Trigeminal evoked potentials and sensory deficits in atypical facial pain - a comparison with results in trigeminal neuralgia

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Accepted for publication: July 24, 2002

Summary

Trigeminal evoked potentials (TEPs) and sensory deficits in eighty-three patients admitted for first surgical treatment of facial pain were retrospectively analysed. Thirty-seven patients suffered from trigeminal neuralgia (TN), 10 from symptomatic TN (sTN), and 36 from atypical facial pain (AFP). Eighteen percent of the TN patients reported sensory deficits on the pain side, but 35% had delayed ipsilateral N13 waves. Of the sTN patients, 60% had either sensory deficits or a pathological corneal reflex and 62.5% a pathological N13. Of the AFP patients, 61% complained of sensory deficits, but only 31% had a pathological N13. The percentage of pathological P19 waves was slightly lower (20%, 50%, and 11%, respectively), but showed a similar trend. Normal TEPs were found even in the presence of a sensory deficit (reported only in the AFP group). These findings may add weight to the hypothesis of underlying psychiatric disorders in AFP.

KEY WORDS: Atypical facial pain, sensory evoked potentials, trigeminal nerve, trigeminal neuralgia.

Introduction

Unlike trigeminal neuralgia (TN), which is classified as a clinical entity with a relatively uniform symptomatology and absence of neurological deficits (1), the definition of "atypical facial pain" (AFP), or "atypical facial neuralgia" (2) is still controversial. In this dishomogeneous pain syndrome, the character of the pain is often burning, severe, and unresponsive to analgesic medication (3). Patients report duration of 2 to 3 hours of mostly unilateral pain. The majority of patients report trigger mechanisms like chewing, head movements and cold stimuli. The maxillary nerve is involved in 76% of patients (3). Patients are predominantly female (69% to 88%), normally over the age of 50 years (3,4). Most patients report facial sensory deficits: numbness in 31%, dys- or paraesthesia in 63% (3,4). Radiological examinations, including magnetic resonance imaging, are normal. Minor surgical or dental interventions or a trauma without nerve affection often precede the clinical symptoms (3-5). Some authors stress the importance of an underlying psychopathology (6-8). In contrast to TN, the pharmacotherapy of AFP should start with antidepressants (3). However, the rate of pain-free or improved patients when pharmacotherapy is undertaken is much lower in AFP than in TN. There is strong evidence that surgical therapy should be avoided in AFP (2,4,9,10). Thus, its differential diagnosis vs TN is important.

Somatosensory evoked potentials following stimulation of the trigeminal nerve (TEPs) have proven to be an objective, non-invasive measurement of facial proprioception. Even subclinical impairment of sensory function can be detected and quantified. Normal values have been established (11).

Because studies of TEPs in patients with AFP are scarce and fail to produce uniform findings (12,13), this retrospective study was carried out to evaluate our findings in patients with TN versus AFP, with respect to clinical symptomatology.

Materials and methods

In a retrospective study, the records of patients admitted to our department for treatment of TN were reviewed, taking into consideration clinical findings and the results of TEP and neuroradiological investigations. The diagnosis of either TN or AFP was based on clinical criteria, considering the history, pain anamnesis and results of neurological examination. The criteria used were those given in the current literature (1-4).

Every patient had been examined by neurosurgeons or neurologists who, given the large number of patients suffering from facial pain referred to our department (14), are experienced in the diagnosis and treatment of facial pain syndromes. Dentogenic pain and temporomandibular joint disorders were excluded by the departments of dental and craniofacial surgery respectively. Patients with symptomatic TN (sTN), caused by tumours of the posterior fossa or by multiple sclerosis, formed a third group. TEPs had, at the time of hospitalisation, been obtained.

Functional Neurology 2002; 17(3): 133-136
(using a Nicolet Spirit, Nicolet, Klein Ostheim, Germany) in 83 patients suffering from facial pain referred to our department for surgical treatment (Table I) and who had not previously been treated surgically. The TEPs were elicited by bipolar perioral stimulation with a square-shaped stimulus of 0.1 ms duration and a frequency of 5.1 Hz. After an offset of 5 ms, 3x500 responses for each side were recorded and averaged. The different electrode was placed contralaterally to the stimulation site at the C5 and C6 electrode positions. The indifferent electrode was positioned at Fz. The stimulus intensity was about 20 to 40 mA and normally three times above the sensory threshold. Contraction of perioral muscles was avoided by decreasing the intensity. Normal values were set according to our data obtained in normal volunteers, in accordance with the literature (11) (Tables II and III). Statistical analysis was performed using the T-test, the Wilcoxon-test and the Chi-square-test, as appropriate.

Results

Neurological findings

Table IV lists the number of patients who reported hyp-, dys-, par, or anaesthesia in the trigeminal region. In contrast to TN, 61% of the patients with AFP reported a sensory dysfunction on examination. The neurological examination revealed only five patients with a pathological corneal reflex; all of them were suffering from sTN.

TSEP findings

As shown (Fig.s 1-3), only the patients suffering from sTN had significantly delayed TEPs on their affected side, compared to the normal values (T-test, p<0.05, for N13 and P19). Also this was the only group in which the difference between the affected and unaffected side exceeded the normal values (mean±2.5 SD) (for N13, P19, and N27).

Table V shows the percentage of latencies above mean±2.5 SD in the different diagnostic groups. All the groups, but predominantly the sTN group, were found to contain a certain percentage of patients with prolonged latencies above the normal values: N13 and P19 revealed more abnormalities than N27.

Discussion

In most cases of facial pain, a careful history and clinical examination will lead to the correct diagnosis (2). However, some patients complain of facial pain that does not fit with certainty into a specific category, and invasive or expensive tests have to be carried out to exclude an organic disease. In addition, our series reveals that the differential diagnosis of facial pain is often unfamiliar to non-neurologists, by whom most of the TN-diagnosed patients were, in fact, referred. As reported in a recent paper, some tumours responsible for sTN remain undiagnosed for years (15). To avoid excessive diagnostic procedures, unsuccessful medical treatment and destructive surgical procedures, non-invasive tests would be useful.

Table I - Characteristics of the sample.

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Mean age</th>
<th>Female: Male</th>
</tr>
</thead>
<tbody>
<tr>
<td>TN</td>
<td>57 yrs</td>
<td>38:31</td>
</tr>
<tr>
<td>sTN</td>
<td>54 yrs</td>
<td>5:7</td>
</tr>
<tr>
<td>AFP</td>
<td>49 yrs</td>
<td>34:8</td>
</tr>
</tbody>
</table>

Abbreviations: TN = trigeminal neuralgia, sTN = symptomatic trigeminal neuralgia; AFP = atypical facial pain.

Table II - Normal values used for the analysis of TEP latencies.

<table>
<thead>
<tr>
<th>Latencies (ms)</th>
<th>N13</th>
<th>P19</th>
<th>N27</th>
</tr>
</thead>
<tbody>
<tr>
<td>mean</td>
<td>12.5</td>
<td>18.5</td>
<td>26.7</td>
</tr>
<tr>
<td>SD</td>
<td>.87</td>
<td>1.51</td>
<td>2.23</td>
</tr>
<tr>
<td>Upper limit</td>
<td>14.7</td>
<td>22.3</td>
<td>32.5</td>
</tr>
</tbody>
</table>

Table III - Normal values used for the analysis of intraindividual side-to-side TEP differences.

<table>
<thead>
<tr>
<th>Side difference of the latencies (ms)</th>
<th>N13</th>
<th>P19</th>
<th>N27</th>
</tr>
</thead>
<tbody>
<tr>
<td>mean</td>
<td>.45</td>
<td>.55</td>
<td>.72</td>
</tr>
<tr>
<td>SD</td>
<td>.35</td>
<td>.55</td>
<td>.63</td>
</tr>
<tr>
<td>Upper limit (mean+2.5 SD)</td>
<td>1.34</td>
<td>1.93</td>
<td>2.29</td>
</tr>
</tbody>
</table>

Table IV - Number of sensory deficits reported in the different groups

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Number of patients</th>
<th>Sensory dysfunction</th>
</tr>
</thead>
<tbody>
<tr>
<td>TN</td>
<td>37</td>
<td>7 (18%)</td>
</tr>
<tr>
<td>sTN</td>
<td>10</td>
<td>3 (30%)</td>
</tr>
<tr>
<td>AFP</td>
<td>36</td>
<td>22 (61%)</td>
</tr>
</tbody>
</table>

Abbreviations: TN = trigeminal neuralgia; sTN = symptomatic trigeminal neuralgia; AFP = atypical facial pain.
while others have found predominantly normal latencies in TN (21,22). Our results showing mostly normal latencies but a proportion of TN patients with prolonged values reflect these heterogeneous findings.

The fact that 35% of the TN patients had delayed N13 peaks on their affected side strengthens the assumption that these patients may have a morphological change of their trigeminal nerve, as described by several authors (20,23). The phenomenon of pathological TEPs on the unaffected side, which in other papers has been described in 27% to 35% of TN patients (12,13,16), was also observed in our study. The cause of this phenomenon has not yet been established.

In agreement with the current literature, a high number of our patients suffering from sTN revealed pathological TEPs. Cruccu et al. (18) found pathological latencies in 80% of patients suffering from sTN. Also the “unaffected

Table V - TEPs: results in the different groups.

<table>
<thead>
<tr>
<th>Parameter/Side</th>
<th>TN</th>
<th>sTN</th>
<th>AFP</th>
</tr>
</thead>
<tbody>
<tr>
<td>N13/affected</td>
<td>35.3</td>
<td>62.5</td>
<td>31.4</td>
</tr>
<tr>
<td>N13/unaffected</td>
<td>17.7</td>
<td>25</td>
<td>8.6</td>
</tr>
<tr>
<td>P19/affected</td>
<td>20.6</td>
<td>50</td>
<td>11.4</td>
</tr>
<tr>
<td>P19/unaffected</td>
<td>8.8</td>
<td>37.5</td>
<td>11.4</td>
</tr>
<tr>
<td>N27/affected</td>
<td>0</td>
<td>12.5</td>
<td>5.7</td>
</tr>
<tr>
<td>N27/unaffected</td>
<td>2.9</td>
<td>12.5</td>
<td>2.9</td>
</tr>
</tbody>
</table>

Abbreviations: TN = trigeminal neuralgia; sTN = symptomatic trigeminal neuralgia; AFP = atypical facial pain.
side" revealed abnormal TEPs, which is not surprising in the case of a multifocal disease like multiple sclerosis. TEP studies in AFP are few. Bennett and Janetta (12) reported no pathological latencies in their AFP patients, whereas Bremerich et al. (13) reported pain intensity-dependent delayed TSEPs in these patients. This discrepancy may be due to the different numbers of patients investigated in these studies (patients in whom an injury of the peripheral trigeminal nerve is the cause of AFP, and patients subsequent to invasive treatment of AFP) (2). A lesion of A-delta and C-fibres probably cannot be excluded by the kind of painless stimuli used in TEP studies. The extent to which depression (7) or other psychiatric disorders contribute to AFP remains unclear. They were found in 62% of patients with AFP in a study by Remick et al. (8). Patients with personality disorders are more prone to developing chronic facial pain (2,24), while AFP patients often score highest in neurotic scales (25). This may explain the main result of our investigation: that AFP patients report a high level of sensory deficits – a finding well known from other studies (3) – despite a low incidence of pathological TEPs in this group. This may constitute an additional argument in favour of the investigation of TEPs in all patients with facial pain. A discrepancy between the neurological testing and the TEPs may support the need for a psychological evaluation in these patients.

Acknowledgments

The authors would like to thank Dr B.A. Haug, of the Department of Clinical Neurophysiology, who supervised most of the evoked potentials investigations.

References