II
FACIAL NERVE COMPRESSION IN SMALL ACOUSTIC NEUROMAS

Introduction

Facial palsy is often a late symptom in patients with acoustic neuromas. The incidence varies from over 50 to 15 % owing to the occurrence of large tumours in the material (Cushing, 1917; Lundborg, 1952; Pulec & House, 1964; Olivecrona, 1967; Pertuiset, 1970; Thomsen & Zilstorff, 1975). The unaffected motor function of the facial nerve contrasts sharply with the surgical finding of a greatly deformed and distorted facial nerve in most cases.

Interrupted or delayed trigemino-facial transmission has been reported in patients with acoustic tumours (Bender et al., 1969; Lyon & Van Allen, 1972; Kimura & Lyon, 1973; Eisen & Danon, 1974). In most patients with confirmed tumour this was large, however, and the morphological relationship between the tumour and the facial and trigeminal nerves was not determined in any case.

Compression of the facial nerve is present, however, when the tumour is still intrameatal since the VIIth nerve intimately accompanies the compartments of the VIIIth nerve, where these lesions originate. A conduction block in the facial nerve is therefore to be expected even at an early stage.

In the present study the conduction of the trigemino-facial pathway has been examined in patients with small acoustic neuromas. The aim is to evaluate if the facial reflex examination can be used to detect the facial nerve involvement in these lesions. The study was in progress during several years, since intracanalicular acoustic neuromas are still not often diagnosed.

Patients

11 unselected patients, 2 women and 9 men, aged 26-68 years, were studied.

Acoustic neuroma was histologically confirmed in all patients, always within the internal auditory meatus and with no or little spread into the cerebello-pontine angle. There was no involvement of the brain stem or trigeminal nerve in any case.

Hearing loss was the initial symptom in all patients, except Case 6, whose first symptom was dizziness. The mean duration of the hearing loss was 6.3 years.

Electronystagmography and audiometric tests showed unilaterally impaired caloric excitability and sensory-neuronal hearing loss in all patients. No clinical signs of impaired motor function of the facial nerve were found. No symptoms or signs of involvement of the brain stem, cerebellum, or the trigeminal or other cranial nerves were present, in conformity with the definition of tumour size.

Radiological examination of the internal auditory meatus, pneumoencephalography (PEG), computerized tomography (CT) and/or positive-contrast cisternography were done. These examinations were also supplemented by vertebral angiography in most cases.

Radiological evidence of widening of the internal auditory meatus as defined by Valvassori (1969) was obtained in all patients. These changes were classified into two groups, Slight and Marked. In the Marked group the canal was either eroded or dilated 4 mm or more in comparison with the corresponding segment on the oppisite side. Patients with canals widened from 2 to 4 mm were assigned to the Slight group.
Changes on PEG, CT and cisternography conformed with the surgical findings. There were no signs of displacement or deformity of the brain stem or of the fourth ventricle.

All patients were operated on by the suboccipital approach. In most cases a microsurgical technique was used.
The internal auditory meatus was drilled out and the tumour extension within the canal was carefully exposed. The deformity of the facial nerve was demonstrated when the tumour was removed step by step. The facial nerve was usually flattened and splayed by the tumour as it entered the canal. Within the meatus the nerve was squeezed against the meatal wall.

The patients were classified into two groups according the degree of facial-nerve deformity and the size of involved nerve segment within the canal (Fig. 7).

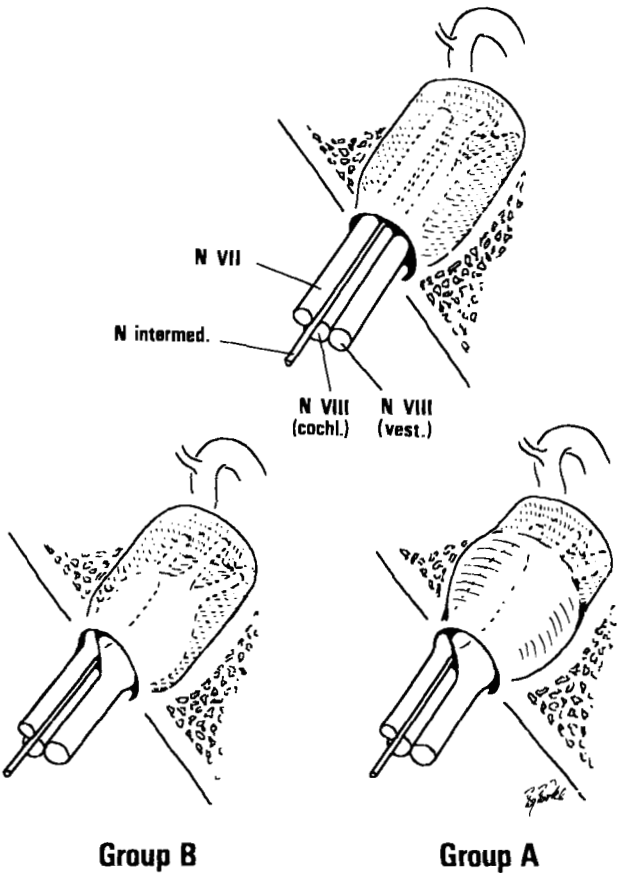

Fig. 7. Diagram of the facial and acoustic nerves in relation to the internal auditory meatus in normal subjects and in patients with acoustic neuromas.
The facial-nerve deformity within the meatus was classified into two groups according to size of the involved nerve segment and degree of involvement: Group A (Marked involvement) and, Group B (Moderate involvement).

In group A most of the intrameatal nerve segment was greatly flattened. In group B the compressive lesion was smaller, and the facial nerve was involved to a lesser extent than in group A.

The clinical, radiological and surgical findings are listed in Table 1, but further comments are called for in 4 patients.

Case 1. The facial nerve showed marked deformity within the meatus (group A), but the nerve was also displaced ventrally and stretched by a cerebello-pontine extension of the tumour.

Cases 8 and 11. There was marked intracanalicular facial-nerve involvement (group A) in both patients. Their facial nerves were also squeezed against the petrous bone just before entering the internal auditoty meatus.

Case 10. The tumour was extensive round the internal auditory foramen, the bony wall of which was eroded, but the lesion did not reach the fundus of the meatus. There was an uninvolved segment of the facial nerve of 5 mm in the lateral part of the canal. The intrameatal nerve involvement in this patients was classified into grup B.

Methods

TEST PROCEDURE

The early response was evoked by repetitive mechanical stimulation at 1 Hz, and recorded by electromyographic technique. For details see Part I. No patient was receiving drugs known to influence the facial reflexes. The test preceded the neuroradiological examination except in one patient, Case 3, where it took place the day after lumbar pneumoencephalography. Serial examinations on 4 separate occasions were done before operation in Case 5 . In 2 patients, Cases 9 and 10, the test was repeated in the early postoperative period.

CHARACTERISTICS ANALYSED

The characteristics of R1 were studied. The measurements were based on c. 100 responses on each side.

The following variables were analysed.

The mean latency (ML), in ms, on both sides,

The mean latency difference (MLD),

The latency distribution (SD),

The pattern of latency.

Table 1. Clinical, radiological and surgical findings on the involved side in patients with small acoustic neuromas.

CASE	SYMPTOMS			AUDIO-VESTIBULAR TESTS		X-RAY CHANGES	SURGICAL FINDINGS	
Sex Age	Duration of Hearing Loss	Tinnitus	Vertigo	Sensory-neuronal Hearing	Caloric Test 44°C and 30°C	Meatus widening [b]	Tumour size	Facial Nerve Involvement in the Internal Auditory Meatus (Group) [a]
1 M 68	10 yrs	0	occasional	RH	RR	M	small pontine angle spread	A
2 F 68	12 yrs	+	occasional	D	RR	S	intrameatal	B
3 M 67	8 yrs	+	occasional	RH	NR	S	intrameatal	A
4 M 57	7 yrs	+	occasional	D	RR	M	intrameatal	A
5 F 54	2 yrs	+	0	RH	NR	M	intrameatal	A
6 M 54	1 yr	+	occasional	RH	NR	S	intrameatal	B
7 M 50	3 yrs	+	0	RH	RR	S	intrameatal	B
8 M 44	4 yrs	+	0	RH	RR	M	small pontine angle spread	A
9 M 43	15 yrs	0	0	D	NR	M	intrameatal	A
10 M 26	5 yrs	0	0	RH	RR	M	intrameatal	B
11 M 53	2 yrs	+	0	RH	NR	M	small pontine angle spread	A

a) For definitions see text
b) Meatus dilated either 2-4 mm (S) or 4 mm or more and/or eroded (M)
Abbreviations. RH = residual hearing, D = deafness, RR = reduced response, and NR = no response

(For definition and description of measurement technique see Part I pp. 16-17).

The investigation included also the mean duration of R1 and the rate of occurrence of R1c. These variables are not further commented in the present study, since they occurred symmetrically and did not differ from values obtained in the control study (see Part I).

The electromyographic interference pattern was also studied. The patients were instructed to close their eyes tightly and the potentials were displayed as described earlier. Obvious differences in the interference between the tumour side and non-tumour side were established from the recordings.
On that occasion recordings at rest were also analysed with the same electrodes (Buchthal, 1965).

Results

The delay of the early response on the tumour side can be ascribed to the facial nerve compression as the trigeminal nerve and the brain stem were proved to be uninvolved. The individual results of the facial reflex examination are shown in Table 2.

The mean latency difference exceeded the upper limit of 0.25 ms in all patients (Fig. 8).
In patients with marked intracanalicular facial-nerve involvement (Group A) the mean delay was 0.80 ms ($n = 4$, SD_{n-1} 0.17). 3 patients, Cases 1, 8 and 11, were excluded from Group A owing to facial-nerve distorsion within the pontine-angle; mean latency difference was about 1.5 ms in these cases. In Group-B patients the mean delay was 0.52 ms ($n = 4$, SD_{n-1} 0.07). The difference in latency delay between groups A and B is significant ($P < 0.025$).

The latency distribution of the early response was within the reference limits (0.49 ± 0.15 ms,) except in Case 3.

The pattern of latency was similar on both sides, and a declining graph was obtained in all patients.

Table 2. Test results in patients with small acoustic neuromas. Mean latency (ML) and SD of the early response (R1) on tumour side (i) and non-tumour side (c) and mean latency difference (MLD) in ms.

CASE	AGE	$ML_i \pm SD$	$ML_c \pm SD$	MLD
1	68	16.23 ± 0.60	14.77 ± 0.56	1.46
2	68	15.05 ± 0.52	14.43 ± 0.61	0.62
3	67	14.18 ± 0.65	13.63 ± 0.79	0.55
4	57	13.64 ± 0.39	12.83 ± 0.57	0.81
5	54	12.96 ± 0.42	12.03 ± 0.47	0.93
6	54	12.97 ± 0.61	12.46 ± 0.58	0.51
7	50	11.73 ± 0.35	11.18 ± 0.53	0.55
8	44	15.09 ± 0.45	13.55 ± 0.55	1.54
9	43	13.63 ± 0.39	12.71 ± 0.37	0.92
10	26	12.07 ± 0.58	11.64 ± 0.46	0.43
11	53	15.59 ± 0.59	13.96 ± 0.46	1.63

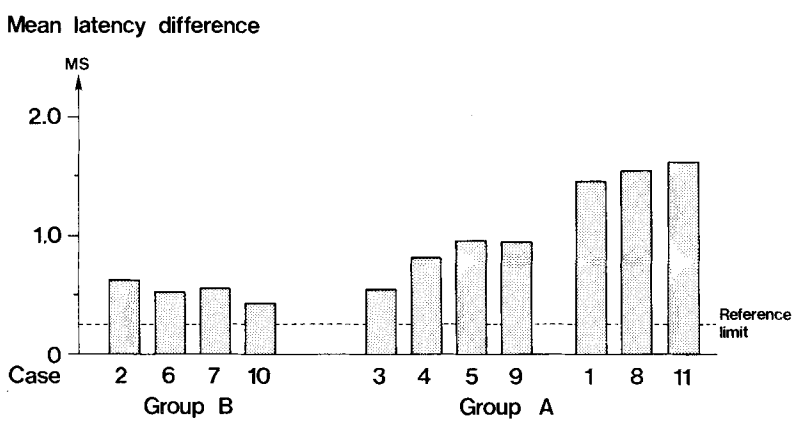

Fig. 8. Change in latency of R1 (mean latency difference) in 11 patients with small acoustic neuromas. The patients were grouped according to the intrameatal facial-nerve involvement. In Cases 1, 8 and 11 the facial nerve was also involved within the pontine angle.

The electromyographic interference pattern showed no obvious differences between the sides, and no electromyographic signs of denervation were present.

The preoperative examinations in Case 5 showed increasing mean latency on the tumour side over a period of two years. The results are shown in Fig. 9.

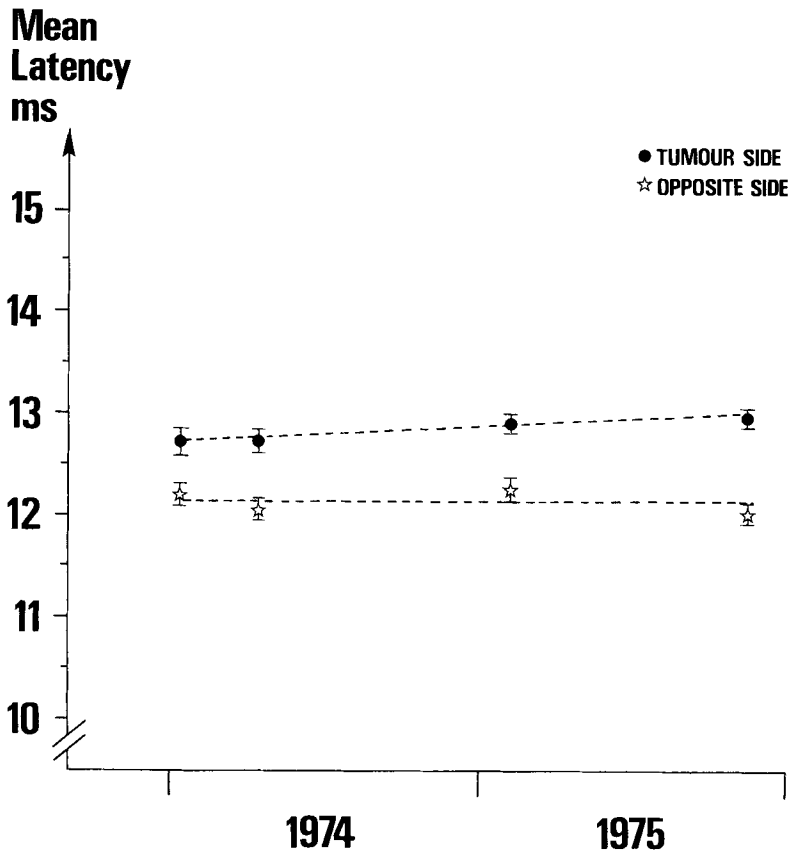

Fig. 9. Serial test results in a patient (Case 5) with an entirely intrameatal acoustic neuroma before operation. The mean latencies of R1 on both sides ± 1.96 . SE are indicated.

The results of re-examination in the early postoperative period in Cases 9 and 10 are shown in Fig. 10.

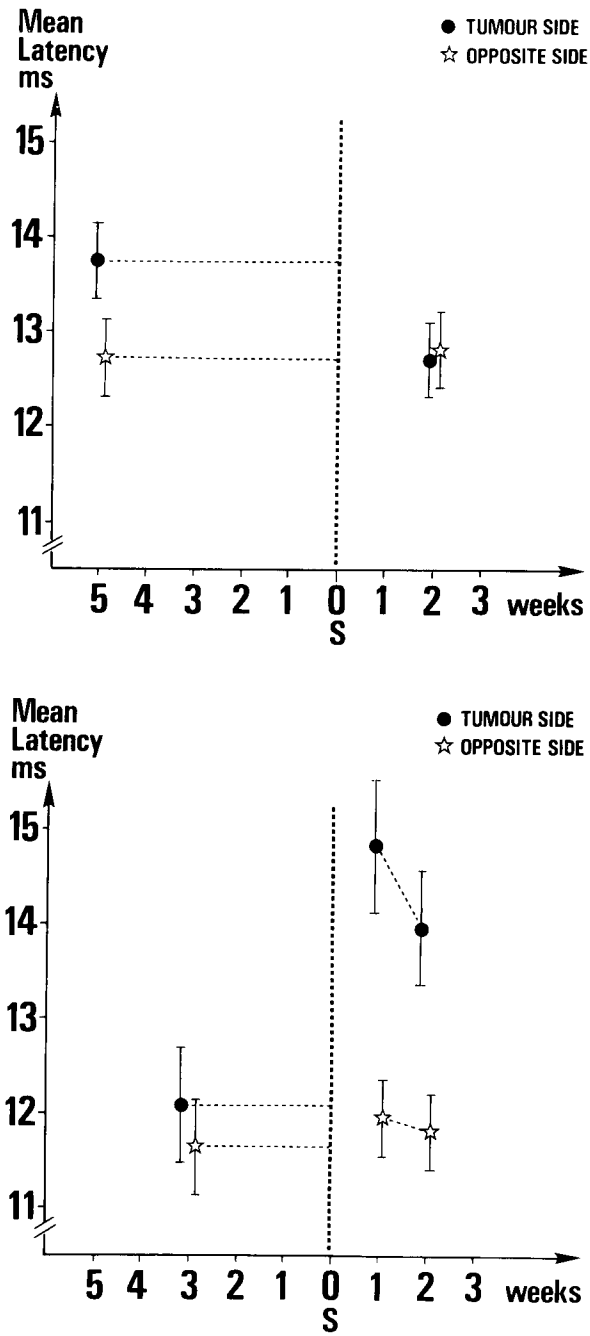

Fig. 10. Change in latency of R1 after removal (S) of an acoustic neuroma in two patients, Case 9 (above) and Case 10 (below). Transient facial palsy with total recovery developed in Case 10. The mean latency of R1 $\pm$ SD is indicated.

Discussion

Most acoustic neuromas originate within the internal auditory meatus, presumably at the junction between the neurolemmal and neuroglial portion of the vestibular nerve (Henschen, 1915; Skinner, 1929; Hardy & Crowe, 1936; Leonard & Talbot, 1970; Neely et al., 1976; Ylikoski et al.,1978a and 1978b). The further development particularly with regard to the facial nerve in enlarging tumours, may proceed as follows. As the tumour grows, the VIIIth nerve is invaded, especially the division where the tumour originates, but the VIIth nerve is displaced and later compressed against the bony wall of the canal. At this stage it is presumed that the vestibular and cochlear branches will announce clinical signs of impaired function, unlike the facial nerve, which is apparently more resistant to pressure. The expanding tumour will also cause resorption of bone with enlargement of the internal auditory meatus. The tumour then spreads to the cerebello-pontine angle, and the facial nerve in this area will also become involved. Later the brain stem and the trigeminal nerve become compressed. This is the subject of a separate study (see Part III). The facial nerve has no perineurium in its intracranial and intrameatal course (Sunderland & Cossar, 1953). The nerve fibres are loosely arranged, and they are not confined within a strong sheath encircling each funiculus. A slowly growing lesion can therefore easily flatten the nerve into a thin band, a deformity often seen. This may also explain the increased resistance of the intracranial part of the facial nerve to nerve degeneration. Progressive facial palsy is often the initial sign at a much earlier stage of a growing tumour of intratemporal segment of the facial nerve, where the fibre bundles are bound together by perineurium (Pulec, 1969; Fisch & Rüttner, 1977; Portmann, 1977).

Delayed nerve conduction in 'entrapment neuropathies' is now well documented following the original discription by Simpson (1956). A slowing of nerve conduction at, distal to, or even proximal to the compressive lesion has been confirmed in several studies in patients with carpal-tunnel and related syndromes (Thomas, 1960; Goodman & Gilliatt, 1961; Johnson & Ortiz, 1966; Kemble, 1968). A local conduction block may occur over the compressed segment with no distal nerve degeneration (Simpson, 1956).

In the present study the conduction time of R1 on the tumour side was

compared with that on the opposite side, which therefore acted as control. The delay of R1 can be ascribed to the facial-nerve involvement. The trigeminal nerve and the brain stem were proved to be uninvolved in all patients.

The mean latency difference was abnormal in all patients in this series. The difference in mean latency conformed with the facial-nerve involvement demonstrated (Fig. 8). The mean latency difference was thus significantly greater ($P < 0.025$) in patients with extensive intrameatal nerve compression (Group A) than in patients with slighter involvement (Group B). The mean latency difference was further increased in 3 patients, whose facial nerves were also involved in the pontine angle.

No patient showed electromyographic signs of denervation with the technique used. It is therefore postulated that a conduction block of the affected facial nerve segment accounts for the slowing in conduction of R1 and this is corroborated by the restoration of the conduction time two weeks after removing the tumour in Case 9.

Factors reflecting the central influence on the early response such as latency distribution and pattern of latency were within the predicted limits, except in Case 3. The increased latency distribution on both sides in this patient was probably due to the fact that encephalography was performed the day before the test. A similar change was observed after PEG in another patient (Case 77, Fig. 14). Apart from Case 3, no signs of abnormal brain-stem function were thus found; this conforms with the neuroradiological and surgical findings.

The results of serial examinations clearly prove the reliability of the test.

The mean latency value on the normal side was reproducible with no significant change on four different occasions in Case 5(Fig. 9); slight increase in latency on the tumour side could thus be shown. An increasing deterioration in hearing also pointed to a growing tumour and these signs constituted indications for surgical treatment in this patient.

The mean latency on the normal side was not significantly changed in Case 9 after removing the tumour (Fig. 10). In Case 10 the mean latency on the affected side became still greater during the early postoperative period and at the same time there was a significant increase on the non-tumour side (Fig. 10). This latter value fell to the preoperative level

two weeks after operation however; the transient change in latency therefore seems to be a consequence of the operation. Facial palsy developed postoperatively. Significant recovery in latency was noted at the second postoperative examination; the facial palsy also showed clinical inprovement which subsequently became complete.

Summary and conclusion

1. The early response (R1) was significantly delayed on the tumour side in all patients with mainly intrameatal acoustic neuromas. The size of the mean latency difference was in concordance with the observed deformity of the facial nerve. There was no clinical or neurophysiological signs of denervation of the orbicularis oculi muscles. It is therefore postulated that a conduction block of the facial nerve accounts for the delay of R1 in patients with small acoustic neuromas. The early recovery in latency after tumour removal supports this view. Facial reflex examination is an important diagnostic tool in association with audio-vestibular tests for early detection of acoustic neuromas; a condition of greatest importance for the outcome of the surgical treatment.
2. The latency distribution and pattern of latency of the early response (R1) were not changed in this study except in one patient who had been subject to pneumoencephalography the day before the test. The presence of unchanged latency distribution and pattern of latency is consistent with the proved condition that no tumour involved the trigeminal nerve and/or brain stem.