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Research Article

NEUROLOGICAL ASSESSMENT USING A QUANTITATIVE SENSORY TEST IN PATIENTS WITH CHRONIC UNILATERAL TEMPOROMANDINULAR PAIN

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ABSTRACT

Background: Quantitative Sensory Testing (QST) can be used to establish sensory assessment in orofacial pain patients. This study evaluated responses to thermal stimuli in subjects with subtypes of orofacial pain such as temporomandibular disorders (TMD) using QST.

Methods: Eighteen participants with unilateral TMD pain (15 females, 3 males; age: 29-66 years) recruited from the Orofacial Pain Clinic, the Royal North Shore Hospital, Sydney. The study followed the Methods of Limits of the German Research Network testing for the Warm Sensation, Cold Sensation, Heat Pain and Cold Pain using a TSA-II Neurosensory Analyser. The results were compared to the unaffected side of the same patient. A single t test statistical analysis was performed; a p value of less than 0.05 was considered significant.

Results: The Mean Difference between the pain side and the non-pain side for Cold Sensation was 0.36($t=2.275$, $p=0.036$),-0.34 for Warm Sensation ($t=-.705$, $p= 0.49$), 3.47 for Cold Pain ($t=4.393$, $p<0.001$), -0.6 for Hot Pain ($t=-1.074$, $p=0.298$).

Conclusion: The TMD patients reacted to thermal stimuli using QST methods in different ways to other orofacial pain conditions. Future studies need to be undertaken to determine sensitivity and specificity of QST in TMD pain.

INTRODUCTION

Chronic orofacial pain may last for decades and can cause immense suffering and psychological distress (Svensson *et al*., 2004). It is a challenging problem for both the patient and the clinician, as there are often problems in determining a definitive aetiology, providing an accurate diagnosis and offering effective treatment (Pigg *et al*., 2010). Temporomandibular disorders (TMDs), as an example, are complicated multifactorial orofacial pain conditions with a wide range of clinical signs and symptoms affecting the temporomandibular joints, the masticatory muscles, or both (Sessle, 2000; Svensson, 2007), and are one of the most common non odontogenic orofacial pain (Goulet, Lavigne & Lund, 1995; Lipton, Ship & Larach-Robinson, 1993; Locker & Slade, 1988; Von Korff *et al*., 1988). Other researchers have confirmed this view (Dworkin *et al*., 1990; Yap *et al*., 2003). However, the similarity of TMDs to other conditions makes diagnosis a complicated issue, as symptoms often overlap with other conditions (Mohl & Ohrbach, 1992). There are a wide variety of muscular, vascular, neurological, rheumatic, inflammatory and psychological diseases that have similar symptoms and cause dysfunction of the masticatory system and similar pain to TMD (Talebzadeh, 1993). Some studies suggest that TMDs have often been misdiagnosed as other head and neck conditions (Look *et al*., 2010) and this resulted in frequent treatment failure (McNeill, 1997).

Some advances have been made in the neurological assessment of pain, including nerve conduction, evoked responses and Quantitative Sensory Testing (QST). The QST methods (e.g., thermal testing and testing of vibration sense) provide a number of potential tools for understanding the characteristic of nociceptive system function and the neurophysiology

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associated with orofacial pain (Cruccu *et al*., 2010; Treede, 2003)

QST has been recognised as a safe reliable and efficient technique(Pigg *et al*., 2010; Rolke *et al*., 2006b; Shy *et al*., 2003; Van Brakel *et al*., 2005) and could be used for clinical purposes (Essick *et al*., 2010). In a recent study(Salame, Blinkhorn & Karami, 2018), the authors concluded that QST methods using thermal stimuli could be used to evaluate sensory dysfunction in orofacial pain patients.

The aim of this study was to evaluate responses to thermal stimuli in subjects with TMD pain, in order to investigate whether QST could be used as a diagnostic tool to differentiate orofacial pain patients, such as those withTMD pain.

MATERIALS AND METHODS

A total of eighteen participants suffering from unilateral TMD pain were recruited by the supervising consultant from patients who attended a weekly Orofacial Pain Clinic at the Pain Management and Research Centre, Royal North Shore Hospital, Sydney, New South Wales, Australia. Of these, fifteen were females and three males (mean age 50.4 years, SD= 11.21, range 29-66 years). Subjects were excluded if they had a history of psychiatric or another illness in which medications (e.g. anticonvulsants, antidepressants, and analgesics) were taken that could affect the pain response. The project was approved by the Human Research Ethics Committee (HREC) of Northern Sydney Central Coast Area Health (NSCCH) and the Human Research Ethics Committee of The University of Sydney. The instrument used to collect the research data was a TSA-II Neurosensory Analyser (*Figure 1*). This device is controlled by designated software that can produce repeatable thermal stimuli (Yarnitsky & Granot, 2006)

Figure 1 TSA II NeuroSensory Analyser - Software, Cooling Unit and Thermode. (*Photo is taken from TSA-II USER and SERVICE GUIDE 2010).*

The study followed the protocol of the German Research Network (DFNS) (Rolke *et al*., 2006a), using the Methods of Limits (Yarnitsky & Granot, 2006) and testing four modalities of thermal thresholds:

- Warm sensation-(1-2°C above 32 °C) to assess C-fibre mediated response
- Cold sensation-(1-2 °C below 32 °C) to assess A-delta fibre mediated response
- Heat induced pain-(around 45 °C) to assess predominantly C-fibre mediated (with some A-delta fibre) response
- Cold induced pain-to assess a combination of both C and A-delta fibre mediated response.

In the method of limits, the intensity of the stimulus changes until it is halted by the patient when the required sensation is felt. The thermode temperature then returns to the adaptation temperature for the next stimulus.

Each participant was given a brief demonstration and instructed to remain still and hold the Thermode (1.6 x 1.6 cm) over the centre of the painful site (i.e. where pain usually begins if the area is touched) or on the area where pain commences. The test starts when the temperature control unit achieves the requested temperature (baseline temperature initially determined by the investigator). The temperature baseline varies between 30 °C and 32 °C and it is determined when the subject feels neither warmth nor cold after a few seconds of skin contact with the Thermode.

The Cold sensation (CS) and warm sensation (WS) rate of temperature change was set at 1°C/second. The cold-induced pain (CP) and heat-induced pain (HP) rate of temperature change was set at 1.5 °C/second. The interval between stimuli (from the end of one stimulus to the onset of the next stimulus) was set at 4-6 sec. in CS and WS and 10 sec. in HP and CP.

Participants were instructed to click the mouse of the computer at exactly the moment they first felt the temperature change (in the case of thermal sensation) or as soon as they felt their pain threshold had been reached (in the case of cold pain/hot pain) and they were informed that a prompt reaction to the temperature change was very important. As soon as they pressed the mouse, the Thermode temperature immediately returned to the baseline temperature for the next stimulus. Four different clusters of stimuli were given, with five stimuli in each cluster (a maximum of 20 stimuli in a test). The whole or part of the test was repeated if the participant inadvertently pressed the key too early or too late.

The test results were compared to the results collected from the unaffected side of the same patienton the same area on the other (control) side of the face. Differences in the test results between one side of the face and the other may indicate peripheral nerve disease or injury.

RESULTS

The results of the four modalities (CS, WS, CP, and HP) were compared again between the pain and the non-pain sides in patients with unilateral TMD pain using the single t test statistical analysis, where a p value of less than 0.05 was considered to be significant. As shown in *Table 1.*

Table 1 Means and Standard Deviations of all scores of LIMITS-1 test for unilateral TMD pain patients.

	N	Mean	Std. Deviation	Std. Error Mean
LCSPNP	18	.36	0.668	.158
LWSPNP	18	-.34	2.073	.489
LCPPNP	18	3.47	3.356	.791
LHPPNP	18	-.60	2.369	.558

LCSPNP: Limits, Cold Sensation, Pain vs Non-pain.
LWSPNP: Limits, Warm Sensation, Pain vs Non-pain.
LCPPNP: Limits, Cold Pain, Pain vs Non-pain.
LHPPNP: Limits, Hot Pain, Pain vs Non-Pain.

The Mean Difference between the pain side and the non-pain side for Cold Sensation was 0.36 (SD = 0.668 Celsius), -0.34 for Warm Sensation (SD =2.073 Celsius), 3.47 for Cold Pain, (SD =3.365 Celsius) -0.6 for Hot Pain (SD =2.369 Celsius).

There were statistically significant differences between the pain and the non-pain side in terms of Cold Sensation (t=2.275, p=0.036) and Cold Pain (t=4.393, p<0.001) and no significant differences for Warm Sensation (t=-.705, p=.0.49) and for Hot Pain (t=-1.074, p=0.298) (*Table 2.* and *Figure 2*).

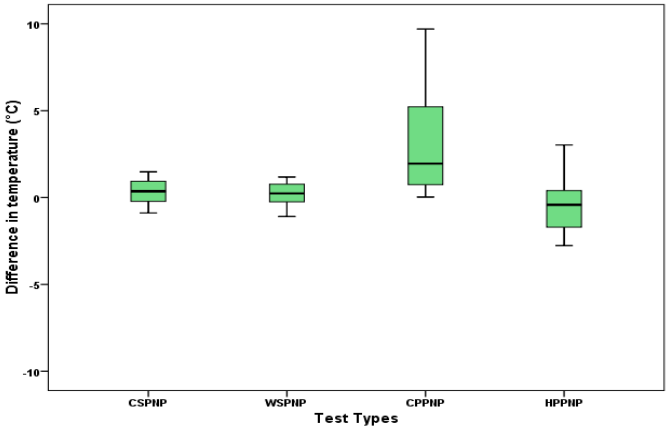

Figure 2 The results of the LIMITS-1 test. The differences in temperature (°C) between pain and non-pain sides of patients with unilateral TMD pain using four different test types (CS, WS, CP and HP).

Table 2 Results of t test analysis of all scores of LIMITS-1 experiments for patients with unilateral TMD pain.

One-Sample Test

	Test Value = 0					
	t	df	Sig. (2-tailed)	Mean Difference	95% Confidence Interval of the Difference	
					Lower	Upper
LCSPNP	2.275	17	.036	.358	.03	.69
LWSPNP	-.705	17	.490	-.344	-1.38	.69
LCPPNP	4.393	17	.000	3.474	1.81	5.14
LHPPNP	-1.074	17	.298	-.599	-1.78	.58

LCSPNP: Limits, Cold Sensation, Pain vs Non-pain.
LWSPNP: Limits, Warm Sensation, Pain vs Non-pain.
LCPPNP: Limits, Cold Pain, Pain vs Non-pain.
LHPPNP: Limits, Hot Pain, Pain vs Non-Pain.

DISCUSSION

The results of this study indicated disruption of thermal processing on the pain side compared to the non-pain side in the presence of TMD pain. This disruption exhibited as lower sensation thresholds to both Cold Sensation and Cold Pain stimuli. Other recent studies on TMD patients using QST methods found disruption of thermal sensitivity(Eliav *et al*., 2003; King *et al*., 2009; Park *et al*., 2010; Pfau *et al*., 2009; Svensson, List & Hector, 2001). However, a detailed assessment of these studies showed different types of thermal sensations malfunctioning. As an example, Park and coworkers showed increased cold pain threshold and decreased heat pain threshold in 39 TMD patients comparing to healthy controls (Park *et al*., 2010). King and colleagues showed that all 14 women with TMD pain exhibited decreased heat pain thresholds compared to controls (King *et al*., 2009). However a QST study on 23 TMD patients showed decreased cold pain threshold only, with no difference in cold sensation, hot sensation and heat pain threshold (Pfau *et al*., 2009); meanwhile another two studies found no significant difference between patients with TMD related pain and controls for heat pain threshold (Svensson, List & Hector, 2001) and (Eliav *et al*., 2003).These last two studies concerned heat pain thresholds only.

Only two components were affected in our study compared to other studies on TMD patients. According to Greenspan "various somatosensory modalities can occur independently to one another" (Greenspan, 2001). In a QST study that appliedcold sensation, warm sensation, cold pain and heat pain on 465 patients with orofacial pain, the authors divided the patients into three groups: thermal hyposthesia, thermal hyperalgesia and both conditions. They found that hyposthesia and hypersthesia may occur individually and there may not be any association between them (Verdugo & Ochoa, 1992). Koltzenburg and his coworkers suggested that cold allodynia occurs with or without heat allodynia (Koltzenburg, Lundberg & Torebjork, 1992).

TMD patients in our study showed hypersensitivity towards cold stimuli only with no changes for heat stimuli. Lower tolerance to a cold sensation was reported in patients with TMD, fibromyalgia syndrome (Berglund *et al*., 2002) and myofascial TMD patients (Fernandez-de-las-Penas *et al*., 2009; Fernandez-de-Las-Penas *et al*., 2011). In myofascial TMD patients sensitivity to cold stimuli is higher that heat stimuli (Fernandez-de-las-Penas *et al*., 2010; Fernandez-de-Las-Penas *et al*., 2011). Results from an animal study (Yamazaki *et al*., 2008) revealed the role of the para-trigeminal nucleus nociceptive neurons in cold sensation. As reported by Berglund, some changes in central processing of pain, cold hyperalgesia can occur (Berglund *et al*., 2002). It was also reported that the severity of cold hyperalgesia was related to the intensity of TMD pain symptoms, and this could explain the role of peripheral nociceptors in the mechanism of sensitization (Fernandez-de-las-Penas *et al*., 2010). Several other studies have shown that C polymodal nociceptors contribute to cold pain (hyperalgesia) (Craig & Bushnell, 1994). Another study(de Medinaceli *et al*., 1997) suggested that cold hyperalgesia is the consequences of peripheral rather than a central process. An investigation of cold hyperalgesia suggested that the central disinhibition (excitement) of C nociceptors is followed by a reduction of cold fibre activity (Beise, Carstens & Kohlloffel, 1998). Other studies (Fruhstorfer, 1984; Wahren, Torebjork & Jorum, 1989; Yarnitsky & Ochoa, 1990) reported that patients with A fibre degeneration in the absence or dysfunction of C nociceptor inhibitors suffered from cold hyperalgesia and had a lower cold pain threshold. It has also been found that a lack of C nociceptor inhibitors as a result of A fibres degeneration explains why patients feel cold pain (Baumgartner *et al*., 2002; Fields, Rowbotham & Baron, 1998).

On the other hand, a study by Bragdon and his coworkers showed decreased heat detection threshold in TMD patients versus controls (Bragdon *et al*., 2002). They proposed that central processing in addition to muscle disorders could be behind these results. Also Park and his colleagues found that patients in three TMD subgroups (according to RDC/TMD) were more sensitive to thermal pain with a higher threshold to cold pain and a lower threshold to hot pain (Park *et al*., 2010). Some studies postulate that heat hyperalgesia found in TMD, chronic tension-type headache and fibromyalgia pain patients are the results of a generalized hyperalgesic response to superficial somatosensory stimulation (Bendtsen, Jensen &

Olesen, 1996; Langemark *et al*., 1989; Lautenbacher, Rollman & McCain, 1994).

Thermal hyperesthesia or allodynia may also occur in paients with inflammatory or musculoskeletal pain such as TMD (Kosek, Ekholm & Hansson, 1996; Kosek & Ordeberg, 2000). Lower pain threshold and tolerance has been reported in patients with unilateral and/or bilateral TMD pain when compared with healthy subjects(Kashima *et al*., 1999; Maixner *et al*., 1998), and these results may be due to inflammation in TMJ (Stegenga, 2001) which would affect the detection thresholds of large afferent fibres (Benoliel *et al*., 2002). An example of this is the complications after third molar extractions; common symptoms are hypersensitivity of the skin covering the chin and of the tongue mucosa due to the inferior alveolar and lingual nerves damage (Eliav & Gracely, 1998). Inflammation could have caused dysfunction in the detection threshold of large myelinated fibres. Such sensitivity may be as a result of an inflammatory reaction in the extraction site as the symptoms usually disappears within a few days. The presence of cytokines I, an inflammatory neurotransmitter, in the TMJ synovial fluids in patients with TMDs was reported in many studies (Shafer *et al*., 1994). Although the heat hyperalgesia in patients with TMD in trigeminal and extra- trigeminal regions have been reported in some studies(Fernandez-de-las-Penas *et al*., 2010; Maixner *et al*., 1998) there are other researchers who disagree even though they could not offer a rational explanation as to why the hot pain threshold is lower in TMD patients when compared to healthy subjects (Raphael *et al*., 2009; Svensson, List & Hector, 2001).

Patients with different types of orofacial pain conditions can show different reactions to thermal assessment according to the duration of the pain, such as longer lasting pain that could cause a higher thermal pain threshold(Ellrich, Ristic & Yekta, 2006). For instance, when sustained noxious heat stimulation was applied to TMD patients, they showed greater thermal C-fibre-mediated temporal summation than controls (Maixner *et al*., 1998). Temporal summation is the increase in pain intensity after repeated stimuli. This can happen if the second stimulus is given less than 3 seconds after the removal of the first stimulus in normal non-pain subjects (Maier *et al*., 2010). However, some orofacial pain patients exhibited more pronounced temporal summation and exaggerated after-sensations of pain compared to controls following repetitive noxious stimulations (Raphael *et al*., 2009). This effect was explained by alterations in the central nervous system processes(Price & Dubner, 1977) or by a central synaptic sensitivity that is influenced by peripheral nociceptive signals (Raphael *et al*., 2009). In this study the time between two trials for the sensation threshold experiments was 6 seconds and for the pain threshold experiments was 10 seconds, which is well above the normal value of 3 seconds. But if the patients suffered increased after-sensation pain as suggested by other investigations, the exaggerated temporal summation could have affected the threshold values for thermal stimuli in these patients.

One possible cause of these variations between the results could be related to methodological issues. It has been suggested that technical and methodological parameters can alter the degree of superficial sensitivity (Svensson, List & Hector, 2001). A simple explanation is related to the way patients in this study were holding the thermode over the painful area and the pressure they were exercising. In discussing their results, Raphael and his associates suggested that different patients could push the thermode with different forces against their skin during the recording process and this could stimulate deep tissues at the test site and exaggerate the pain response and some subjects may not be able to differentiate between pain and sensitivity (Raphael *et al*., 2009). However another study suggested that shallow contact is unlikely to cause stimulation of deep tissues regardless of the pressure exercised (Raphael *et al*., 2009).It may seem impossible to provide an answer to this problem unless a standardised technique for holding the thermode against the skin that does not put “excessive” pressure can be devised. Such discussions highlight the difficulties of undertaking this type of research, as reproducibility of patient responses is a problem and maintaining constant electrode pressure for different patients is subject to wide variation.

This study confirmed that orofacial pain patients with TMD pain reacted to thermal stimuli using QST method. However the pattern of reaction in our study was different to patterns observed in other studies undertaken on other TMD pain patients using similar methods. Many factors could have interfered regarding these differences in the results including biological, social and psychological factors. Technical factors such as small sample size, homogeneity of subjects who were recruited from the same centre and so belonging to a certain demographical background, age and gender and other technical factors related to the equipment themselves could have played a role.

CONCLUSION

This study showed that QST methods using thermal stimuli could be used to evaluate sensory dysfunction in TMD pain patients using specific parameters such as cool and warm sensation and cold and hot pain. However, we could not define a specific pattern for the TMD patients in our study to react to the thermal stimuli that could be used for diagnostic purposes.

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