Objective: To assess the value of clinical (House-Brackmann grading) and neurophysiological (conventional electroneurography) monitoring of the facial nerve before and after (at day 10 and day 80) microsurgical parotidectomy in a group of patients with parotid tumors.

Study Design and Setting: From January 7, 1999, to February 27, 2001, 33 patients were evaluated for parotid neoplasms confirmed by cytologic examination: 27 were benign and 6 were malignant epithelial tumors. All patients underwent preoperative electroneurography of the affected side and the normal contralateral side.

Results: Preoperatively, 27 of 33 patients with benign lesions had normal facial nerve function on clinical and neurophysiological evaluation, while 3 of 6 patients with malignant lesions showed compound muscle action potential abnormalities of amplitude and latency, in the absence of facial nerve deficits. At the first postoperative evaluation, 2 of 6 patients with epithelial cancer and 4 of 27 patients with benign tumors had an absence of voluntary activity and compound muscle action potentials after nerve stimulation at the stylomastoid foramen; 1 patient with a malignant lesion and 5 patients with benign tumors had a transient facial palsy with amplitude reduction or latency prolongation of compound muscle action potential. This abnormality persisted in 2 of 27 patients at the second evaluation performed at day 80 after surgery. In 2 of 6 patients with malignant lesions, the day-80 electroneurogram showed a complete absence of nerve conduction.

Conclusion: Electroneurography is a sensitive tool for monitoring clinically silent facial nerve function deficits in the context of preoperative tumor-induced damage and postsurgical early and late follow-up of nerve function.


T otal parotidectomy with seventh cranial nerve preservation is the treatment of choice for parotid tumors. However, transient facial nerve dysfunction is reported to occur in approximately 27% of patients and permanent facial nerve palsy in 4%.1,2 The absence of a clinically detectable facial nerve deficit does not rule out the presence of subclinical nerve damage. In fact, for a slow-growing tumor to induce a clinically evident neurological abnormality, degeneration phenomena have to involve more than half of the nerve fibers.1,3 Therefore, the detection of subclinical nerve damage may be relevant in patient management.

Electroneurography (ENoG) is capable of detecting and monitoring such clinically silent abnormalities.3 The percentage of damaged nerve fibers is correlated with motor response reduction relative to the contralateral uninvolved nerve.3,4 Recent studies4-7 on normal subjects have shown limited variability between the 2 sides of the face, with mean differences between 3%7 and 32.5%.9 The objectives of this study were to assess preoperative and postoperative electroneurographic changes in the seventh cranial nerve in a group of patients with space-occupying lesions of the parotid and to estimate the sensitivity and specificity of electrophysiological investigations relative to the clinical neurological assessment.

METHODS

From January 7, 1999, to February 27, 2001, 33 patients were evaluated at the Department of ENT of the University of Ferrara Medical School (17 men and 16 women; age range, 24-74 years). The diagnostic workup included parotid region ultrasonography and fine-needle aspiration cytologic examination. The tumor types were 19 mixed tumors, 7 cystoadenolymphomas, 3 mucoepidermoid carcinomas, 2 adenocarcinomas, 1 lipocytic lipoma, and 1 non-Hodgkin lymphoma.

A total microsurgical parotidectomy with seventh cranial nerve preservation was per-
formed in 30 of 33 patients; the remaining 3 underwent revision parotidectomy for tumor recurrence. Facial nerve function was assessed clinically preoperatively and postoperatively using House-Brackmann grading (H-B).10,11; all patients underwent bilateral comparative electroneurographic evaluation on postoperative days 10 and 80. The facial nerves were investigated by electrically stimulating the nerve along its course and recording the direct motor response (compound motor action potential [CMAP]) from the orbicularis oculi and orbicularis oris muscles through surface electrodes.12 This wave was used to calculate amplitude (peak to peak) and latency. The latter variables are appropriate indicators of nerve function, with the following normal reference values: (1) CMAP amplitude of 3.1±1.0 mV when derived from the orbicularis oculi muscle and 2.2±0.8 mV from the orbicularis oris muscle and (2) CMAP latency of 3.2±0.7 milliseconds at a standard distance (10.5 cm) from the orbicularis oculi muscle and 3.0±0.6 milliseconds from the orbicularis oris muscle.13

An early electrodiagnostic study, performed on day 7 to day 10 after nerve injury, may be useful to delineate conduction block (neurapraxia) from axonotmesis.14-16 In fact, Wallerian degeneration typically occurs about 9 days after injury.17 Therefore, at day 10, the amplitude of the CMAP is roughly proportional to the number of surviving axons. We have compared injured and contralateral CMAP amplitudes and latencies to estimate the degree of axon loss. For evidence of reinnervation, a 2- to 4-month waiting period is recommended.18

### RESULTS

Facial nerve function on the preoperative clinical examination was normal (H-B grade I) in all 33 patients.

On the preoperative ENoG, all 27 patients with benign tumors had normal nerve function, while 3 of 6 patients with malignant lesions (1 of whom had a recurrent mucoepidermoid carcinoma) had abnormalities of CMAP amplitude (1 patient, ≤75%, and 2 patients, 100%, compared with the contralateral nerve) and latency (minimum latency prolongation greater than the mean±2.5 SD) (Figure).

The clinical examination performed 10 days after surgery showed that 22 (81%) of 27 patients with benign tumors had mild clinically detectable deficits (H-B grade II), while the remaining 5 (19%), including 2 with mixed tumor relapses and 1 with a postoperative parotid region hematoma, had substantial deficits (H-B grade III-IV). Among the group with malignant neoplasms, 3 of 6 had a mild nerve deficit (H-B grade II), and the remaining 3, who demonstrated electoneurographic alterations at the preoperative evaluation, had a severe deficit (H-B grade III-IV) (1 patient had a recurrent mucoepidermoid carcinoma and the other 2 had adenocarcinoma adhesive to facial nerve fibers).

In 17 (63%) of 27 patients with benign lesions (H-B grade II), ENoG performed on postoperative day 10 showed CMAP variations that were within normal limits (<50% compared with the normal contralateral nerve). In 6 (22%) of the 27, a latency prolongation (greater than the mean±2.5 SD) and CMAP amplitude decrease (>50% compared with the normal contralateral nerve) were seen. In the remaining 4 (15%), no voluntary activity or CMAPs as a response to nerve stimulation were detectable.

Among the 6 patients with malignant tumors, the day-10 examination showed no voluntary activity or CMAPs in 2 patients, 1 with adenocarcinoma and 1 with mucoepidermoid carcinoma. In the patient with adenocarcinoma, the examination showed CMAP amplitude decrease and prolongation of the motor response latency. These 3 patients had electoneurographic alterations at the preoperative examination. In the remaining 3 patients, the day-10 examination findings were normal.

On clinical examination performed on postsurgical day 80, 4 (15%) of 27 patients with benign neoplasms who had shown an absence of nerve activity at the first postoperative ENoG had a persistent complete deficit of facial motility. In 2 of 6 patients with malignant lesions, who at the first postoperative ENoG had an absence of nerve activity, the clinical evaluation demonstrated a substantial and persistent deficit (H-B grade III-IV). In 4 of 27 patients with benign lesions (1 patient had a mixed tumor relapse) who showed an absence of nerve activity at the first postoperative examination, day-80 ENoG showed a persistence of tracing abnormalities (CMAP amplitude decrease to <50% compared with
the contralateral side, with normal latency). In the 2 patients with an absence of nerve activity at the first ENoG, the alteration persisted also at day 80 and showed complete denervation (Table).

**COMMENT**

Facial expression is one of the most important means of nonverbal communication. Therefore, seventh-nerve palsy, by abolishing facial motor control, may profoundly interfere with social interactions and self-image.11

The facial nerve is the cranial nerve most frequently involved in functional deficits, largely of the peripheral type. Its long and winding path is mostly located in a nonexpandable tunnel (bony facial canal) in the parenchyma of the parotid gland and in superficial tissues; this may account for its vulnerability as a consequence of trauma, inflammatory processes, and neoplasms.19

Facial nerve damage is frequently an inevitable complication of parotid surgery, including treatment of benign lesions and especially when dealing with large masses or lesions of the deep lobe of the parotid. Postoperative palsy, even if transient, may represent a severe problem for the patient, who should be thoroughly informed about the risks and clinical course of this complication. Laccourreye et al,20 in a study of 247 patients who underwent total parotidectomy with seventh-nerve preservation, reported a 68.3% incidence of postoperative transient paralysis of the cranial nerve and a 3.6% incidence of permanent damage. In comparison, Dulguerov et al,1 in 70 patients operated on for parotidectomy, reported a 27% rate of transient deficits and a 4% rate of permanent damage.

Electroneurographic methods allow a precise and specific assessment of abnormalities of the extrapetrous portion of the seventh nerve.7 Electrophysiological studies of the motor conductivity of the facial nerve have an increasingly important role in the quantitative assessment of nerve damage secondary to parotid masses or surgical procedures. The amplitude of the CMAP provides some guidance relative to prognosis.14 In facial nerve lesions, patients with CMAP amplitudes within 30% or more of those of the contralateral side have an excellent outcome,21 those with 10% to 30% have good but incomplete recovery, and those with less than 10% have a poor outcome.22 The evaluation methods of facial nerve CMAP amplitude and latency change proposed in this study are reliable for patient monitoring, especially during postsurgical follow-up. The information allows monitoring of nerve conduction deficits and accurate assessment of their time course.

Although our study included a limited number of patients, we showed that abnormalities in latency and amplitude of evoked responses may be detected preoperatively even in the absence of clinical signs of nerve damage, as seen in 3 patients with malignant tumors. At postoperative follow-up evaluations of the patients with benign lesions, latency and amplitude evaluations showed a transient and partial block of neural transmission in 6 of 27 patients; these 6 had mild clinically detectable deficits that normalized in approximately 3 months. In the other 21 subjects, we detected mild clinically detectable deficits, in the absence of ENoG alteration. These patients fully recovered within 1 month. These results suggest that ENoG is able to distinguish between neurapraxia or mild axonotmesis of the facial nerve (associated with a good prognosis) and severe or moderate axonotmesis (associated with a prolonged or incomplete recovery).23

At the first postoperative follow-up in the patients with malignant lesions, latency and amplitude evaluations showed a complete block of neural transmission in 2 of 6 patients, which was confirmed at the second follow-up evaluation by the persistence of clinically evident facial paralysis. One of these 6 patients had a transient partial block of neural transmission accompanied by a mild clinical deficit that improved somewhat in approximately 3 months. Our results show an overall frequency of transient defects or abnormalities persisting at 3 months of 12% (4/33). These 4 patients, with benign tumors, underwent several electroneurographic evaluations that demonstrated a recovery within 12 months after nerve injury. Of the 3 patients with malignant tumors who showed alterations of nerve conduction at the preoperative ENoG, 1 with alteration of the CMAP at day 10 showed a complete recovery at day 80. In the other 2 patients, a persistent absence of nerve activity was revealed by the day-80 ENoG and by evaluations performed after 6 and 12 months.

The most relevant factors involved in the cause and pathogenesis of neural damage are nerve stretching, compression, and anoxia secondary to ischemia that may occur during surgery. Electrophysiological evaluation has shown to be a sensitive tool to monitor facial nerve function. In particular, electrodiagnostic studies can be useful for the assessment of clinically silent presurgical damage. On the basis of our results, this damage is a negative prognostic factor. Furthermore, these methods provide information to distinguish between neurapraxia and neurotmesis in postoperative follow-up evaluations and to demonstrate the degree of recovery of facial nerve function during postsurgical follow-up.
This tool may yield substantially more information than many sophisticated imaging technologies that provide little functional information. Electrophysiology is simple, rapid, and minimally invasive, and well tolerated by patients.

In conclusion, electrophysiological studies should be the investigation of first choice for the assessment of damage to nerve structures.

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