Iatrogenic injury and regeneration of the facial nerve after parotid gland tumour surgery: a pilot study with clinical and neurophysiological assessment

Agnieszka Wiertel-Krawczuk

Department of Pathophysiology of Locomotor Organs, Poznan University of Medical Sciences

http://orcid.org/0000-0002-4587-0822

Juliusz Huber

University of Medical Sciences, Poznań, Poland
Department of Pathophysiology of Locomotor Organs

http://orcid.org/0000-0002-8671-0497

Corresponding author: juliusz.huber@ump.edu.pl

ABSTRACT

Introduction. Benign tumour surgery of the parotid gland may cause iatrogenic injury of the facial nerve, with results of postoperative treatment depending on the type of injury. The study aimed to clarify the mechanism of facial nerve injury after benign tumour surgery of parotid gland.

Materials and Methods. The effectiveness was verified preoperatively and 1, 3, 6 and 17 months postoperatively. House-Brackmann scales, electroneurography, blink reflex study and needle electromyography were performed. Pharmacological treatment (Galantamine, Cocarboxylase, Dexamethasone, Triamcinolone) and supervised physiotherapeutic procedures (Facial-Oral-Tract-Therapy, Proprioceptive neuromuscular facilitation) were applied for six months.

Results. Tumour removal led to the total paralysis of the left facial nerve, IV, III and III House-Brackmann grades were ascertained at the subsequent 3rd–5th periods of observation. In postoperative studies, electroneurography results showed full functional recovery of the frontal branch and incomplete regeneration in the marginal mandibular branch. Blink reflex examination showed proper parameters of evoked potentials only during preoperative and the last observation period. Residual voluntary activity of the frontal muscle and weak voluntary activity of orbicularis oris muscle were recorded in the needle electromyography examination. Contracture of mimic muscles at rest and improvement of their voluntary activity on the left side was observed six months after surgery compared to the early period of observation.

Conclusion. Consecutive studies showed the predominant axonal type of injury in the marginal mandibular branch and neuropraxia effect of the facial nerve, allowing the creation of a rehabilitation programme optimal for the functional recovery of the nerve.

Introduction

Iatrogenic injury of the facial nerve accompanied by benign tumours surgery occurs rarely [1], with the size and location of the tumour within the parotid gland determining the scope and duration of surgery which may contribute to postoperative complications [2]. Nerve oedema and ischaemia during surgery, as well as mechanical and thermal manipulations within surrounding tissues may result in persistent or temporary facial nerve injuries in the postoperative period [3,4], which can lead to axonotmesis or neuropraxia, conditioning the degree of facial nerve damage. Neuropraxia is present when a conduction block appears without macroscopic nerve damage, which may reflect a demyelinating lesion without a Wallerian degeneration distal to the injury. In the case of axonotmesis, the nerve fibers, including motor axons are damaged, but the epineurium and perineurium remain intact, with the Wallerian degeneration occurring distal to the zone of injury [5]. Data regarding the type of iatrogenic injury, neuropraxia or axonotmesis dominating during parotid gland tumour surgery is lacking. Elmadawy, Hegab, Alahmady and Shuman [6] pointed to electromyographic (EMG) and electroneurographic (ENG) assessment as valuable tools for the evaluation of facial nerve function after different surgeries. Axonotmesis and conduction block in motor fibers may lead to degenerative changes in muscles which can be detected in needle electromyographic recordings. Conduction block as result of neuropraxia can be functionally reversible. Similarly, in the case of axonotmesis, the regeneration process is possible due to the surviving proximal stump of the nerve. The improvement of motor function in facial nerve and face muscles after 12 weeks of conservative treatment may indicate a functional recovery [6,7]. Additionally, late complications related to improper functional regeneration of the facial nerve are crucial for successful treatment [8,9].

Aim

This study aimed to explain the mechanism of facial nerve iatrogenic injury by means of axonotmesis versus neuropraxia taking into consideration results of clinical and neurophysiological observations performed 1, 3, 6 and 17 months after benign parotid gland tumour surgery. Also, conservative treatment results with pharmacotherapy which may influence nerve functional recovery in one patient with the iatrogenic facial palsy are presented.

Material and Methods

The pilot study included thirteen patients with a benign parotid gland tumour, which was recognised based on clinical and ultrasonographic examinations. The study group consisted of thirteen patients, including six women and seven men aged 16 to 67 (the average age was 47), with tumours of small or medium size, from 1.5 cm to 3.5 cm, located in the superficial lobe of the parotid gland (N = 12) except one patient, where the tumour was located in both lobes. Results of histopathological examination, surgical treatment as well as the clinical and neurophysiological studies of this particular patient are presented and discussed in detail in this paper. The surgeon removed the tumour located in the lower pole of the left parotid gland in this particular case. Ultrasound Doppler imaging revealed poorly demarcated and rich vascularised pathological structure 1.5 cm under the skin, 35 x 30 mm in size measured transversally and 40 mm sagittally in computer tomography, without signs of the destruction of the mandible and mastoid process (Figure 1). The anteromedial part of the tumour closely adhered to the left pterygoid muscle; a clear border between them was not visible in several scans. The tumour did not adhere to the masseter muscle, external auditory meatus and lymph nodes, as well as other salivary glands showed no pathologies.

Observations including clinical and neurophysiological examinations of the facial nerve function were performed preoperatively and 1, 3, 6 months postoperatively. Additionally, a patient with deep localisation of the tumour and facial nerve palsy underwent a neurophysiological examination of the facial nerve 17 months after nerve damage. The investigation took place as part of the routine diagnostics ordered by the physician. The stage of injury was also determined by the House-Brackmann (H-B) scale [10], which is used for the clinical evaluation of the

Figure 1. Photograph of CT scan with the left parotid gland tumour marked by a white arrow, the mandible is marked by a black arrow

degree of nerve damage in facial nerve palsy. The measurement is determined by analysis of the face muscle movement, symmetry, and muscle tone, with the presence of deficits like synkinesis, contracture of face muscle, or hemifacial spasm also determining the degree of nerve dysfunction. The scale is based on functional impairment, ranging between I (normal function) and VI (no movement, total paralysis).

Neurophysiological tests were recorded with a Keypoint system (Medtronic A/S, Skovlunde,

Denmark). Standard (AgCl) 5 mm2 surface electrodes were used for recording compound muscle action potentials (CMAP) from frontal and orbicularis oris muscles (Figure 2 A and B) during ENG examination. The active electrode (cathode) was placed on the muscle belly, with the reference electrode positioned on contralateral frontal muscle or the chin, the ground electrode on the neck. A stimulating bipolar electrode was applied to the stylomastoid foramen area along the anatomical passage of the facial nerve [11]. The time base was set at 5 ms/D, recording sensitivity at 2 mV/D, 20 Hz upper and 10 kHz lower filters of recorder amplifier were used during ENG.

The intensity of the single electrical stimuli (with 0.2 ms duration and 1 Hz frequency) was increased from 30 mA to a value evoking the potential with the largest amplitude. Values of amplitude [12] and standardised latency [13-15] were analysed in CMAP recordings. The decrease of CMAP amplitude was the result of axonal loss (axonotmesis), whereas an increase in the standardised latency parameter was initiated by the demyelination process (neuropraxia). Twenty-one healthy volunteers (fourteen females and seven males) aged 21 to 51 years (mean age was 28) underwent neurophysiological tests to establish reference values.

The Blink reflex examination (BR) was used as a supplementary method for facial nerve testing. The blink reflex test is an essential element of a comprehensive neurophysiological assessment of the facial nerve peripheral damage with 81% sensitivity and 94% specificity. The improvement in parameters of the recorded potentials
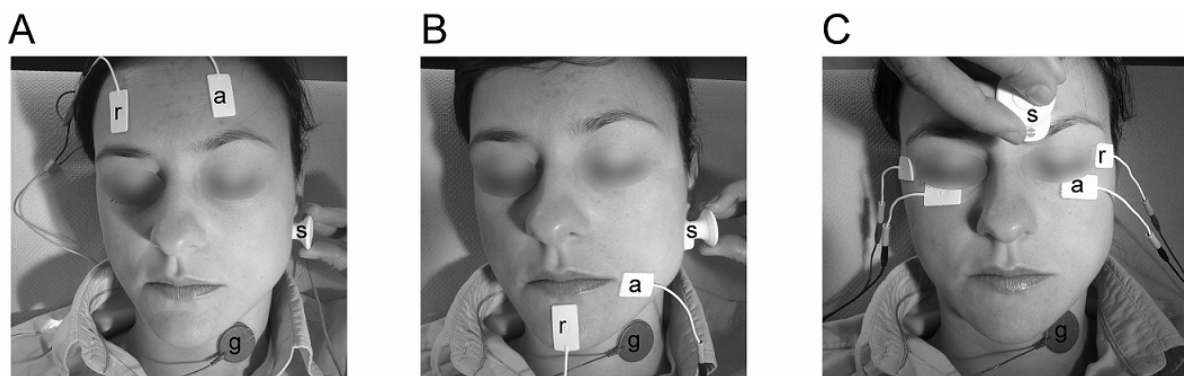

Figure 2. Photographs illustrating principles of electroneurographic studies: location of stimulating (s), ground (g) and recording (a-active, r-reference) electrodes during the examination of neural transmission in motor fibers of the facial nerve within the frontal branch (A), marginal mandibular branch (B) and the blink reflex study (C).

to the reference values suggests that the nerve impulse conduction disorder concerns the myelin sheath of the nerve without signs of axon damage. The blink reflex study is also an indicator of the improvement in the function of the facial nerve fibers. Recording electrodes were placed bilaterally on the orbicularis oculi muscles and a ground electrode on the neck. Electrical stimulus was applied over the supraorbital nerve (Figure 2 C) and required rectangular stimulus at 1 Hz with intensity of 20 mA and 0.5 ms duration. The short-latency ipsilateral R1 response and two long-latency R2 responses were analysed in recordings both ipsi- and contralaterally [11,16].

Statistical calculations for neurophysiological tests were performed using the STATISTICA version 9.1 programme (StatSoft Inc., 2009). The compliance of parameters with the normal distribution was checked by the Shapiro-Wilk test. Descriptive statistics of measurable parameters concerned minimum and maximum values, arithmetic means and standard deviation values. Simple comparison of parameters was used in all observation periods.

Confirmation of axonal changes in the facial nerve required needle EMG examination from the frontal and orbicularis oris muscles [11]. The study included the analysis of the muscle spontaneous activity at rest and evaluation of twenty motor unit action potentials (MUAPs) parameters (amplitude in mV, duration in ms and Size Index in mV/ms) recorded during weak voluntary contraction [15]. Two parameters of MUAPs with values greater than reference values determined the neurogenic advancement of their injury and reinnervation process in examined muscle. The last stage of EMG examination included measurements of amplitude and frequency of motor unit recruitment during maximal voluntary contraction lasting 5 seconds [17]. Conducting the two last stages of EMG examination was possible only in face muscles presenting voluntary activity.

The modified Blair's skin incision was applied, with patients (N = 12) treated surgically by the extracapsular tumour removing technique. General anaesthesia with volatile anaesthetics was applied during the surgery in all patients, with no complications in patients during anaesthesia. Superficial parotidectomy with tumour removal was performed in one patient where the tumour was located in the lower parotid pole in the superficial and deep lobes. The facial nerve was identified and dissected using an anterograde technique due to its location in the parotid gland. Facial nerve branches were located on the surface of tumour and were "peeled off" during the intervention which lasted 180 minutes.

In one patient with the tumour located in both lobes, the pharmacological treatment included injections with Galantamine (5 mg per day, for 30 days), Cocarboxylase (50 mg per day, for 10 days), Dexamethasone (4 mg twice a day, for 10 days) and Triamcinolone (orally 4 mg twice a day, for 3 weeks). The above conservative treatment, as well as physiotherapy, was used because of iatrogenic facial nerve palsy which occurred in this particular patient.

Physiotherapeutic treatment was introduced two days after the surgery, performed twice a day for eight days. Two therapeutic procedures were applied, the Facial-Oral-Tract-Therapy (F.O.T.T.) [18], which stimulated muscle receptors on the paralysed side together with relaxation and stretching, as well as with muscle tension decrease on the asymptomatic side, and Proprioceptive neuromuscular facilitation (PNF) [19], which included activation of facial muscles on both sides to evoke their synergy, similar to the conditions of their normal innervation. Proper face muscle function on the asymptomatic side influences contractile properties of muscles on the paralysed side. The patient continued the exercises at home (2–3 times a day for 10 minutes) using a facial mirror biofeedback technique, focusing on the symmetry of facial movements. Supervised treatment was continued for the next 6 months.

The remaining patients did not receive the treatment, as mentioned earlier, due to the lack of complications after surgery in the form of facial nerve palsy.

Ethical considerations were according to the Helsinki Declaration. The research was approved by the Bioethical Committee of the University of Medical Sciences including studies on healthy people. Each patient was informed about the aim of study and provided written consent for examinations and data publication. Statistical analysis included comparison of mean values and ranges of parameters with reference to the tumour size and analysed results of neurophysiological studies.

Results

In nine out of thirteen patients, the histopathological study revealed pleomorphic adenoma, adenolymphoma (Warthin's tumour) in three patients and myoepithelioma was recognised in the preliminary histological study in one patient. The type of tumour was indicated by the immunohistochemical examination. Tumour was not malignant; its total excision was not confirmed. The anatomical tumour-nerve relation and surgery difficulty were factors determining the iatrogenic facial nerve injury.

In twelve patients, the clinical examination using the H-B scale showed the proper function of the facial nerve bilaterally (H-B I) in all periods of observations. One of the co-existing symptoms was analgesia of auricle; this symptom remitted 6 months after surgery.

The data presented in Table 1 concerns one patient with a tumour located in both lobes of the parotid gland, which shows that before the surgery, despite proper facial nerve function (H-B grade I), the only weakness of the orbicularis oculi muscle was found on the symptomatic side. Xerostomia, as well as numbness and itching of the left cheek, were reported by the patient. Tumour removal led to the total paralysis of left facial nerve fibers (grade VI of H-B scale), which was accompanied by additional symptoms such as hypoesthesia of auricle and numbness. Recovery of the facial nerve function (H-B grades IV, III and III, respectively) was found at the subsequent 3rd–5th periods of observation. Late complications such as Frey's syndrome and oral-ocular synkinesis were detected 3 months after surgery (Table 1). The H-B score was IV, although the improvement of facial nerve function was detected in ENG (Figure 6) and BR examination (Table 3), as well as in the residual bioelectrical activity of mimic muscles (Table 4). Following (4th and 5th) periods of observation, the H-B score was III with further improvement of nerve function and muscle activity. Moreover, contracture of mimic muscles on the symptomatic side was observed in these observation periods. The patient reported regression of auricle hypoesthesia in the 5th period (Table 1).

The results of the ENG and BR examination in twelve patients were within physiological limits (Table 2), with no significant differences between the symptomatic and asymptomatic sides in the four periods of observation.

In the case of one patient, data in Figure 3 shows that the amplitudes of CMAP evoked after stimulation of the frontal branch were comparable to the reference values at all periods of observation. In postoperative studies, CMAP amplitudes recorded after the marginal mandibular branch stimulation were always lower compared to refer-

Table 1. Clinical assessment of facial nerve function on the tumour side according to the H-B scale as well as the coexisting symptoms in one particular patient as an example

Periods of observation	Coexisting symptoms	H-B scale
1st	Slightly weaker eye clinching against the resistance Xerostomia Numbness and itching of the left cheek	I
2nd	Hypoesthesia of the auricle Numbness on the left side of the face	VI
3rd	Hypoesthesia of the auricle Oral-ocular synkinesis Frey's syndrome	IV
4th	Hypoesthesia of the auricle Oral-ocular synkinesis Frey's syndrome Contracture of the facial muscles	III
5th	The correct sense of touch in the auricle Oral-ocular synkinesis Frey's syndrome Contracture of the facial muscles	III

H-B scale: (House-Brackmann scale): *I* normal function, *II* slight dysfunction, *III* moderate dysfunction, *IV* moderately severe dysfunction, *V* severe dysfunction, *VI* total dysfunction. Periods of observation: *1st* before the surgery, *2nd* one month after the surgery, *3rd* three months after the surgery, *4th* six months after the surgery, *5th* seventeen months after the surgery.

ence values. Additionally, the dispersion of CMAP potential duration recorded from the orbicularis oris muscle was noted 6 months after surgery, which coexisted with mimic muscles contracture on the symptomatic side (Table 1). Values of CMAP standardised latencies after stimulation of both nerve branches were higher when compared to the controls, indicating decreasing conduction velocity of the nerve impulses in subsequent postoperative periods. Values of the standardised latency were normal 17 months after surgery only in studies of the frontal branch.

The value of CMAP amplitude evoked after stimulation of the frontal branch on the symp-

Table 2. Summary of normative values (mean and range) for the clinical neurophysiology studies in the control group of healthy subjects (N = 21)

Type of neurophysiological examination	Type of parameters	
	Amplitude (mV)	Standardized latency (ms/cm) or latency (ms)
ENG – CMAP from frontal branch	1.53 mV 0.7–2.3 mV	0.29 ms/cm 0.23–0.35 ms/cm
ENG – CMAP from marginal mandibular branch	4.37 mV 2.2–6.2 mV	0.28 ms/cm 0.24–0.32 ms/cm
BR – R1 response	NA	11.06 ms 9.4–12.56 ms
BR – R2 response	NA	30.05 ms 25–35 ms
BR – R2c response	NA	30.6 ms 25–36 ms

ENG – electroneurography, CMAP - compound muscle action potential, EMG – electromyography, BR– blink reflex, R1 - short-latency ipsilateral response, R2 - long-latency ipsilateral response, R2c - long-latency contralateral response, NA - not analyzed.

Table 3. Latencies of the blink reflex potentials recorded on the side symptomatic side in all periods of observation in one particular patient as an example

Evoked potential latency (ms)	Periods of observation				
	1st	2nd	3rd	4th	5th
R1	9.8	No potential	Residual potential	15.7	9.2
R2	29.2	No potential	43.8	37.7	28.3
R2c	28.2	No potential	45.7	35.5	28.3

Periods of observation: 1st before the surgery, 2nd one month after the surgery, 3rd three months after the surgery, 4th six months after the surgery, 5th seventeen months after the surgery.
R1 and R2 - ipsilaterally recorded potentials, R2c – contralaterally recorded potential.

Table 4. Results of EMG studies recorded from frontal and orbicularis oris muscles in the four periods of observation in one particular patient as an example

	Periods of observation				
	1st	2nd	3rd	4th	5th
Frontal muscle					
Spontaneous activity	Not examined	Numerous F + PSV	F + PSV	Single F + PSV	Single F + PSV
Voluntary activity	Not examined	No Activity	Residual activity	Moderate activity Neurogenic MUAPs	Moderate activity Neurogenic MUAPs
Orbicularis oris muscle					
Spontaneous activity	Not examined	Numerous F + PSV	F + PSV S	Single F + PSV S	Single F + PSV S
Voluntary activity	Not examined	No activity	Weak activity	Moderate activity Neurogenic MUAP MC	Moderate activity Neurogenic MUAP MC

Periods of observation: 1st before the surgery, 2nd one month after the surgery, 3rd three months after the surgery, 4th six months after the surgery, 5th seventeen months after the surgery.
F – fibrillations, PSV – positive sharp waves, S – synkinesis, MC – muscular contracture.

tomatic side was 38% lower compared to the asymptomatic side one month after surgery. Despite the lack of difference in amplitude, above 50% between sides, the result of H-B score (VI), the presence of denervation signs and no voluntary activity in EMG examinations from frontal muscle confirmed an axonal injury within the frontal branch of the facial nerve. Furthermore, values of CMAP amplitudes were comparable to those recorded on the asymptomatic side in subsequent periods of observation. Amplitudes of CMAPs after stimulation of the marginal mandibular branch were lower at 86%, 58%, 47% and 27% in subsequent postoperative observation periods in comparison to the values recorded on the asymptomatic side.

According to Esslen [20], CMAP amplitude value reflects a 1:1 proportion of active axons within the examined nerve branch. In the case of one patient, we ascertained the progression of nerve regeneration based on the results of CMAP amplitude variability (expressed in percentages) after marginal mandibular branch stimulation in subsequent postoperative observation periods compared to the preoperative recording. These values were 14%, 43%, 49% and 68%, indicating the advancement of the regeneration process

which was most prominent at 29% between the first and third month after surgery.

The Blink reflex examination showed proper parameters of evoked potentials only during the preoperative and the final observation periods (17 months after surgery; Table 3, Figure 4 AB1, 4). One month after tumour resection, R1 and R2 ipsilateral evoked potentials were not recorded on the symptomatic side (Figure 4 A2), nor was the R2 contralateral response after stimulation on the asymptomatic side (Figure 4 B2). This reflects the consequences of injury in the efferent pathway of the reflex arc (the facial nerve on the symptomatic side). In addition, low amplitude potentials with prolonged latencies in the BR study were recorded three months after surgery (Figure 4 AB3), indicating the recovery of nerve impulses transmission.

Data presented in Table 4 for a case of one patient proves the recovery of mimic muscles activity, which was observed 3 months after surgery. Residual voluntary activity of the frontal muscle and weak voluntary activity of the orbicularis oris muscle were recorded in the EMG examination. Analysis of MUAP parameters 6-months after surgery showed properties of neurogenic changes secondary to nerve injuries. Positive sharp waves
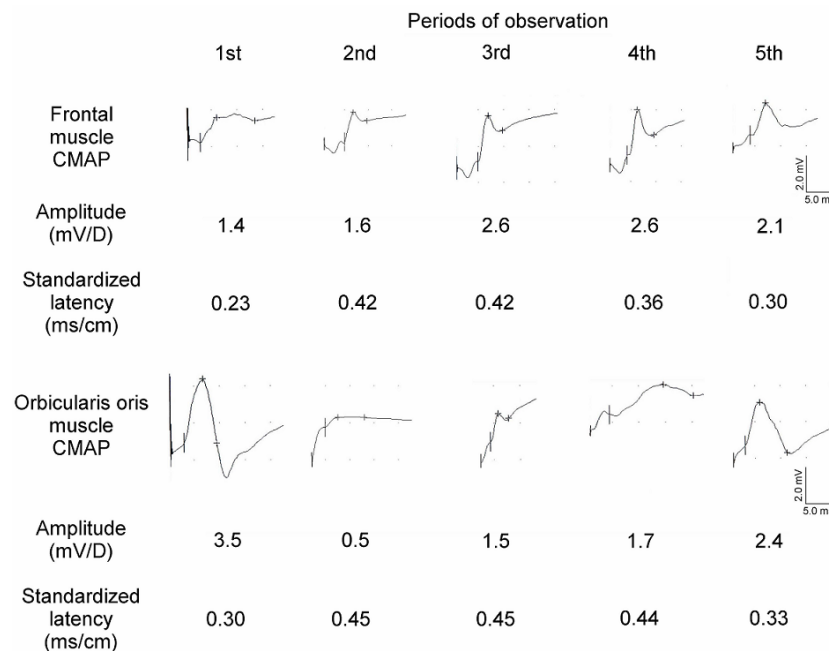

	Periods of observation				
	1st	2nd	3rd	4th	5th
Frontal muscle CMAP					
Amplitude (mV/D)	1.4	1.6	2.6	2.6	2.1
Standardized latency (ms/cm)	0.23	0.42	0.42	0.36	0.30
Orbicularis oris muscle CMAP					
Amplitude (mV/D)	3.5	0.5	1.5	1.7	2.4
Standardized latency (ms/cm)	0.30	0.45	0.45	0.44	0.33

Figure 3. Results of ENG study on the symptomatic side with examples of CMAP recordings from the frontal and orbicularis oris muscles in the five stages of observation. CMAP: compound muscle action potential; Periods of observation: 1st before the surgery, 2nd one month after the surgery, 3rd three months after the surgery, 4th six months after the surgery, 5th seventeen months after the surgery.

and fibrillation potentials indicating the denervation of face muscles were recorded in the remaining postoperative observation periods. This activity was particularly frequent in the 1st and 3rd months after surgery, thus confirming the acute neurogenic injury. Signs of denervation process were weaker in other postoperative observation periods (Figure 5).

Assessment of EMG spontaneous activity 6 and 17 months after surgery was particularly complicated in terms of the orbicularis oris muscle due to the coexistence of oral-ocular synkinesis and facial muscles contracture (Figure 6). The synkinesis on the symptomatic side appeared after 3 months, whereas muscles contracture

was present 6 months after the parotid gland tumour surgery (Table 1).

A decrease of asymmetry between the symptomatic and asymptomatic side was observed in the long-lasting postoperative observation, being visible during the total relaxation of facial muscles. During attempts of voluntary contraction, the asymmetry increased particularly in the lower part of face. Asymmetry was deepened by the muscle contracture on the symptomatic side six months after surgery. Weak voluntary activity and the clearly visible narrowing of the eyelid fissure during activation of orbicularis oris muscle (oral-ocular synkinesis) were observed.
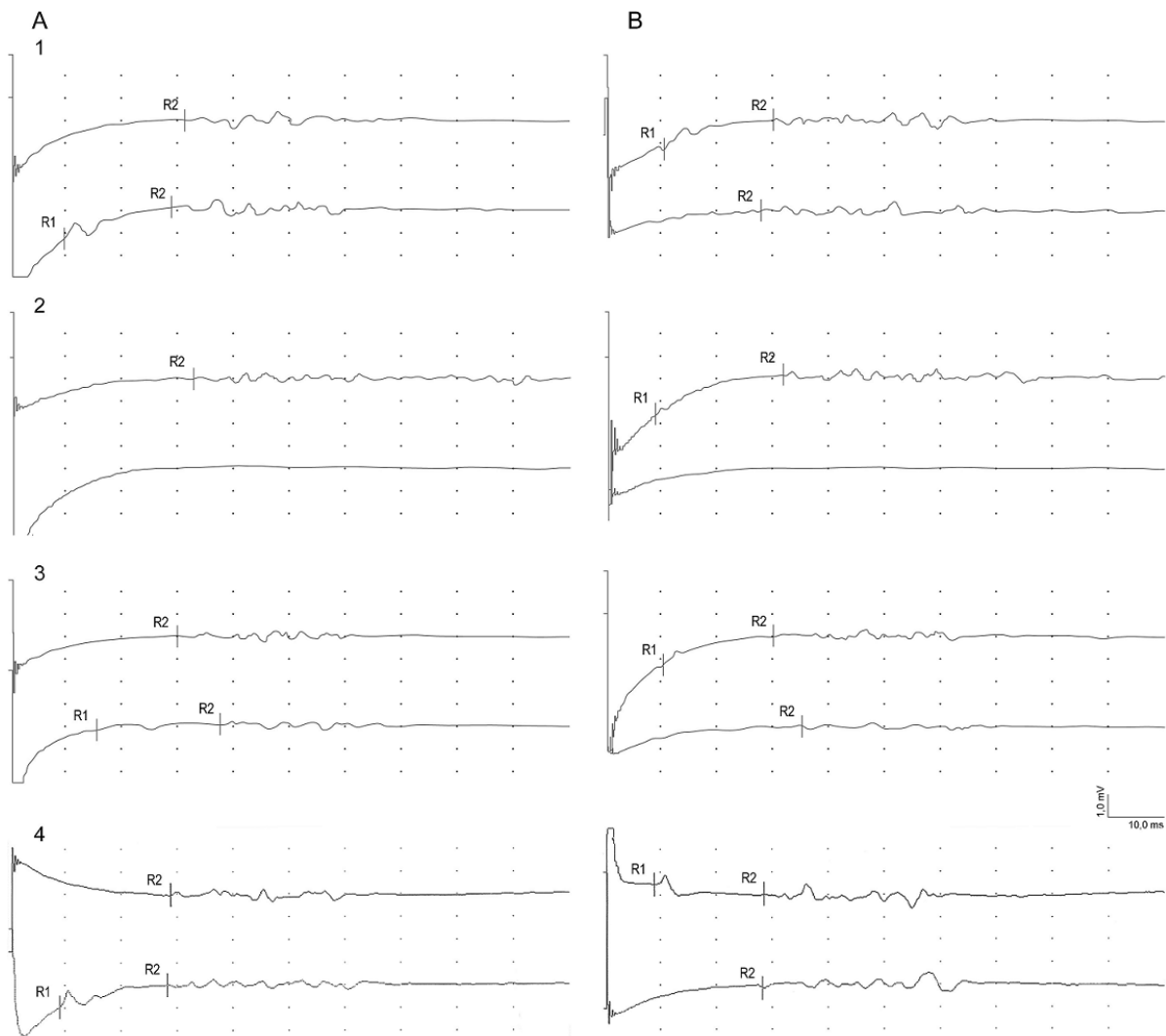

Figure 4. Examples of blink reflex studies in the three stages of observation: before surgery (1), one month after surgery (2), three months after surgery (3) and seventeen months after surgery (4) recorded on the symptomatic (A) and asymptomatic side (B)
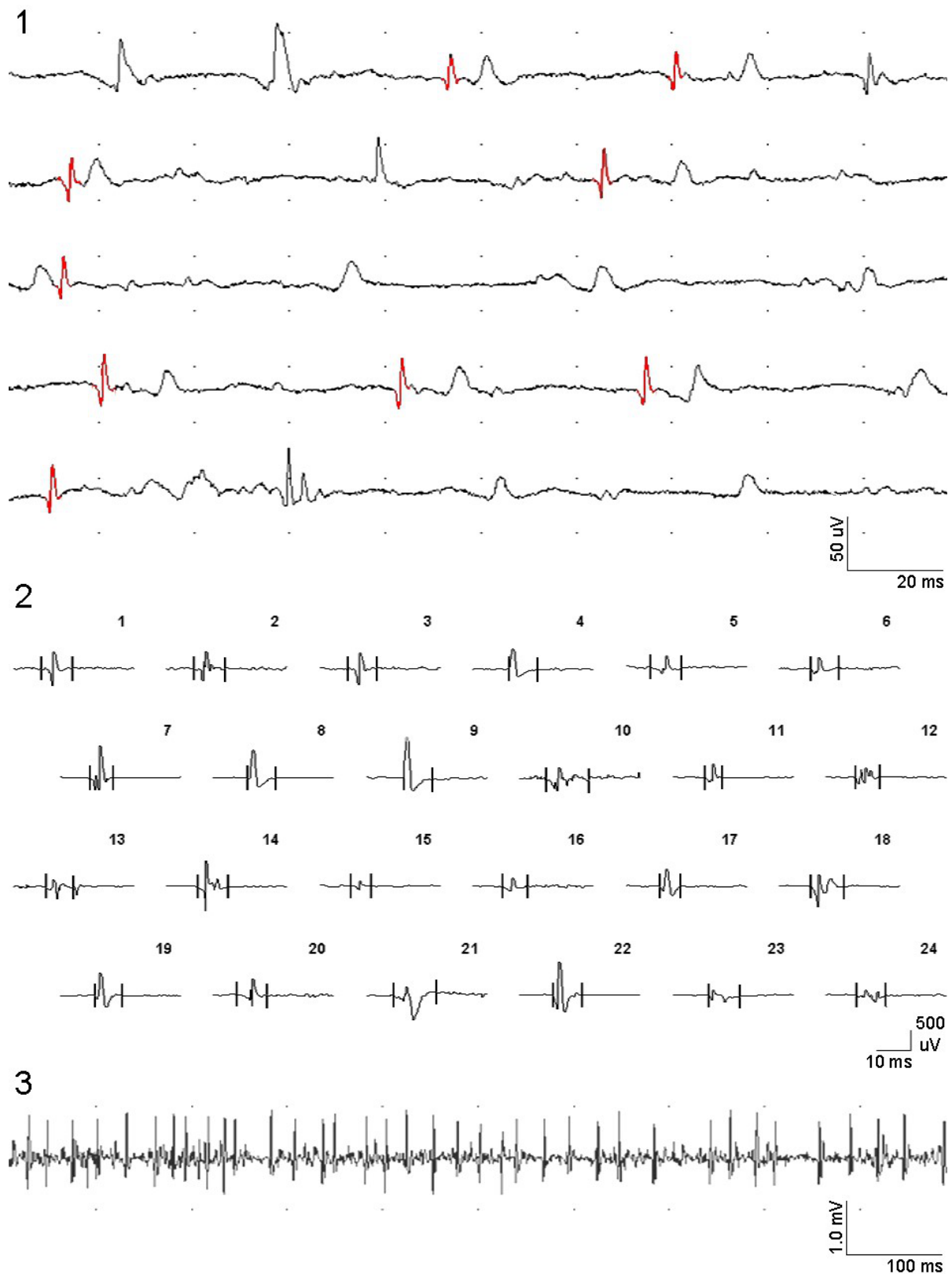
Figure 5. Examples of recordings in the EMG study from the orbicularis oris muscle 6 months after the parotid gland tumour removal showing the spontaneous denervation activity at rest with fibrillations marked in red (1), motor unit action potentials (MUAP) during attempts of voluntary contraction (2) and frequency pattern of MUAPs discharges during the maximal contraction (3). Note the increase in duration in single MUAPs (2) and frequency decrease of MUAPs recruitment (3) confirming the advancement of neurogenic pathology.
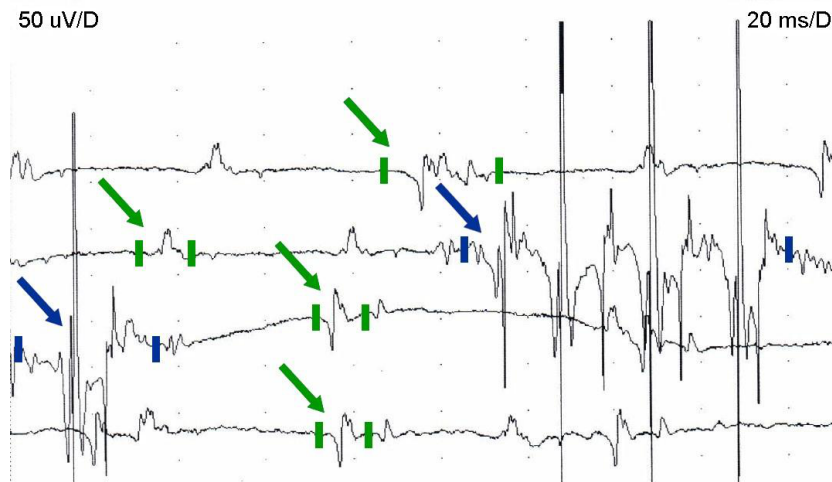
Figure 6. Example of the spontaneous activity recorded from the orbicularis oris muscle at rest. Muscle spasms associated with the presence of oral-ocular synkinesis (marked with the blue arrows) and single MUAP discharges resulting from orbicularis oris contractures (indicated by the green arrows) are presented.

Discussion

Differentiation of the facial nerve injury type presented in this study based only on ENG results is difficult. CMAP amplitude may be reduced in the case of axonotmesis and conduction block. The latter refers to neuropraxia with decreasing axonal conductive properties at the site and above compression. Possibilities of regeneration differ in these types of injuries, according to Hughes et al. [21], 50% reduction of CMAP amplitude recorded proximally to the site of nerve block confirms this phenomenon. If the distal point of facial nerve stimulation overlaps with the site of parotid gland tumour surgery, ascertaining the conduction block is not possible. In our study, the analysis of facial nerve regeneration progress with its clear exponent of CMAP amplitude increase in the period between the 1st and the 3rd month suggests that the most probable mechanism of nerve injury was coexistence of conduction block and mild axonotmesis. Diminished conduction block in the 3rd month after surgery resulted in a significant increase of CMAP amplitude which, in the case of frontal branch examination, reached a value comparable to normal. The increase in CMAP amplitude recorded from the marginal mandibular branch in all periods of observation was also recorded, but this value has never reached the reference, confirming mild axonotmesis. The advancement in facial nerve regeneration was also observed in the results of the blink reflex examination, validated by recordings of R1 and R2 responses on the symptomatic side 3 months after surgery.

The rate and dynamics of changes recorded in the frontal and orbicularis oris muscles during EMG studies also indicate the possibility of coexistence of both axonotmesis and neuropraxia. The spontaneous activity at rest showing denervation and lack of muscle voluntary activity was observed 1 month after surgery. The appearance of voluntary activity of face muscles, which was the symptom of nerve regeneration with still existing denervation, were recorded 3 months after surgery. Similar to observations of Martin and Helsper [22], in our study, the first symptoms of reinnervation were better expressed in muscles located closer to the injured area. At the same time, oral-ocular synkinesis was also observed in EMG, suggesting an axonotmesis mechanism of nerve injury and reflects the abnormal proliferation of axons during regeneration. Considering the location of the tumour in the parotid gland and possible mechanical and thermal reasons of injury during surgery, it is likely that axonotmesis involved the marginal mandibular branch to a greater extent than the whole nerve trunk. This can be validated by a better function of the frontal than marginal mandibular branch ascertained in the neurophysiological examination. The marginal mandibular branch is located nearest to the area of surgery, hence it may be exposed to more damage than another branch of the facial nerve.

Moreover, the marginal mandibular branch has the least anatomical connections between other branches of the facial nerve, so the regenerative capacity may be limited. Similar results pointing to the iatrogenic injury of the marginal mandibular branch during parotid gland tumour surgery were described in other studies [2,23].

The results presented in this study and findings of Reddy et al. [24] indicate the importance of physiotherapeutic treatment introduced immediately after surgery for creation of the appropriate conditions for facial nerve regeneration and muscle atrophy prevention. Spontaneous regeneration is successful when nerve branches undergo intensive afferent and efferent functional stimulation, consequently accelerating neurogenesis and activating the cortical centres in a feedback way. Exercises to activate face muscles promote regenerating axonal endings to create new connections at neuromuscular junction [25].

The study findings also suggest that precautions undertaken during surgery, including intraoperative monitoring of facial nerve function, and application of appropriate conservative treatment are essential for the final nerve regeneration. Neurophysiological examination performed 1, 3, 6 and 17 months after surgery allowed for objective determination of the facial nerve injury type, the progress of its regeneration and face muscles reinnervation. Comparative studies showed the predominant mild axonotmesis, especially with reference to the marginal mandibular branch. This type of injury was also confirmed by detecting post-regenerative deficits previously mentioned by other authors [26-29]. In this study, complications after surgical removal of the parotid gland tumour affected only one patient. The rarity of this phenomenon in the case of benign parotid tumours prompted the authors to present this case and propose optimal therapeutic and diagnostic processes.

In the case of axsonotmesis, spontaneous regeneration of the nerve may be incomplete, therefore, a detailed neurophysiological diagnosis is non-invasive and a valuable diagnostic and prognostic tool for assessing facial nerve function.

Acknowledgements

References

1. Aimoni C, Lombardi L, Gastaldo E, Stacchini M, Pastore A. Preoperative and Postoperative Electroneurographic Facial Nerve Monitoring in Patients With Parotid Tumors. Archives of Otolaryngology–Head & Neck Surgery. 2003 Sep 1;129(9):940. https://doi.org/10.1001/archotol.129.9.940
2. Wiertel-Krawczuk A, Huber J, Wojtysiak M, Golusiński W, Pieńkowski P, Golusiński P. Correlations between the clinical, histological and neurophysiological examinations in patients before and after parotid gland tumor surgery: verification of facial nerve transmission. European Archives of Oto-Rhino-Laryngology. 2014 Apr 17;272(5):1219-1229. https://doi.org/10.1007/s00405-014-3032-4
3. Koide C, Imai A, Nagaba A, Takahashi T. Pathological Findings of the Facial Nerve in a Case of Facial Nerve Palsy Associated With Benign Parotid Tumor. Archives of Otolaryngology - Head and Neck Surgery. 1994 Apr 1;120(4):410-412. https://doi.org/10.1001/archotol.1994.01880280038006
4. Marchesi M, Biffoni M, Trinchi S, Turriziani V, Campana FP. Facial Nerve Function After Parotidectomy for Neoplasms with Deep Localization. Surgery Today. 2006 Mar 24;36(4):308-311. https://doi.org/10.1007/s00595-005-3146-9
5. Ozgur A, Semai B, Hidir UU, Mehmet Fatih O, Tayfun K, Zeki O. Which electrophysiological measure is appropriate in predicting prognosis of facial paralysis?. Clinical Neurology and Neurosurgery. 2010 Dec;112(10):844-848. https://doi.org/10.1016/j.clineuro.2010.07.001
6. Elmadawy A, Hegab A, Alahmady H, Shuman M. Clinical and electromyographic assessment of facial nerve function after temporomandibular joint surgery. International Journal of Oral and Maxillofacial Surgery. 2015 Oct;44(10):1275-1280. https://doi.org/10.1016/j.ijom.2015.04.013
7. Martins RS, Bastos D, Siqueira MG, Heise CO, Teixeira MJ. Traumatic injuries of peripheral nerves: a review with emphasis on surgical indication. Arquivos de Neuro-Psiquiatria. 2013 Oct;71(10):811-814. https://doi.org/10.1590/0004-282x20130127
8. Terzis JK, Karypidis D. Therapeutic Strategies in Post–Facial Paralysis Synkinesis in Adult Patients. Plastic and Reconstructive Surgery. 2012 Jun;129(6):925e-939e. https://doi.org/10.1097/prs.0b013e318230e758
9. Campbell WW. Evaluation and management of peripheral nerve injury. Clinical Neurophysiology. 2008 Sep;119(9):1951-1965. https://doi.org/10.1016/j.clinph.2008.03.018
10. House JW. Facial nerve grading systems. The Laryngoscope. 1983 Aug;93(8):1056-1069. https://doi.org/10.1288/00005537-198308000-00016
11. Lee HJ, DeLisa JA. Manual of nerve conduction study and surface anatomy for needle electromyography. Philadelphia: Lippincott Williams & Wilkins; 2005.

12. Di Bella P, Logullo F, Lagalla G, Sirolla C, Provinciali L. Reproducibility of normal facial motor nerve conduction studies and their relevance in the electrophysiological assessment of peripheral facial paralysis. Neurophysiologie Clinique/Clinical Neurophysiology. 1997 Sep;27(4):300-308. https://doi.org/10.1016/s0987-7053(97)85828-5

13. Kimura J. Electrodiagnosis in Diseases of Nerve and Muscle: Principles and Practice (4 ed.). Oxford University Press; 2001. https://doi.org/10.1093/med/9780199738687.001.0001

14. Preston D, Shapiro B. Electromyography and neuromuscular disorders. Clinical-electrophysiologic correlations. 2nd Ed. Elsevier, Butterworth & Heinemann; 2005.

15. Rubin DI, Daube JR. Clinical neurophysiology. Oxford University Press; 2009.

16. Brown W, Bolton C, Aminoff M. Neuromuscular function and disease. Basic, clinical and electrodiagnostic aspects. Vol. 1. Philadelphia: W.B. Saunders; 2002.

17. Stlberg E, Falck B. The role of electromyography in neurology. Electroencephalography and Clinical Neurophysiology. 1997 Dec;103(6):579-598. https://doi.org/10.1016/s0013-4694(97)00138-7

18. Nusser-Müller-Busch R. Die Therapie des Facio-Oralen Trakts. F.O.T.T. nach Kay Coombes. Heildelberg: Springer Medizin Verlag; 2011.

19. Adler SS, Beckers D, Buck M. PNF in Practice. Heidelberg: Springer Medizin Verlag; 2008.

20. Esslen E. The Acute Facial Palsies. Investigations on the Localization and Pathogenesis of Meato-Labyrinthine Facial Palsies. Berlin: Springer; 1977.

21. Hughes GB, warnock GR, glasscock ME, jackson CG, ray WA, sismanis A. Clinical electroneurography. The Laryngoscope. 1981 Nov;91(11):1834-1846. https://doi.org/10.1288/00005537-198111000-00007

22. Martin H, helsper JT. Spontaneous Return of Function Following Surgical Section or Excision of the Seventh Cranial Nerve in the Surgery of Parotid Tumors. Annals of Surgery. 1957 Nov;146(5):715-727. https://doi.org/10.1097/00000658-195711000-00001

23. Bendet E, Talmi YP, Kronenberg J. Preoperative electroneurography (ENoG) in parotid surgery: Assessment of facial nerve outcome and involvement by tumor—A preliminary study. Head & Neck. 1998 03;20(2):124-131. https://doi.org/10.1002/(sici)1097-0347(199803)20:2<124::aid-hed5>3.0.co;2-4

24. Reddy PG, Arden RL, Mathog RH. Facial nerve rehabilitation after radical parotidectomy. The Laryngoscope. 1999 06;109(6):894-899. https://doi.org/10.1097/00005537-199906000-00010

25. Shafshak T. The Treatment of Facial Palsy From the Point of View of Physical and Rehabilitation Medicine. Europa Medicophysica. 2006;42(1):41-7. PMID 16565685

26. Dulguerov P, Quinodoz D, Cosendai G, Piletta P, Marchal F, Lehmann W. Prevention of Frey Syndrome During Parotidectomy. Archives of Otolaryngology–Head & Neck Surgery. 1999 Aug 1;125(8):833. https://doi.org/10.1001/archotol.125.8.833

27. Marchese-Ragona R, De Filippis C, Marioni G, Staffieri A. Treatment of Complications of Parotid Gland Surgery. Acta Otorhinolaryngol Ital. 2005 Jun;25(3):174-8. PMID 16450773

28. Pourmomeny AA, Asadi S. Management of synkinesis and asymmetry in facial nerve palsy: a review article. Iran J Otorhinolaryngol. 2014 Oct;26(77):251-6. PMID 25320703

29. Peitersen E. Bell's Palsy: The Spontaneous Course of 2,500 Peripheral Facial Nerve Palsies of Different Etiologies. Acta Oto-laryngologica. Supplementum. 2002;(549):4-30. PMID 12482166