Masseter muscle function after percutaneous balloon compression of trigeminal ganglion for the treatment of trigeminal neuralgia: A neurophysiological follow-up study

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A B S T R A C T

Objective: We aimed to evaluate by longitudinal neurophysiological examinations the natural course of masseter muscle weakness that developed after percutaneous balloon compression (PBC) of the trigeminal ganglion for the treatment of idiopathic trigeminal neuralgia.

Methods: The affected side of 15 patients (mean age 69.5 ± 4.5 years) who underwent unilateral PBC were studied before, 1 month, 6 months and 12 months after surgery by means of: (1) motor evoked potentials (MEPs) of the masseter muscle elicited by transcranial magnetic stimulation of the contralateral motor cortex and of the ipsilateral trigeminal motor branch; and (2) concentric needle electromyography of masseter muscle.

Results: The latencies of MEPs to cortical and nerve stimulation became significantly prolonged 1 month after PBC, whereas, thereafter, they demonstrated a gradual shortening towards preoperative values. The interference electromyographic pattern 1 month post-PBC study was reduced in all patients, but it improved in follow-up, returning 12-months postoperatively to complete in 13 and nearly complete in two patients.

Conclusion: Masseter muscle weakness should be expected in all cases after PBC of the trigeminal ganglion.

Significance: As verified by repeated studies, the results of which favored a focal demyelination process of trigeminal motor branch, muscle dysfunction appears to be reversible over a period of 6–12 months.

1. Introduction

When conservative means fail to confront paroxysmal pain of trigeminal neuralgia or patients cannot tolerate long-term polypharmacy and adverse reactions of drugs, neurosurgical intervention is required to release the pressure on the nerve from a vascular loop or simply to interrupt sensory inputs by ablative procedures at the trigeminal ganglion. A large variety of neurosurgical techniques, more or less invasive, and their modifications have been proposed and tested aiming to determine the most effective one with the lowest morbidity and recurrence rate (Lopez et al., 2004; Jellish et al., 2008).

Based on the concept that a restricted mechanical injury of the trigeminal ganglion is expected to suppress the transition ofafferent (sensory) signals, including painful ones, from periphery to brain centres, several interventional techniques have been explored. Percutaneous balloon compression (PBC), is a well-established method which, like other percutaneous techniques, is recommended for elderly patients who have excessive surgical risk or are not willing to undergo microvascular decompression (Toda, 2008). PBC offers initial pain relief at a rate similar to that of percutaneous radio-frequency thermocoagulation but with lower incidence of disturbing anaesthesia dolorosa and vision-threatening corneal keratitis (Brown and Pilitsis, 2005). However, amongst the percutaneous procedures, PBC has the highest rate of masseter muscle weakness (Omeis et al., 2008; Tatli et al., 2008), which implies that motor fibres carried by the trigeminal nerve are particularly vulnerable to this technique. The exclusive aim of the present study is to investigate by serial neurophysiological examinations the type and severity of the trigeminal nerve motor-branch injury and follow-up the natural course of the corresponding masseter
weakness associated with PBC of the trigeminal ganglion in patients with idiopathic trigeminal neuralgia.

2. Methods

2.1. Subjects

Fifteen patients (10 males and five females; aged 65–80 years, mean age 69.5 ± 4.5 years) fulfilling the criteria for idiopathic trigeminal neuralgia (Nurmikko and Eldridge, 2001) were recruited to the study with their informed consent. They were derived from a larger group of 70 patients with idiopathic or secondary trigeminal neuralgia refractory to medication or recurrent after failure of a surgical procedure, who was referred for neurosurgical consultation in our Institution between 2007 and 2009. On the whole, 20 patients underwent microvascular decompression, of whom most elderly and high-risk patients were operated upon using a percutaneous procedure (18 glycerol rhizotomy and 27 PBC); in the remaining five patients, surgical options were rejected. The present study was approved by the University Hospital of Patras ethics committee and the procedures were in accordance with the Declaration of Helsinki. Seven patients experienced pain on their right side; in 12 patients a single trigeminal division was affected (eight patients had V3 and four patients had V2), whereas more than one division was affected in the remaining three patients (V1 and V2 in one case, V2 and V3 in two cases). All patients received neurological examination and brain magnetic resonance imaging preoperatively, to exclude cases with symptomatic neuralgia. Surgical option was offered only to those patients with pain refractory to medication or with drug-related severe adverse reactions.

2.2. Experimental procedure

All baseline electrophysiological studies took place during the morning prior to surgery and included: (1) conduction time to transcranial magnetic stimulation (TMS) of the ipsilateral (affected) trigeminal nerve and conduction time to TMS of the contralateral motor cortex; and (2) electromyographic (EMG) examination of the masseter muscle. Each participant was comfortably seated with his/her head and neck supported. TMS was applied using Magstim 200 stimulator (The Magstim Company, Whitland, Dyfed, UK) equipped with a circular coil of 90-mm diameter, a 2.0 T maximum magnetic field strength and a stimulus frequency of 0.2–0.3 Hz. The magnetic field direction in the coil was counter-clockwise when viewed from above (side A visible) to preferentially stimulate the left hemisphere and clockwise (side B visible) to activate the right hemisphere. Motor evoked potentials (MEPs) were recorded from the masseter muscle of the affected side with bipolar surface electrodes firmly attached over the muscle belly (active electrode) and at the mandibular angle (reference electrode) on a Dandec Keypoint EMG apparatus (Medtronic-Dantec Electronics, Skovlunde, Denmark) with a filter band-pass of 10 Hz–2 kHz. Based on previously described methods (McMillan et al., 1998; Guggisberg et al., 2001), to define the optimal stimulation position (hot spot) for masseter muscle activation, the coil was placed over the contralateral cortex, approximately 6–7 cm lateral and 2–4 cm anterior to the vertex (Cz) with the handle pointing backwards and 45° laterally from the midline line, and it was sifted to lateral and anterior–posterior directions by 1-cm steps until the largest MEP from the resting masseter was obtained. Stimulus intensity was initially set at 70% of maximum output and once the hot spot was determined, and marked with red ink, the intensity was further increased by 2% steps up to 90% to ensure supramaximal stimulation. Subsequently, five consecutive stimuli were delivered with the coil holding at the hot spot and the evoked MEPs were stored for off-line analysis. Similarly, for TMS of the ipsilateral trigeminal nerve, the hot spot and the appropriate stimulus intensity (ranging from 50% to 80% of maximum output) were defined prior to the recording of five consecutive MEPS. The optimal site for stimulation of the trigeminal motor branch was found approximately 8–9 cm lateral to the vertex and 1 cm anterior to the biauricular line (Cruccu et al., 1989). Finally, EMG examination of the masseter was performed using a concentric bipolar needle to search for spontaneous activity and visually assess the interference pattern during maximal voluntary effort. Throughout the study, muscle condition (resting or contraction) was ensured by audiovisual EMG feedback.

2.3. Operative technique and post-operation follow-up

The procedure of PBC has been previously described (Constantoyannis et al., 2008). In brief, unilateral PBC was performed under general anaesthesia employing a 14-gauge needle with stylet, which was inserted approximately 2 cm lateral to the angle of the mouth, advanced parallel to the sagittal plane until it was entered into the foramen ovale and was targetting, under fluoroscopy, the junction point of the clivus and petrous apex lines. A No. 4 Fogarty catheter was introduced through the needle and the balloon of the catheter was then inflated with 0.75 ml of contrast medium, while the inflation and the shape of the balloon (elliptical or pear shape) are continuously monitored radiologically (Fig. 1). After 2–5 min of constant pressure application, the balloon was deflated and withdrawn together with the needle.

The patients were discharged the day after the operation and were regularly evaluated for acute pain relief, surgery-related complications and pain recurrence every 3–6 months. In addition, for the purposes of the present study, the above-described TMS stimulation procedures and EMG recordings were repeated at intervals of 1 month, 6 months and 12 months post-PBC. To ensure that the hot spot on the scalp for TMS stimulation remained the same in all the four examinations of each subject, records were kept for the exact positions in reference to the 10–20 international system (Rossini et al., 1994). Moreover, in every post-PBC visit, medical records for the degree and persistence of pain relief and the possible surgical complications or side effects, including clinical manifestation of masseter muscle weakness, were maintained.

Fig. 1. Schematic diagram of the surgical procedure of percutaneous balloon compression showing the catheter direction and the balloon shape and position within the trigeminal ganglion.
2.4. Statistical analysis

MEP latency was measured from stimulus artefact to onset of negative peak and, in each set of five responses, the shortest MEP latency was selected for statistical analysis. The repeated-measures analysis of variance (ANOVA) test was used for comparison of MEP latency data across the pre-PBC, 1 month, 6 months and 12 months post-PBC groups, followed by the Levene test to examine the equality of group variances and Tukey post hoc test for pairwise multiple comparisons. Level of significance was set at 5%. All analysis was completed using SPSS version 15 (SPSS Inc., Chicago, IL, USA).

3. Results

PBC was successfully performed in all patients leading to pain relief within 24 h after surgery. Apart from mild facial numbness over all branches of the trigeminal nerve, no major side effects or complications such as corneal anaesthesia, keratitis and anaesthesia dolorosa, were observed in any of the patients. Eleven patients remained pain free until the end of the 12-month follow-up period, three patients required, after the first 6 months, the addition of oral medication to achieve complete pain relief, whereas one patient experienced 8-months’ post-PBC recurrent pain refractory to medication.

Reproducible MEPs were obtained from every subject in the pre-PBC and the three post-PBC examinations. The proper adjustment of coil position enabled the production of potentials with clear-cut latency onset in all cases, although accurate measurement of amplitude was not always achieved due to magnetically induced artefacts (Fig. 2). At the 1-month post-PBC examination, MEPs latencies to cortical (contralateral) and neural (ipsilateral) stimulation were prolonged, being significantly different from the pre-PBC values as well as from the 6-month and 12-month post-PBC values (Fig. 3). Thereafter, a continuous decrease of values towards normal was observed. For each person and session, the MEPs latency difference (cortex–nerve latency) was calculated. Mean latency differences were not significantly changed across the follow-up examinations (mean pre-PBC ± SD 1.6 ± 0.6; 1-month 1.7 ± 0.8; 6-months 1.6 ± 0.6; 12-months post-PBC 1.7 ± 0.6; \( p > 0.3 \)), suggesting a parallel increase of cortical and neural latencies at the first post-PBC examination and, thereafter, a progressive decrease towards the preoperative values.

EMG showed absence of denervation potentials (fibrillations and positive sharp waves) in all patients and sessions. At the pre-PBC study, normal recruitment of motor unit potentials and full interference pattern (100%) on maximal voluntary contraction was shown in all individuals. At the 1-month post-PBC session, incomplete interference was shown in all patients (nine had a discrete pattern <20% of normal, five had 30–70% and one had 80%). At the 6-month post-PBC session the pattern, was moderately reduced (50–80%) in 10 patients, whereas, in five patients it returned to normal (100%). At the 12-month post-PBC session, the recruitment of motor units was much improved, with 80% interference pattern in two patients and 100% in 13 patients. On questioning, 10 patients, including those nine with a discrete EMG pattern, admitted difficulty in chewing on the affected side 1-month post-PBC and in five of them, noticeable masseter weakness persisted until the 6-month follow-up.

4. Discussion and conclusion

Motor fibres, which originate from the trigeminal motor nucleus in the mid-pons, on their way to the masseter and the other mastication muscles, pass beneath the trigeminal ganglion and unite with the sensory mandibular division just after its exit through the foramen ovale. Therefore, it is conceivable that the motor branch could be compressed together with the sensory ganglion, resulting in variable degree of neurogenic dysfunction of the corresponding masseter muscle. In the present study, the significantly delayed MEPs latencies in the first post-PBC examination as compared with the baseline (pre-PBC) ones indicated slowing of motor conduction along the examined neural paths, that is, cortex-to-masseter muscle and trigeminal motor branch-to-muscle. The fact that MEP latencies to nerve and cortex stimulation demonstrated a similar numeric increase suggesting that the assumed delay took place at a site common for both paths and, obviously, this was the trigeminal ganglion and it was caused by the PBC procedure. Absent MEP was not observed in any of the cases, suggesting that complete motor conduction block did not occur. Thereafter, the nerve conduction test, repeated twice for each patient, showed a parallel and progressive decrease of latencies to nerve and cortex stimulation revealing the benign, demyelinating
nature of the pathophysiological process. Finally, the absence of denervation potentials taken together with the time pattern of recovery (6–12 months) and the degree of recovery (full) as shown in the EMG examinations implied that loss of trigeminal motor axon continuity (axonal damage) did not contribute to masseter muscle dysfunction. Therefore, the possibility of a direct trauma to the main motor tract or to the isolated masseteric nerve by the penetrated needle at the level of the foramen ovale (Alvernia et al., 2010), leading to axonotmesis and, consequently, incomplete recovery is not supported by our findings. The low incidence of corneal keratitis observed in PBC as opposed to the other precutaneous procedures is not surprising because the corneal reflex is served by small-diameter unmyelinated fibres (Brown and Pilitsis, 2005), which, unlike motor fibres, are less vulnerable to pressure. Unfortunately, the reversible nature of the demyelinating injury caused by balloon compression could also explain the recurrence of pain several months later, following nerve repair.

Our findings suggest that all patients undergoing PBC should expect to manifest mild or more severe masseter muscle weakness. The impressive percentage of this particular complication herein is in line with literature data. Masseter muscle weakness is described as a common PBC-related consequence with an incidence reaching 66% or even 100% of the cases in different series (Brown and Pilitsis, 2005; Tatli et al., 2008). With respect to the final outcome, a single report has shown permanent mastication weakness in 4% of 50 patients undergoing PBC for idiopathic trigeminal neuralgia (Park et al., 2008), whereas another study described mild weakness in 24% of 56 patients that resolved within 12 months (Brown and Pilitsis, 2005). Neurophysiological follow-up studies of the trigeminal motor branch in the context of the PBC procedure have never been performed before.

According to the classical view regarding cortical representation of the masseter muscle, the trigeminal motor nucleus is connected via corticobulbar fibres with both hemispheres. However, a recent, TMS-based study demonstrated that the majority of low-threshold motor neurons receives excitatory projections from the contralateral hemisphere alone (Pearce et al., 2003). Therefore, we only applied cortical TMS on the side opposite to the trigeminal neuralgia. In our patients, the baseline MEP latency measurements were not far from the lower reported values: the mean latency to cortical stimulation was 4.6 ms as compared with literature values ranging from 5.1 to 6.2 ms (Guggisberg et al., 2001), and the mean latency to direct trigeminal nerve stimulation was 3.0 ms as compared with the previously reported value of 3.5 ms (McMillan et al., 2001). This latency deviation could be attributed to methodological differences, particularly the use of a round coil in the present study instead of a figure-of-eight coil that was employed in the other studies.

The amplitude of MEPs was not measured for three reasons: (1) the great variability that characterises this parameter in a series of consecutive responses even when healthy individuals are examined (Zanette et al., 1995), makes it unsuitable for longitudinal studies; (2) the amplitude of MEPs elicited by cortical stimulation depends on the synchronisation of descending impulses via a multisynaptic motor pathway and therefore, could not be directly compared to that of MEPs elicited by immediate, no-synapses-involved stimulation of the lower motor neurons axons; and (3) a hypothetical amplitude reduction alone could not distinguish neuromapraxia from axonotmesis. The next step of our investigation would be to examine the relationship between the severity of masseter weakness and operational parameters, such as the duration of pressure and balloon shape. Such statistical estimation was not attempted in the present study, because, for the purpose of multivariable analysis, the sample size was not large enough to allow solid conclusions to be drawn. The methodology used in the present study although is simple, painless and applicable by standard EMG apparatuses, it has a limitation. As shown in Fig. 2, the accurate determination of latency onset and amplitude measurements of the MEPs recordings from facial muscles could be sometimes difficult due to the magnetic field–related artefacts.

No neurosurgical approach for the management of trigeminal neuralgia is free from risk or limitations. Choosing the most suitable one on an individual basis would depend not only on the incidence of pain relief but also on the frequency and severity of potential complications inherent in each particular technique. The neurophysiological findings presented herein confirmed that masseter muscle weakness should be expected in all patients after the PBC procedure. However, this complication is not a major drawback, at least under the described conditions and surgical parameters, since the trigeminal motor nerve suffered a demyelinating injury associated with temporary masseter weakness followed by full recovery of muscle strength 6–12 months postoperatively.

References