis; timing not specified	Retrospective series of 111 children <16 yr with simple depressed cranial fractures to examine outcome with respect to the occurrence of seizures, neurological dysfunction, and cosmesis between surgically and nonsurgically managed patients.	•No difference in seizures, neurological dysfunction, cosmetic deformity between surgical and nonsurgical treatment. Groups differed with respect to maximum depression of fracture ($P = 0.000$), but not to duration of coma, age. •Dural laceration was significantly associated with neurological deficit.
van den Heever and van der Merwe (16)	284	III	Unknown	Surgery versus nonsurgical	Septic complication; outcome of focal neurological abnormalities; mortality	Retrospective review of 284 patients whose primary injury was nonmissile depressed cranial fracture to compare outcome between surgical and nonsurgical groups. Surgical indications detailed.	•5.3% infection rate—2.8% conservative versus 8% surgical. •5% incidence of deterioration of neurological deficit—4% conservative versus 7% surgical. •1.4% mortality, all in surgical group—selection bias noted. Note: cannot separate patients with CT. Paper included to show comparable outcome between surgical and nonsurgical management of depressed cranial fracture.
Wylen et al. (17)	32	III	GCS 6–15	Bone fragment replacement	Infection, average 22 mo	Retrospective review of 32 patients surgically treated with primary bone fragment replacement for compound depressed cranial fracture within 72 h of injury to examine infection rate.	•No instances of infectious complications or new/worsened neurological deficit following primary bone fragment replacement within 72 h of injury. •25% incidence of persistent neurological deficit at 6 mo.

	No. of patients	Infection (%)
Initially clean, injury to OR <36 h	283	3.9[1]
Initially clean, injury to OR >48 h	21	4.8
Injury to OR >48 h or never	76	36.5[2]
Bone fragment replacement	166	3.6
Bone fragment removal	135	4.4

[1] P = n.s, [2] $P < 0.001$

	No. of patients	Infection (%)
Ampicillin + sulphonamide	107	1.9
No antibiotics	19	10.5[1]
All other antibiotics	45	15.6[2]

[1] $P < 0.05$, [2] $P < 0.01$, compared with ampicillin + sulphonamide

	No. of patients	Seizures (%)	Cosmetic deformity (%)	Residual deficit (%)
Surgery	19	10.5	21	0
Nonsurgical	23	4.3	17.4	0

	No. of patients	Infection (%)	Mortality (%)
Nonsurgical	139	2.8	0
Surgery	124	8	3.2

	No. of patients	Deterioration (%)
Nonsurgical	62	4

	No. of patients	Infection (%)
Replacement	32	0

[a] GCS, Glasgow Coma Scale; GOS, Glasgow outcome score; GR, good recovery; N/A, not applicable; CT, computed tomographic scan; SAH, subarachnoid hemorrhage; D, death; CNS, central nervous system; OR, operation; n.s., not significant.