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## **Idiopathic, herpetic, and HIV-associated facial nerve palsies: abnormal MR enhancement patterns.**

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# Idiopathic, Herpetic, and HIV-Associated Facial Nerve Palsies: Abnormal MR Enhancement Patterns

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**PURPOSE:** To determine specific criteria that can be used to define normal versus abnormal MR contrast enhancement of the facial nerve. **METHODS:** Twenty-three patients with acute unilateral inflammatory peripheral facial nerve palsy were examined on a 1.5-T MR using multiplanar T1-weighted spin-echo sequences before and after injection of gadopentetate dimeglumine. These MR patterns were compared with those of healthy control subjects. **RESULTS:** The normal facial nerve usually showed a mild to moderate enhancement of the geniculate ganglion and the tympanic-mastoid segment. The intracanalicular-labyrinthine segment did not enhance. All patients showed abnormal enhancement of the distal intracanalicular and the labyrinthine segment. An intense enhancement could be observed in the geniculate ganglion and the proximal tympanic segment, especially in herpetic palsy. Associated enhancement of the vestibulocochlear nerve was seen in herpetic and idiopathic palsy. Enhancement of the inner ear structures was detected only in herpetic palsy. **CONCLUSIONS:** Abnormal contrast enhancement of the distal intracanalicular and the labyrinthine facial nerve segment is observed in all patients and is the only diagnostically reliable MR feature proving an inflammatory facial nerve lesion. The intense enhancement of the geniculate ganglion and the proximal tympanic segment is possibly correlated with the reactivation of the latent infection in the sensory ganglion. The abnormal enhancement results from breakdown of the blood-peripheral nerve barrier and/or from venous congestion in the venous plexuses of the epi- and perineurium.

**Index terms:** Nerves, facial (VII); Nerves, magnetic resonance; Bell palsy; Acquired immunodeficiency syndrome (AIDS); Herpes zoster

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Idiopathic (Bell palsy) and herpetic (Ramsay Hunt syndrome) facial nerve palsies together account for 86% of all unilateral lower motor neuron facial palsies (1). The incidence of Bell palsy in the general population is 17 to 19 in 100,000 per year and increases in each decade, reaching 30 to 35 in 100,000 per year after the age of 60 (1). Facial nerve palsy in herpes zoster infection, however, is six times less common (1). Bell disease affects all ages and does not show a sex prevalence (1, 2). A spontaneous recovery usually occurs in all inflammatory palsies. Only

in cases of more than 95% nerve fiber degeneration within 14 days is surgical decompression of the edematous nerve at the origin of the fallopian canal in the fundus of the internal auditory meatus (called *meatal foramen*) recommended (3).

Although magnetic resonance (MR) examinations of facial nerves in idiopathic and herpetic facial nerve palsy have been performed several times (4-10), no distinct and diagnostic patterns of nerve damage could be recognized. T1-weighted MR sequences after injection of gadopentetate dimeglumine showed variable degrees of homogeneous and smooth enhancement in different segments of the facial nerve, including the distal intracanalicular segment, but without obvious predilection for a specific part of the facial nerve.

Homogeneous enhancement of the facial nerve itself is not specific for Bell palsy or herpetic facial nerve palsy: it is also reported in postoperative or posttraumatic facial nerve palsy (9).

Breakdown of the blood-peripheral nerve barrier allows leakage of contrast material and results

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in abnormal enhancement of the facial nerve. This increased permeability of the blood-peripheral nerve barrier is observed in posttraumatic, inflammatory, and ischemic lesions of peripheral nerves (11–14).

In our prospective study we have tried to compare the contrast-enhanced MR findings of patients with idiopathic, herpetic, and human immunodeficiency virus (HIV)-associated acute lower motor neuron facial nerve palsy with healthy control subjects to determine specific criteria for distinguishing these two groups. In addition, the patterns of abnormal enhancement in idiopathic, herpetic, and HIV-associated facial palsy were evaluated for specific differences.

### Materials and Methods

Twenty-three patients (12 men and 11 women) with a mean age of 45.1 years (range, 12 to 85 years) were examined on a 1.5-T MR imager. An idiopathic facial nerve palsy was diagnosed in 14 patients and a herpetic palsy in 8. The diagnosis of herpetic palsy was established based on laboratory parameters (elevated IgG and IgM antibodies against varicella-zoster virus) and typical clinical settings (auricular vesicles in the external auditory meatus, ear pain, facial palsy, and possible eighth cranial nerve involvement with resulting tinnitus, hearing loss, and vertigo). One patient presented with a facial nerve palsy associated with HIV infection.

Twenty-two patients presented with complete peripheral facial nerve palsy. The mean duration of their symptoms before the MR examination was 6.7 days (range, 1 to 21 days). One patient had already completely recovered from a peripheral nerve palsy 4 months earlier.

According to Fisch et al (3, 29), evoked electroneurography helps to determine the degree of the nerve fiber degeneration in patients with peripheral facial nerve palsy. In 50% of all patients with more than 94% of nerve fiber degeneration within the first 2 weeks of palsy, an incomplete spontaneous recovery has to be expected unless the nerve is surgically decompressed at the meatal foramen. More than 94% maximal nerve fiber degeneration was measured in 8 of our patients. In 6 patients, the facial nerve was surgically decompressed at the meatal foramen; early postoperative MR examinations within 2 weeks were obtained in 2 of these patients. T1-weighted spin-echo sequences were of 500 to 640 msec repetition time, 20 to 25 msec echo time, and had 4 excitations. A temporal-bone surface coil (7.6- or 14-cm diameter) was applied in 22 patients. The head coil was used in one patient only. Thin sections (2.5 to 3 mm) in the

transverse (interleaved and/or overlapping sections) and coronal and occasionally in the Stenver plane of the section were performed. Precontrast transverse T1-weighted sequences were obtained in 12 patients. Gadopentetate dimeglumine was injected in all patients (40-mL intravenous bolus, high-dose protocol).

In a retrospective, nonblinded analysis, the enhancement pattern (localization and intensity) was visually evaluated by two neuroradiologists and compared with a series of 20 contrast-enhanced facial nerves without any evidence of facial nerve disease. These control subjects had been evaluated for suspected acoustic neuroma, but MR did not prove any abnormality. Identical technical parameters were applied for the examination of the control subjects. Furthermore, a concomitant enhancement of the vestibulocochlear nerve and of the cochlea and/or vestibule itself was occasionally observed. Its frequency was evaluated in relation to the underlying cause.

### Results

Table 1 shows the intensity and location of the contrast enhancement of the facial nerve in all patients of our series with lower motor neuron facial nerve palsy and in the group of control subjects evaluated for suspected acoustic neuroma without clinical evidence of facial nerve disease.

The normal facial nerve usually shows a moderate enhancement of the geniculate ganglion and the proximal tympanic segment. A mild enhancement is observed in the distal tympanic and the mastoid segment (Fig 1).

A moderate to intense enhancement of the distal intracanalicular segment of the facial nerve is always observed in patients with facial palsy (Figs 2 and 3, *arrow 1*). A mild enhancement of the labyrinthine segment is common (Figs 2 and 3, *arrow 2*). The geniculate ganglion and the proximal tympanic segment usually show a moderate to marked enhancement (Figs 2 and 3). The distal tympanic and the mastoid segment, however, do not often present an abnormal enhancement.

Early postoperative MR examinations in 2 patients performed within 2 weeks after the operative decompression of the facial nerve at the meatal foramen show a reduced enhancement of the facial nerve in the distal intracanalicular segment compared with their preoperative examinations. The intensity of the enhancement of the labyrinthine segment, however, is increased compared with the preoperative MR (Fig 4). An additional enhancement of the vestibulochochlear nerve is observed in 3 patients with idiopathic

TABLE 1: Enhancement pattern of the facial nerve in 23 patients with lower motor neuron facial nerve palsy and in 20 control subjects

Enhancement	Facial Nerve Segment					
	Distal intrameatal	Labyrinthine	Ganglion	Tympanic		Mastoid
				Proximal	Distal	
Idiopathic palsy (n = 14)						
Intense	8	-	6	3	1	2
Moderate	6	-	6	9	-	5
Minimal to mild	-	14	2	2	13	7
Herpetic palsy (n = 8)						
Intense	5	-	6	4	2	1
Moderate	2	-	2	4	1	2
Minimal to mild	1	8	-	-	5	5
HIV-associated palsy (n = 1)						
Intense	-	-	-	-	-	-
Moderate	1	-	1	1	-	1
Minimal to mild	-	1	-	-	1	-
Normal facial nerve						
Intense	-	-	-	-	-	-
Moderate	-	-	20	20	-	-
Minimal to mild	-	-	-	-	20	20

Note.—Control subjects were evaluated for suspected acoustic neuroma; they had normal MR findings and no evidence of facial nerve disease.

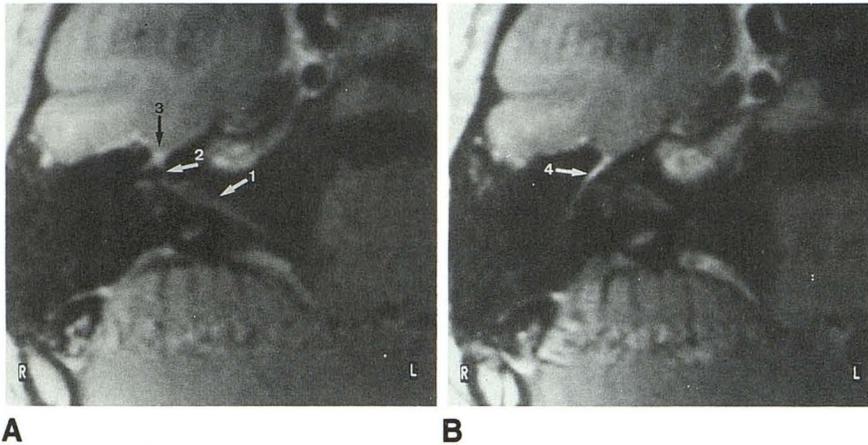


Fig. 1. Transverse postcontrast MR of a normal facial nerve with moderate enhancement of the geniculate ganglion (3), proximal tympanic (4) and mastoid segment. Absent enhancement of the distal intracanalicular (1) and the labyrinthine (2) segment.

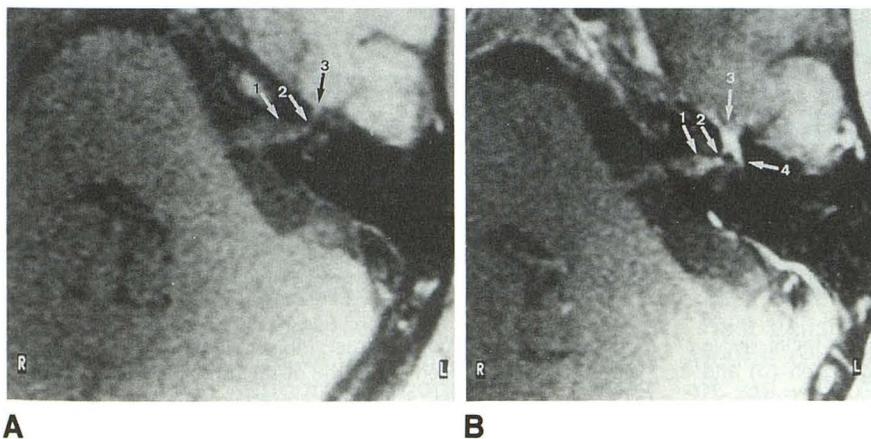


Fig. 2. Transverse precontrast (A) and postcontrast (B) MR in herpetic facial nerve palsy. Intense enhancement of the geniculate ganglion (3), proximal tympanic (4), and distal intracanalicular (1) segment of the facial nerve. Minimal enhancement in the labyrinthine segment (2).

Fig. 3. Transverse precontrast (A) and postcontrast (B) MR in idiopathic facial nerve palsy. Intense enhancement of the distal intracanalicular (1) and proximal tympanic (4) segment of the facial nerve and of the geniculate ganglion (3). Minimal enhancement of the labyrinthine segment (2).

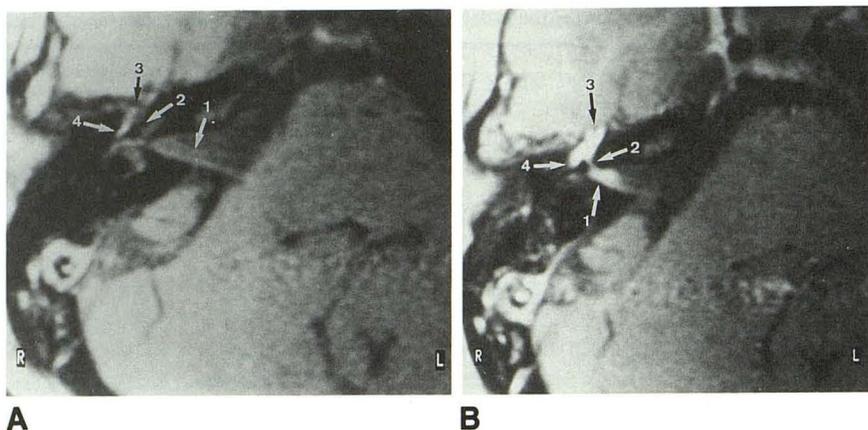


Fig. 4. Postoperative precontrast (A) and postcontrast (B) transverse MR in idiopathic facial nerve palsy. Decompressed facial nerve at the dilated meatal foramen. Decreased enhancement of the distal intracanalicular segment (1) of the facial nerve compared to the preoperative MR. Moderate enhancement of the labyrinthine segment (2) postoperatively.

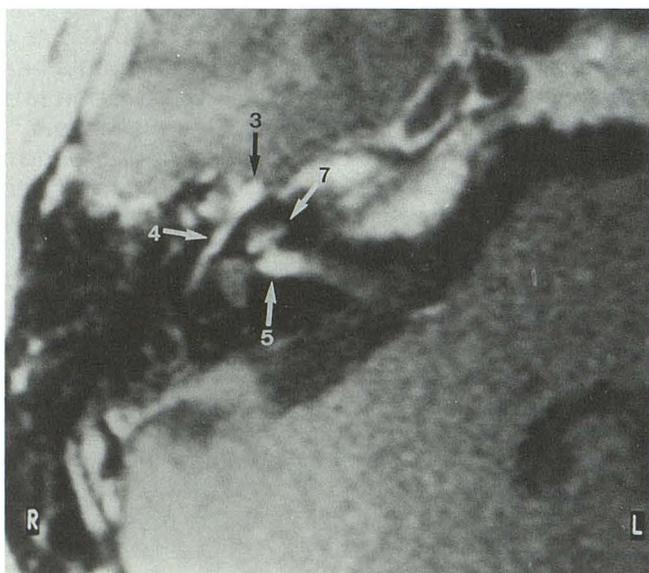
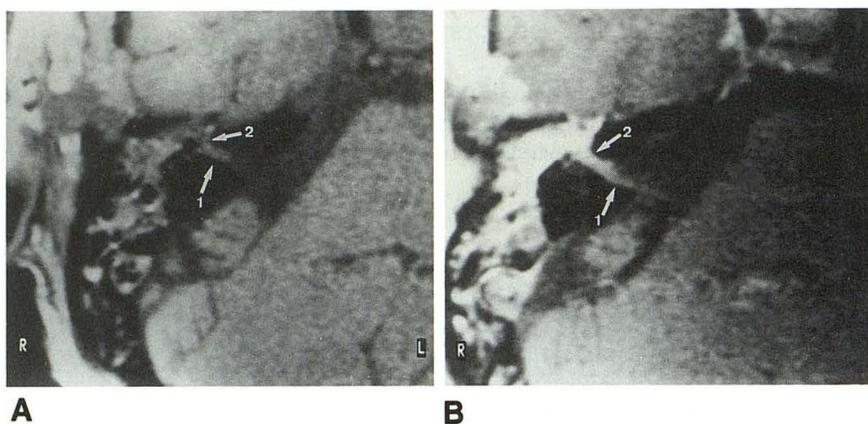


Fig. 5. Transverse postcontrast MR in herpetic facial nerve palsy. Intense enhancement of the geniculate ganglion (3) and the proximal tympanic (4) segment of the facial nerve. Enhancement of the vestibulocochlear nerve (5) and of the cochlea (6).

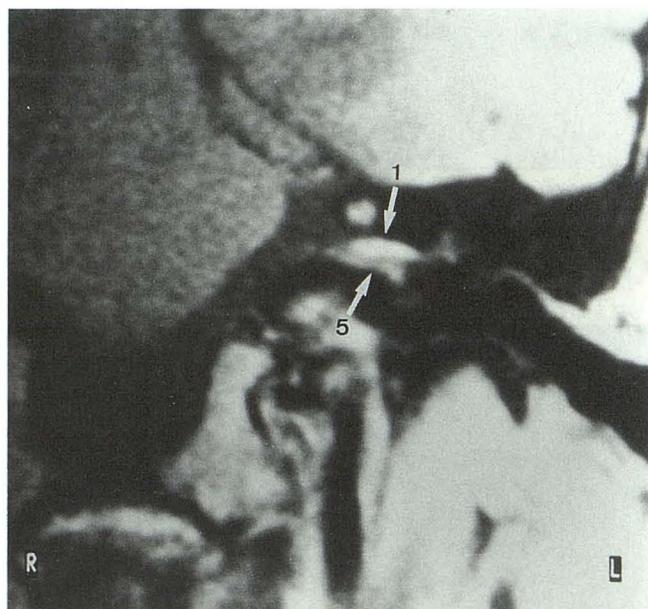


Fig. 6. Coronal postcontrast MR in idiopathic facial nerve palsy. Intense enhancement of the distal intracanalicular segment (1) of the facial nerve. Additional enhancement of the vestibulocochlear nerve (5).

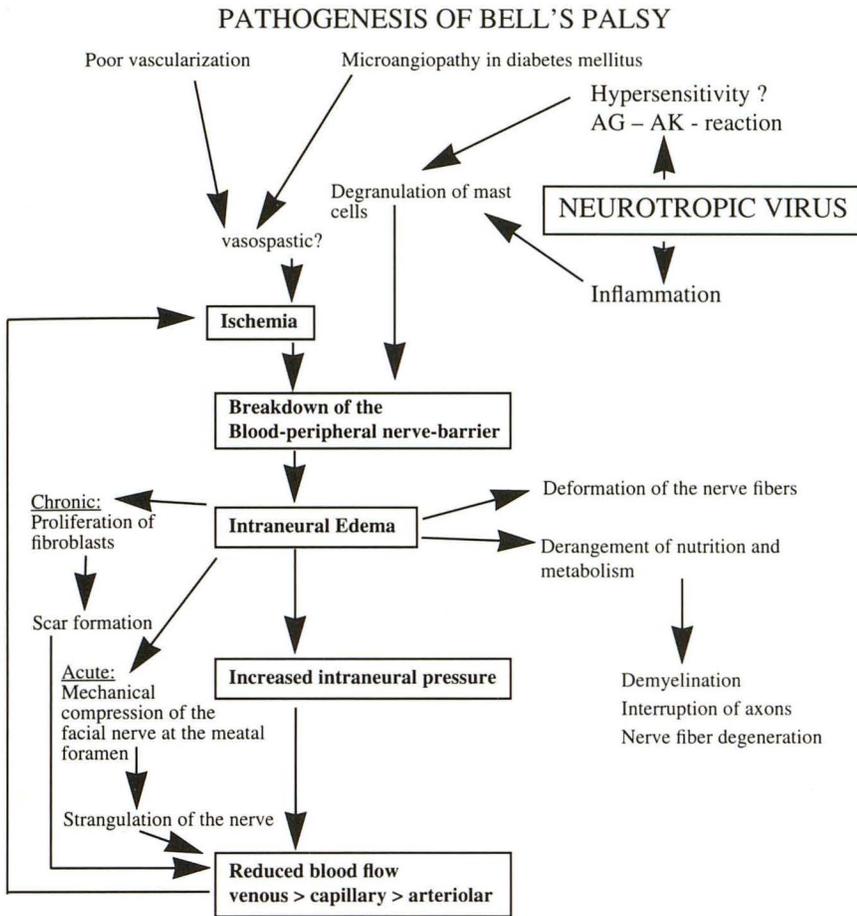


Fig. 7. The complex pathogenesis in Bell, herpetic, and HIV-associated facial nerve palsies.

palsy and in 4 patients with herpetic palsy (Figs 5 and 6, *arrow 5*). An enhancement of inner ear structures (cochlea, Fig 5, *arrow 7*, and vestibule) is seen in the same 4 patients with herpetic palsy. Three of the patients also presented with vertigo and hearing loss, referring to inner ear or vestibulocochlear deficits. Only 1 of the 3 patients with Bell palsy and enhancement of the vestibulocochlear nerve, however, presented with subjective hyperacusis but had a normal audiogram.

**Discussion**

Idiopathic (or Bell) palsy and herpetic facial nerve palsy (Ramsay Hunt syndrome) mainly cause a unilateral lower motor neuron facial nerve palsy (1). In herpetic palsy the varicella-zoster virus causes peripheral nerve palsy (15, 16), and involvement of other cranial nerves (predominantly of the vestibulocochlear and the trigeminal nerve) can be observed (1, 2, 15, 32).

In Bell palsy the exact causes of this mostly monosymptomatic nerve palsy are uncertain, but recent data also support the concept of a viral infection (1, 2, 14, 17, 18).

Neurotropic viruses (especially the herpesvirus group, including varicella-zoster, herpes simplex, cytomegaly, and Epstein-Barr virus) silently infect the sensory cells of peripheral nerves (eg, the geniculate ganglion). The virus may be reactivated at any time and may move centrally and peripherally along the nerve fibers.

The complex pathogenesis (19) in Bell, herpetic, and HIV-associated facial nerve palsies is shown in a schematic in Fig 7. The formation of an intraneural edema seems to be the main factor in determining the pathophysiological process: the reactivation of the latent viral infection probably produces an intraneural edema in the peri-, epi-, and endoneural sheaths. The edema itself results either from a breakdown of the blood-peripheral nerve barrier or from venous congestion of the epi- and perineural venous plexuses (13). The blood-peripheral nerve barrier is formed by the endothelium of the endoneural capillaries and by the flattened inner perineural cells covered with basement laminae (11, 13). Mast cells are frequently found along the intraneural capillaries and may degranulate because of an allergic-immunologic (hypersensitivity?) (16, 20, 21) or direct inflammatory process. The

released substances increase the permeability of the blood-peripheral nerve barrier with consecutive spread of liquid into the endoneural space, intraneural edema formation, and nerve fiber swelling. On the other hand, these substances are also well-known vasoactive mediators and may possibly break down the blood-nerve barrier by a vasospastic-ischemic process. The vasospastic effect is pronounced in cases with preexisting diabetes mellitus and associated microangiopathy (1, 17). However, even in healthy persons, the nutrition especially of the labyrinthine segment is always very poor because of the limited vascularization in this area (1, 2, 22-24).

Intraoperative evoked electromyography locates the site of the nerve involvement at the meatal foramen in 94% of all patients. This foramen is the narrowest site of the entire osseous fallopian canal (3, 25). The edematous and swollen facial nerve is compressed at the foramen. This mechanical compression and the increased intrafascicular pressure resulting from the endoneural edema can lead to a reduced endoneural capillary and peri- and epineural arteriolar/venular flow with subsequent hemorrhagic/ischemic infarction of the nerve. Ischemia and mechanical compression together are accompanied by a further local increase of the permeability of the blood-peripheral nerve barrier. The resulting edema spreads along the endoneural space and triggers a vicious circle of facial nerve damage (13, 26-29).

The neural dysfunction in the acute stage of the facial nerve palsy is caused by a derangement of the endoneural metabolism attributable to the endoneural edema, by the ischemic infarction, by the impaired axoplasmic flow followed by demyelination, interruption of axons, and nerve fiber degeneration (1, 3, 13). In the chronic stage the intraneural edema brings about the formation of an intraneural scar, interfering with the restoration of the nerve function (12, 13).

Currently, MR seems to be the ideal technique to delineate the facial nerve from the brain stem to the stylomastoid foramen (30). The normal facial nerve always shows a moderate degree of enhancement in the geniculate ganglion and in the proximal tympanic (Fig 1, arrows 3 and 4) segment. A mild enhancement is seen in the distal tympanic and the mastoid segments. This normal enhancement most probably results from pooling of contrast material within the venous plexuses at the peri- and epineurium. The absence of enhancement in the labyrinthine and intracanalicular nerve segments (Fig 1, arrows 1 and 2) is explained by the absence of the peri- and epineurium in these nerve segments (1, 3, 26).

The intense, homogeneous, and smooth enhancement of parts of the facial nerve in cases of herpetic, idiopathic, or HIV-associated peripheral facial nerve palsy possibly is caused by a breakdown of the blood-peripheral nerve barrier and subsequent diffusion of contrast material into the endoneural space. Venous congestion in the epi- and perineural plexuses, on the other hand, could also cause an enhancement on the basis of an increased vascular pool of contrast material (11, 13). It is not known why the intense enhancement is limited to certain segments of the facial nerve, especially in the region of the geniculate ganglion. Perhaps it is related to the reactivated viral infection in the geniculate ganglion by neurotropic viruses and secondary spread of the inflammatory process along the endoneural space proximally and distally.

The markedly reduced flow of contrast material into the compressed endoneural capillaries in the labyrinthine segment seems to prevent a severe leakage of contrast material into this segment and the resulting enhancement is minimal. After surgical decompression of the meatal foramen, however, the mechanical compression of the nerve is decreased, the capillaries are reopened, and the still-impaired blood-peripheral nerve barrier allows a further flow of contrast material into the endoneurium, resulting in a moderate enhancement of this segment postoperatively. This phenomenon was observed in 2 patients (1 with idiopathic and 1 with herpetic palsy) in an early postoperative control MR within 2 weeks after the surgical decompression. The enhancement of the distal intracanalicular segment of the facial nerve, however, was reduced or absent in the early postoperative control MR, indicating a rapid postoperative improvement. Although the patients still had complete facial nerve paralysis, there were already signs of reinnervation on electromyography in this early postoperative period. Three months after surgical decompression, a complete clinical and electrophysiological recovery of the nerve function was observed in all patients.

Although a moderate to severe enhancement of the tympanic segment and the geniculate ganglion and sometimes of the mastoid segment is regularly observed in cases with idiopathic and herpetic facial nerve palsy, these enhancement patterns are not diagnostic. It depends on the experience of the neuroradiologist to distinguish between a normal enhancement and one that indicates disease. It also must be emphasized that the present study used a high dose of gadolinium. At standard doses of 0.1 mmol/kg, the enhancement patterns may be somewhat less intense than observed in our series, although we would

not expect the distribution or appearance to change significantly. The only distinct and diagnostically reliable feature of an inflammatory nerve lesion is a typical moderate to intense, homogeneous, and smooth enhancement of the facial nerve in the fundus of the internal auditory canal. This may be caused by a leakage of contrast material into the endoneural space. It may also be related to the intraoperatively observed engorgement of the axoplasmic flow in the nerve segments shortly proximal to the mechanical compression (3, 26, 31).

An additional enhancement of the vestibulocochlear nerve can be observed in patients with idiopathic or herpetic facial nerve palsy (29, 32). In idiopathic palsy, the asymptomatic involvement of the vestibulocochlear nerve is clinically well known, but the frequency of the involvement differs in reports from various authors. It is possibly caused by compression of the vestibulocochlear nerve by the swollen facial nerve within the tight osseous borders of the internal auditory canal.

In herpetic palsy, however, the enhancement of the vestibulocochlear nerve is accompanied by an enhancement of cochlea and/or vestibulum in mostly symptomatic patients and correlates with the phenomenon of multiple cranial nerve involvement in this disease (1, 32).

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