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T2-Weighted Three-dimensional Fast Spin-Echo MR in Inflammatory Peripheral Facial Nerve Palsy

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PURPOSE: **Our objective was to identify histologically and intraoperatively verified focal nerve thickening of the distal intrameatal segment on three-dimensional fast spin-echo (FSE) T2-weighted MR images as a new diagnostic criterion in patients with inflammatory peripheral facial nerve palsy.**

METHODS: **Twenty-two patients with clinically diagnosed unilateral (n = 20) or bilateral (n = 2) inflammatory peripheral facial nerve palsy were examined on a 1.5-T MR imager using noncontrast and contrast-enhanced T1-weighted SE sequences and 3-D T2-weighted FSE sequences with secondary reformations. Abnormal contrast enhancement and possible focal nerve thickening of the distal intrameatal segment, labyrinthine nerve segment, and geniculate ganglion region were analyzed prospectively.**

RESULTS: **In all patients, the T1-weighted postcontrast SE images showed characteristic smooth, linear, abnormally intense contrast enhancement of the distal intrameatal segment, indicating peripheral inflammatory nerve palsy. In 23 nerves (96%) a focal bulbous nerve thickening of the distal intrameatal segment was observed on 3-D T2-weighted FSE images. In 100% of patients with peripheral inflammatory facial nerve palsy, postcontrast T1-weighted SE images showed a smooth, linear, and abnormally intense contrast enhancement of the distal intrameatal segment; reformatted very thin 3-D T2-weighted FSE images showed a focal bulbous nerve thickening of the distal intrameatal segment in 96% of patients. These findings corresponded to intraoperative and histologic findings.**

CONCLUSION: **Three-dimensional T2-weighted FSE sequences are fast and cheap compared with T1-weighted postcontrast images, but secondary reformations are time-consuming and require exact anatomic knowledge for careful analysis of the different nerve segments.**

In patients with intraoperatively and histologically established inflammatory peripheral facial nerve palsy, a focal short-segmented bulbous swelling of the distal intrameatal nerve segment of the facial nerve can be observed near the entrance of the bony canal, resulting from inflammatory edematous nerve thickening and from damming of the axoplasmic flow (1–4). However, to date, neither two-dimensional T2-weighted spin-echo (SE) images with a section thickness of 2 to 3 mm (and therefore limited spatial resolution) nor T1-weighted postcontrast SE images have been able to depict this focal nerve thickening. Although postcontrast T1-weighted SE images show a characteristic linear, smooth, and abnormally intense contrast enhancement of the distal intrameatal and labyrinthine segments, often with associated abnormal enhancement of the geniculate ganglion region (5–11), the focal nerve thickening cannot be reliably assessed, because after contrast enhancement, structures are known to appear larger than they really are.

In our prospective study we tried to determine whether the intraoperatively and histologically observed focal nerve swelling of the distal intrameatal segment can be depicted on very thin three-dimensional high-resolution T2-weighted fast spin-echo (FSE) images and whether the T1-weighted postcontrast images showing the characteristic abnormal nerve enhancement of the distal intrameatal segment can be replaced by the faster and cheaper T2-weighted 3-D FSE images in establishing an adequate and reliable diagnosis of inflammatory peripheral facial nerve palsy.

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Methods

Twenty-two patients (eight men and 14 women; mean age, 46 years; range, 18 to 72 years) with unilateral (n = 20) or

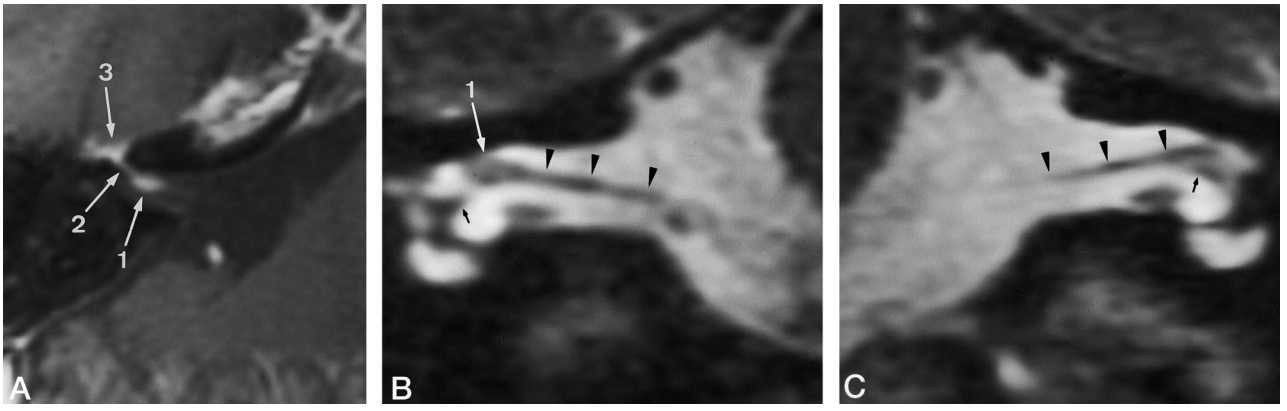

FIG 1. A 68-year-old man with a typical idiopathic peripheral facial nerve palsy on the right side.

A, Transverse contrast-enhanced T1-weighted SE image (500/16/4) shows enhancing distal intrameatal segment (*1*) and labyrinthine segment (*2*) and intensely enhancing geniculate ganglion region (*3*).

Oblique coronal 3-D T2-weighted FSE images (4000/150/1) of the abnormal right nerve (*B*) and the healthy left nerve (*C*), reconstructed parallel to the nerve course (*black arrowheads*), show a typical focal bulbous nerve swelling of the distal intrameatal segment (*1*) on the abnormal side and a normal-sized nerve on the healthy side. A small osseous crest separates the superior from the inferior part of the internal auditory canal (*black arrow*).

bilateral (n = 2) clinically typical inflammatory peripheral facial nerve palsy were examined prospectively on a 1.5-T MR unit. Seventeen patients had idiopathic or Bell palsy and five had herpetic palsy as determined by clinical signs and symptoms and laboratory evaluation. Seven of the patients with Bell palsy had surgery for progressive neuronal degeneration, over 94% within 14 days, as established at electroneurography (1, 2); and an inflammatory nerve lesion was confirmed intraoperatively.

A 5-inch-diameter phased-array receiver dual coil was applied to both ears. T2-weighted FSE images were acquired before intravenous injection of contrast agent by using a 3-D FSE protocol with 40 to 60 coronal sections. Imaging parameters were as follows: 4000/150/1 (repetition time/echo time/excitations), 0.7-mm section thickness, 200-mm field of view (FOV) with three-quarter FOV in the phase direction, 512 × 256 matrix, echo train length of 48, three overlapping slab sections, and a scan time of 8:30 minutes. On an independent General Electric Sun workstation, secondary multiplanar reformations were performed perpendicular and parallel to the course of the distal intrameatal and labyrinthine segments as well as parallel to the course of the tympanic segment, resulting in oblique transverse, sagittal, and coronal images with a section thickness of 0.4 mm. Effective spatial resolution (voxel size) of the reformatted images was 0.4 × 0.4 × 0.6 mm.

For comparison, noncontrast T1-weighted SE images were acquired with imaging parameters of 500/minimum/3, a section thickness of 3 mm with a gap of 0.5 mm, an FOV of 170 mm, and a scan time 4:51 minutes. Postcontrast transverse and coronal overlapped T1-weighted SE images were obtained after injection of a bolus of 20 mL of contrast material with imaging parameters of 500/minimum/4, a section thickness of 3 mm with a 1-mm overlap, and an FOV of 160 to 170 mm, resulting in four series, each with a scan time of 6:28 minutes, for a total scan time of 26 minutes.

For the T1-weighted postcontrast SE images, two neuroradiologists independently assessed the presence or absence of abnormal contrast enhancement of the distal intrameatal and labyrinthine segments and the geniculate ganglion region. Additionally, they looked for focal nerve thickening of the distal intrameatal segment and of the geniculate ganglion region.

For the 3-D FSE-T2-weighted images, the presence or absence of focal nerve thickening of the distal intrameatal segment or geniculate ganglion region was evaluated. The maximum nerve diameter as well as the maximum length of the nerve thickening were measured on reformatted oblique sagittal and coronal maximally zoomed images. The measurements were repeated three times for each nerve segment and the average was calculated. Minimal blurring of the facial nerve with secondary indistinct nerve margins on the intensely zoomed images resulted in submillimeter measurement faults of 0.1 to 0.3 mm.

For comparison, the maximum nerve diameter of the contralateral normal facial nerve was measured in all patients.

Results

T1-weighted postcontrast SE images showed an abnormal, smooth, and linear enhancement of the distal intrameatal and labyrinthine segments in all patients (Figs 1A and 2A), corresponding to the findings reported in previous studies (5–6). The geniculate ganglion region showed abnormally intense enhancement in 13 nerves (54%). In one patient (4%), the geniculate ganglion region seemed enlarged (Fig 2A) relative to the normal contralateral region (Fig 2B). The thickening of the distal intrameatal and labyrinthine segments could not be assessed reliably on the postcontrast T1-weighted SE images, because the contrast agent caused an intense blurring of the margins of the facial nerve.

On the 3-D T2-weighted FSE images, 23 nerves (96%) showed thickening of the distal intrameatal segment (Figs 1B, 2C–F, and 3A and B). In one patient, the distal intrameatal nerve segment was not thickened, despite the typical inflammatory linear contrast enhancement on the T1-weighted postcontrast SE images.

In 22 nerves the very distal intrameatal nerve segment was thickened over a distance of 3 to 4 mm, with a maximum nerve diameter of 2 to 2.3 mm (Fig 3A and B). In one patient, the nerve was thickened over a distance of 6 mm, with a maximum nerve diameter of 3.5 mm (Fig 2C–E). In accordance with previous anatomic reports, the contralateral normal facial nerve measured about 1 to 1.2 mm in the distal intrameatal segment (6, 12) (Fig 3C). In one patient, a prominent thickening of the geniculate ganglion region was present, with a diameter of 6 × 3.4 mm

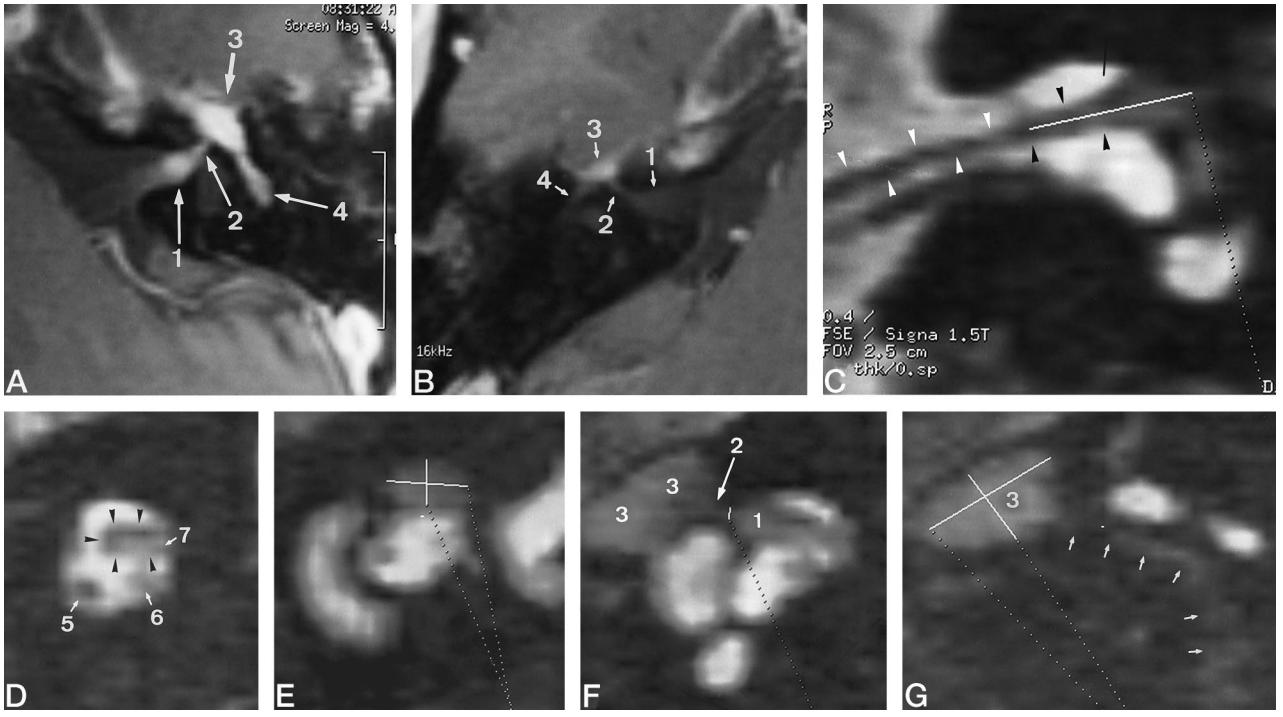

FIG 2. A 37-year-old woman with idiopathic facial nerve palsy on the left side.

A, Transverse contrast-enhanced T1-weighted SE image (500/16/4) shows enhancing distal intrameatal segment (*1*) and labyrinthine segment (*2*) and intensely enhancing and strongly thickened geniculate ganglion region (*3*) and tympanic segment (*4*) on the left side.

B, Same type of image as in *A* shows normal distal intrameatal segment, labyrinthine segment, geniculate ganglion, and tympanic segment on the right side.

C, Reformatted oblique coronal 3-D T2-weighted FSE image (4000/150/1) perpendicular to the intrameatal course of the facial nerve (*arrowheads*) shows a focal bulbous nerve swelling over a distance of 6.1 mm.

D, On oblique sagittal 3-D T2-weighted FSE image (4000/150/1) perpendicular to the very distal intrameatal segment, the facial nerve (*arrowheads*) is apparently enlarged, with a maximum nerve diameter of 2 × 3.6 mm.

E, The nerve is seen even more distally (ie, nearer to the meatal foramen) than in *D*. The cochlear nerve (*5*) and inferior vestibular nerve (*6*) are depicted. The superior vestibular nerve (*7*) is directly adjacent to the facial nerve.

F, Oblique sagittal 3-D T2-weighted FSE image (4000/150/1) parallel to the course of the labyrinthine segment shows the distal intrameatal segment (*1*), the meatal foramen (with a diameter of 0.5 mm measured on the image) with the labyrinthine segment (*2*), and the geniculate ganglion region (*3*).

G, On oblique sagittal 3-D T2-weighted FSE image parallel to the course of the tympanic segment, the thickened geniculate ganglion region (maximum diameter, 3.4 × 6 mm) is visible (*3*) with a normal nerve diameter along the tympanic and mastoid segments (outlined by *arrows*).

(Fig 2G). For comparison, the normal geniculate ganglion region measured not more than 1 to 3 mm in diameter; however, measurements of the normal geniculate ganglion region were very difficult, since, on the reformatted images, the margins of this area could not be easily defined in all patients.

Discussion

Inflammatory peripheral facial nerve palsy can be reliably diagnosed on postcontrast T1-weighted SE images, since the distal intrameatal and labyrinthine segments always have abnormal linear, smooth, and homogeneous contrast enhancement, often accompanied by intense enhancement of the geniculate ganglion and the tympanic segment (5, 6). In our department, we routinely acquire one series of 3-mm-thick noncontrast images (3:14 minutes) and four series of 3-mm-thick and overlapped (gap, negative 1 mm) contrast-enhanced T1-weighted SE images (each, 6:28 minutes) of both ears in the transverse and coronal planes to demonstrate the entire course of the facial nerve. This results in a complete MR protocol with and without contrast enhancement of five series of T1-weighted SE images, for a total examination time of 29 minutes (5, 6). Given this lengthy examination time, we hypothesized that very thin T2-weighted SE images acquired as a 3-D sequence might replace the T1-weighted contrast-enhanced SE images in the diagnosis of inflammatory peripheral facial nerve palsy. The 3-D acquisition is much faster (less than 9 minutes), allows secondary multiplanar and very thin reformations, and does not require an injection of expensive paramagnetic contrast agent.

Intraoperative and histologic observations in patients with inflammatory peripheral facial nerve palsy have revealed a prominent but focal bulbous swelling of the facial nerve along the distal intrameatal segment just proximal to the entrance of the facial nerve at the meatal foramen into the narrowest segment of the bony canal; namely, the labyrinthine segment (1–4, 7). How can this nerve swelling be explained?

In patients with inflammatory peripheral nerve palsy (ie, Bell or herpetic palsy), an edematous swell-

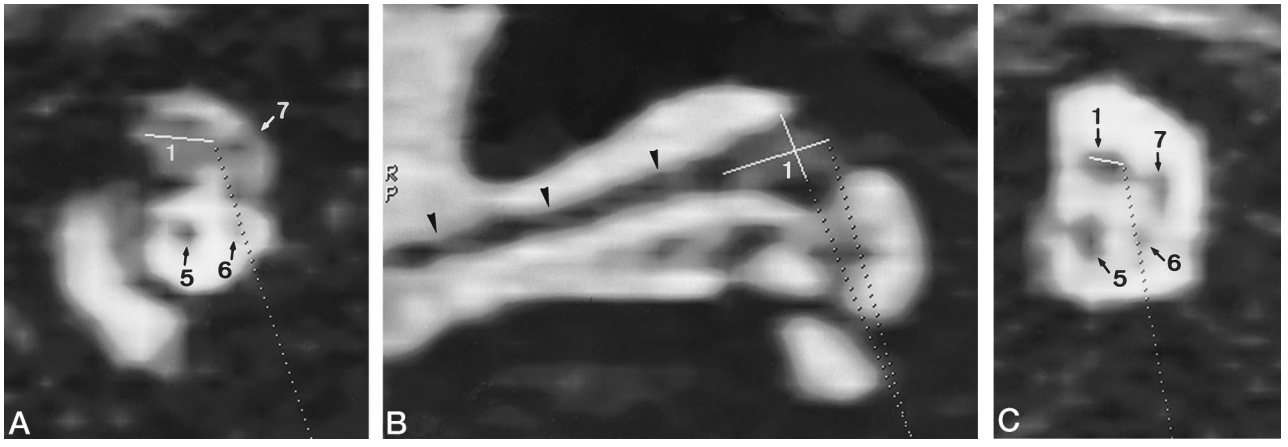

FIG 3. A 50-year-old man with unilateral idiopathic Bell palsy of the left facial nerve.

Oblique sagittal (*A*) and coronal (*B*) reformatted 3-D T2-weighted FSE images (4000/150/1) perpendicular and parallel to the nerve course within the internal auditory canal (*arrowheads*) show the focal swelling of the facial nerve in its very distal intrameatal segment (*1*) over a distance of 3.3 mm with a diameter of 2.1 mm.

C, For comparison, oblique sagittal 3-D T2-weighted FSE image shows the facial nerve (*1*), the cochlear nerve (*5*), and the inferior (*6*) and superior (*7*) vestibular nerve within the internal auditory canal. The normal facial nerve (distal intrameatal segment) has a diameter of 1.0 mm.

ing of the nerve can be seen histologically, probably resulting from breakdown of the blood/peripheral nerve barrier after release of inflammatory mediators by (often sparse) inflammatory cell infiltrates (3, 4, 7, 13–16) with secondary leakage of fluid into the perineural, epineural, and endoneural spaces (5). Secondarily, the swollen nerve is compressed within the narrowest part of the bony canal of the facial nerve, at the meatal foramen and within the labyrinthine segment. Compression of the nerve leads to a secondary damming of the neuroplasm (17, 18). Therefore, the bulbous nerve swelling is caused by both the edematous nerve swelling and the damming of the axoplasmic flow proximal to the nerve compression. The axoplasmic flow includes a unidirectional fast and slow transport system, which carries down protein and particulates from their site of synthesis in the neuron cell bodies to the periphery (18) with the help of a specialized transporting filament to which other proteins and particulates are bound. The fast transport works at a rate of 400 mm per day (17, 18). In experimental studies (2, 17), local constriction of a peripheral nerve has been shown to cause damming of the neuroplasm on the proximal side of the bottleneck along with reduction of the nerve's diameter distally, resulting proximally in a massive bulbous swelling of the nerve that tapers off in the proximal direction (17).

On T1-weighted postcontrast SE images, the diameter of the nerve cannot be measured reliably, because contrast material makes structures look larger than they really are. Therefore, reliable measurements of the nerve diameter can be made only on T2-weighted images. However, to date, nerve swelling of the distal intrameatal segment has not been demonstrated on fast 2-D T2-weighted SE MR images. The recent development of a 3-D T2-weighted FSE sequence that allows the acquisition of MR data in the submillimeter region (from 0.7 to 1 mm) and reformations in multiplanar directions with a 0.4-mm section thickness and a voxel size of $0.4 \times 0.4 \times 0.6$ mm enabled us to observe focal nerve thickening of the distal intrameatal segment proximal to the meatal foramen on T2-weighted noncontrast images in 96% of our patients. In 4% of the patients, however, no nerve thickening was observed, despite the presence of an inflammatory nerve palsy. Further studies are necessary to explain this unexpected absence of nerve thickening despite the presence of the typical inflammatory contrast enhancement on postcontrast T1-weighted SE images. Theoretically, a correlation between the extent of nerve thickening and the patient's clinical status, characterized by degree of neuronal degeneration or duration of the inflammatory process, may be assumed and should be evaluated in future investigations. In our study, measurements of nerve degeneration by electroneurography were obtained only in a few patients.

Currently, in our department, both T1-weighted contrast-enhanced images and 3-D T2-weighted FSE images are acquired in patients with suspected inflammatory peripheral facial nerve palsy, since the diagnostic reliability of the 3-D T2-weighted FSE sequence needs to be improved before it can replace the postcontrast T1-weighted SE sequence, which has a diagnostic reliability of 100%. For daily clinical examinations, however, we recommend the acquisition of only T1-weighted postcontrast SE images in patients without the typical focal bulbous swelling of the distal intrameatal segment (suggestive of an inflammatory nerve palsy) on the 3-D T2-weighted FSE sequence, since, when carefully done, acquisition of secondary multiplanar reconstructions is very time-consuming, but they are necessary to ensure optimal analysis of the 3-D T2-weighted FSE sequences. Additionally, thorough knowledge of the anatomic course of the facial nerve is mandatory in order to reliably appreciate the nerve thickening.

The focal inflammatory nerve thickening visible on reformatted 3-D T2-weighted FSE images along the very distal intrameatal segment has to be carefully distinguished from a focal nerve thickening of the same nerve segment caused by a tumorous lesion (ie, a facial nerve schwannoma). Usually, inflammatory and tumorous lesions of the peripheral facial nerve have different clinical presentations, with rapid onset of the palsy in inflammatory lesions and a slowly progressive palsy in tumorous lesions. However, there is a certain overlap in the clinical presentation, which prevents a definitive diagnosis of an inflammatory facial nerve palsy on the basis of clinical characteristics alone. In our study, the intraoperative findings in seven patients who had surgery for the inflammatory palsy reliably helped to prove the diagnosis of an inflammatory lesion and to exclude a tumorous lesion. In the other 15 patients, however, the diagnosis of an inflammatory lesion was based first on the typical clinical presentation (which is especially characteristic in patients with herpetic palsy) and second on the abnormal enhancement pattern of the various facial nerve segments seen on postcontrast T1-weighted images; namely, the typical smooth, linear, and abnormal contrast enhancement along the distal intrameatal segment.

However, the difficulties in differentiating a tumorous from an inflammatory lesion are illustrated in the MR images of a 37-year-old woman in our study (Fig 2). In this patient, the preoperative diagnosis of facial nerve schwannoma was suggested on noncontrast and contrast-enhanced T1- and T2-weighted MR images despite the typical clinical presentation of an inflammatory palsy, since not only the distal intrameatal segment but also the geniculate ganglion region was apparently thickened and intensely enhancing (Fig 2). In patients with inflammatory palsy, abnormal contrast enhancement of the distal intrameatal segment and often of the labyrinthine segment and geniculate ganglion can be observed, but obvious thickening of the geniculate ganglion region has not yet been described (5, 6). Owing to rapid clinical deterioration, with neuronal degeneration of more than 94% within 4 days, this patient was operated on, and the intraoperative diagnosis of an inflammatory nerve lesion was proved by an intense swelling of the distal intrameatal segment and geniculate ganglion region. Additional help for differentiating tumorous from inflammatory nerve lesions could be gained from repeat MR studies showing the facial nerve returning to normal size in patients with clinical improvement; however, in our patients, repeat MR studies were not done.

Conclusion

Inflammatory peripheral facial nerve palsy appears as a typical smooth, linear, and abnormal contrast enhancement of the distal intrameatal segment on contrast-enhanced T1-weighted SE images and as a focal bulbous nerve swelling of the distal intrameatal segment on very thin T2-weighted 3-D FSE images. Both morphologic criteria can be seen in inflammatory nerve palsy but, in our study, the postcontrast T1-weighted images showed the abnormality slightly more often than did the 3-D T2-weighted FSE images. The 3-D T2-weighted FSE sequence allows the depiction of nerve structures in the submillimeter region; therefore, for the first time, correlations with microscopic views are possible.

Compared with noncontrast and contrast-enhanced T1-weighted SE images, the new unenhanced 3-D T2-weighted FSE sequence saves the cost of the contrast material and presumably requires the patient to spend less time in the scanner; however, it needs careful and often time-consuming postprocessing for reliable assessment and evaluation of the images, resulting in additional costs.

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