Bitemporal head crush injuries: clinical and radiological features of a distinctive type of head injury

José González Tortosa, M.D., Juan F. Martínez-Lage, M.D., and Máximo Poza, M.D., Ph.D.

Regional Service of Neurosurgery, “Virgen de la Arrixaca” University Hospital, Murcia, Spain

Object. Most craniocerebral injuries are caused by mechanisms of acceleration and/or deceleration. Traumatic injuries following progressive compression to the head are certainly unusual. The authors reviewed clinical and radiological features in a series of patients who had sustained a special type of cranial crush injury produced by the bilateral application of rather static forces to the temporal region. Their aim was to define the characteristic clinical features in this group of patients and to assess the mechanisms involved in the production of the cranial injuries and those of the associated cerebral and endocrine lesions found in this peculiar type of head injury.

Methods. Clinical records of 11 patients were analyzed with regard to the state of consciousness, cranial nerve involvement, findings on neuroimaging studies, endocrine symptoms, and outcome. Furthermore, an experimental model of bitemporal crush injury was developed by compressing a dried skull with a carpenter’s vice.

Seven of the 11 patients were 16 years old or younger. All patients presented with a characteristic clinical picture consisting of no loss of consciousness (six patients), epistaxis (nine patients), otorrhagia (11 patients), peripheral paralysis of the sixth and/or seventh cranial nerves (10 patients), hearing loss (five patients), skull base fractures (11 patients), pneumocephalus (11 patients), and diabetes insipidus (seven patients). Ten patients survived the injury and most recovered neurological function.

Conclusions. Static forces applied to the head in a transverse axis produce fractures in the skull base that cross the midline structures without producing significant cerebral damage. Stretching of cranial nerves at the skull base explains the nearly universal finding of paralysis of these structures, whereas an increase in the vertical diameter of the skull accounts for the occurrence of diabetes insipidus in the presence of an intact function of the anterior pituitary lobe. The association of clinical, endocrine, and neuroimaging findings encountered in this peculiar type of head injury supports the idea that this subset of injured patients has a distinctive clinical condition, namely the syndrome of bitemporal crush injury to the head.

Key Words • skull injury • skull base • otorrhagia • epistaxis • sixth cranial nerve • seventh cranial nerve • nerve injury • diabetes insipidus • pneumocephalus

Most traumatic craniocerebral lesions seen in daily practice are caused by accidents involving an acceleration or deceleration mechanism. Injuries to the head due to the accidental application of bilateral temporal progressive crushing forces are rare. We report on 11 patients who suffered this type of head injury and analyze their characteristic presentation. In addition, we developed an experimental model of a crush injury to the skull in an attempt to explain the clinical and radiological features encountered in this condition. The combination of clinical and radiological features we report are unique to this type of bitemporal crush injury and constitute a distinctive clinical entity.

Clinical Material and Methods

The medical records of those patients with head injuries treated at our institution between 1980 and 2000 were searched for cases of cranial crush injuries. We identified 11 patients whose head injury had been caused by bilateral temporal compression of the skull at the time of the accident, and these patients are the subject of this study. Patients with crushing head injuries that did not involve the bitemporal axis were excluded. Seven patients have already been reported on in the Spanish literature.

Epidemiological data, clinical presentation, neuroimaging studies, and outcomes were analyzed in all patients.

Results

Most patients with crush injuries to the head were found to share a constellation of signs and symptoms, which are summarized in Table 1.

Illustrative Case Report: Case 1

This 10-year-old boy sustained a head injury when his head became trapped in a packing machine. He did not lose consciousness, but he was bleeding from his nose and ears. On initial examination, the boy was fully alert, although he displayed bilateral paralysis of the sixth and seventh cranial nerves and thus was unable to produce saliva and tears.

Abbreviation used in this paper: CSF = cerebrospinal fluid.
Skull radiographs demonstrated air in the basilar cisterns and the ventricles (Fig. 1 right). Additional images revealed diastasis of the sphenoidocipital synchondrosis (Fig. 2 upper), together with bilateral fractures of the sphenoid sinus walls, which appeared to be occupied (Fig. 2 lower). An electroencephalography study revealed delta waves from both temporal lobes and bursts of slow waves—up to two cycles per second and 100 mV on the posterior cerebral regions—which were believed to represent disturbed function of the basilar cerebral structures. A few hours after admission, the boy was found to have diabetes insipidus and a right CSF otorrhea, which subsided spontaneously. Assessment of the boy’s hypothalamus–adenohypophysial axis endocrine function indicated normal results. At the latest follow-up visit 2 years after the accident, the boy exhibited normal body constitution, secondary sexual features, and full-range ocular movements together with return of salivation and tears. Nonetheless, he continues to suffer bilateral peripheral facial paralysis (House–Brackmann\textsuperscript{13} Grade II) and is taking desmopressin to control the diabetes insipidus.

### Clinical Features

Seven of the 11 patients were children or adolescents with ages between 10 and 16 years (Table 1). In all cases, the patient’s head had been compressed in a bitemporal direction by a moving object against an immobile surface (Table 2), although an additional acceleration or deceleration component was present in some instances. Six patients never lost consciousness. All individuals exhibited blood discharge from the ear, being bilateral otorrhagia in eight patients. Epistaxis was recorded in all but two cases. Ten patients were found to have a sixth cranial nerve palsy, which occurred bilaterally in eight. A peripheral facial nerve paralysis was noted in 10 patients (bilateral in six and unilateral in four). Impaired tear production was observed in seven patients; five patients experienced hearing loss, which was bilateral in one patient.

### Neuroradiological Findings

Skull radiographs, which had been obtained in all patients, exhibited air within the cranial cavity, generally in the vicinity of the sphenoid sinus (Fig. 3), and within the intraventricular region in six patients (Table 3). Nine injured patients exhibited a fracture of the temporal squama. Of the seven patients with an age of 16 years or younger, a fracture in the sphenoidocipital synchondrosis was demonstrated in three, whereas a fracture of the lateral wall of the sphenoid sinus was visualized in six. A longitudinal fissure of the petrous bone was observed in seven cases along with a fracture of the sellar floor in four (Fig. 4). No patient had an associated brain lesion.

### Early Evolution

Shortly after admission, four patients experienced CSF otorrhea and one developed rhinorrhea, which was transient in all five of them. Seven individuals developed diabetes insipidus, which became permanent in four. An adult patient showed a carotid–cavernous fistula that required endovascular treatment. One of the patients, who had been admitted while in a coma, died of sepsis following aspiration pneumonia.

![Fig. 1. Case 1. Left: Skull radiograph revealing air within the basilar cisterns and ventricles. Right: Skull radiograph demonstrating a fracture of the temporal squama radiating longitudinally to the petrous bones (arrows).](image-url)
Patient Outcomes

Nine patients underwent a follow-up review at 18 months postinjury. Among eight patients who initially experienced a facial paralysis, the condition was ameliorated and a House–Brackman Grade II paresis was demonstrated in five; a right-sided Grade II and a left-sided Grade V paresis remained in one patient. Hearing loss, which had been initially present in five patients, persisted in two of them, although the deficit was only 5% in one of these patients. With the exception of one patient, all individuals who had presented with paralysis of the sixth cranial nerve recovered. The patient with a carotid–cavernous fistula (Case 8) still complained of diplopia, although no objective deficit in ocular movements was noted on examination. Only four of seven cases of early diabetes insipidus still required treatment with desmopressin. Endocrine tests for anterior pituitary lobe function were normal.

Experimental Model

We developed an experimental model of crush injury to the skull. A dried skull was submitted to bitemporal compressive forces delivered by a carpenter’s vice (Fig. 5). Skull compression was stopped as soon as the first traces of producing a skull fracture were noted. The application of these forces caused a posterior and inward avulsion of both petrous bones, which became separated from their anterolateral attachments (Fig. 6). The fracture line extended behind the spinous and ovale foramina and widened the foramen lacerum to join with a contralateral fracture across the sphenoorbitocranial synchondrosis. The tips of both petrous bones became approximated, overriding the basilar portion of the occipital bones. In addition to causing diminution of the bitemporal diameter, the vertical diameter of the skull was increased by 8 mm (Fig. 7).

Discussion

In a review of the neurosurgical literature published in English, we found 18 additional cases of crush injuries to the head.14,31,35 Apparently, an additional two cases reported in the study of Prasad, et al.,26 were also caused by bitem-
poral compression of the skull according to descriptions of the patients’ cranial and cerebral lesions. The injuries found in the study by Duhaime, et al.,9 indicate that skull compression was produced either frontally or in an anteroposterior direction. We have not included the “kolhu injury,”20 a type of crush injury caused by a machine used in India for pounding sugar cane, because all reported cases represent a completely different type of head injury from the one described here. Perhaps with a kolhu injury, a sharp surface of the machinery applies an acute load of energy onto a small surface, which is capable of causing a depressed skull fracture and a subjacent cerebral lesion before producing an important cranial deformation.

The results of our experimental model of skull crush injury are in agreement with those in the study by Russell and Schiller31 on postmortem adult skulls. The findings indicate that bilateral critical pressure applied to the skull always produces a fracture that runs in the same direction as the applied force. This fracture line joins another on the opposite side and traverses the dorsum and sellar floor or the sphenooccipital synchondrosis.37 The tympanic cavity was also involved in half of the cases reported by Russell and Schiller.31

State of Consciousness

A striking feature in this type of head trauma is the absence, or the short duration, of the loss of consciousness with which these patients present, especially taking into account the severe appearance the patients manifest. In fact, the patients may bleed from the ears and nose, show extracranial motility deficits and facial paralyses, and present with pneumocephalus and disturbances of the hypothal-

<table>
<thead>
<tr>
<th>Finding</th>
<th>No. Positive Cases/Cases Studied</th>
</tr>
</thead>
<tbody>
<tr>
<td>fracture of temporal squama</td>
<td>9/10</td>
</tr>
<tr>
<td>fracture of petrous bone</td>
<td>7/8</td>
</tr>
<tr>
<td>fracture of sphenooccipital</td>
<td>3/7</td>
</tr>
<tr>
<td>synchondrosis</td>
<td></td>
</tr>
<tr>
<td>fracture of sphenoid sinus</td>
<td>6/7</td>
</tr>
<tr>
<td>fracture of sellar floor</td>
<td>4/10</td>
</tr>
<tr>
<td>pneumocephalus</td>
<td>11/11</td>
</tr>
<tr>
<td>pneumoventricle</td>
<td>6/11</td>
</tr>
</tbody>
</table>

amis–hypophysial axis. The explanation for these occurrences is that the head remained fixed between the hitting or compressing object and another surface that prevented head rotation. Denny-Brown and Russell8 demonstrated how easy it is to produce a brain concussion in experimental animals when the head is unrestrained and, conversely, how difficult it is when the skull is immobilized. In the latter condition, the brain is hardly displaced,27 and, accordingly, rotational lesions responsible for brain concussion do not occur.12,24 Mechanical forces by themselves are capable of killing patients, but they generally do not produce brain concussion. In addition to the bitemporal compression, mixed mechanisms may be involved in certain cases. In the latter scenario, acceleration and/or deceleration forces participate in producing abrupt shifts of intracranial structures, which cause the typical alterations seen in ordinary head injuries.

Sixth Cranial Nerve Paralysis

With raised intracranial pressure39 or acceleration or deceleration head injuries,52 it has been hypothesized that upward or downward displacement of the brainstem is responsible for the occurrence of lesions of the sixth cranial nerve. The nerve is stretched against the rigid edge of the petrous bone or against the Gruber ligament that fastens it from above, although Takagi, et al.,58 have pointed out that the dura mater is the structure that fixes the nerve against the apex of the petrous pyramid underneath this ligament. We agree with Russell and Schiller51 in that the true cause of the injury of the sixth cranial nerve is the displacement of the petrous apex proper (Fig. 8). In fact, the incidence of sixth cranial nerve paralysis in several series on head injuries was 1 to 3%,51 and 6.6% in a study of 75 fractures of the petrous bone.58 These incidences contrast markedly with the high

Fig. 4. Skull radiograph depicting a fracture of the sellar floor.

Fig. 5. Photograph obtained during an experimental model of a crush injury to the head by using a dried skull.
incidence of paralysis of the abducens nerve in cases of bitemporal crush injuries, that is 90.9%. It is apparent that the biomechanics in this type of injury are directly responsible for the production of lesions of this cranial nerve. A similar pathogenic mechanism might also explain the rare reported cases of complete injury of the fifth cranial nerve, because this nerve enters the Meckel cave just above the vertex of the pyramid. In the reviewed literature, we read about only two patients with a selective lesion of the third division of the trigeminal nerve. Most likely in these two cases, the site of the injury was situated at the foramen ovale and was directly involved by the fracture.

Cochlear and Facial Nerve Lesions

The presence of otorrhagia, CSF otorrhea, transmission deafness, and paralysis of the facial nerve point toward the tympanic region as the meeting place for all these lesions. This suspicion is confirmed by the experimental compression of the skull. Nonetheless, the absence of tear production, in the cases in which we searched for it, indicates that the lesion of the facial nerve cannot be distal to its genu unless the fracture line also affects the great petrosal nerves, which run in the same direction and situation as the bone fissure. If this were the case, there would exist a double lesion involving one side of the facial nerve and the petrosal nerves on the other. In addition, the peculiar rotation and avulsion of the petrous bones produced in this type of injury results in widening of the foramen lacerum (Figs. 6 right and 8), which is traversed by the petrosal nerves up to the vidian foramen at the base of the pterygoid apophysis. The nerves could thus be injured by a stretching mechanism that in turn would pull the facial nerve at its genu and cause a motor facial paralysis.

Carotid–Cavernous Fistula

The association of carotid artery lesions with fractures of the skull base is well known. Resnick, et al., reported carotid artery injuries in 7.6% of their patients with basilar fractures traversing the foramen lacerum. In our series, only one patient with a fracture of the sphenoid sinus and the sellar floor developed a unilateral carotid–cavernous fistula (9%). We attribute the occurrence of this arterial lesion to the force that widened the foramen lacerum after having avulsed the petrous bone.

Diabetes Insipidus

The pathogenesis of the diabetes insipidus in the cases of bitemporal crush injuries to the head is more difficult to explain. Its overall incidence in traumatic brain injury is less than 1%, although in another series it was 2.8%. Surprisingly, 63.6% of our patients manifested this complication. Histopathological findings in individuals dying of head trauma have consisted of infarction of the anterior hypophysis, hemorrhages in the neurohypophysis, and lesions of the supraoptic and paraventricular nuclei and of the infundibulum. Such findings are usually reported in cases of extreme acceleration or deceleration traumatic brain injuries. It is noteworthy that whereas the neurohypophysis receives a rich vascular supply from the hypophysial arteries, the anterior pituitary lobe is almost exclusively supplied by the portal vessels coursing down through the pituitary stalk. Following stalk transection, an infarction...
of variable extent of the pars distalis develops, which may even account for 90% of the gland volume when the lesion is very caudal. Together with the failure of the hypothalamic hormones to reach the adenohypophysis, there is an intrinsic inability of cellular response of the gland leading to panhypopituitarism associated with diabetes insipidus.

In traumatic crush injuries of the head, it seems reasonable to think that there is no shearing of the pituitary stalk as occurs in ordinary head injuries. Furthermore, none of the seven patients with diabetes insipidus in our series developed an anterior lobe pituitary insufficiency, which indicates that this was not the level of the lesion. On the other hand, massive destruction of the posterior lobe within the pituitary fossa seems to be unlikely in the absence of a shearing mechanism and given that only three patients with diabetes insipidus had an associated fracture of the sellar floor. In addition, fractures involving the floor of the sella are not associated with a greater incidence of pituitary lesions. There is only one report of a hematoma within the neurohypophysis and diabetes insipidus following a mild head injury.

In the experimental model, skull deformation also occurred in its vertical diameter, which was increased by 8 mm (Fig. 7). This fact lends support to the view that in living individuals, especially in children, even greater cranial deformations might occur because of a higher cranial elasticity in comparison with that in a dried skull. This deformation of the cranial bones explains the upward shift of the hypothalamus, which, by being tethered to the skull base by the pituitary stalk, might suffer a bilateral stretch at the supraoptic and paraventricular nuclei (Fig. 9). This sequence of events would easily explain the development of isolated diabetes insipidus and the sparing of the remaining function of the anterior pituitary lobe.

Intraventricular air has been reported in 2% of cases of traumatic pneumocephalus, which contrasts with the 54.5% incidence demonstrated in our patients. In a series of 77 patients with posttraumatic pneumocephalus, the presence of air within the ventricles constituted a rare event despite the frequent occurrence of fractures of the sphenoid sinus and petrous bones. This overwhelming difference in the incidence of pneumoventriculus in bitemporal cranial crush injuries supports the notion of the existence of a hypothalamic tear, which would permit the intraventricular passage of air from the suprasellar cisterns, without having the air to traverse the foramina of Luschka and Magendie, as happens in ordinary head injuries. Vascular injury to the floor of the third ventricle caused by the stretch of pial or perforating vessels might explain the occurrence of other features of hypothalamic involvement, but we did not observe this occurrence.

**Conclusions**

We have reported on 11 patients who suffered a special type of head injury due to the application of bilateral compressing forces to the skull. With this type of head injury, loss of consciousness frequently does not occur and there is a high incidence of otorrhagia, epistaxis, bilateral palsies of the sixth and seventh cranial nerves, transverse fractures of the skull base, and pneumocephalus. The combination of all these clinical and neuroimaging findings constitutes a characteristic entity, namely the syndrome of bitemporal crush injury of the head. This clinical entity is easily identifiable and should indicate to the attending physician diabetes insipidus and, less often, carotid–cavernous fistula.

**References**

2. Born JD, Hans P, Smitz S, et al: Syndrome of inappropriate secre-
Bitemporal crush injuries


Address reprint requests to: J. González Tortosa, M.D., Regional Service of Neurosurgery, “Virgen de la Arrixaca” University Hospital, E-30120 El Palmar, Murcia, Spain. email: jtortosa@arrixaca.huva.es.