AJNR

# Contrast-enhanced MR of the facial nerve in patients with posttraumatic peripheral facial nerve palsy.

S Sartoretti-Schefer, M Scherler, W Wichmann and A Valavanis



*AJNR Am J Neuroradiol* 1997, 18 (6) 1115-1125
http://www.ajnr.org/content/18/6/1115

# Contrast-Enhanced MR of the Facial Nerve in Patients with Posttraumatic Peripheral Facial Nerve Palsy

Sabine Sartoretti-Schefer, Martin Scherler, Werner Wichmann, and Anton Valavanis

**PURPOSE:** To estimate the value of noncontrast and contrast-enhanced T1-weighted MR imaging in detecting the underlying mechanisms of injury and regeneration in immediate- or delayed-onset posttraumatic peripheral facial nerve palsy. **METHODS:** Twenty-four patients with posttraumatic peripheral facial nerve palsy were examined on a 1.5-T MR imaging unit with precontrast and postcontrast T1-weighted spin-echo and gradient-echo sequences. **RESULTS:** Abnormal enhancement of the distal intrameatal nerve segment was visible in 92% of the patients up to 2 years after their initial trauma. A hematoma within the geniculate ganglion was seen in 33% of the patients with a longitudinal fracture. The greater superficial petrosal nerve (in 32% of patients) and the geniculate ganglion (in 48% of patients) were thick and intensely enhancing. Hematoma within the cochlea/vestibule or enhancement of the cochlea/vestibule and the vestibulocochlear (eighth) nerve was observed in transverse fractures. **CONCLUSION:** MR images can show long-lasting abnormal nerve enhancement, especially in the distal intrameatal nerve segment, related to the long-lasting breakdown of the blood/peripheral nerve barrier associated with nerve degeneration and regeneration after traumatic stretching of the greater superficial petrosal nerve. Additionally, intraoperatively observed perineural and intraneural scar formation leads to thickening and intense enhancement of the affected nerve segments on MR images. A hematoma in the region of the geniculate ganglion can be seen in some but not all patients. Associated damage of the inner ear structures in patients with transverse fractures is also visible on MR images.

**Index terms:** Nerves, facial (VII); Nerves, magnetic resonance

*AJNR Am J Neuroradiol* 18:1115–1125, June 1997

A posttraumatic peripheral facial nerve palsy can develop after either longitudinal or transverse fracture of the temporal bone (1–5). Diagnosis of temporal bone fracture can be accurately established with computed tomography (CT) (6–10). Axial and coronal CT scans can show the exact course of the fracture line in relation to the bony facial nerve canal as well as associated injuries (ie, disruption of the ossicular chain, hematotympanun, the site of leakage of cerebrospinal fluid in patients with otorhinoliquorrhea, and injury of the temporomandibular joint) (6–10).

In patients with posttraumatic peripheral facial nerve palsy, CT may correctly show the course of the fracture line through the bony facial nerve canal. It occasionally depicts hematoma in the region of the geniculate ganglion and compression of the facial nerve by an adjacent bony fragment (7), but the facial nerve itself is not visible, and therefore CT only indirectly delineates the nerve lesion. Magnetic resonance (MR) imaging, however, allows direct visibility of the injured peripheral facial nerve in patients with posttraumatic facial nerve palsy.

Several intraoperative investigations have established different mechanisms for posttraumatic facial nerve damage (2, 3, 11). First, the nerve can be transected. Second, adjacent bony fragments and/or hematoma can compress the nerve. Third, an intraneural hematoma can develop as a result of traction of the greater su-

Received September 4, 1996; accepted after revision January 2, 1997.

From the Institute of Neuroradiology (S.S-S., W.W., A.V.) and ENT (M.S.), University Hospital of Zürich (Switzerland).

Address reprint requests to Sabine Sartoretti-Schefer, MD, Institut für Neuroradiologie, Universitätsspital Zürich, Frauenklinikstrasse 10, CH-8091 Zürich, Switzerland.

AJNR 18:1115–1125, Jun 1997 0195-6108/97/1806–1115

perficial petrosal nerve (GSPN) associated with formation of intraneural edema that extends in a retrograde direction. This leads to compression of the facial nerve at the meatal foramen with secondary ischemia, nerve degeneration, and, occasionally, regeneration via a pathogenic mechanism similar to that described in inflammatory palsy (2, 3, 12–16).

Histologic examination of facial nerve biopsy specimens in patients who have had surgery for posttraumatic peripheral facial nerve palsy reveals simultaneous degeneration and regeneration processes with intraneural scar formation within the facial nerve (17).

In our retrospective study, we tried to estimate the value of noncontrast and contrast-enhanced MR imaging in patients with posttraumatic peripheral facial nerve palsy in terms of its ability to identify the location of the nerve injury, the underlying mechanism of injury (ie, intraneural hematoma/edema, nerve compression by bony fragment, or hematoma and nerve transection) (2, 3, 17), and the regeneration processes as known from histologic examinations.

The intraoperative findings in eight patients who had surgery were correlated with the preoperative MR abnormalities to identify the fracture line and its course as well as to determine associated injuries of the inner ear structures in patients with transverse fractures.

## Materials and Methods

We retrospectively evaluated the pathologic and radiologic findings in 24 patients (16 male, eight female; mean age, 38 years; range, 13 to 79 years) with posttraumatic peripheral facial nerve palsy.

All patients were examined on a 1.5-T MR unit using a 5-inch surface coil centered over the external ear. Noncontrast and contrast-enhanced T1-weighted spin-echo sequences were performed in all patients, and fast T2-weighted spin-echo images were obtained in 12 patients. A standard MR protocol with T1-weighted spin-echo sequences (500–640/15–21/2–3 [precontrast] or 4 [postcontrast], [repetition time/echo time/excitations]), overlapped 2- to 3-mm-thick sections, and a 160- to 170-mm field of view (FOV) was used in all patients. In two patients, a gradient-echo three-dimensional T1-weighted (fast field-echo) sequence (19/8.4/4) with a flip angle of 35°, section thickness of 0.9 mm, 30 sections, and FOV of 160 mm was added. T2-weighted images were acquired with parameters of 2500–4000/80–120/4 and a section thickness of 2 to 3 mm.

Sections were obtained in the axial (precontrast and postcontrast T1- and T2-weighted) and coronal (postcontrast T1-weighted) planes as well as occasionally in an oblique sagittal plane (postcontrast T1-weighted) along the axis of the tympanic segment (in four patients). Contrast material was injected intravenously using a bolus of 0.5 mmol/kg body weight (a high-dose protocol, used routinely in our department). MR imaging was performed immediately after injection of the contrast agent.

Axial and coronal CT studies of the affected ear were obtained with a section thickness/table feed of 1 mm/460 mA in all patients to ascertain the presence or absence of an associated transverse or longitudinal fracture of the temporal bone.

On CT scans, a longitudinal fracture of the temporal bone was diagnosed in 17 patients, a unilateral fracture was present in 14 patients, and a bilateral fracture in three patients. Only in one patient with bilateral longitudinal fracture was a bilateral MR examination done. In the other two patients with bilateral longitudinal fractures and bilateral facial nerve palsy, only the more severely affected side was examined with MR imaging. Therefore, 18 MR studies of longitudinal fractures and associated peripheral facial nerve palsies were evaluated. A transverse fracture of the temporal bone was visible on CT scans in three patients. In four patients, no fracture was identified at CT.

A total of 25 posttraumatically paretic peripheral facial nerves were examined on MR images. The peripheral facial nerve palsy was of immediate onset (occurring within 24 hours after the trauma) in 16 facial nerves; these were associated with a transverse fracture in three patients, a longitudinal fracture in 12 patients, and with no fracture (at CT) in one patient. A palsy of delayed onset (occurring more than 24 hours after the trauma) was diagnosed in nine facial nerves; these were associated with a longitudinal fracture in six patients and with no visible fracture (at CT) in three patients.

In three patients with transverse fractures, an additional loss of inner ear function was demonstrated by auditory and vestibular function tests. In 23 patients, single MR examinations were performed. In another patient, the first examination was on the 38th day after trauma and the second was on the 65th day after trauma. The earliest MR examination was obtained 7 days after trauma; the latest, 2 years after trauma. The mean interval between trauma and MR examination was 75.5 days. Table 1 shows the delay in days between the MR examination and the trauma itself and correlates the maximal percentage of nerve fiber degeneration on electroneurography with day of MR examination.

The intensity of the contrast enhancement within the distal intrameatal, labyrinthine, geniculate ganglion, proximal tympanic, distal tympanic, and mastoid segments was evaluated. Evaluation consisted of visual inspection and classification of the degree of contrast enhancement into one of three grades: intense, moderate, or minimal. The region-of-interest method for objectively measuring the signal intensity of the facial nerve could not be applied owing to the small dimensions of the bony facial nerve canal (1.02 mm in the labyrinthine segment, 1.53 mm in the proximal tympanic/distal tympanic segments, and

**TABLE 1: Delay between MR examination and trauma and correlation with maximal percentage of nerve fiber degeneration at electroneurography**

| | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Days between MR and trauma | 7 | 8 | 13 | 16 | 30 | 36 | 38 | 44 | 45 | 56 | 71 | 83 | 142 | 195 | 457 | 730 |
| No. of patients | 1 | 3 | 2 | 2 | 1 | 1 | 2 | 1 | 3 | 1 | 2 | 1 | 1 | 1 | 1 | 1 |
| Percentage of nerve degeneration at electroneurography | 40 | 35<br>100<br>100 | 87<br>97 | 8<br>79 | 68 | 100 | 75<br>100 | 100 | 100<br>100 | 100 | 84<br>100 | 100 | 100 | 45 | 100 | 100 |

**TABLE 2: Intensity of contrast enhancement of the various facial nerve segments in patients with posttraumatic peripheral facial nerve palsy**

| Degree of Enhancement | Facial Nerve Segment (25 Nerves) | | | | | |
|---|---|---|---|---|---|---|
| | DIS | LS | GG | PTS | DTS | MS |
| Intense | **2** | | **18** | **10** | **1** | **1** |
| Moderate | **12** | **5** | *7* | *14* | **7** | **6** |
| Minimal | **9** | **19** | | *1* | *16* | *14* |
| Not visible | *2* | *1* | | | 1 | 4 |

Note.—Bold numbers indicate abnormally intense enhancement not observed in normal facial nerves. According to our previous study (13), in normal facial nerves a normal enhancement pattern can be seen in different nerve segments. Usually, the geniculate ganglion shows a moderate enhancement and the PTS, DTS, and MS a moderate or minimal enhancement. Therefore, the numbers in italics remind the reader that these enhancement intensities are also normally seen in healthy nerves and do not indicate an abnormal enhancement pattern. DIS indicates distal intrameatal segment; LS, labyrinthine segment; GG, geniculate ganglion; PTS, proximal tympanic segment; DTS, distal tympanic segment; and MS, mastoid segment.

1.48 mm in the mastoid segment) and of the smallness of the facial nerve itself (0.85 mm in the labyrinthine segment, 1.12 mm in the proximal tympanic/distal tympanic segments, and 0.94 mm in the mastoid segment) as compared with the smallest region of interest available (16, 18). Other possible abnormalities of the temporal bone evaluated on MR images were as follows: 1) visibility of the fracture line itself and the course of the fracture line in relation to the different facial nerve segments; 2) presence of an associated hematoma within the bony facial nerve canal or within the facial nerve itself, especially in or adjacent to the geniculate ganglion; 3) presence of a transection or compression of the facial nerve by an adjacent bony fragment; 4) presence of dural enhancement along the anterior border of the petrous bone or within the internal auditory canal as a possible indirect sign of an osseous microfracture or macrofracture, even if the fracture line could not be identified radiologically; 5) presence on late posttraumatic MR images of a thick and intensely enhancing GSPN and of a thick and intensely enhancing geniculate ganglion (both over 1 mm in diameter) (18, 19), related to scar formation as proved by facial nerve biopsy samples (17); and 6) presence of a hematoma within the inner ear spaces (cochlea and vestibule) or of abnormal contrast enhancement of the inner ear spaces or of the vestibulocochlear (eighth) nerve in patients with transverse fractures.

Eight patients had surgery for immediate-onset posttraumatic facial nerve palsy. Two patients had a transverse fracture (operated on 49 and 1095 days, respectively, after the trauma); five patients had a longitudinal fracture (operated on 53, 62, 72, 85, and 480 days, respectively, after the trauma); and one patient had no radiologically visible fracture (operated on 9 days after the trauma). The intraoperative findings were compared with the preoperative abnormal findings on MR images.

**TABLE 3: Visibility and course of fracture line on MR images in longitudinal and transverse fractures of the temporal bone**

| | Longitudinal Fracture (n = 18) | Transverse Fracture (n = 3) |
|---|---|---|
| Fracture line visible on MR | 12 | 3 |
| Course of the fracture line through: | | |
| Internal auditory canal | . . . | 2 |
| Vestibule | . . . | 1 |
| GG | 5 | . . . |
| Slightly lateral to GG with extension to GSPN | 5 | . . . |
| PTS/DTS | 2 | 1 |

Note.—GG indicates geniculate ganglion; GSPN, greater superficial petrosal nerve; PTS, proximal tympanic segment; and DTS, distal tympanic segment.

## Results

Tables 2 through 5 present the pathologic findings in our 24 patients with posttraumatic peripheral facial nerve palsy.

In 23 paretic nerves (92% of patients) with a mean interval between trauma and MR examination of 88 days, abnormal contrast enhance-

**TABLE 4: Abnormal MR findings in patients with posttraumatic peripheral facial nerve palsy with or without temporal bone fracture**

| | Longitudinal Fracture (n = 18) | Transverse Fracture (n = 3) | No Fracture (n = 4) |
|---|---|---|---|
| Hematoma within GG (hyperintense on T1-weighted images; subacute stage) | 6 | . . . | . . . |
| Hematoma lateral to GG | 3 | . . . | 2 |
| Transection of seventh cranial nerve | 1 | . . . | . . . |
| Compression of seventh cranial nerve by adjacent bony fragment | . . . | . . . | . . . |
| Enhanced and thickened GSPN | 6 | . . . | 2 |
| Enhanced and thickened GG | 7 | 3 | 2 |
| Thickened PTS | 1 | 2 | |
| Enhancing scar in IAC | 2 | . . . | . . . |
| Dural enhancement along anterior border of temporal bone | 14 | . . . | 3 |

* Questionable.

Note.—GG indicates geniculate ganglion; GSPN, greater superficial petrosal nerve; PTS, proximal tympanic segment; and IAC, internal auditory canal.

**TABLE 5: No. of associated abnormalities of the inner ear spaces/eighth cranial nerve after transverse fracture of the temporal bone**

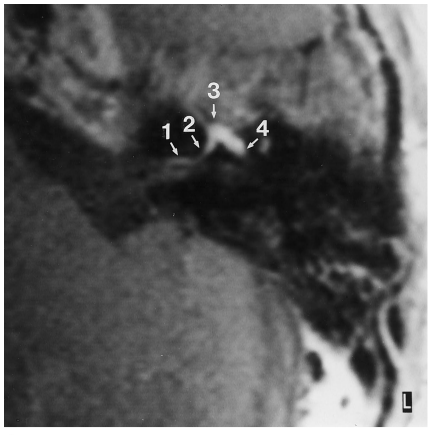
| | Hematoma | | Abnormal Contrast Enhancement | |
|---|---|---|---|---|
| | Fracture Line through the Inner Ear | | | |
| | Yes | No | Yes | No |
| Cochlea | | 1 | | 3 |
| Vestibule/semicircular canals | 1 | | 1 | 2 |
| Eighth cranial nerve | | | | 2 |

ment with variable intensity was seen in the distal intrameatal segment (Figs 1–8). This enhancement was visible even up to 2 years after the trauma. In two patients with longitudinal fractures, with an interval between trauma and MR examination of 36 and 457 days, respectively, no pathologic enhancement of the distal intrameatal segment could be identified, despite complete peripheral facial nerve palsy (Fig 9). In the other nerve segments, abnormally intense contrast enhancement was observed in the labyrinthine segment in 96% of affected nerves (Figs 1, 3, and 4), in the geniculate ganglion in 72% (Figs 1, 2, 4, and 9), in the proximal tympanic segment in 40% (Figs 1, 2, 4, 8, and 9), in the distal tympanic segment in 32% (Fig 4), and in the mastoid segment in 28%, as compared with normal facial nerves (13). No correlation could be established between the degree of enhancement of the nerve segment and the maximal percentage of nerve degeneration at electroneurography (Table 1).

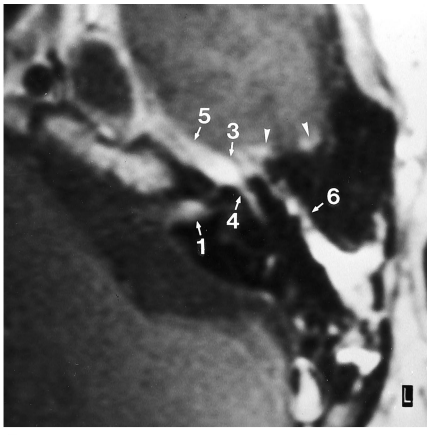
The fracture line itself was visible in 100% of the patients with transverse fractures (Figs 7 and 8) and in 66% of the patients with longitudinal fractures (Figs 2, 5, 6, and 9). Usually, the fracture line coursed either through the geniculate ganglion or slightly lateral to the geniculate ganglion with extension through the GSPN (Figs 2, 5, and 6) or through the proximal tympanic segment (Fig 9) in longitudinal fractures and through the internal auditory canal (Fig 7) or the inner ear (Fig 8) and possibly through the proximal tympanic segment (Fig 8) in transverse fractures.

A hematoma within the geniculate ganglion was observed in 33% of the patients with longitudinal fractures (Fig 6), but compression of the nerve by an adjacent bony fragment was never established (Table 4). Transection of the nerve was suspected in one patient (case 7); nerve enhancement stopped at the transition from the proximal to the distal tympanic segments, and no enhancement was visible in the distal tympanic and mastoid segments (Fig 9). In the second patient with intraoperatively proved nerve transection, the transection was not demonstrable on MR images (Tables 6 and 7).

The GSPN appeared as an intensely enhancing and thickened nerve in eight (32%) of the patients (Fig 2). A thickened geniculate ganglion (Figs 1, 2, 4, and 9) was visible in 12 patients (48%). In patients with transverse fractures of the temporal bone, an associated hematoma within the inner ear (Fig 7) and/or abnormal enhancement of the inner ear spaces (Figs 7 and 8) and of the eighth cranial nerve (as a result of nerve damage) (Fig 7) could be seen.



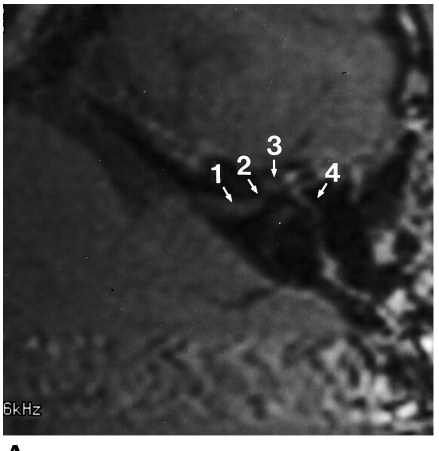

1



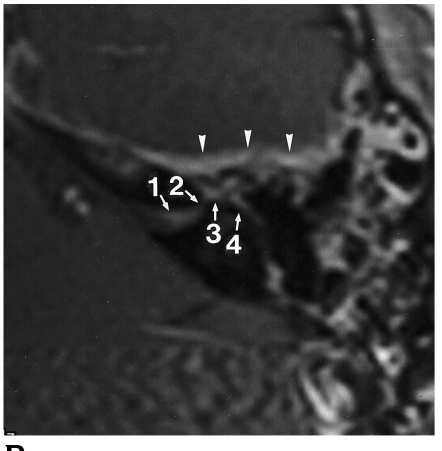

2

Fig 1. Contrast-enhanced transverse T1-weighted (560/16/4) MR image 142 days after delayed posttraumatic peripheral facial nerve palsy shows no visible fracture line. Minimal enhancement of the distal intrameatal segment (*1*), moderate enhancement of the labyrinthine segment (*2*), and intense enhancement and thickening of the geniculate ganglion (*3*) and proximal tympanic segment (*4*) are related to long-lasting damage of the blood/peripheral nerve barrier and formation of perineural fibrosis in the region of the geniculate ganglion.

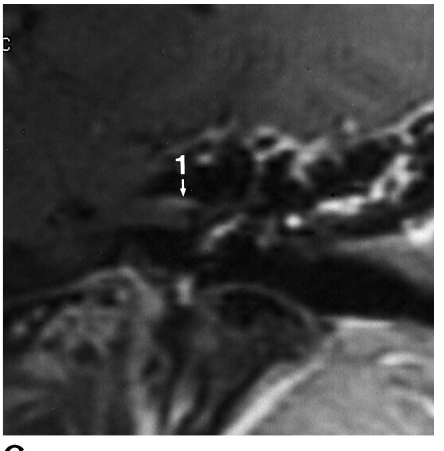
Fig 2. Case 3: Contrast-enhanced transverse T1-weighted (600/20/4) MR image 45 days after longitudinal fracture of the petrous bone and immediate-onset peripheral facial nerve palsy shows intense enhancement and thickening of the geniculate ganglion (*3*) and GSPN (*5*). The proximal tympanic segment is intensely enhanced (*4*). The longitudinal fracture line (*6*) courses through the geniculate ganglion and GSPN. The distal intrameatal segment (*1*) is moderately enhanced. Minimal dural enhancement is seen along the rostral border of the temporal bone (*arrowheads*).




A




B




C

Fig 3. Noncontrast (500/15/2) (*A*) and contrast-enhanced (600/20/4) (*B*) transverse and coronal (*C*) T1-weighted MR images in a patient with delayed posttraumatic peripheral facial nerve palsy without associated fracture of the temporal bone, obtained 7 days after trauma, show moderate enhancement of the distal intrameatal segment (*1*) and minimal enhancement of the labyrinthine segment (*2*). The geniculate ganglion (*3*) and proximal tympanic segment (*4*) are normally enhanced. Intense dural enhancement is seen along the rostral border of the temporal bone, indicating possible microtrauma of both the temporal bone and the adjacent dura (*arrowheads*).

Dural enhancement along the rostral border of the temporal bone was observed in 68% of patients either with or without a visible fracture line (Figs 2–4). Tables 6 through 8 compare intraoperative and preoperative MR findings in the eight patients who had surgery for posttraumatic peripheral facial nerve palsy. In patients with intraoperatively proved scar formation, especially of the GSPN and geniculate ganglion, the preoperative MR examination showed a correspondingly thickened and intensely enhancing nerve segment.

## Discussion

The facial nerve is the only cranial motor nerve commonly affected by trauma to the head. A fracture of the temporal bone must be assumed in patients with posttraumatic facial nerve palsy even when a fracture cannot be identified radiologically (1).

The commonly observed dural enhancement along the rostral border of the temporal bone on MR images in patients with or without temporal bone fracture may prove to be radiologically

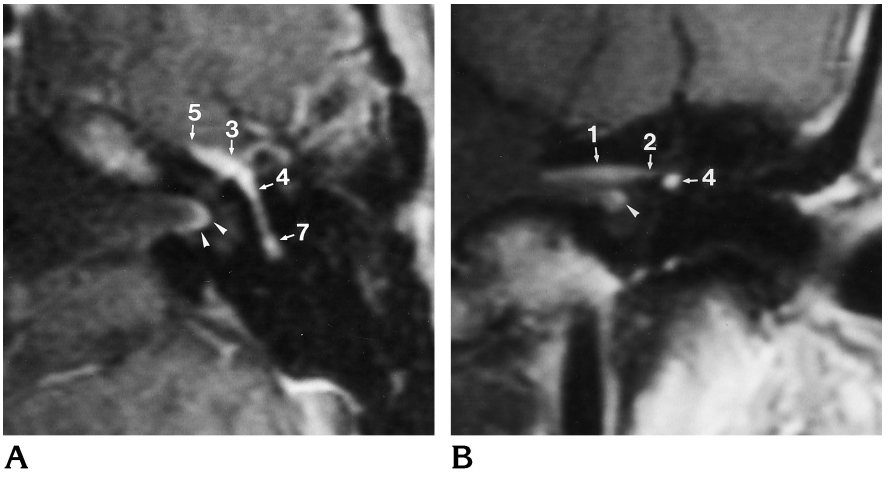



Fig 4. Contrast-enhanced transverse (*A*) and coronal (*B*) T1-weighted (640/21/4) MR images, obtained 195 days after longitudinal fracture of the temporal bone and immediate-onset peripheral facial nerve palsy, show intense enhancement of the distal intrameatal (*1*), proximal tympanic (*4*), and distal tympanic (*7*) segments and of the thick geniculate ganglion (*3*) and the thick GSPN (*5*). The labyrinthine segment (*2*) is moderately enhanced. Dural enhancement is seen within the fundus of the internal auditory canal (*arrowheads*) that blends with the enhancement of the distal intrameatal nerve segment.

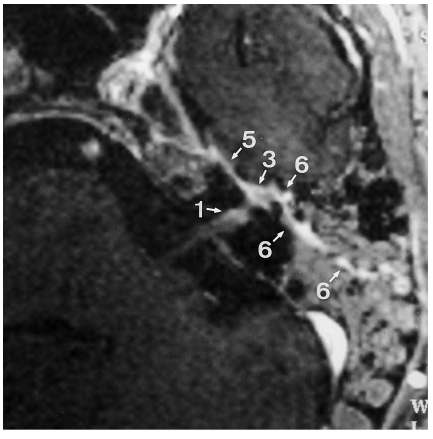



Fig 5. Case 5: Contrast-enhanced transverse fast field-echo (gradient-echo) T1-weighted (19/8.4/4, 35°) MR image, obtained 71 days after longitudinal fracture and immediate-onset facial nerve palsy, shows the longitudinal fracture line (*6*) coursing through the geniculate ganglion (*3*). The distal intrameatal segment (*1*) and the GSPN (*5*) are abnormally moderately enhanced.

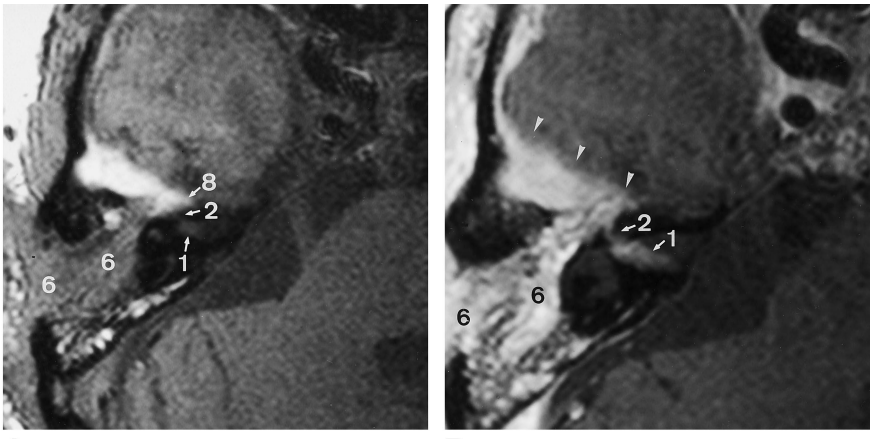



Fig 6. Noncontrast (500/15/2) (*A*) and contrast-enhanced (640/21/4) (*B*) transverse T1-weighted images, obtained 44 days after longitudinal fracture and immediate-onset palsy, show subacute hyperintense hematoma (*8*) within the geniculate ganglion, an enhancing broad longitudinal fracture line (*6*), and abnormal enhancement of the distal intrameatal (*1*) and labyrinthine (*2*) segments. Note associated subdural hematoma (*arrowheads*).

invisible microfractures of the temporal bone associated with microtears of the adjacent dura. Fractures of the temporal bone are subdivided into longitudinal (80% to 90%, with associated peripheral facial nerve palsy in 10% to 20% of the patients) and transverse (10% to 20%, with secondary loss of inner ear function and associated peripheral facial nerve palsy in 38% to 50%, depending on the course of the fracture line through the temporal bone) fractures (1–5).

Posttraumatic partial or complete peripheral facial nerve palsy can be classified as either immediate onset (occurring within 24 hours after the trauma and often caused by nerve transection) or delayed onset (occurring more than 24 hours after the trauma, caused by intraneural hematoma/edema after stretching of the GSPN, with or without associated fracture, or by nerve compression) (1, 2, 20).

Surgically verified posttraumatic facial nerve lesions associated with a longitudinal fracture are located in the region of the geniculate ganglion in 64% of patients, in the GSPN in 25%, in the distal tympanic/mastoid segment in 7%, and in the labyrinthine segment in 4% (2). In transverse fractures, the nerve lesion is located within the internal auditory canal in 10% of patients, within the labyrinthine segment in 80%,

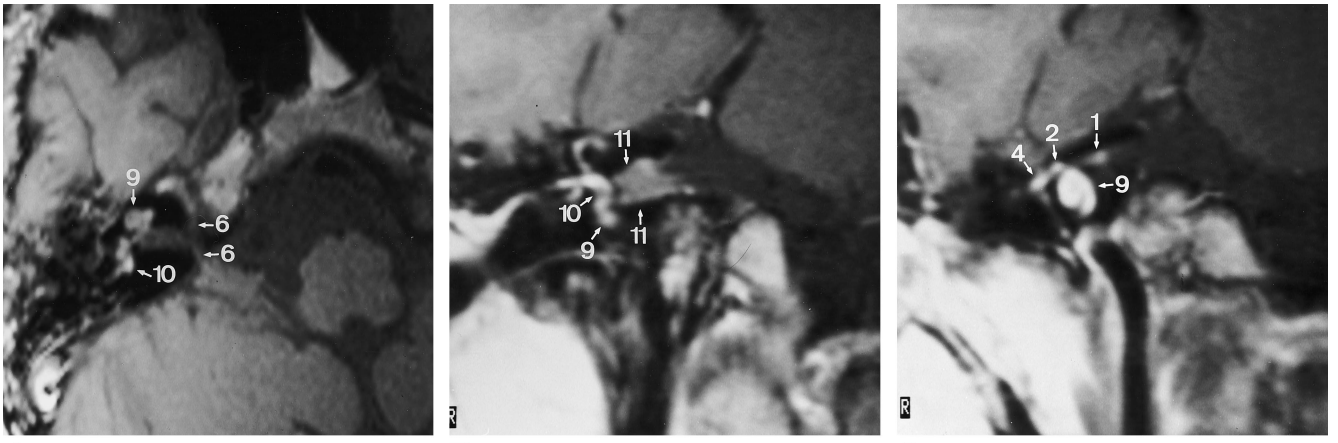



Fig 7. Noncontrast transverse (500/15/2) (*A*) and contrast-enhanced coronal (600/20/4) (*B* and *C*) T1-weighted images, obtained 38 (*A*) and 65 (*B* and *C*) days after transverse fracture, show a fracture line (*6*) coursing through the internal auditory canal and associated palsy of immediate onset. A hyperintense hematoma within the cochlea (*9*) and vestibule (*10*) was seen 38 days after trauma (*A*). Sixty-five days after trauma, the distal intrameatal segment (*1*) and superoinferior vestibulocochlear nerve (*11*) within the internal auditory canal show intense enhancement. The cochlea (*9*) and vestibule/semicircular canals (*10*) are also intensely enhanced. (The noncontrast images, not depicted, no longer showed the hyperintense hematoma within the cochlea/vestibule.) Note intense enhancement of the labyrinthine (*2*) and proximal tympanic (*4*) segments.

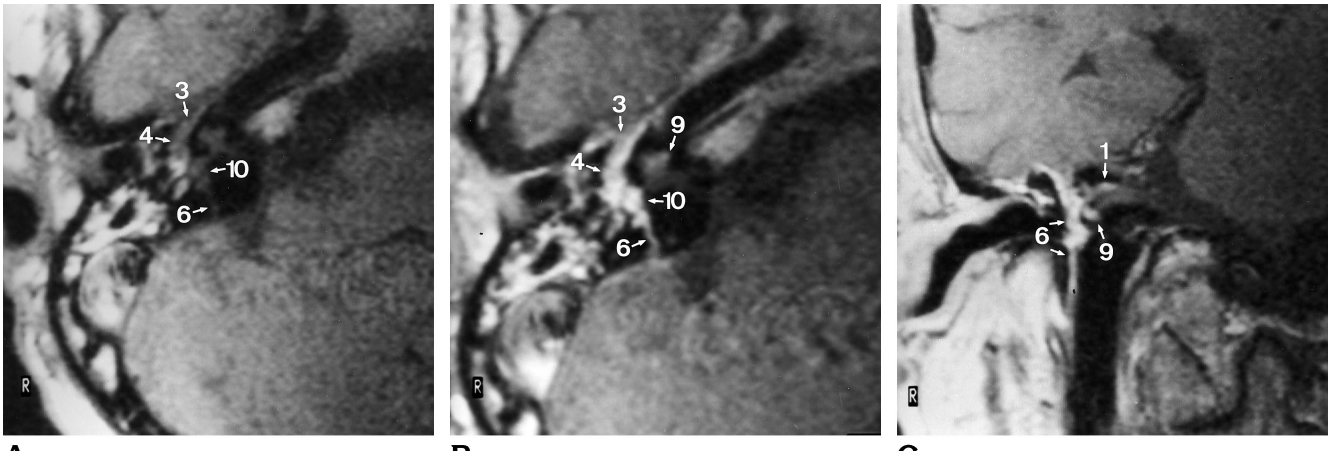



Fig 8. Noncontrast transverse (500/15/2) (*A*) and contrast-enhanced transverse (*B*) and coronal (*C*) T1-weighted (640/21/4) images, obtained 45 days after transverse fracture and immediate-onset palsy, show abnormal enhancement in the distal intrameatal segment (*1*). The contrast-enhancing fracture line (*6*) crosses the vestibule and the thickened proximal tympanic segment (*4*) of the facial nerve. The cochlea (*9*) and vestibule (*10*) show moderate enhancement. The geniculate ganglion (*3*) is thickened and enhanced.

and in the region in or around the geniculate ganglion in 10% (2), corresponding to the results obtained in our study (Table 3).

Different mechanisms of injury to the peripheral facial nerve are possible and can be observed intraoperatively. In longitudinal fractures, most commonly an intraneural hematoma at the level of the geniculate ganglion is present (in about 50% of the patients) (3). A bony fragment compressing the adjacent nerve is visible in 17% to 20% of the patients, and a transection of the nerve occurs in 26% to 30% of the patients (3). In a few patients, no definite injury can be identified intraoperatively (2, 3, 17). Severe traction and stretching of the GSPN are present in all patients, and lead to the formation of an intraneural hematoma and secondary edema that extends in a retrograde direction along the proximal nerve (3, 14).

Facial nerve injury sustained after stretching of the facial nerve during cerebellopontine angle operations is said to result from movements of the nerve within its immobile sheath in the preganglionic segment, subsequent rupture of

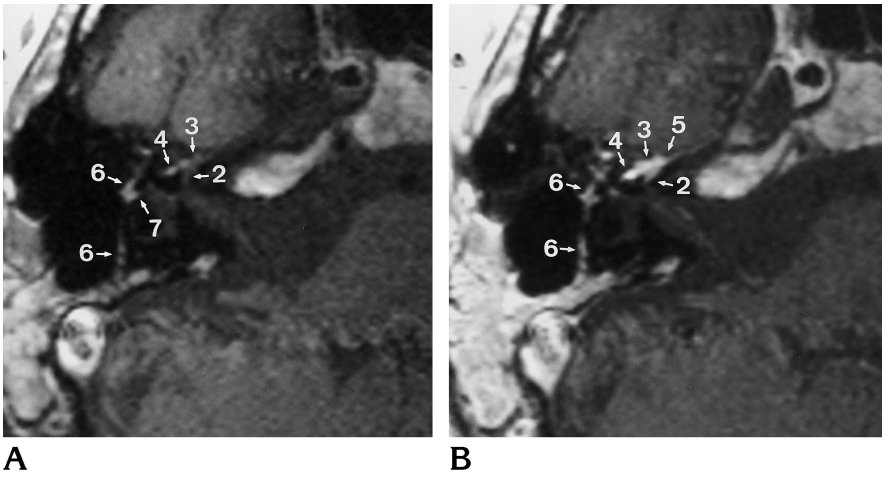



Fig 9. Case 7: Transverse noncontrast (500/15/2) (*A*) and contrast-enhanced (600/20/4) (*B*) T1-weighted spin-echo images, obtained 457 days after longitudinal petrous bone fracture and immediate-onset palsy, show nerve transection (proved intraoperatively). The contrast-enhancing fracture line (*6*) courses through the tympanic segment (*7*). The intensely enhanced and thickened geniculate ganglion (*3*) and proximal tympanic segment (*4*) may be attributed to possible scar formation. The distal tympanic and mastoid segments are not depicted reliably. The very proximal GSPN is minimally thickened (*5*). The labyrinthine segment is abnormally enhanced (*2*).

**TABLE 6: Preoperative MR findings**

| Case | Delay between Trauma and MR, d | Abnormal Enhancement of Seventh Cranial Nerve Segments | | | | Thickened Seventh Nerve Segment | | Hematoma in GG | Seventh Nerve Transection | Abnormal Enhancement | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | DIS | LS | GG | PTS | GSPN | GG | | | V/C | Eighth Nerve |
| 1 | 45 | ++ | + | +++ | +++ | ... | + | ... | ... | + | ... |
| 2 | 730 | +++ | ++ | +++ | +++ | ... | + | ... | ... | + | + |
| 3 | 45 | ++ | + | +++ | +++ | + | + | ... | ... | ... | ... |
| 4 | 45 | + | + | ++ | ++ | ... | + | + | ... | ... | ... |
| 5 | 71 | ++ | + | +++ | +++ | + | + | ... | ... | ... | ... |
| 6 | 83 | + | + | +++ | ++ | + | + | ... | ... | ... | ... |
| 7 | 457 | ... | + | +++ | +++ | +* | + | ... | +* | ... | ... |
| 8 | 8 | ++ | + | +++ | +++ | ... | ... | ... | ... | ... | ... |

* Questionable enhancement in PTS.

Note.—Cases 1 and 2 had surgery for transverse fracture, cases 3 through 7 had surgery for longitudinal fracture, case 8 had no radiologically visible fracture. +++ indicates intense enhancement; ++, moderate enhancement; +, minimal enhancement; DIS, distal intrameatal segment; LS, labyrinthine segment; GG, geniculate ganglion; PTS, proximal tympanic segment; GSPN, greater superficial petrosal nerve; and V/C, vestibule and cochlea.

the supplying blood vessels to the facial nerve, and formation of intraneural hemorrhage and edema (21). The swollen facial nerve is secondarily compressed and damaged at the meatal foramen (this is the canalicular entrance of the facial nerve into the bony labyrinthine segment). An identical mechanism (namely, compression of the edematous nerve) is also responsible for the development of facial nerve palsy in inflammatory palsy (ie, Bell palsy or herpetic palsy) (12, 13). Therefore, identical enhancement patterns of the different facial nerve segments can be assumed in patients with inflammatory (13) and posttraumatic peripheral facial nerve palsy; and identical enhancement patterns are observed independent of the exact site of the nerve injury along the course of the facial nerve in posttraumatic palsy. Abnormal nerve enhancement is always or nearly always observed in the distal intrameatal segment and is commonly seen in the labyrinthine segment and the proximal tympanic segment as well as in the geniculate ganglion. The distal tympanic and mastoid segments more rarely show abnormally intense enhancement as compared with a normal facial nerve (13). In our series, only two patients with posttraumatic peripheral facial nerve palsy (imaged 36 and 457 days, respectively, after trauma) did not have the commonly observed abnormal enhancement of the distal intrameatal segment, indicating damage to the nerve itself (13, 32), on contrast-enhanced MR images, despite clinical persistence of the palsy. In both patients, however, abnormally intense enhancement of the geniculate ganglion and proximal tympanic segment was observed. We have no reasonable explanation for the absence of

**TABLE 7: Intraoperative findings in eight patients**

| Case | Delay between Trauma and Surgery, d | Nerve Transection | Fresh or Organized Hematoma/Scar in Different Nerve Segments | | | | | Bone within Fallopian Canal | Other Findings |
|---|---|---|---|---|---|---|---|---|---|
| | | | LS | GG | PTS | GSPN | V/C | | |
| 1 | 49 | | + | + | + | . . . | + | . . . | |
| 2 | 1095 | . . . | . . . | . . . | . . . | . . . | . . . | . . . | Seventh nerve atrophy |
| 3 | 53 | . . . | . . . | + | . . . | + | . . . | . . . | |
| 4 | 62 | . . . | . . . | + | . . . | + | . . . | GG | |
| 5 | 72 | + (PTS) | + | + | +* | +* | . . . | . . . | |
| 6 | 85 | . . . | + | + | + | + | . . . | GG | |
| 7 | 480 | + (PTS) | + | + | + | . . . | . . . | . . . | |
| 8 | 9 | . . . | . . . | . . . | . . . | . . . | . . . | . . . | Traction of GSPN, retrograde edema, compression at meatal foramen |

Note.—Cases 1 and 2 had transverse fracture, cases 3 through 7 had longitudinal fractures, and case 8 had no radiologically visible fracture. See Table 6 for abbreviations.

**TABLE 8: Comparison between preoperative MR findings and intraoperative findings**

| Finding | Preoperative MR Imaging | Surgery |
|---|---|---|
| Hematoma within GG | 1 | 6 |
| Bony fragment in GG | . . . | 2 |
| Nerve transection | 1* | 2 |
| Thickened GSPN (scar) | 4 | 4 |
| Thickened GG (scar) | 7 | 6 |

* Questionable.

Note.—GG indicates geniculate ganglion; GSPN, greater superficial petrosal nerve.

nerve enhancement in the distal intrameatal segment in these two patients. Further examinations are probably necessary to accumulate more information on the pathophysiology of posttraumatic nerve palsy.

Additionally, no correlation can be established between nerve enhancement and maximal percentage of nerve fiber degeneration at electroneurography (Table 1); a circumstance that has already been described in connection with inflammatory nerve palsy (32). In our retrospective study, a hematoma in or around the region of the geniculate ganglion was seen on MR images in six (33%) of 18 patients with longitudinal fractures of the temporal bone. In the eight patients who had surgery, seven had intraoperative findings of a hematoma in the geniculate ganglion. However, only in one patient was this hematoma identified correctly on the preoperative MR study. Therefore, it must be assumed that MR imaging is not able to show the hematoma in the geniculate ganglion in all affected patients.

In two patients (cases 5 and 7), a transection of the nerve was identified intraoperatively in the proximal/middle tympanic segment. Despite the transection, continuous enhancement of the facial nerve was seen on MR images in case 5; in case 7, however, the discontinuity of the nerve was only suspected on MR images, since the very distal tympanic and mastoid segments of the nerve were not identified reliably. All the same, it must be assumed that detection of a nerve transection with the help of MR imaging is either impossible or very difficult, since nerve transection was suspected in only one (5%) of our patients with longitudinal fractures, whereas from previous surgical studies (3) it is known that intraoperatively proved nerve transection occurs in up to 30% of patients with such fractures (3).

The same difficulties were encountered in cases of transverse fractures. Intraoperatively, a transection of the nerve was seen within the internal auditory canal or the labyrinthine segment in all patients (2, 17); however, it was not seen on the MR images, because in all three patients with transverse fractures, continuous enhancement of the facial nerve was observed. Similarly, a second, more distal, facial nerve injury that is not visible on MR images often may be proved intraoperatively in patients with either longitudinal or transverse fractures (17).

Histologic examination of facial nerve biopsy specimens, obtained intraoperatively in patients who have surgery for posttraumatic peripheral facial nerve palsy after longitudinal fracture (17), reveal intense retrograde degeneration of myelinated fibers in the facial nerve proximal to the geniculate ganglion and proximal to the site of the nerve injury related to stretching of the nerve after traumatically induced deformation of the bone. This nerve fiber degeneration is accompanied by a simultaneous regeneration

in the proximal nerve segments. The phase of nerve fiber regeneration may persist up to 48 months after the trauma (17, 22). Additionally, endoneural and perineural fibrosis of variable extent, partly seen as thick scar surrounding the regenerating axons, especially in the distal labyrinthine segment or in the proximal tympanic segment, is seen both early and late after injury, but fibrosis formation starts as early as 5 weeks after the trauma.

In accordance with these histologic findings, the thickening (defined as a specific nerve segment with a diameter of more than 1 mm on contrast-enhanced MR images compared with a neuronal diameter of less than 1 mm seen in healthy volunteers [13, 18, 19]) and intense enhancement of the GSPN and/or the geniculate ganglion on contrast-enhanced T1-weighted MR images result from this posttraumatic endoneural and perineural fibrosis (as proved in several of the patients in our study who had surgery [Tables 6–8]). The long-lasting abnormal contrast enhancement of various facial nerve segments (especially the distal intrameatal segment, the labyrinthine segment, the proximal tympanic segment, and the geniculate ganglion) observed on MR images in several patients up to 2 years after trauma can be explained by long-lasting damage to the blood/peripheral nerve barrier (23–31). According to experimental studies in animals, the complex process of degeneration and regeneration following nerve injury is accompanied by a vasogenic response associated with a two-phase breakdown in the blood/peripheral nerve barrier formed by endoneurium and perineurium (23–26, 28, 29, 32). The first phase occurs during wallerian degeneration. It is early, rapid, and associated with a breakdown of the endoneural barrier, leading secondarily to macrophage infiltration and removal of debris. The second phase occurs during nerve regeneration. It is late in onset but lasts for months. It is associated with a breakdown of the perineural barrier and provides for the increased transfer of metabolic substrates to the regenerating nerves. These experimental observations can also be applied to the facial nerve, since this nerve behaves histologically and electrophysiologically as a peripheral nerve. Therefore, long-lasting damage to the blood/peripheral nerve barrier related to degeneration and regeneration of nerve fibers as well as to formation of perineural fibrosis has to be suspected as the underlying pathophysiologic mechanism that explains the long-lasting contrast enhancement of the facial nerve observed in our study (24–29, 32).

In summary, intraoperative findings show that posttraumatic peripheral facial nerve palsy results from nerve transection, from compression by a bony fragment/hematoma, or from formation of an intraneural hematoma/edema after stretching of the GSPN with secondary compression of the swollen nerve within the bony facial nerve canal. MR images show long-lasting abnormal nerve enhancement, especially in the distal intrameatal segment but often also in the labyrinthine and proximal tympanic segments and in the geniculate ganglion related to the long-lasting breakdown of the blood/peripheral nerve barrier associated with nerve degeneration and regeneration after injury. Intraoperatively observed perineural and intraneural scar formation leads to thickening and intense enhancement of the affected nerve segments on MR images. MR imaging is able to show the hematoma in or around the region of the geniculate ganglion in some but not all patients, but it is not able to depict nerve compression reliably by an adjacent bony fragment or the nerve transection itself. Most associated injuries of the inner ear and of the eighth cranial nerve as well as the course of the fracture line are visible on MR images.

## References


1. Harker LA, McCabe BF. Temporal bone fractures and facial nerve injury. *Otolaryngol Clin North Am* 1974;2:425–428
2. Fisch U. Facial paralysis in fractures of the petrous bone. *Laryngoscope* 1974;84:2141–2154
3. Fisch U. Management of intratemporal facial nerve injuries. *J Laryngol Otol* 1980;94:129–134
4. McCabe BF. Injuries to the facial nerve: symposium on trauma in otolaryngology. *Laryngoscope* 1972;82:1891–1896
5. McGovern FH. Facial nerve injuries in skull fractures. *Arch Otolaryngol* 1968;88:102–108
6. Yamaki T, Yoshino E, Higuchi T, Horikawa Y, Hirakawa K. Value of high-resolution computed tomography in diagnosis of petrous bone fracture. *Surg Neurol* 1991;26:551–556
7. Schubiger O, Valavanis A, Stuckmann G, Antonucci F. Temporal bone fractures and their complications: examination with high resolution CT. *Neuroradiology* 1986;28:93–99
8. Valavanis A, Schubiger O, Stuckmann G, Antonucci F. CT-Diagnostik traumatischer Läsionen des Felsenbeines. *Radiologe* 1986; 26:85–90
9. Dolan KD. Temporal bone fractures. *Semin Ultrasound CT MR* 1989;10:262–279
10. Holland BA, Brant-Zawadzi M. High-resolution CT of temporal bone trauma. *AJR Am J Roentgenol* 1984;143:391–395

11. Murakami M, Ohtani I, Aikawa T, Anzai. Temporal bone findings in two cases of head injury. *J Laryngol Otol* 1990;104:986–989
12. Fisch U, Felix H. On the pathogenesis of Bell's palsy. *Acta Otolaryngol* 1983;95:532–538
13. Sartoretti-Schefer S, Wichmann W, Valavanis A. Gadolinium-enhanced MR in patients with idiopathic, herpetic and HIV-associated facial nerve palsies: abnormal enhancement patterns compared with normal individuals. *AJNR Am J Neuroradiol* 1994;15: 479–485
14. Anderson J, Awad IA, Hahn JF. Delayed facial nerve palsy after temporal lobectomy for epilepsy: report of four cases and discussion of possible mechanisms. *Neurosurgery* 1991;28:453–456
15. Morello G, Bianchi M, Migliavacca F. Combined extra-intradural temporal rhizotomy for the treatment of trigeminal neuralgia: results in 409 patients. *J Neurosurg* 1971;34:372–379
16. Fisch U. Surgery for Bell's palsy. *Arch Otolaryngol* 19891;107: 1–11
17. Felix H, Eby TL, Fisch U. New aspects of facial nerve pathology in temporal bone fractures. *Acta Otolaryngol (Stockh)* 1991;111: 332–336
18. Miehlke A, Fisch U. Fazialislähmungen im labyrinthären, meatalen und intrakraniellen Bereich. In: Berendes J, Link R, Zöllner F, eds. *Hals-Nasen-Ohrenheilkunde in Praxis und Klinik*. Stuttgart, Germany: Thieme; 1979: 21.1–21.62
19. Gulya AJ, Schuknecht HF. Neuroanatomy: the facial nerve. In: Gulya AJ, Schuknecht HF, eds. *Anatomy of the Temporal Bone with Surgical Implications*. New York, NY: Parthenon; 1995:161–177
20. Potter JM. Facial palsy following head injury. *J Laryngol Otol* 1964;78:654–657
21. Sekiya T, Iwabuchi T, Okabe S. Occurrence of vestibular and facial nerve injury following cerebellopontine angle operations. *Acta Neurochir (Wien)* 1990;102:108–113
22. Ylikoski J, Brackmann DE, Savolainen S. Facial nerve abnormalities after acoustic tumor removal: morphological and clinical study of seven patients with postoperative facial paralysis. *Arch Otolaryngol* 1982;108:795–800
23. Mellick RS, Cavanagh JB. Changes in blood vessel permeability during degeneration and regeneration in peripheral nerves. *Brain* 1968;91:141–160
24. Bush MS, Reid AR, Allt G. Blood-nerve barrier: ultrastructural and endothelial surface charge alterations following nerve crush. *Neuropathol Appl Neurobiol* 1993;19:31–40
25. Podhajsky RJ, Myers RR. The vascular response to nerve crush: relationship to wallerian degeneration and regeneration. *Brain Res* 1993;623:117–123
26. Weerasuriya A, Hockman CH. Perineurial permeability to sodium during wallerian degeneration in rat sciatic nerve. *Brain Res* 1992; 581:327–333
27. Latker CH, Wadhwani KC, Balbo A, Rapoport SI. Blood-nerve barrier in the frog during wallerian degeneration: are axons necessary for maintenance of barrier function? *J Comp Neurol* 1991; 309:650–664
28. West NR, Collins GH. Relationship of wallerian degeneration to regrowing axons. *J Neuropathol Exp Neurol* 1991;50:693–703
29. Sparrow JR, Kiernan JA. Endoneurial vascular permeability in degenerating and regenerating peripheral nerves. *Acta Neuropathol* 1981;53:181–188
30. Bradbury M. *The Concept of the Blood-Brain-Barrier*. New York, NY: Wiley; 1979:127–135, 351–375
31. Lundborg G. Structure and function of the intraneural microvessels as related to trauma, edema formation and nerve function. *J Bone Joint Surg Am* 1975;57-A:938–948
32. Sartoretti-Schefer S, Brändle P, Wichmann W, Valavanis A. Intensity of MR-contrast enhancement does not correspond to clinical and electroneurographic findings in acute inflammatory facial nerve palsy. *AJNR Am J Neuroradiol* 1996;17:1229–1236