B-ENT, 2016, **12**, Suppl. 26/1, 193-201

Basilar skull fractures: the petrous bone

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Key-words. Skull fracture; basilar; petrous bone; cranial neuropathies; traumatic; cerebrospinal fluid otorrhoea

Abstract. *Basilar skull fractures: the petrous bone. Objectives*: to provide suggestions for the management of three of the most dangerous or important lesions (internal carotid artery lesions, cerebrospinal fluid leaks and facial nerve paralysis) associated with the petrous part of basilar skull fractures, thereby trying to assess categories of evidence and determine strengths of recommendation.
Methodology: A PubMed-based literature review was carried out, as well as a consultation of online sources as encountered in the literature review. Also, a non-systematic search of chapters of well-known books dealing with the subject of temporal bone traumata was conducted.
Results: Specific levels of evidence and/or strength of recommendation can be retrieved from the literature, but only with respect to the prophylactic use of antibiotics, the prescription of antithrombotic medications and the indications for angiography.
Conclusion: The ample amount of available literature allows for sound management decisions, with reference made to algorithms when available in the literature. Nevertheless, for most of the management/search questions, categories of evidence and strength of recommendation are low or lacking.

Introduction

Five bones form the base of the skull: the ethmoid, sphenoid, occipital, frontal and temporal bones. The temporal bone consists of four parts (Figures 1a and 1b): the squamous, mastoid, petrous and tympanic parts. The squamous part is the largest and most superiorly positioned relative to the rest of the bone. Posteroinferior to the squamous is the mastoid part. The tympanic part is relatively small and lies inferior to the squamous part, anterior to the mastoid part and superior to the styloid process.

Traumata of the middle and posterior skull base often involve the temporal (petrous) bone, as opposed to traumata of the anterior skull base, which frequently affect the paranasal sinuses. It is important to diagnose a basilar skull fracture, since even small linear non-displaced fractures can be associated with (potentially life-threatening) complications. Internal carotid artery injury, tearing of the meninges with cerebrospinal fluid (CSF) leakage and meningitis, facial nerve paralysis and deafness are among the many possible complications. These four mentioned complications are either life-threatening or resort specifically to the responsibility of the ENT surgeon. Deafness due to inner ear trauma is most often irremediable (except by the eventual use of cochlear implants or, even more exceptionally, by brain stem implants), while traumatic middle ear lesions are covered elsewhere in the current Annual Report. Therefore, the present paper will focus on the management of internal carotid artery injury, CSF leakage and facial nerve paralysis.

Methodology

In order to perform a literature review, we searched PubMed (1964 to April 2015) with "Skull fracture, basilar" as MeSH® keywords. From 299 titles and abstracts, only 80 were consulted. Titles and abstracts not in English, titles without an abstract, titles and abstracts that were considered not to be relevant to the subject, isolated case reports or reports dealing with traumata of animals were eliminated. In addition, subtopics (such as late complications and hearing loss due to penetrating traumata), which were covered by other authors

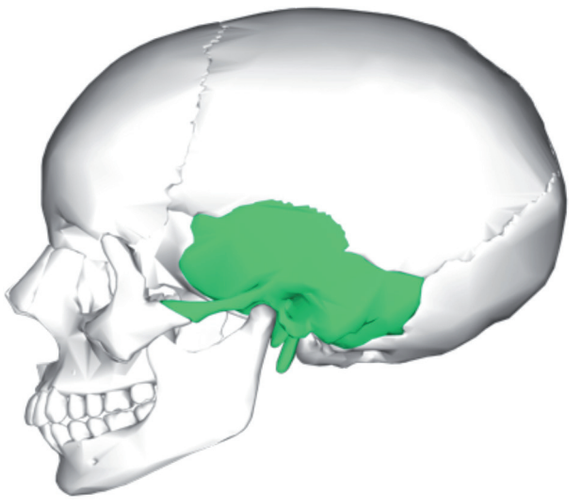

Figure 1a
Position of the temporal bone

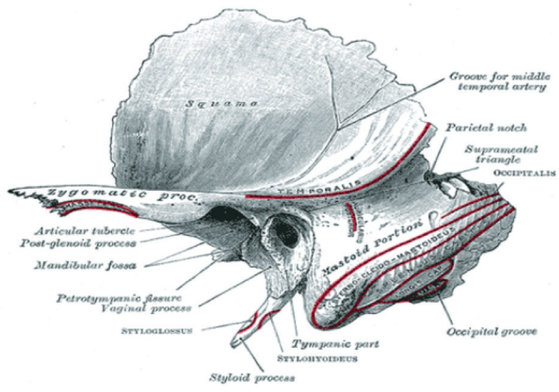

Figure 1b
Structure of the temporal bone

contributing to the present Annual Report, were discarded. Full-text articles were obtained as needed.

Additionally, the Cochrane Library (Cochrane Database Systematic Review, 2015, April 28;4), UpToDate® ("Emergency medicine, Adult trauma, Skull fractures in adults, Skull fractures in children") and the Eastern Association for the Surgery of Trauma ("Practice Management Guidelines, Blunt Cerebrovascular Injury and Mild Traumatic Brain Injury, Evaluation and Management of") were accessed on 11 December 2015. Only the first and last one of these online sources provided information discussed in the present paper.

Also, some well-known books were consulted for general background information on specific surgical procedures.

Prevalence and classification of temporal bone fractures

By means of an eight-year review involving 4,398 patients diagnosed with blunt-mechanism facial fractures, which were identified by the International Classification of Diseases (ICD-9) and by a review of CT scans, 7.6% sustained basilar skull fractures.[1] Temporal bone fractures are a frequent manifestation of head trauma. During a time span of two years, out of a total of 2,977 patients seen at a university trauma centre, 90 (3%) presented a temporal bone fracture.[2]

Traditionally, temporal bone fractures are categorized as either longitudinal or transverse, based on the orientation of the fracture line with respect to the axis of the temporal bone.[3] This classification is still useful, since it allows for some straightforward generalizations: longitudinal fractures are the most common ones (80% of temporal bone fractures), with bleeding from the ear and lesions to the tympanic membrane. The facial nerve is less frequently affected (in 5% to 30% of patients). Transverse fractures are the result of more forceful blows and present with more severe complications: lesions of the internal auditory canal, the cochlea and vestibular system, producing immediate profound sensorineural hearing loss and vertigo. Immediate and severe facial nerve paralysis is present in 50% of these transverse fractures.[3] This traditional classification has been questioned, while new classifications, based on a sparing or violation of the otic capsule, have been thoroughly assessed,[2,4] specifically with respect to associated clinical sequelae and intracranial complications. In a group of 90 patients with temporal bone fractures, the longitudinal-transverse classification could not be determined, while the otic capsule-sparing versus -violating classification seemed to be more appropriate. Patients with otic capsule-violating fractures were two times more likely to develop facial nerve paralysis, four times more likely to develop CSF leaks and seven times more likely to develop profound hearing loss, as well as more likely to sustain intracranial

complications (epidural haematoma, subarachnoid haemorrhage).[2] However, at a level I trauma centre, over a five-year time span, 202 patients presented with temporal bone fractures. The most prevalent subtypes were longitudinal (44%) and otic capsule-sparing fractures (96%). Concurrent intracranial injuries (ICI) were seen among 91% of the patients and cervical spine injuries (CSI) were seen in 9%. However, neither the longitudinal-transverse-mixed nor the otic capsule-sparing versus -involving classification correlated with these outcomes.[4]

Part I: general signs and basic principles of management of skull base trauma

Skull base fracture can give rise to numerous and diverse complications, both in the short and in the long term: (future) CSF leaks, meningoencephalocele, blepharocele, loss of consciousness, (acute) brain herniation, long-term neuroendocrine sequelae, permanent neuro(psychological) sequelae, pontomedullary lacerations, stroke and mortality. Therefore, management of skull base traumata should be assumed by a multidisciplinary effort (emergency staff members, experienced head and neck radiologists, neurosurgeons, maxillofacial surgeons, ENT surgeons, neurologists and paediatricians).

The patient's airway, breathing and circulatory system should be stabilized first. Furthermore, due to the high frequency of cervical spine injuries and head injuries, the neck-head victim should be online immobilized until spinal injury is ruled out. A rising pulse rate and falling blood pressure suggest acute blood loss, while a slowing pulse rate and rising blood pressure suggest increased intracranial pressure.[3] Only after stabilization of the patient's condition should a careful neuro-otologic examination be performed to search for signs and symptoms of temporal bone fractures. Signs of temporal bone fractures are, in descending order of frequency, as follows: haemotympanum, bleeding ear canals, hearing loss, CSF otorrhoea and/or (very rarely) rhinorrhoea, facial nerve paralysis, and (rarely) vestibular symptoms. In addition, mastoid ecchymosis (Battle's sign) and air in the temporomandibular joint fossa on a CT scan are possible signs of temporal bone fractures. As discussed below, it is critical to assess facial nerve function as soon as possible. In comatose patients, painful stimuli will induce a grimace.[5]

Part II: management of internal carotid artery lesions

Skull base fractures can cause a variety of vascular lesions: internal carotid artery injuries, carotid cavernous fistulas (suggested by chemosis, proptosis, pulsating exophthalmos, diplopia, ophthalmoplegia, orbital pain, blindness etc.), venous (transverse and sigmoid) sinus thrombosis, extracranial (but nonetheless due to skull base fractures) internal carotid artery pseudoaneurysms and vertebral artery injuries. The latter occur with a similar incidence rate as internal carotid artery injuries and are generally lead to catastrophic outcomes (brain stem dysfunction);[6] however, they are beyond the scope of the present paper. Transverse/sigmoid venous sinus thrombosis, due to temporal and occipital bone fractures, might be more frequent, as a retrospective recollection of five case reports suggests,[7] but will not be addressed either.

A carotid *canal* fracture does not necessarily mean that a carotid *artery* injury (CAI) did occur. The carotid canal is most frequently fractured at the lacerum-cavernous junction (the spheno-occipital suture). True vascular injury, however, is most often seen in fractures through the petrous segment of the canal.[8] The prevalence of CAI, as estimated by diagnostic angiography, is 2%, while the prevalence of carotid canal fracture might be 31%.[9] When considering all blunt trauma admissions, the incidence of CAI is estimated to be 0.5%, but rises in the presence of a basilar skull fracture.[10] CAI incidence (2.0%) is higher with basilar skull fractures than without. Meanwhile, even though skull fractures occur more often in males (80% of temporal bone fractures),[4] CAI is strongly associated with the female sex.[9] In an attempt to differentiate carotid artery injuries from carotid canal fractures, several potential risk factors and different imaging techniques have been analysed. Specific facial fracture patterns (bilateral, complex midface, Le Fort I, subcondylar), especially in association with basilar skull fractures, give rise to an increased risk for blunt CAI.[10] The presence of pneumocephalus, petrous carotid canal fractures and sphenoid bone fractures on CT scans of the head and cranial base (as compared with angiography) provide an 85% specificity and an 80% negative predictive value for CAI (but with a lower sensitivity).[9] However, other authors using a similar methodology, but with

different CT scan criteria, obtain a specificity of 67% and a sensitivity of 60% for the detection of injury to the internal carotid artery passing through the canal.[11] Stroke-related mortality for CAI is 13%, while the stroke rate (hemiplegia/hemiparesis) for CAI is 31%.[6] Many discrepancies were noted with respect to anticoagulation therapy (with cerebral contusion or other intracranial lesions, this may present an unacceptable risk for intracranial hemmorrhage[12]), but the stroke rate with CAI decreases in a statistically significant way to 6.8% when treated with heparin and antiplatelet therapy, as opposed to 64% in untreated vessels.[6]

Excellent information with respect to this subject can be found in the Practice Management Guidelines for Blunt Cerebrovascular Injury of the Eastern Association for the Surgery of Trauma (available at: https://www.east.org/education/practice-management-guidelines/blunt-cerebrovascular-injury; accessed on 11 December 2015), which are based on the Biffl criteria[28]. The guidelines state that patients presenting with any neurologic abnormality, which is unexplained by a diagnosed injury, or blunt trauma patients presenting with epistaxis from a suspected arterial source after trauma should be evaluated for blunt cerebrovascular injury (BCVI) (level of recommendation II) [note that the levels of recommendation and classes of quality of the evidence are somewhat different from those of Shekelle et al.[13]]. Additional risk factors in asymptomatic patients with significant blunt head trauma for evaluation for BCVI are further enumerated (level of recommendation III). Diagnostic four-vessel cerebral angiography remains the gold standard for the diagnosis of BCVI (level of recommendation II). In the absence of contraindications, grades I (intimal irregularity with <25% narrowing) and II (dissection or intramural haematoma with >25% narrowing) injuries should be treated with antithrombotic agents, such as aspirin or heparin (level of recommendation II). Details about patients for whom a more aggressive approach (surgery or angio-interventional) is needed are also outlined. No recommendations can be given for how long antithrombotic therapy should be administered, but follow-up angiography is recommended in grades I to III injuries. According to the Eastern Association for the Surgery of Trauma, this should be performed at seven days post-injury in order to reduce the incidence of angiography-related complications.

Part III: management of cerebrospinal fluid leaks

Skull base fractures can lead to meningitis due to an open connection between the central nervous system and bacteria present in the middle ear, the paranasal sinuses and nasopharynx. The occurrence of CSF leaks in the setting of skull base fractures, however, is uncommon, while that of meningitis is even less common.[14] By means of a cross-sectional analysis of a state-wide database over a 15-year time span, 3,563 paediatric and 10,761 adult patients were included.[14] The rates of CSF leaks were similar in children and adults (2.33% and 1.75%, respectively). The rates of in-hospital meningitis were 0.48% and 0.64%. The rates of CSF leaks and meningitis at 90 days were even lower, as well as similar for children and adults. Even later in life, patients can develop meningitis.[15,16] With otic capsule-disrupting fractures, there is not only a fourfold increase in CSF fistula,[2] but these fractures may also carry a higher risk of delayed meningitis due to the inability of the otic capsule enchondral bone to remodel.[5]

Laboratory tests (beta-trace protein or beta-2 transferrin) should confirm CSF in the presence of otorrhoea and/or rhinorhoea.[15] Localization of the origin of the CSF leak is, contrary to frontobasal leaks, rarely possible in temporal bone fractures by endoscopic means (with or without intrathecal, rather than topical, application of fluorescein).[15] Imaging techniques are therefore often needed. Exact radiological localization of the fistula, due to the complexity of basilar skull fractures, is always difficult.[17] In a series of 23 surgically confirmed CSF leaks, MR cisternography might yield slightly better results than high-resolution CT scans (eventually with three-dimensional reconstruction) in predicting the exact fistula location.[17]

In the presence of a CSF leak, two questions must be answered: does antibiotic prophylaxis prevent meningitis, and when is surgical closure of a CSF leak needed? The answer to the former question is thoroughly investigated by a very recent update by the Cochrane Library. By means of five randomized clinical trials (RCTs) (including 208 subjects) and 17 non-RCTs (including 2,168 patients), comparing different types of antibiotic prophylaxis with placebo or no intervention, the authors conclude: "Currently available evidence from RCTs does not support prophylactic antibiotic use in patients with basilar skull fractures, whether there is evidence

of CSF leakage or not. Until more research is available, the effectiveness of antibiotics in patients with basilar skull fractures cannot be determined because studies published to date are flawed by biases. Large, appropriately designed RCTs are needed."[18] By means of the GRADE approach, the quality of trials was assessed as moderate.[18]

The latter question, about when to surgically close a CSF leak, is the more difficult one to answer. Some authors[16] state that the "majority" of traumatic CSF leaks can be managed conservatively (elevation of the bed's head, bed rest, stool softeners), while those persisting beyond a week should be managed surgically to avoid the risk of meningitis. Other authors[16] provide more elaborate information and state that patients are already at risk of developing meningitis with leaks that persist longer than 24 hours. By means of a 15-year review of medical records, 51 patients with CSF leaks of more than 24 hours after traumatic head injury were identified. Spontaneous resolution was observed in 55% of the patients, after an average of five days. In 45% of the cases, surgery was required. Recurrent meningitis, 6.5 years after trauma on average, was due to an occult CSF leak. In 16% of the cases, delayed (with an average of 13 days post-trauma) onset of CSF was noted.[16] It is important to note that the most common fractures in this series involved the frontal sinuses, while, according to the authors of this present paper, rhinological CSF leaks are usually more aggressively tackled. Moreover, by means of prophylactic antibiotic administration, and contrary to the recommendations of the Cochrane Collaboration, the risk of meningitis is claimed to be halved (10% versus 21%).[16] According to Yilmazlar et al.,[19] CSF drainage is an option prior to surgical closure of a leak: in 39.5% of 81 patients, a CSF leak resolved spontaneously. Among 24 (29.6%) patients, CSF drainage was applied; seven of these patients thereafter required a surgical closure of the leak, bringing the total of surgical closures to 25 patients (30.9%). These authors offer also a treatment algorithm. Discussion of the different surgical techniques or materials to be used for closing CSF leaks go behind the scope of the present paper. Nevertheless, Brodie[5] provides a useful algorithm for the management of CSF leaks, while Jackler[20] provides colourful images of the possible surgical approaches/techniques: if a leak persist for seven to 10 days, in spite of conservative measures and lumbar drainage, surgical closure is advised[5]; in the presence of an otic capsule-violating temporal bone fracture, resection of the external ear canal and tympanic membrane, together with obliteration of the middle ear, the Eustachian tube and mastoid (exclusion/obliteration), is suggested.[5,20] In the presence of an otic capsule-sparing temporal bone fracture, with brain herniation, an attico-antro-mastoidectomy with mini-cranial fossa craniotomy is proposed. In the absence of brain herniation, a transmastoidal approach is suggested[5] (Figure 2).

Part IV: management of facial nerve paralysis

Although not life-threatening, as opposed to the complications of temporal bone fractures outlined above, sequelae of facial nerve paralysis can nevertheless be severely disfiguring.

With anterior and middle (temporal bone) fractures, all cranial nerves (except cranial nerves 9 to 12) are at risk: specifically for temporal bone fractures, the facial nerve is the first to sustain injury.[21] As outlined above, patients with otic capsule-violating fractures are two times more likely to develop a facial nerve paralysis;[2] fortunately, however, these otic capsule-violating fractures represent an absolute minority[2,4,5] of temporal bone fractures: 2.5% to 5.8%. The incidence of facial nerve paralysis is, therefore, estimated to be between 30% and 50% in otic capsule-disrupting fractures as opposed to between 6% and 13% in the otic capsule-sparing ones.[2,5] Brodie[5] elaborates on the sampling errors that lead to an overestimation of facial nerve paralysis and determines, by means of her own study, including 820 temporal bone fractures, that 7% of temporal bone fractures result in facial paralysis, of which 25% are complete.

Management of facial nerve injuries remains controversial and should be guided by clinical presentation, neuroradiological findings (high-resolution CT scans of the fallopian canal and MRI scans in the presence of a paralysis without positive CT scan findings[22]) and electrodiagnostic tests. One of the main issues is a correct identification of those patients who should be surgically explored. Two of the most important predictors for recovery are the timing of the onset (delayed versus immediate onset) of facial paralysis and the severity of the injury. The former predictor is questioned by Adegbite et al.,[23] as outlined below. Among 25 patients with a post-traumatic facial nerve palsy, and followed over a period of 18 months at regular intervals, 95% of

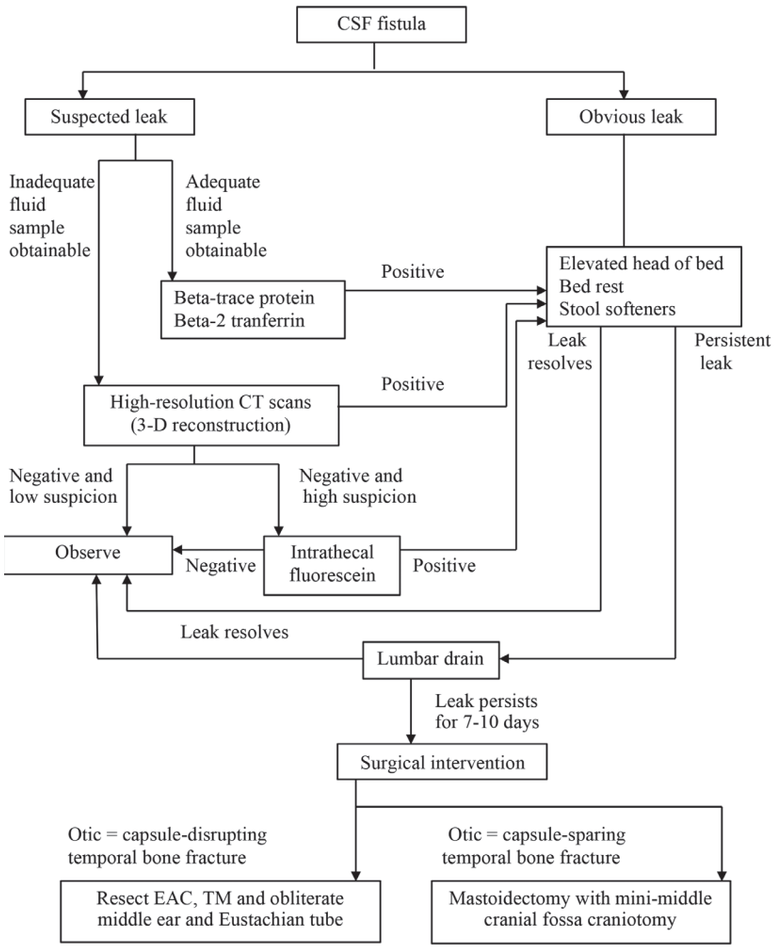

Figure 2

Suggestions for the management of cerebrospinal fluid leaks (adapted from Brodie et al.,5 according to Wang et al.,17 and Ratilal et al.18)

the patients ultimately experienced partial recovery of the facial nerve function.[23] Moreover, these authors found no statistically significant difference in recovery between immediate- and delayed-onset facial nerve palsies. The degree of palsy, however, had a statistically significant influence on the recovery of facial nerve function. These authors endorse a conservative approach to facial nerve injuries and suggest surgery for patients in whom complete facial nerve paralysis persists for 12 to 18 months after trauma.[23] With respect to documenting facial nerve recovery, the House-Brackman grading system[24] is most often used, while the more recent Sunnybrook facial grading system[25] is claimed to be more sensitive and less prone to inter-observer variation. Based on the House-Brackmann grading system, Brodie[5] reviewed the facial nerve outcome from three non-operative studies (including 59 patients) and five operative studies (including 162 subjects). A review of this literature argues strongly against surgical decompression of delayed post-traumatic facial paralysis. Facial function recovery among these patients is almost always to a House-Brackmann grade I or II. Demonstrating that decompression of a post-traumatic, non-severed nerve is efficacious remains to be proven in a randomized, prospective study.[5] Cases in which the onset of paralysis is not known should be managed in a similar way to the immediate-onset group.[5] Thereby, only patients with complete paralysis of immediate or unknown onset are considered for surgical exploration. Severity of facial nerve injury

can be assessed clinically and with electrodiagnostic testing. Once more, it is Brodie[5] who provides a concise review of these tests. Electroneuronography (ENoG) is considered the most useful. Traditionally, the outcome in patients in whom degeneration on ENoG reaches 90% within six days of the onset of traumatic facial paralysis seems to be poorer, such that the facial nerve should be decompressed in these cases. However, other authors are unable to confirm this. A similar controversy is found with respect to the presence of voluntary activity on electromyography (EMG): if present during the acute post-injury period, the probability of good recovery is high. However, only a 62% accuracy for predicting a favourable result was obtained.[5] In an extensive review, Chang and Cass[26] conclude: "Patients who should not require surgical intervention include those who have: documented normal facial nerve function after injury regardless of progression, presentation with incomplete facial nerve paralysis with no progression to complete paralysis, and degeneration <95% on ENoG. The remaining patients presumably have a poorer prognosis for return of facial nerve function although it remains unclear exactly how poor the return of function will be. Decompression surgery likely has a beneficial effect if performed within 14 days of injury, so those patients with expected poor natural outcomes may be offered this intervention. Late decompression surgery is not recommended. Late exploratory surgery is recommended only in those patients who do not experience adequate recovery of facial nerve function and likely require nerve repair. Despite the availability of a relatively large volume of published data, there remain many unanswered questions."

With all these data consulted, Brodie[5] proposes an algorithm for traumatic facial nerve paralysis (Figure 3). Although adequate literature data are once more lacking, based on the assumption that neural oedema is a primary factor in the progression of neural injury, all patients with delayed-onset facial paralysis are placed on a two-week course of systemic corticosteroids (except in the presence of medical contraindications). Patients with complete immediate-onset paralysis are submitted to electrodiagnostic tests between three and seven days post-injury. If loss of stimulability or 95% degeneration on ENoG within 14 days is observed, surgical facial nerve exploration is suggested. With otic-sparing fractures, and in the presence of a well-aerated mastoid or ossicular discontinuity, a transmastoid/supralabyrinthine total facial nerve decompression is advised (with a great auricular nerve cable or direct anastomosis, if a severed facial nerve is encountered). In the presence of otic-violating fractures, a translabyrinthine total facial nerve decompression is recommended (with a great auricular nerve cable or direct anastomosis, if a severed facial nerve is encountered). With poorly pneumatized mastoids and/or with inappropriate decompression/exposure, a combination with a middle cranial fossa approach might be necessary. It might be of importance to remember that about 80% of injuries of the facial nerve occur at the level of the geniculate ganglion and 20% in the mastoid

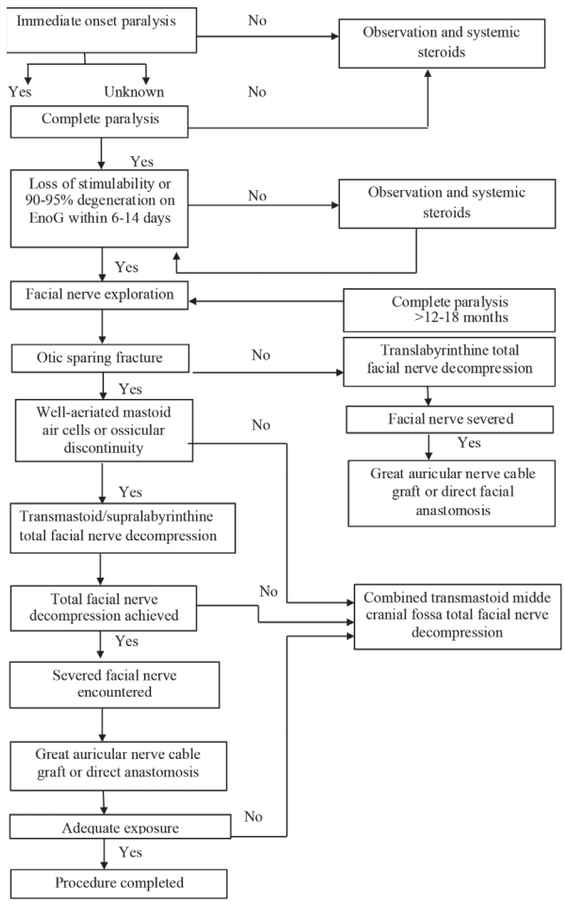

Figure 3

Suggestions for the management of traumatic facial paralysis (adapted from Brodie *et al*.,[5] according to Adegbite *et al*.[23] and Chang and Cass[26])

course. With respect to the former localization, exposed by a middle fossa approach, care has to be taken not to enter the superior semicircular canal.[5]

By means of an RCT among 50 patients with a facial nerve paralysis for more than nine months, mime therapy improved facial symmetry by 20.4 points on the Sunnybrook facial grading system, while reducing the severity of paresis with a 0.6 grade on the House-Brackmann grading system.[27]

Conclusions

Take-home messages

1. The traditional longitudinal-transverse classification of temporal bone fractures allows for a good theoretical understanding of the development of intratemporal lesions, but is hard to apply in reality. Although more modern otic capsule-sparing versus -violating classification provides apparently more suited information with respect to the likelihood of developing intratemporal complications, neither classification correlates (well) with concurrent intracranial complications or cervical spine injuries.
2. Carotid artery injury is rare, but specifically linked to temporal bone fractures. Increased awareness and aggressive screening allows for a lower stroke-related mortality and morbidity. CT scans should, therefore, be checked for pneumocephalus, petrous carotid canal fractures, sphenoid bone fractures, brain contusion, subarachnoid haemorrhage, basilar skull fracture, subdural haematoma, soft tissue swelling, sphenoid sinus air-fluid level and other skull fractures. These findings may help to diagnose carotid artery injury, but sensitivity and specificity figures are, according to the relevant literature, not uniform and too low. Diagnostic four-vessel cerebral angiography remains the gold standard for the diagnosis of BCVI in specific patients (level of recommendation II). Instauration of anticoagulation/antiplatelet treatment before ischaemia develops might lead to a near tenfold decrease in hemiplegia/hemiparesis. In the absence of contraindications, grades I (intimal irregularity with <25% narrowing) and II (dissection or intramural haematoma with >25% narrowing) vascular injuries should be treated with antithrombotic agents, such as aspirin or heparin (level of recommendation II).
3. Treatment decisions with respect to CSF leaks must be taken in relation to the general and neurological status of each individual patient and should account for associated intracranial lesions. Timing for eventual CSF drainage and surgical closure must be decided with care. Available algorithms can guide the decisions. Currently, there is no evidence to support prophylactic antibiotic use in the presence of a basilar skull fracture, whether there is evidence of CSF leakage or not (recommendation based on trials of moderate quality).
4. Many unanswered questions remain with respect to the management of post-traumatic facial nerve paralyses. On a theoretical basis, patients with delayed-onset facial paralysis are placed on a two-week course of systemic corticosteroids, at least if no medical contraindications are present. It seems easier to determine which patients do not need surgical facial nerve exploration than those who might benefit from surgery: patients with documented normal facial nerve function after injury, regardless of progression later on, presentation with incomplete facial nerve paralysis with no progression to complete paralysis, and degeneration <95% on ENoG should not be submitted to surgery. For patients who might eventually benefit from surgical exploration, reference is made in the text to an algorithm. The ENT surgeon who wants to surgically address post-traumatic facial nerve paralysis should master all necessary surgical approaches to the temporal bone, with the inclusion of a middle fossa approach (or plan surgery together with a neurosurgeon). Controversy with respect to late (decompression) surgery is unresolved.
5. The management of temporal bone fractures is surrounded by several controversies. A Cochrane Database Systematic Review addresses the question with respect to antibiotic prophylaxis in depth; even for this subject large, appropriately designed RCTs are needed. The Practice Management Guidelines of the Eastern Association for the Surgery of Trauma provide level II recommendations for blunt cerebrovascular injury. However, in spite of a systematic literature review about the management of facial nerve injury due to temporal bone trauma,[26] for (nearly) all other management questions, the available literature comprises retrospective reviews of charts, medical records, registries or, at best, larger databases, most often without any control

group and hampered by small numbers (probably inherent to the rare nature of temporal bone fractures). With the exceptions as detailed in the text, categories of evidence are, at best, for level IIB; hence, strength of recommendation C.

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