U.PORTO
FACULDADE DE DESPORTO
UNIVERSIDADE DO PORTO

The role of physiotherapy in the global burden of Temporomandibular Disorders.

Maria Amélia Alves do Paço

Dissertação apresentada às provas para a obtenção do grau de Doutor em Fisioterapia, nos termos do Despacho nº 13169/2011, publicado em Diário da República, 2.ª série, N.º 189, de 30 de Setembro de 2011, orientada pela Professora Doutora Teresa Maria Costa Pinho e co-orientada pelo Professor Doutor José Alberto Ramos Duarte.

Porto, 2016

CIAFEL

Paço, M. (2016). *The role of physiotherapy in the global burden of Temporomandibular Disorders*. Doctorate dissertation in Physiotherapy, presented at the Faculty of Sport, University of Oporto.

KEY-WORDS: TEMPOROMANDIBULAR DISORDERS, GLOBAL BURDEN, PHYSIOTHERAPY

Sei que o melhor de mim está para chegar…

Acknowledgements

Esta dissertação é o culminar de um percurso de 4 (BONS) anos que resultaram num crescimento não só científico mas também pessoal que me marcarão para a vida. E porque já diz o ditado: “*sozinhos vamos mais rápido, mas juntos vamos mais longe*”, efetivamente eu tive a felicidade de contar com a ajuda, apoio, amizade e amor de muitas pessoas que não posso deixar de referir:

- A Senhora Professora Doutora Teresa Maria da Costa Pinho, minha orientadora e minha maior mentora em todo este processo. Quero manifestar-lhe o meu reconhecimento e profundo agradecimento por todo o apoio, disponibilidade, dedicação, confiança e motivação constantes. Por ser um exemplo de profissionalismo, ética, capacidade científica, generosidade, humildade e por ter um empenho capaz de contagiar todos em seu redor e em tudo aquilo que faz. Toda a aprendizagem, não só do ponto de vista científico como também humano, representará sempre um exemplo a seguir na minha vida académica e profissional. Muito obrigada pela amizade, a estima e o carinho com que me tem distinguido.

- Ao Senhor Professor José Alberto Ramos Duarte, meu co-orientador, quero manifestar a minha profunda gratidão pela sua prontidão e generosidade sempre que recorri aos seus sábios conselhos, conhecimentos e esclarecimentos. Foi sem dúvida uma contribuição fundamental em todo o percurso e representará sempre uma referência a seguir na minha vida académica e professional.

- A todos os meus colegas da Escola Superior de Saúde Vale do Ave, em especial a Daniela, Marta, Francisco, Liliana e João, por toda a ajuda, compreensão e motivação ao longo deste período. O vosso apoio foi fundamental para a conclusão desta etapa da minha vida.

- À Rita e ao Francisco, meus colegas e acima de tudo amigos, pelo apoio, motivação, disponibilidade e por serem importantes referências na minha vida.

- À minha querida amiga Paula, porque sem ela o caminho teria sido sem dúvida nenhuma, mais longo e mais difícil. Tenho que lhe agradecer tudo, principalmente a sua amizade e carinho inesgotáveis. Muito obrigada!

- Aos meus pais que sempre me ensinaram que o conhecimento não ocupa espaço e que deveria procurar ser sempre mais e melhor, procurando evoluir sempre como pessoa e como profissional. Quando eu for grande quero ser como vós!

- A toda a minha família, em especial às minhas irmãs, Lô e Piwi e ao Marco, pelo carinho, apoio e estarem sempre do meu lado.

E *the last but not the least*, quero agradecer com todo o coração ao meu querido David, meu companheiro de todas as viagens, que está sempre comigo e que me apoia incondicionalmente qualquer que seja o próximo desafio a que me proponho. Muito obrigada por estares sempre comigo e por seres o meu Porto de Abrigo… E claro que o meu pequeno Diogo também merece um agradecimento, por se ter portado tão bem nesta reta final de conclusão da tese, deixando a mãe trabalhar tranquila.

A todos, o meu mais sincero OBRIGADA!

General Index

Index of Figures

Index of Tables

RESUMO

O objetivo desta tese foi identificar a carga global das disfunções temporomandibulares verificando como o papel do fisioterapeuta pode ser potenciado, considerando o modelo biopsicossocial. Realizou-se uma revisão sistemática e metanálise sobre a efetividade da fisioterapia no tratamento da disfunção temporomandibular. Foi também levado a cabo um estudo transversal analítico, no qual os participantes, selecionados através de um método de amostragem de bola de neve, completaram um questionário *on-line*. Isto permitiu avaliar a prevalência de disfunção temporomandibular na população portuguesa assim como, avaliar os seus fatores de risco. Com este estudo foi validada uma escala, posteriormente utilizada para avaliar o estado do conhecimento da população em geral sobre as disfunções temporomandibulares. Realizou-se um estudo *quasi*-experimental, longitudinal e retrospetivo, com uma amostra não probabilística de conveniência, constituída pelos registos clínicos de pacientes com disfunção temporomandibular, submetidos a tratamento ortodôntico. Recorreu-se à cefalometria, no sentido de analisar as variáveis cranio-cervico-faciais, quando comparados os valores pré-tratamento com os valores pós-tratamento ortodôntico e os da fase de contenção. Por fim, foi realizado um estudo tipo série de casos, com três casos de pacientes com disfunção temporomandibular submetidos a tratamento ortodôntico, com resultados distintos. Este estudo teve como objetivo compreender quais as características que podem beneficiar da fisioterapia e os seus resultados imediatos. Concluiu-se que a fisioterapia é mais efetiva na redução da dor do que as outras modalidades de tratamento com as quais foi comparada, no tratamento da disfunção temporomandibular. Os principais fatores de risco associados foram: sexo feminino, impulsividade, cefaleia tensional, enxaqueca, ansiedade, trauma facial e hábitos parafuncionais. A escala criada é válida e fiável e a população Portuguesa tem um conhecimento global sobre a disfunção temporomandibular positivo. O tratamento ortodôntico produziu diferenças significativas na postura craniocervical, sendo propenso a retornar aos valores basais.

PALAVRAS-CHAVE: DISFUNÇÃO TEMPOROMANDIBULAR, CARGA GLOBAL, FISIOTERAPIA

ABSTRACT

The aim of this thesis was to identify the global burden of temporomandibular disorders ascertaining how the physiotherapist's role may be enhanced, in the light of the biopsychosocial model. A systematic review and meta-analysis regarding physiotherapy effectiveness in the management of temporomandibular disorders was performed. An analytical cross-sectional study was carried out, in which the participants, selected through a snow-ball sampling method, completed an online questionnaire. This allowed the assessment of the prevalence of temporomandibular disorders in the Portuguese population and the assessment of its risk factors. With this study a scale was validated that enabled to assess common knowledge about temporomandibular disorders as well as evaluate the status of the Portuguese population concerning this condition. A quasi-experimental, longitudinal and retrospective study, with a non-probabilistic convenience sample of the clinical records from patients with temporomandibular disorders and submitted to orthodontic treatment was performed. A cephalometric analysis was performed, in order to verify if there were changes in the cranio-cervico-facial variables when comparing pre orthodontic treatment with post orthodontic and contention phase values. At last, a case series was carried out, with three cases of patients with temporomandibular disorders that were submitted to orthodontic treatment, but had distinct outcomes. This study aimed to understand the different characteristics presented that may benefit from physiotherapy and its' immediate effectiveness. It was concluded that physiotherapy is more effective in pain reduction than the other treatment modalities to which it was compared to, in the management of temporomandibular disorders. The main risk factors associated were: female gender, impulsiveness, tension-type headache, migraine, anxiety, facial trauma and parafunctional habits. The knowledge scale is psychometrically valid and reliable and the Portuguese population have an overall positive knowledge about temporomandibular disorders. Orthodontic treatment produced significant differences in the craniocervical posture, being prone to return to basal values.

KEY-WORDS: TEMPOROMANDIBULAR DISORDERS, GLOBAL BURDEN, PHYSIOTHERAPY

List of Abbreviations

AA	[Most anterior point of atlas vertebra]
ANB	[Angle between A point, *nasion* and B point]
C0	[Base of the occipital bone]
C1	[First cervical vertebra]
C2	[Second cervical vertebra]
C3	[Third cervical vertebra]
CI	[Confidence interval]
CV	[Craniovertebral angle]
CVT	[Represents the line between OT point and the most posteroinferior aspect of the fourth cervical vertebra]
DD	[Disc displacement]
DERS	[Difficulties in emotion regulation scale]
EMG	[Electromyographic]
ESC	[Education and self-care]
FAI	[Fonseca anamnestic index]
FMA	[Frankfort-Mandibular plane angle]
H	[The most anterior point of the hyoid bone]
H1	[Represents the line between the third cervical vertebra and *retrognation*]
ICC	[Intraclass correlation coefficient]
IMT	[Intraoral myofascial therapy]
IQR	[Interquartile range]
KMO	[Keisser-Meyer Olkin]
MeSH	[Medical subject headings]
NSL	[Represents the line between *sela turcica* and *nasion*]
OCD	[Obsessive-compulsive disorder]
OPT	[Represents the line between OT point and the most posteroinferior aspect of the odontoid process]
OR	[Odds ratio]
OT	[Orthodontic treatment]
PCA	[Principal component analysis]

PNS	[Posterior nasal spine]
PPT	[Pressure pain threshold]
RCTs	[Randomized Controlled Trials]
RDC-TMD	[Research diagnostic criteria for temporomandibular disorders]
Rgn	[*Retrognation*]
ROM	[Range of movement]
SD	[Standard deviation]
SMD	[Standardized mean difference]
SPSS	[Statistical package for the social sciences]
TMDs	[Temporomandibular disorders]
TMJ	[Temporomandibular joint]
VAS	[Visual analogue scale]
VC	[Variation coefficient]
WHO	[World health organization]

CHAPTER 1

INTRODUCTION

"Temporomandibular disorders" (TMDs) is a term that comprises a variety of conditions affecting the anatomic and functional characteristics of the temporomandibular joint (TMJ). Historically, it has been attributed to mechanisms related to dental or structural abnormalities, but with considerable controversy and little solid evidence (Clark, 1991; Tallents, 1991). An otorhinolaryngology surgeon, J. B. Costen was the first to report a relationship between occlusion and temporomandibular joint function, by suggesting that changes in dental condition led to anatomical changes in the temporomandibular joint, creating ear symptoms (Costen, 1934). For this reason, this disorder has also been called Costen's syndrome and is also known as craniomandibular disorder or even craniofacial dysfunction (Durham, 2013; Langdon, 1994; Michelotti & Iodice, 2010; Nicolakis et al., 2000; von Piekartz, 2007).
TMDs concerns an heterogeneous group of pathologies that manifest in the orofacial region, head and neck, and result from the dysfunctional interrelationship between TMJs, masticatory and cervical muscles, teeth and dental tissues as well as the central and peripheral nervous systems (Magnusson, Egermark, & Carlsson, 2005). It is defined by the American Academy of Orofacial Pain as a group of musculoskeletal and neuromuscular conditions that involve the TMJs, the masticatory muscles and all associated structures in a reciprocal interaction and influence (Leeuw & Klasser, 2013). This disorder results in one or more signs and symptoms: orofacial pain (at rest or during mouth movement), masticatory muscle pain or a combination of both. Other symptoms include impaired mandibular range of motion, joint noises associated with function, muscle and joint tenderness as well as head and neck pain (Cairns, 2010b; Liu & Steinkeler, 2013; Suvinen, Reade, Kemppainen, Könönen, & Dworkin, 2005a).

The identification of a universal and unambiguous cause of TMDs is lacking. Notwithstanding, aetiological concepts have been suffering a paradigm shift through the years, evolving from biomedical theories (solely mechanical causes and specific anatomical changes) to a multifactorial theory based on the biopsychosocial concept, which encompasses integrative and multidisciplinary models. This evolution shows that there has been no single aetiological factor identified for TMDs and it is accepted that its aetiology is complex and multifactorial (Greenberg, Glick, & Ship, 2008; Greene, 1995; Gremillion, 2000b; Liu & Steinkeler, 2013; Melis & Di Giosia, 2016a; Oral, Bal Küçük, Ebeoğlu, & Dinçer, 2009; Suvinen et al., 2005a). Some of the aetiological factors described are: trauma, occlusal factors, muscular factors, hormonal factors, psychosocial factors, parafunctions and hereditary factors (Chisnoiu, Chisnoiu, Moldovan, Lascu, & Picos, 2016; Chisnoiu et al., 2015; Maydana, Tesch, Denardin, Ursi, & Dworkin, 2010; Suvinen et al., 2005a)

TMDs represents the most common chronic orofacial pain condition with prevalence studies demonstrating that this dysfunction affects between 10% to 25% of the population (Gremillion, 2000b; LeResche, 1997b; Manfredini et al., 2011b; Oral et al., 2009) with an annual incidence rate between 2% and 4% (Slade et al., 2007; Slade et al., 2016; Von Korff, Le Resche, & Dworkin, 1993). The prevalence of TMDs related pain is low until adolescence, not differing the frequency between males and females. However, there is a peak occurrence between 20-40 years, with women being more prone to TMDs than men (Ferreira, Silva, & Felício, 2015; LeResche, 1997b; Liu & Steinkeler, 2013). It represents a considerable socio-economic burden on the population. Overall, the annual TMDs management cost in the United States of America, not including imaging, has doubled in the last decade to $4 billion (National Institute of Dental and Craniofacial Research, 2016). It has been estimated that TMDs results in almost 18 million total work days lost per year for every 100 million working adults in the United States of America and that approximately 85% of the cost of treating TMDs is associated with the treatment of only a little percentage of patients with persistent pain and dysfunction (Maixner et al., 2011).

The complexity intrinsic to TMDs is evident when analysing the underlying pathophysiology. The pathophysiological mechanisms differ whether the problem lies on the articular component, muscular component or other components.

Concerning articular pain, regardless of the underlying condition, as the TMJ degenerates, a variety of morphological and functional deformities can lead to a significant loss of function and joint pain (Zarb & Carlsson, 1999). The joint capsule, synovial membrane and articular disc are innervated by myelinated and unmyelinated nerve fibres with free nerve endings. Some of these contain neuropeptides as calcitonin gene-related peptide and substance P (Asaki, Sekikawa, & Kim, 2006), that are related with pain and inflammation. This means that TMJ may be excited by noxious mechanical and chemical stimuli, resulting in pain. The TMJ is considered a heavily loaded structure and when the ability of the disc to redistribute joint stresses is limited, the susceptibility of the joint to damage due to overloading is highly enhanced (Gallo, 2005). Excessive loading of the joint may result from mild derangements and lead to progressive joint injury or cause local hypoxia in TMJ tissues, and thus the increase in calcitonin gene-related peptide and substance P may be a compensatory mechanism (Tanaka, Detamore, & Mercuri, 2008). Having this, peripheral mechanisms, that may be caused by overloading of the TMJ, cause pain by the mechanical stimulation of nociceptors, increased release of neuropeptides and substance P or by local hypoxia. In addition to peripheral mechanisms, central mechanisms may also play a role on TMJ pain. A sustained nociceptive input from painful TMJs may lead to a sensitization of the central nervous system, which in turn is thought to contribute to lower pain thresholds and also provide a neural mechanism that could underlie the development of referred pain and generalized pain sensitivity in patients with TMDs (Cairns, 2010b). Furthermore, pain in the TMJ may cause reflex masticatory muscle spasm (Tanaka et al., 2008) which is a protective jaw muscle reflex that may increase the masticatory muscles' pain.

A different pathophysiology is present when TMDs' pain is localized in the masticatory muscles and is worsened on muscle palpation and during function. This represents myofascial pain, and there is less evidence regarding pathophysiological changes to the masticatory muscle tissues of patients with myofascial TMDs (Cairns, 2010b). Pain in the masticatory muscles might be caused by the reflex masticatory muscle spasm (Tanaka et al., 2008), that reduces jaw mobility as a protective contraction when pain is present. It is hypothesized that an increase in muscle tone results in jaw muscle pain, which further increases muscle tone resulting in muscle spasm and / or fatigue that increases pain in a cyclic reinforcing manner (“vicious cycle theory”) (Murray & Peck, 2007). Another theory, the “pain adaptation model”, proposes that pain does not necessarily occur as a result of muscle hyperactivity but rather to other causes, and that alterations of muscle activity may be a response to pain to limit movement, protecting the jaw from further damage and promoting healing (Lund, Donga, Widmer, & Stohler, 1991; Murray & Peck, 2007). Murray & Peck (2007) also introduced the “integrated pain adaptation model”, that takes into account the inter-individual variability and proposes that homeostasis is maintained and pain minimized by individually unique motor strategies, that result from a combination of the sensory and affective components of pain.

Furthermore, the masticatory muscles are innervated by myelinated and unmyelinated trigeminal afferent fibres with non-specialized endings that are activated by noxious mechanical and ⁄ or chemical stimuli, which appear to function as polymodal nociceptors (Cairns, 2010b). These masticatory muscle nociceptors have receptors for algogenic substances, such as serotonin and glutamate, and contain neuropeptides (calcitonin gene-related peptide and substance P) (Ambalavanar et al., 2006). Moreover, repetitive strain injury to the muscle, as happens in parafunctional activities, induces localized tissue ischaemia and / or release of algogenic substances (serotonin, glutamate) which sensitizes muscle nociceptors. Beyond this, ongoing masticatory muscle pain is also effective in inducing central sensitization, which further amplifies pain (Ernberg, Hedenberg-Magnusson, Kurita, & Kopp, 2006; Sarlani, Grace, Reynolds, & Greenspan, 2004; Sarlani & Greenspan, 2003). In addition, it is also speculated that TMD patients are not only more pain sensitive but also demonstrate reduced ability to inhibit pain, possibly because of dysfunction of endogenous pain inhibition systems (King et al., 2009).

Considering the pathophysiological mechanisms underlying TMDs, there appears to be an interplay of central and peripheral nociceptive mechanisms that contribute to some manifestations of TMDs, while other mechanisms reflect gene-environment interactions (Cairns, 2010b). This notion of distinct pathways supports the ideas underlying a targeted treatment approach and physiotherapy may play an important role on addressing TMDs impairments. One of the interventions used by physiotherapists in the management of TMDs is manual therapy, which may contribute to pain reduction, muscular tension reduction and range of movement improvement (Fonseca, Paço, & Oliveira, 2016). These effects may be explained by peripheral, neurophysiological, spinal, and supraspinal mechanisms (Bialosky, Bishop, Price, Robinson, & George, 2009a). In response to injury, the peripheral nociceptors and inflammatory mediators act together, and manual therapy may directly affect this process (Bialosky et al., 2009a). In addition, manual therapy has been proven to trigger mechanical hypoalgesia as well as other changes related to the activation of the sympathetic nervous system, suggesting a mechanism mediated by the periaquaductal gray and the spinal dorsal horn (Bialosky et al., 2009a; Schmid, Brunner, Wright, & Bachmann, 2008b). Moreover, Schmid et al. (2008) found strong evidence to support the involvement of the central nervous system in mediating the response to manual therapy treatment. These are the mechanisms underlying physiotherapy interventions, notwithstanding the effectiveness of physiotherapy in the management of TMDs is still unclear because, despite the existence of several studies that aimed to assess physiotherapy effects' (Fernández-Carnero et al., 2010; La Touche et al., 2009; Nascimento et al., 2013; Tuncer, Ergun, Tuncer, & Karahan, 2013) most of the studies performed presented methodological issues, which make difficult to draw conclusions (McNeely, Olivo, & Magee, 2006; Medlicott & Harris, 2006b).

One of the problems often found in the literature, and that may explain the different results regarding the different interventions' effectiveness, is the difficulty and the variability on TMDs diagnosis. The diagnose is essentially clinical through subjective examination (information regarding pain, other symptoms, traumas, oral habits, parafunctional habits, previous treatments) and physical examination (range of movement, articular noises, occlusal examination, joint and muscles palpation). There are several questionnaires and indexes described and validated to diagnose TMDs and its severity, as the Helkimo Index (Helkimo, 1974; Van Der Weele & Dibbets, 1987), Fonseca Anamnestic Index (Campos, Carrascosa, Bonafé, & Maroco, 2014a; Fonseca, Bonfante, Valle, & Freitas, 1994b) and, more recently, the International Research Diagnostic Criteria for TMDs Consortium Network, has developed the Diagnostic Criteria for TMDs (Peck et al., 2014; Schiffman et al., 2014). This is one of the most advocated instruments to diagnose TMDs, not only for research purposes but also to use in clinical settings, in order to allow comparisons of patient populations in different studies and also to provide a common conceptual framework to use in the clinic (Leeuw & Klasser, 2013; Schiffman et al., 2014). In this classification, TMDs are divided into TMJ disorders and masticatory muscle disorders, headache disorders and there is also a subgroup classified as associated structures (Leeuw & Klasser, 2013; Schiffman et al., 2014).

Well-defined operationalized diagnostic criteria are central to accurately identify the cause(s) of pain as well as other relevant characteristics of the patient that could influence the management, and thus the outcome of the intervention. This assumes a particular importance since the longer the pain persists, the greater the potential for emergence and amplification of cognitive, behavioural and psychosocial risk factors. As such, and because there is a high rate of scientific development in the field of orofacial pain, there is a substantial need to identify the risk factors that lead to the onset and maintenance of TMDs. Female gender, facial trauma, parafunctional habits and psychological factors are frequently reported risk factors (Dıraçoğlu et al., 2016b; Fillingim et al., 2011b; Huang, LeResche, Critchlow, Martin, & Drangsholt, 2002; Magalhães et al., 2014a; Michelotti, Cioffi, Festa, Scala, & Farella, 2010b; Ohrbach et al., 2011a; Poveda Roda, Bagan, Díaz Fernández, Hernández Bazán, & Jiménez Soriano, 2007). However, due to the complexity and multifactorial aetiology and to the scientific growing knowledge in the field, further highlighting about risk factors is still needed. The information regarding risk factors is vital, because the correct identification of these factors, will allow the health professional to address those issues or even refer to the most adequate professional, in a multidisciplinary approach. If this does not happen the pain will persist, the risk factors will amplify the pain, resulting in enhanced pain sensitivity, further pain persistence and consequently, reduced probability of success from standard treatments.

Another potential risk factor proportionally related with health level, not often reported in the literature but highly advocated by the World Health Organization (WHO), is the level of knowledge the person has about the condition he/she suffers (World Health Organization, 2016a) since it influence the attitude towards health choices. There is a raising concern with the identification of health determinants, so that public policies may address those determinants in order to promote and guide behaviour changes towards healthy behaviours and build up a healthier community. One of the health determinants found, and in which physiotherapy plays an important role, is literacy. This was defined by the WHO as the cognitive and social skills of the individual, that determine the motivation and ability to access, understand and use effectively the information to promote the improvement and maintenance of his health (World Health Organization, 2016b). Having this in mind, and considering that physiotherapists are professionals specialists in health promotion, they are in an ideal position to influence the health of the individual (European Region - World Confederation for Physical Therapy, 2016). In order to do so, it is important to ascertain the level of knowledge about TMDs in the population where the physiotherapist acts. Only by understanding the literacy level, the physiotherapist will be able to define the best strategy to educate and empower the patient, promoting the development of active coping strategies. Considering the chronicity often associated with TMDs, this empowerment seems crucial for a better prognosis, since a successful self-management program allows healing and prevents further injury to the musculoskeletal system (Leeuw & Klasser, 2013). It may even be enough to control the problem (Randolph, Greene, Moretti, Forbes, & Perry, 1990).

The correct diagnosis and identification of potential risk factors will be central to outline the best intervention. Differential diagnosis is possibly the main challenge due to the amount of anatomical surrounding structures, the referral pain mechanisms, the importance attributed to the face as well as the emotional and psychosocial issues involving TMDs (Okeson, 1996; Okeson & de Leeuw, 2011). Nonetheless, management goals for TMDs sufferers are similar to those for other musculoskeletal disorders. This include, in general, decrease pain, decrease joint overloading, restoration of functionality and, very importantly, resumption of

normal daily activities. The described goals are best achieved by a well-established program designed to treat not only the physical impairments but also to reduce or eliminate the effects of all contributing factors (Leeuw & Klasser, 2013). Studies of the natural history of many TMDs suggest that they may be transient, self-limiting and tend to improve or resolve over time (Greene, 2010). Little is known about which signs and symptoms will progress to more serious conditions in the natural course of TMDs, thus a more aggressive and irreversible approach should be avoided as a first treatment option (Leeuw & Klasser, 2013).
Regarding treatment modalities, when managing TMDs there are several health professionals that may act together, in a multidisciplinary approach, so that the impairments and contributing factors found can be fully addressed and provide the best outcome (Leeuw & Klasser, 2013; Okeson, 2013). Treatment modalities include patient education and self-management, cognitive behavioural therapy, pharmacotherapy, physical therapy, speech therapy, orthodontics, occlusal therapy and also maxillofacial surgery (Al-Riyami, Moles, & Cunningham, 2009; de Toledo, Silva, de Toledo, & Salgado, 2012; Leeuw & Klasser, 2013; List & Axelsson, 2010; Okeson, 2013; Piekartz, 2009).

Among the treatment modalities described, orthodontic treatment seems to be one of the most sought by patients in the management of TMDs (Luther, Layton, & McDonald, 2010b; Macfarlane et al., 2009). The literature regarding orthodontic treatment effects is somewhat controversial, with several studies reporting good results on the TMDs resolution or, at least, on reducing the risk of the patient to develop it, while other studies suggest that orthodontic treatment increases the risk of onset of signs and symptoms of TMDs (Egermark, Blomqvist, Cromvik, & Isaksson, 2000b; Egermark, Carlsson, & Magnusson, 2005; Henrikson, Nilner, & Kurol, 1999; Imai et al., 2000; Leite, Rodrigues, Sakima, & Sakima, 2013; Luther, 2007b; Nielsen, Melsen, & Terp, 1990; Ohlsson & Linquist, 1995). Despite this controversy, it seems currently accepted that orthodontic treatment is not a risk factor for TMD (Akhter et al., 2008a; Bourzgui, Sebbar, Nadour, & Hamza, 2010; Egermark et al., 2005; Kim, Graber, & Viana, 2002a; Leite et al., 2013; Manfredini et al., 2016a). Orthodontic treatment aims to restore the normal occlusion and obtain occlusal stability. In order to accomplish that, it requires teeth movement, to correct position anomalies and structural malformations, as well as the adaptation of the position and dimension relations between teeth and facial bones through the application of forces and/or by stimulating and redirecting the functional forces within the craniofacial complex (American Association of Orthodontists, 1997). However, the close relationship between the craniomandibular system and the craniocervical system (Armijo Olivo, Magee, Parfitt, Major, & Thie, 2006; Gomes, Horta, Gonçalves, & Santos-Pinto, 2014; Rosa, 2012) raises the doubt that if the mechanical transformation of the craniofacial region, often accompanied by skeletal changes, may occur without altering the muscular and articular stability of the surrounding structures, namely the craniocervical region. The implications of orthodontic treatment in the craniocervical posture could justify the apparent contradictory results attributed to orthodontic treatment in the management of TMDs. Accepting the fact that recent evidence suggests that orthodontic treatment does not contribute to the development of TMDs, the potential craniocervical alterations underlying should not be neglected, since they may have the potential to enhance the risk of developing or aggravating the condition. So, it seems important to ascertain if concomitantly with the orthodontic treatment there are any changes regarding craniocervical posture. Indeed, the presence of such alterations does not necessarily means TMDs signs or symptoms, but it may promote favourable conditions for it.

Taking into account the potential consequences of postural changes associated with orthodontic intervention, the clinician should be aware of the implications that these may have not only in the interpretation of signs and symptoms but eventually in a clinical relapse, after orthodontic treatment. Thus, it is important to identify what characteristics are present in cases where relapse (occlusal and/or TMDs symptoms) occurs and, considering the impairments found, to clarify what intervention is best suited to address those impairments.

Considering the lack of consensus in the literature regarding physiotherapy effectiveness on TMDs, and acknowledging that the physiotherapist performance may be constrained by lack of mastering its management, due to the complexity of the condition (multifactorial aetiology, risk factors identification, differential diagnosis, patients' empowerment, multitude of available treatment modalities) the main aim of this work was to identify the global burden of TMDs in order to ascertain how the physiotherapist's role may be enhanced, in the light of the biopsychosocial model. Taking this into account, the specific objectives defined were:

1. To analyse the methodological quality, summarize the findings, and perform a meta-analysis of the results from randomized controlled trials that assessed the effects of physiotherapy management of TMDs.
2. To quantify the severity and prevalence of temporomandibular disorders in the Portuguese general population and also to assess the association between TMD's severity and demographic, medical and oral risk factors.
3. To develop and validate a scale to assess common knowledge about TMDs in the general population as well as to evaluate the status of TMDs knowledge in the Portuguese population.
4. To compare craniocervical posture, hyoid bone position and craniofacial morphology before, after orthodontic treatment, and also in the contention phase and verify if the presence of condylar displacement, the skeletal Class or the facial biotype interfere with the above mentioned outcomes.
5. To understand the different skeletal, muscular, facial and occlusal characteristics presented by TMDs patients that may benefit from a particular intervention, namely physiotherapy and its immediate effectiveness.

The present thesis is structured accordingly with the scandinavian model and is divided in six chapters.

The first chapter concerns the introduction about TMDs definition, aetiology, incidence, economic impact, pathophysiology, diagnosis and intervention. This chapter ends with the objectives of the research project. The second chapter, entitled "State of the Art" encompasses a published systematic review and meta-analysis regarding Physiotherapy effectiveness on TMDs. The third chapter is entitled "Original research manuscripts" and is composed by four original studies aiming to answer the proposed objectives. In the fourth chapter, the “Discussion” chapter, a general and integrated discussion regarding the results of the original studies is presented. The fifth chapter encompasses the main conclusions from the obtained results and also some perspectives for future research. The last chapter presents the bibliographic references that support the first and fourth chapters.

CHAPTER 2

STATE OF THE ART

STUDY I

The Effectiveness of Physiotherapy in the Management of Temporomandibular Disorders: A Systematic Review and Meta-analysis

Reprint with permission from

Journal of Oral & Facial Pain and Headache

The Effectiveness of Physiotherapy in the Management of Temporomandibular Disorders: A Systematic Review and Meta-analysis

Maria Paço, MSc
PhD Student
CESPU
Instituto de Investigação e Formação Avançada em Ciências e Tecnologias da Saúde
Gandra PRD, Portugal

Bárbara Peleteiro, PhD
Professor
EPI Unit
Institute of Public Health
Department of Clinical Epidemiology, Predictive Medicine and Public Health
University of Porto
Porto, Portugal

José Duarte, PhD
Professor
CIAFEL
The Research Centre in Physical Activity, Health and Leisure
Faculty of Sport
University of Porto
Porto, Portugal

Teresa Pinho, PhD
Professor
CESPU
Instituto de Investigação e Formação Avançada em Ciências e Tecnologias da Saúde
Gandra PRD, Portugal
IBMC
Instituto de Biologia Molecular e Celular
i3S
Instituto de Investigação e Inovação
University of Porto
Porto, Portugal

Correspondence to:
Dr Teresa Pinho
Rua Central de Gandra, 1317, 4585-116
Gandra PRD, Portugal
Phone: +351224157151
Email: teresa.pinho@iscsn.cespu.pt

Aims: To analyze the methodologic quality, summarize the findings, and perform a meta-analysis of the results from randomized controlled trials that assessed the effects of physiotherapy management of temporomandibular disorders. **Methods:** A literature review was performed using the electronic databases PubMed, Science Direct, and EBSCO. Each article was independently assessed by two investigators using the Physiotherapy Evidence Database (PEDro), Jadad scales, and the Cochrane Risk of Bias tool. A meta-analysis was conducted by using the DerSimonian-Laird random-effects method to obtain summary estimates of the standardized mean differences (SMD) and the corresponding 95% confidence intervals (95% CI). Between-study heterogeneity was computed and publication bias was assessed. **Results:** Seven articles met the inclusion criteria and were used in the analysis, corresponding to nine estimates of SMD. The meta-analysis showed that for pain reduction, the summary SMD favored physiotherapy (SMD = −0.63; 95% CI: −0.95 to −0.31; number of studies = 8; I^2 = 0.0%), while for active range of movement (ROM) the differences between the intervention and control groups were not statistically significant (SMD = 0.33; 95% CI: −0.07 to 0.72; number of studies = 9; I^2 = 61.9%). **Conclusion:** Physiotherapy seems to lead to decreased pain and may improve active ROM. However, the results are not definitive and further studies and meta-analyses are needed before these results can be considered fully generalizable. *J Oral Facial Pain Headache 2016;30:210–220. doi: 10.11607/ofph.1661*

Keywords: *mandibular function, pain, RCT*

Temporomandibular disorders (TMD) consist of a group of pathologies affecting the masticatory muscles, the temporomandibular joint (TMJ) and associated structures, or both.[1,2] The etiology of TMD is not clear,[3,4] but these disorders are the most common chronic orofacial pain conditions with prevalence studies demonstrating that TMD can affect from 10 to 25% of the population.[5,6] The presence of persistent pain is the main reason that TMD patients seek medical aid.[4] Other signs and symptoms usually manifested by TMD sufferers are impaired range of mandibular movement, joint sounds, and muscle and joint tenderness as well as head and neck pain.[7] This variety of signs and symptoms reveals the complexity of the condition, which has a multitude of risk factors.[8]

Currently, TMD may be managed by a combination of physiotherapy, splint therapy, orthodontics, pharmacotherapy, counseling, and surgery, among others.[9–13] Noninvasive treatments tend to be the first option for approximately 85 to 90% of TMD patients.[12] In the case of physiotherapy, two systematic reviews performed in 2006[14,15] concluded that the studies reviewed had methodologic problems that affected any possible conclusions about the effectiveness of physiotherapy in treating TMD. Since then, new studies[16–18] attempting to overcome these problems have been conducted, but the effectiveness of physiotherapy interventions in the management of TMD is still unclear. Thus, the aim of this systematic review was to analyze the methodologic quality, summarize the findings, and perform a meta-analysis of the results from randomized controlled trials (RCTs) that assessed the effects of physiotherapy management of TMD.

Materials and Methods

Data Sources

The following electronic databases were searched from their inceptions up to August 2014: PubMed, EBSCO, and Science Direct. The search expression used was built according to medical subject headings (MeSH) terms [("craniomandibular disorders" OR "temporomandibular disorders" OR "orofacial pain" OR "temporomandibular joint dysfunction") AND (physiotherapy OR "physical therapy" OR rehabilitation OR exercises OR "manual therapy")] and restricted to articles published in English, Portuguese, French, or Spanish. In addition, a manual search for further relevant articles in the references of all the included studies was performed.

Study Selection

Types of Studies. This systematic review included RCTs that assessed the effects of physiotherapy treatment regardless of blinding.

Types of Participants. The review included studies with subjects diagnosed with TMD by any specified diagnostic criteria regardless of their age, gender, or race. Studies evaluating patients with TMD found to be caused by psychogenic, neurologic, or metabolic disorders were excluded, as well as those with patients who had undergone TMJ surgery.

Types of Interventions. Interventions performed by therapists and within the scope of physiotherapy practice (ie, manual therapy, dry needling, exercise therapy) were included. Studies with nonphysiotherapy interventions, acupuncture, solely home-physical therapy or electrical modalities, and interventions involving passive range of movement (ROM) devices were excluded, along with studies with mixed treatments (physiotherapy combined with other forms of treatment).

Outcome Measures

Studies were not included in the analysis if they did not assess at least one of the following outcomes: pain and/or mandibular function.

Data Extraction and Quality Assessment

Two independent reviewers (P.M.; P.T.) screened the titles and abstracts of retrieved articles to determine their eligibility according to the criteria listed above. Quality assessment was performed using the Cochrane Risk of Bias tool,[19] the Physiotherapy Evidence Database (PEDro) scale,[20] and the 5-point Jadad scale.[21] The Cochrane Risk of Bias tool assesses six domains: *(1)* selection bias (random sequence generation, allocation concealment); *(2)* performance bias (blinding of participants and personnel); *(3)* detection bias (blinding of outcome assessment); *(4)* attrition bias (incomplete outcome data); *(5)* reporting bias (selective reporting); and *(6)* other bias. The PEDro scale was developed to rate the methodologic quality of trials and includes 11 items. While the first item evaluates external validity and is not used to calculate the PEDro score, the following 8 items deal with a trial's internal validity, and the last 2 items are relevant to the trial's statistical reporting. The PEDro score ranges from 0 (poor quality) to 10 (high quality). The 5-point Jadad scale has been previously validated[21] and focuses on three dimensions of internal validity: quality of randomization, double-blinding, and withdrawals. The score ranges from 0 (poor quality) to 5 (high quality). A trial scoring at least 3 out of 5 is considered to be of strong quality while scores lower than 3 indicate poor quality.

When discrepancies occurred between reviewers on whether the study should be included in the review, the reasons for disagreement were analyzed, the trial report was consulted, and a consensus was achieved. The procedure was the same regarding data extraction.

Meta-analysis

The standardized mean difference (SMD) of each individual study was calculated by determining the difference between the mean outcomes of the intervention's effectiveness and, in the control group, dividing by the pooled standard deviation. If data were not in a form suitable for quantitative pooling, trial authors were contacted for additional information. When necessary, transformations were performed by using the method described by Hozo et al in order to pool data.[22] Summary SMDs and the corresponding 95% confidence intervals (95% CI) were computed with STATA, version 11.2, using the DerSimonian-Laird random-effects method.[23] Heterogeneity between the studies was quantified by using the I^2 statistic.[24] Visual inspection of the funnel plots and Egger's regression asymmetry tests were used to assess publication bias.[25] A P value of $< .05$ was considered to reflect statistical significance.

Results

The search identified 3,243 potentially relevant studies, 3,218 of which were excluded after screening the titles and/or abstracts. After the full-text reading, only seven studies fulfilled all inclusion criteria and were used in the qualitative and quantitative analysis (Fig 1). For the quantitative analysis, the intervention group in the study by Carmeli et al[26] was divided into two subgroups (B1 [pain-dominant patients] and B2 [impaired ROM–dominant patients]) and one of the studies by Kalamir et al[17] had two intervention groups

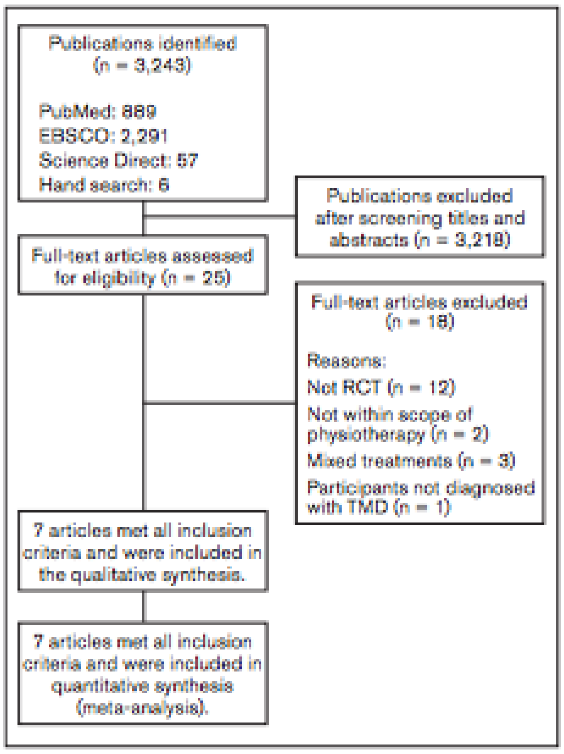


Fig 1 PRISMA flowchart of study selection process.

(intraoral myofascial therapy [IMT] and intraoral myofascial therapy plus education and self-care [IMT + ESC]); these data were analyzed independently.

A total of 329 patients were included in these studies (mean sample size: 47 participants). The main data are summarized in Table 1.

Diagnosis

In six of the seven studies, the TMD diagnostic method used was the Research Diagnostic Criteria for TMD (RDC/TMD);[16,17,27–30] the seventh study (Carmeli et al[26]) made the diagnosis according to the patients' medical history, radiographs, and medical and dental examinations. All subjects in the study by Carmeli et al[26] were diagnosed as having anterior displaced discs. In the studies classified according to the RDC/TMD, one study had patients diagnosed within groups IIb and IIc (disc displacement with and without limitations of mouth opening, respectively),[27] four had group I (muscle disorder) patients,[16,17,28,29] and the study by Tuncer et al[30] had patients from groups I (muscle disorders) and IIa (disc displacement with reduction). The duration of TMD was generally more than 12 weeks (chronic TMD), although this parameter was not described in two studies[17,29] and one study described the duration being from several weeks to years without quantifying the period.[27]

Groups at Baseline

Except for the study by Carmeli et al,[26] all studies reported the baseline comparisons of TMD symptoms. The comparisons showed similarities between groups in three of the studies.[27,28,30] The baseline measurements by Craane et al[16] showed that the intervention group had significantly higher pressure pain threshold (PPT) levels for the affected masseter muscle and temporalis compared with the control group. Differences between groups at baseline were also found in both studies by Kalamir et al; in the first[17] the intervention group had a greater opening range and in the second[29] the intervention group had a greater opening range in addition to higher average pain scores.

Description of Interventions

The duration of total treatment ranged from 1 day to 6 weeks (mean = 5 weeks). Of the included studies, one used a single treatment to test immediate effects of dry needling,[28] three studies performed a 5-week protocol (one with 15 treatments[26] and two with 10 treatments[17,29]), one performed a 4-week protocol (12 treatments),[30] and two had an intervention period of 6 weeks which was comprised of nine treatment sessions.[16,27] Two trials evaluated manual therapy with additional exercise,[26,27] three trials assessed manual therapy combined with home physical therapy,[16,17,30] one trial studied the effect of manual therapy alone,[29] and one trial studied the effect of dry needling.[28]

Manual mobilization and active exercises were compared with an individually designed polyethylene soft occlusal repositioning splint[26] and with a control group.[27] The participants who underwent manual therapy combined with home physical therapy were compared with a control group,[16] a wait-list control group, and two groups of participants who underwent manual therapy alone[17] or home physical therapy alone.[30] Manual therapy alone was compared with ESC[29] and the study by Fernández-Carnero et al[28] compared the effect of dry needling on active trigger points with a sham intervention.

Adverse Events

Only three studies stated that there were no adverse events.[17,29,30] The others failed to mention either the presence or absence of adverse events.

Methodologic Quality

The methodologic quality of the included studies varied. Figure 2 represents the data from the Cochrane Risk of Bias tool analysis. When assessed with the Jadad scale, six of the seven studies were shown to have strong methodologic quality (score higher than 3). Those six studies also had PEDro scores of strong quality (scores higher than 7).[16,17,27–30]

Outcomes

The seven studies utilized nine different outcome measures. The outcome measures found in the studies were visual analog scale (VAS), pain physiopathology instrument scale, 11-point graded chronic pain scale (CPS), 10-cm numeric pain rating scale (NPRS), McGill Pain Questionnaire (MPQ), PPT, mandibular function impairment questionnaire (MFIQ), 7-point global reporting of changes, and jaw opening (interincisal distance).

Pain. The included studies used different instruments to assess pain. All seven studies used "at rest" or "current" to describe the pain. Other measures included pain "with stress" (chewing),[30] "upon maximal active opening" and "upon clenching,"[17,29] and also the "worst" and the "lowest" levels of pain experienced in the preceding 24 hours.[28]

All studies evaluated pain at baseline and after the total treatment protocol, and some also at 3 weeks after baseline (during the treament protocol),[16,27] 6 weeks posttreatment,[16,27] 20 weeks posttreatment,[16,27] 24 weeks posttreatment,[17] 46 weeks posttreatment,[16,27] and 1 year posttreatment.[17]

VAS for Pain Intensity at Rest. Three studies assessed pain intensity through a VAS.[16,27,30] One study showed that physiotherapy resulted in significant pain reduction ($P < .01$)[30] while the studies by Craane et al[16,27] found no significant differences between the physiotherapy and control groups ($P > .05$).

MPQ. Two studies assessed pain by using the MPQ[16,27] and both found no significant differences between the physiotherapy and control groups ($P > .05$).

PPT. Three studies assessed the participants' PPTs over the masseter muscle[16,27,28]; two of these studies found no significant differences between groups ($P > .05$)[16,27] while the study by Fernández-Carnero et al[28] found greater improvements in the intervention group (dry needling) when compared with the control group (sham) ($P < .001$).

Other Pain Measurements. Other pain measurements included the pain physiopathology instrument, which showed that physiotherapy was significantly better than splint therapy in reducing pain ($P < .05$).[26] The studies by Kalamir et al[17,29] utilized an 11-point graded CPS and found a significant difference between the IMT and ESC groups that favored the IMT group ($P < .001$), although this difference was not clinically significant.[29] A significant difference between the treatment groups (IMT and IMT + ESC) and control group was also found with the 1-year assessment showing significantly lower pain scores in the IMT + ESC group when compared with both the IMT group and the control group.[17] The study by Fernández-Carnero et al[28] used a 10-cm NPRS and showed significant differences favoring the intervention (dry needling) when compared with the sham group ($P < .001$).

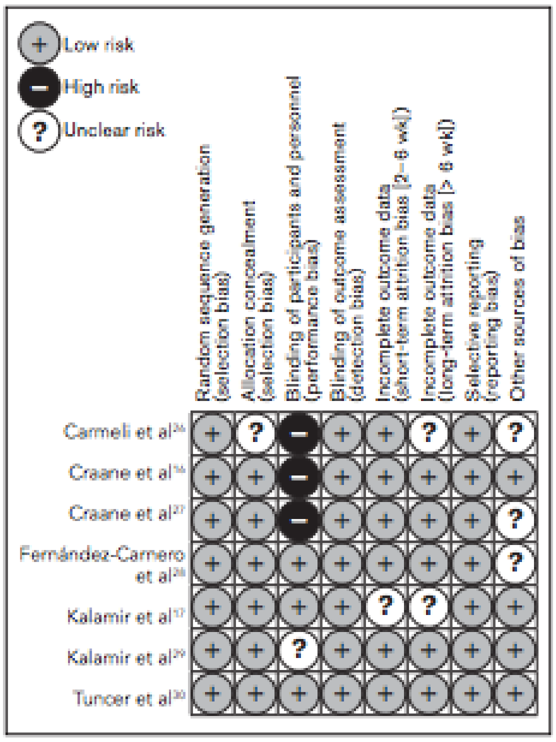


Fig 2 Risk of bias summary.

Meta-analysis Regarding Pain at Rest. Figure 3 represents the meta-analysis of pain at rest in all the included studies except for the study by Fernández-Carnero et al,[28] as their outcome measure was through PPT, a very different instrument whose data could not be grouped with the rest of the data.

The summary SMD showed that globally, there was a statistically significant improvement favoring intervention (SMD = −0.63; 95% confidence intervals [CI]: −0.95 to −0.31). The I^2 result showed no heterogeneity between studies.

When a sensitivity analysis was performed restricting the analysis to studies that presented the same diagnostic criteria, the estimated summary remained similar (SMD = 0.59; CI: −0.98 to −0.21; number of studies = 6; I^2 = 20.5%).

Figure 4 represents the funnel plot concerning the publication bias for pain at rest. Egger's regression asymmetry test shows no evidence of publication bias ($P = .264$).

Mandibular Function. Mandibular function was assessed through the MFIQ,[16,27] passive jaw opening,[16,27] and also by maximum active jaw opening in all included studies.

Table 1 Data Extracted from the Included Studies

Study ID	Objective	Method/ study design	No. of participants	Diagnoses	Interventions	Outcome measures: Outcome	Outcome measures: Scale/Instrument
Carmeli et al[26] (2001)	To compare the results of two treatment protocols (mobilization with active exercises and soft repositioning splint) for the management of ADTMD syndrome.	RCT	36	ADTMD	G1: Soft flat plane occlusal repositioning splint (n = 18) G2: Manual mobilization and active exercises (n = 18)	1. Active ROM of mouth opening 2. Pain	1. Fabric measuring tape 2. PPI scale
Craane et al[27] (2012)	To investigate the effect of physical therapy on pain and mandibular function in patients with ADD-R of the TMJ in a randomized controlled trial.	RCT	49	TMD (IIb and IIc according to RDC/TMD)	G1: Physical therapy (n = 23) G2: Control (n = 26)	1. Pain 2. Mandibular function 3. MMOa 4. MMOp	1. MPQ, VAS, and PPT 2. MFIQ 3. Interincisal distance at MMO (plastic ruler, mm) 4. Interincisal distance at mouth opening (plastic ruler, mm)
Craane et al[16] (2012)	To investigate the effect of physical therapy on pain and mandibular function in patients with masticatory muscle pain (with RDC-TMD Axis I, Ia, or Ib diagnosis) using a randomized and controlled design.	RCT (single-blind, randomized, controlled trial with a 1-year follow-up)	53	TMD (I, Ia, or Ib according to RDC/TMD)	G1: Treatment (n = 26) G2: Control (n = 27)	1. Pain 2. Mandibular function 3. MMOa 4. MMOp	1. MPQ, VAS, and PPT 2. MFIQ 3. Interincisal distance at MMO (plastic ruler, in mm) 4. Interincisal distance at mouth opening (plastic ruler, in mm)
Kalamir et al[17] (2012)	To investigate whether IMT and IMT + ESC treatments are superior to no treatment and to investigate whether IMT + ESC is superior to IMT over the course of 1 year.	RCT	93	Chronic myogenous TMD (according to RDC/TMD)	G1: Control (n = 31) G2: IMT (n = 31) G3: IMT + ESC (n = 31)	1. Pain (at rest, upon MMOa, upon clenching) 2. Interincisal range of opening 3. Global reporting of changes	1. 11-point GCPS 2. Vernier calipers (in mm) 3. 7-point global reporting of changes
Kalamir et al[29] (2013)	To compare the short-term effects of ESC to those of IMT on pain and opening ROM in participants with chronic myogenous TMD.	RCT	46	Myogenous TMD (according to RDC/TMD)	G1: ESC (n = 22) G2: IMT (n = 23)	1. Jaw pain at rest 2. Jaw pain upon MMOa 3. Jaw pain upon clenching 4. Maximal voluntary interincisal opening range (in mm)	1, 2, and 3: 11-point NPRS 4. N/A
Tuncer et al[30] (2013)	To compare the short-term effectiveness of MT + HPT and HPT alone.	RCT	40	TMD, (I, IIa according to RDC/TMD)	G1: HPT (n = 20) G2: MT + HPT (n = 20)	1. Pain intensity at rest 2. Pain intensity with stress 3. Pain-free MMO	1. VAS 2. VAS 3. Measured the interincisal distance (in mm)
Fernández-Carnero et al[28] (2010)	To investigate the effects of dry needling over active trigger points in the masseter muscle in patients with TMD.	RCT, crossover	12	TMD, myofascial pain (according to RDC/TMD)	G1: Deep dry needling (n = 12) G2: Placebo (n = 12)	1. Pain intensity (current, worst, lowest) 2. Pain-free MMO	1. 10-cm NPRS, PPT 2. The distance between the upper and lower central dental incisors (in mm).

ADTMD = anterior displaced temporomandibular disc syndrome; ADD-R = anterior disc displacement without reduction; IMT = intraoral myofascial therapy; ESC = education and self care; MT = manual therapy; HPT = home physical therapy; ROM = range of movement; MMOa = maximal active mouth opening; MMOp = maximal passive mouth opening; PPI = pain physiopathology instrument; MPQ = McGill pain questionnaire; VAS = visual analog scale; PPT = pressure pain threshold; MFIQ = mandibular function impairment questionnaire; GCPS = graded chronic pain scale; NPRS = numeric pain rating scale.

Results	Authors' conclusion	Methodologic quality PEDro	Methodologic quality Jadad
Manual mobilization and exercises demonstrated a significant decrease in total average pain level ($P < .05$) for the patients in G2; occlusal splints did not demonstrate a significant decrease ($P > .05$) in G1. Concerning active ROM of mouth opening, no significant increase was found in G1 ($P > .05$), but a significant increase was found in G2 ($P < .05$). The comparison between groups showed that G2 was significantly better than G1 in reducing pain ($P < .05$). No results on the comparison between groups concerning ROM.	Manual mobilization and active exercises are more effective for treatment of pain and ROM deficits associated with ADTMD than soft repositioning occlusal splint therapy.	5	1
No differences between groups at baseline. All pain variables decreased and all function variables increased significantly over time for both groups. The interaction between time and treatment group was not significant.	Physical therapy had no significant additional effect in patients with ADD-R.	8	3
At baseline there were no significant differences between groups, except in PPT levels for the affected masseter and temporalis muscles, which were significantly higher in the treatment group. Both groups improved significantly over time for VAS pain intensity, PPT, function by MMOa and MMOp, and by MFIQ ($P < .05$), but no significant differences between groups was found.	These findings indicate that independent of the treatment provided, all participants improved over time. There was no specific therapeutic effect of physical therapy on MMO.	7	3
At baseline there were no significant differences between groups except for opening ROM, which was greater in both treatment groups. Both treatment groups had significantly lower pain scores than the control group after baseline. By the 1-year assessment, the IMT + ESC group had significantly lower pain scores than the IMT group, which was not apparent at the 6-week or 6-month assessment. Global reporting of changes showed significant differences in change in scores between the groups, with the IMT + ESC group showing the best scores at 1 year. In both treatment groups, outcomes remained significantly different from the control group even at 1 year.	IMT + ESC can be safely used and may be superior to no treatment as well as IMT alone at 1 year.	8	5
Results for the pain scores (at rest, maximal opening, and clenching) indicated statistically significant differences between groups ($P < .001$); however, this difference was not clinically significant. Results for opening range showed that the difference between groups was not significant ($P = .416$), although the difference intragroup was significant in both ESC and IMT groups ($P = .025$ and $.032$, respectively).	IMT showed significantly lower mean pain scores when compared with ESC, and there were significantly higher odds of IMT achieving a 2 or more point decrease in pain scores in myogenous TMD sufferers. Both treatments indicated positive effects over time; however, the short duration of the trial suggests that the results should be interpreted with caution.	7	5
VAS scores (for pain at rest and pain with stress) significantly decreased in both groups over time ($P < .01$). Time*treatment effect as well as treatment effect were significant only for pain with stress in the MT + HPT group ($P < .01$). On the VAS, mean change scores for pain at rest were 34.6% on HPT and 59.2% on MT + HPT, and for pain with stress there was a decrease of 35.7% in the HPT group and 91.3% in the MT + HPT group ($P < .01$) Pain-free MMO significantly increased in both groups ($P < .01$). Time* treatment effect had a greater increase in the MT + HPT group compared with the HPT group ($P = .009$).	In the short term, MT in conjunction with HPT is more effective than HPT alone for the treatment of TMD, particularly with regard to decreasing pain and increasing pain-free mouth opening.	10	5
The ANOVA detected a significant interaction between intervention and time for PPT for PPT levels in the masseter muscle ($P < .001$) and in the condyle ($P < .001$) and pain-free MMO ($P < .001$). Subjects showed greater improvements in all the outcomes when receiving the deep dry needling compared with the sham dry needling ($P < .001$).	The application of dry needling into active trigger points in the masseter muscle induced significant increases in PPT levels and MMO when compared to sham dry needling in patients with myofascial TMD.	8	4

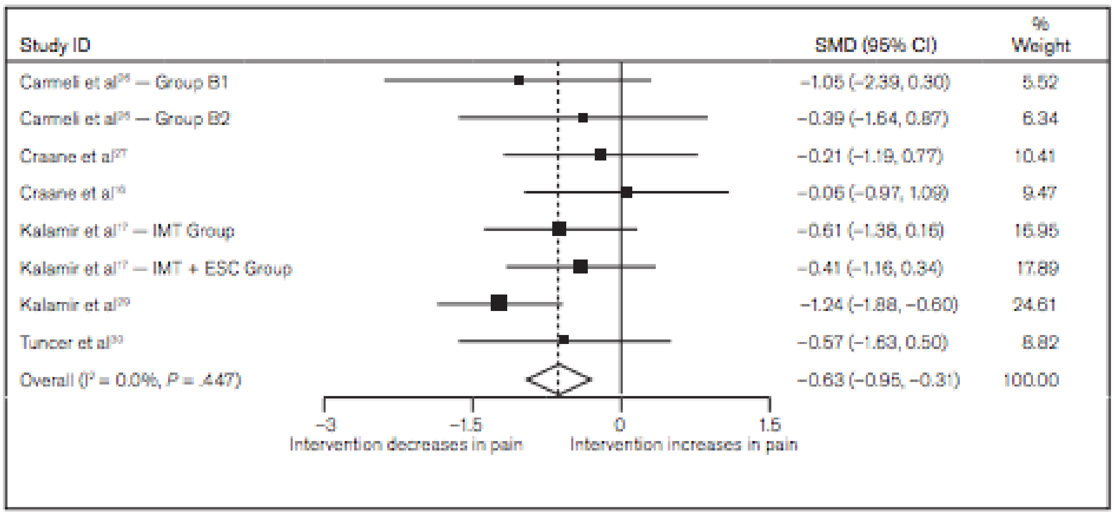


Fig 3 Forest plot of pain at rest.

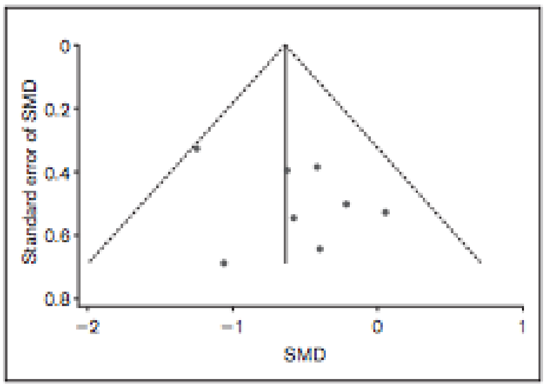


Fig 4 Funnel plot of publication bias for pain at rest. SMD = standardized mean difference.

MFIQ. Two studies assessed mandibular function by using the MFIQ and found no significant differences between the physiotherapy and control groups ($P > .05$).[16,27]

Passive Jaw Opening. Two studies assessed passive jaw opening and found no differences between the physiotherapy and control groups ($P > .05$).[16,27]

Active Jaw Opening. All seven studies assessed maximum active jaw opening. In the study by Carmeli et al,[26] results showed a significant increase in the experimental group ($P < .05$) while the control group failed to demonstrate a significant difference ($P > .05$). Notwithstanding, the results of the comparison between ROM data from the different groups were not found and could not be included in the analysis. Three studies found no differences between the physiotherapy and control groups ($P > .05$).[16,27,29] The study by Tuncer et al[30] revealed that pain-free mouth opening significantly increased in both experimental groups ($P < .01$), and that the time*treatment effect was greater for the experimental group (physiotherapy + home physical therapy) than for the control group (home physical therapy) ($P = .009$). Fernández-Carnero et al[25] also reported a greater improvement in the experimental group (dry needling) when compared with the sham group ($P < .001$), as did Kalamir et al,[17] who found the intervention was superior to the control group even after 1 year ($P < .001$).

Meta-analysis of Active ROM. Figure 5 represents the meta-analysis of active ROM data in all the included studies. The summary SMD shows that globally there was an improvement favoring intervention, although the differences found were not significant (SMD = 0.33; 95% CI: −0.07 to 0.72). The I^2 result revealed moderate heterogeneity between the studies.

When a sensitivity analysis was performed restricting the analysis to studies that presented the same diagnostic criteria, the estimated summary remained (SMD = 0.38; 95% CI: −0.08, 0.85; number of studies = 7; I^2 = 70.1%).

Figure 6 represents the funnel plot of publication bias for active ROM. Egger's regression asymmetry test shows no evidence of publication bias ($P = .575$).

Other Outcomes Measured. One study assessed the participants' perceptions of improvement through a 7-point global reporting of changes[17] and concluded that there were significant differences in the change in scores between the groups, with the

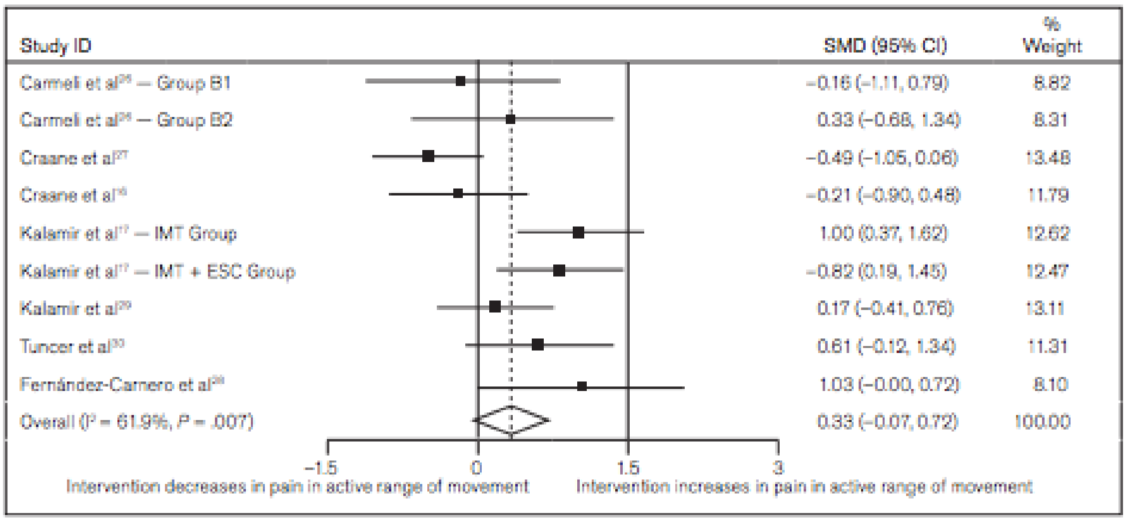


Fig 5 Forest plot of active range of movement.

IMT + ESC group showing the best scores at 1 year compared with the IMT group and the control group.

Discussion

A total of seven RCTs tested the effects of physiotherapy interventions compared with other interventions or control/placebo groups.

The methodologic quality based on the Cochrane Risk of Bias tool and the Jadad and PEDro scales was good, with an overall low risk of bias for all studies, except for the study by Carmeli et al,[26] which had a lower-quality score. All studies used an appropriate sequence generation, which reduced their risk of selection bias. Additionally, six studies employed allocation concealment;[16,17,27–30] however, in the study by Carmeli et al,[26] the risk of selection bias was unclear as the authors included no description of the allocation. Of the seven included studies, only three used double-blinding methods.[17,28,30] In the study by Kalamir et al,[29] there was an unclear risk because the blinding was incomplete, meaning that the participants were not blinded. This is a bias often found in physiotherapy intervention studies due to the difficulties of blinding not only the participant but also the therapist. Future studies should try to find ways to address these problems and assess and report the effectiveness of blinding. Four studies performed power analyses to calculate the required sample size[16,17,27,29] and three of these accounted for possible dropouts,[16,27,29] making these studies less susceptible to type II error.

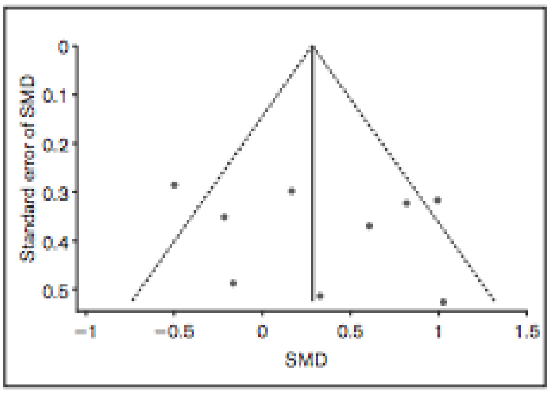


Fig 6 Funnel plot of publication bias for active range of movement. SMD = standardized mean difference.

Almost all the included studies reported details on dropouts,[16,17,27–30] diminishing the possibility of exclusion or attrition bias. Only the study by Carmeli et al[26] had no specific reference to dropouts, although throughout the text it is implicit that there were none. Additionally, the dropout rates of the included studies were very low, ranging from 0%[26,28,30] to 15%.[16] This may be due to the short duration of the trials (the longest trial had the highest dropout rate[16]) and even to the benign nature of the interventions. The dropout rates in the included studies were lower than in similar studies, which reported dropout rates from 15% to 30%.[31,32] Some of the dropout reasons cited were impatience with being on the waiting list,[17] changes in professional and personal life,[16,27,29] illness,[16,27] and insufficient decrease in patient complaints.[16]

The amount of physiotherapy treatment (ie, time per session and number of sessions) is an important clinical consideration and is quite variable. This variability is related to the patient's response to the treatment and the treatment technique selected, as there are so many different techniques within the scope of physiotherapy. This variability and the fact that there are several studies in which physiotherapy is performed by medical assistants or is considered to be any exercise or movement of the jaw is the reason why the present systematic review set the inclusion criterion that the treatment must be performed by a therapist and excluded studies that were solely hands-off. Additionally, while performing the review, several studies were found that used the word "exercise" to describe simply opening and closing the mouth. Therefore, the reviewers had a discussion in order to reach a consensus on what this analysis would consider to be physiotherapy; the definition included manual therapy techniques that are often performed not only by physiotherapists but also by chiropractors, osteopaths, and massage therapists, and also included exercise therapy, although studies in which opening or closing the jaw was considered an exercise without further consideration on how the movement was performed were excluded. In order to highlight the effectiveness of physiotherapy interventions unbiased and unmasked by the cumulative effects of other techniques, studies encompassing mixed treatments were excluded. This aspect narrowed the results but allowed a better understanding of the effects of physiotherapy in isolation and of the contribution of physiotherapy interventions in TMD management.

Since pain is one of the clinical signs of TMD and one of the main reasons that patients seek assistance,[2,7,33] this systematic review assessed the effects of physiotherapy interventions on pain and revealed findings that a physiotherapy intervention was significantly better in reducing pain than home physical therapy alone,[30] sham dry needling,[28] soft occlusal splints,[26] waiting-list control,[17] and ESC[29] (although in this last case, despite being statistically significant, the difference was not clinically relevant). It has been suggested that in patients with myogenous TMD, a change in pain of 24.2 mm on a VAS represents a clinically significant change;[34] and changes greater than 24.2 mm in patients with myogenous TMD were seen in the two studies that used a VAS.[16,30] Additionally, Farrar et al[35] found that a 27.9% decrease (or a 1.74-point decrease when assessed with an 11-point NPRS) in pain represents a clinically significant difference in patients with chronic pain; in the included studies that used this scale, the average change scores supported the clinical effectiveness of the intervention in pain.[17,29]

The meta-analysis results on pain at rest showed that a physiotherapy intervention produced a significant reduction in pain at rest. This reduction may be explained by peripheral and central mechanisms.[36] In response to injury, peripheral nociceptors and inflammatory mediators may act together, and manual therapy may directly affect this process.[36] In addition, manual therapy has been shown to trigger mechanical hypoalgesia, changes related to the activation of the sympathetic nervous system, and to the lessening of temporal summation, suggesting mechanisms that involve the periaqueductal gray and the spinal dorsal horn.[36,37] Schmid et al[37] found strong evidence to support the involvement of the central nervous system in mediating the response to manual therapy treatment. Several studies have been performed in order to study the mechanisms underlying manual therapy; however, none of these studies did so with an intervention directed at a TMD population.

The studies included in this review had many different control methods. Pertaining to ROM, physiotherapy was found to be superior to home physical therapy,[30] to sham dry needling,[28] and to the wait-list control group.[17] However, this difference was not found in the remaining studies. ROM did increase from baseline in the remaining studies,[16,27,29] showing a tendency for physiotherapy interventions to improve ROM, but the improvement was not statistically significant.

Each of the studies reported ethical approval of the study, although only three studies reported the absence of adverse events.[17,29,30] The fact that most of the studies did not report adverse events is alarming and should be taken into account in future studies.

Despite the increase in trials regarding the use of physiotherapy in the management of TMD, most of the published trials were not RCTs. Thus, the findings of these studies are less conclusive and generalizable as the risk of bias is much higher. It is therefore important for future RCTs to be performed in order to assess the effectiveness of physiotherapy in the management of TMD. It is also important to standardize assessment of the outcomes. Although almost all the included studies measured pain, ROM, and other mandibular function outcomes as dependent variables, the measurement instruments used were very different. Standardization of the outcome assessment instruments would allow researchers to pool data from multiple studies and to thereby draw consistent conclusions for the efficacious management of TMD. It will also be important to study further the pain mechanisms underlying physiotherapy interventions. In order to do this, investigations should include outcome measures designed to evaluate the multisystem effects of treatment, such as quantitative sensory testing protocols.[37–39]

One of the possible biases in the review process was the chosen definition of physiotherapy. The present researchers intended this definition to reflect the effects of "hands-on" physiotherapy interventions. Consequently, other potential studies with very different interventions may have been missed. However, all the included interventions were within the scope of physiotherapy and therefore reflect the effectiveness of physiotherapy.

Despite the small number of included articles, the meta-analysis assessed articles of high and very high quality and low risk of bias, allowing the reader to reach an evidence-based decision. However, the reader should take into account that the low number of included studies in the meta-analysis does not allow for definitive conclusions and that further studies are needed.

Conclusions

This systematic review and meta-analysis produced evidence that physiotherapy interventions are more effective than other treatment modalities and sham treatment in the management of TMD for pain reduction and that there was a tendency toward improved active ROM. However, these results are not definitive and should be interpreted with caution, mostly due to the small number of included studies and to the variability of the instruments used to assess the outcomes. Therefore, large-scale, high-quality, experimental studies with a standardized treatment protocol are needed to establish whether physiotherapy is effective and has real therapeutic value in the management of TMD.

Acknowledgments

The authors would like to thank the Institute of Research and Advanced Training in Health Sciences and Technologies. The authors report no conflicts of interest.

References

1. McNeill C. History and evolution of TMD concepts. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1997;83:51–60.
2. Okeson JP. Orofacial Pain: Guidelines for Assessment, Diagnosis and Management. Chicago: Quintessence, 1996.
3. Greenberg MS, Glick M, Ship JA (eds). Burket's Oral Medicine, ed 11. Hamilton, Ontario: BC Decker, 2008.
4. Oral K, Bal Küçük B, Ebeoğlu B, Dinçer S. Etiology of temporomandibular disorder pain. Ağrı 2009;21:89–94.
5. Gremillion HA. The prevalence and etiology of temporomandibular disorders and orofacial pain. Tex Dent J 2000;117:30–39.
6. LeResche L. Epidemiology of temporomandibular disorders: Implications for the investigation of etiologic factors. Crit Rev Oral Biol Med 1997;8:291–305.
7. Suvinen TI, Reade PC, Kemppainen P, Könönen M, Dworkin SF. Review of aetiological concepts of temporomandibular pain disorders: Towards a biopsychosocial model for integration of physical disorder factors with psychological and psychosocial illness impact factors. Eur J Pain 2005;9:613–633.
8. Poveda Roda R, Bagan JV, Díaz Fernández JM, Hernández Bazán S, Jiménez Soriano Y. Review of temporomandibular joint pathology. Part I: Classification, epidemiology and risk factors. Med Oral Patol Oral Cir Bucal 2007;12:E292–E298.
9. Ingawalé S, Goswami T. Temporomandibular joint: Disorders, treatments, and biomechanics. Ann Biomed Eng 2009;37:976–996.
10. Kalamir A, Pollard H, Vitiello AL, Bonello R. Manual therapy for temporomandibular disorders: A review of the literature. J Bodyw Mov Ther 2007;11:84–90.
11. List T, Axelsson S. Management of TMD: Evidence from systematic reviews and meta-analyses. J Oral Rehabil 2010;37:430–451.
12. Scrivani SJ, Keith Da, Kaban LB. Temporomandibular disorders. N Engl J Med 2008;359:2693–2705.
13. Yuasa H, Kino K, Kubota E, et al. Primary treatment of temporomandibular disorders: The Japanese Society for the temporomandibular joint evidence-based clinical practice guidelines, ed 2. Jpn Dent Sci Rev 2013;49:89–98.
14. McNeely ML, Olivo SA, Magee DJ. A systematic review of the effectiveness of physical therapy interventions for temporomandibular disorders. Phys Ther 2006;86:710–725.
15. Medlicott MS, Harris SR. A systematic review of the effectiveness of exercise, manual therapy, electrotherapy, relaxation training, and biofeedback in the management of temporomandibular disorder. Phys Ther 2006;86:955–973.
16. Craane B, Dijkstra PU, Stappaerts K, De Laat A. One-year evaluation of the effect of physical therapy for masticatory muscle pain: A randomized controlled trial. Eur J Pain 2012;16:737–747.
17. Kalamir A, Bonello R, Graham P, Vitiello AL, Pollard H. Intraoral myofascial therapy for chronic myogenous temporomandibular disorder: A randomized controlled trial. J Manipulative Physiol Ther 2012;35:26–37.
18. Mulet M, Decker KL, Look JO, Lenton PA, Schiffman EL. A randomized clinical trial assessing the efficacy of adding 6 x 6 exercises to self-care for the treatment of masticatory myofascial pain. J Orofac Pain 2007;21:318–328.
19. Higgins JPT, Green S. Cochrane Handbook for Systematic Reviews of Interventions, ed 5. http://handbook.cochrane.org . Accessed 19 July 2016.
20. de Morton NA. The PEDro scale is a valid measure of the methodological quality of clinical trials: A demographic study. Aust J Physiother 2009;55:129–133.
21. Jadad AR, Moore RA, Carroll D, et al. Assessing the quality of reports of randomized clinical trials: is blinding necessary? Control Clin Trials 1996;17:1–12.
22. Hozo SP, Djulbegovic B, Hozo I. Estimating the mean and variance from the median, range and the size of a sample. BMC Med Res Methodol 2005;5:13.
23. DerSimonian R, Laird N. Meta-analysis in clinical trials. Control Clin Trials 1986;7:177–188.
24. Higgins JP, Thompson SG. Quantifying heterogeneity in a meta-analysis. Stat Med 2002;21:1539–1558.
25. Sterne JA, Gavaghan D, Egger M. Publication and related bias in meta-analysis: Power of statistical tests and prevalence in the literature. J Clin Epidemiol 2000;53:1119–1129.
26. Carmeli E, Sheklow SL, Bloomenfeld I. Comparative study of repositioning splint therapy and passive manual range of motion techniques for anterior displaced temporomandibular discs with unstable excursive reduction. Physiotherapy 2001;87:26–36.

27. Craane B, Dijkstra PU, Stappaerts K, De Laat A. Randomized controlled trial on physical therapy for TMJ closed lock. J Dent Res 2012;91:364–369.
28. Fernández-Carnero J, La Touche R, Ortega-Santiago R, et al. Short-term effects of dry needling of active myofascial trigger points in the masseter muscle in patients with temporomandibular disorders. J Orofac Pain 2010;24:106–112.
29. Kalamir A, Graham PL, Vitiello AL, Bonello R, Pollard H. Intra-oral myofascial therapy versus education and self-care in the treatment of chronic, myogenous temporomandibular disorder: A randomised, clinical trial. Chiropr Man Therap 2013;21:17.
30. Tuncer AB, Ergun N, Tuncer AH, Karahan S. Effectiveness of manual therapy and home physical therapy in patients with temporomandibular disorders: A randomized controlled trial. J Bodyw Mov Ther 2013;17:302–308.
31. De Laat A, Stappaerts K, Papy S. Counseling and physical therapy as treatment for myofascial pain of the masticatory system. J Orofac Pain 2003;17:42–49.
32. Michelotti A, Steenks MH, Farella M, Parisini F, Cimino R, Martina R. The additional value of a home physical therapy regimen versus patient education only for the treatment of myofascial pain of the jaw muscles: Short-term results of a randomized clinical trial. J Orofac Pain 2004;18:114–125.
33. de Leeuw R, Klasser GD. Orofacial Pain: Guidelines for Assessment, Diagnosis, and Management, ed 5. Chicago: Quintessence, 2013.
34. van Grootel RJ, van der Glas HW. Statistically and clinically important change of pain scores in patients with myogenous temporomandibular disorders. Eur J Pain 2009;13:506–510.
35. Farrar JT, Young JP Jr, LaMoreaux L, Werth JL, Poole RM. Clinical importance of changes in chronic pain intensity measured on an 11-point numerical pain rating scale. Pain 2001;94:149–158.
36. Bialosky JE, Bishop MD, Price DD, Robinson ME, George SZ. The mechanisms of manual therapy in the treatment of musculoskeletal pain: A comprehensive model. Man Ther 2009;14:531–538.
37. Schmid A, Brunner F, Wright A, Bachmann LM. Paradigm shift in manual therapy? Evidence for a central nervous system component in the response to passive cervical joint mobilisation. Man Ther 2008;13:387–396.
38. Schmid AB, Nee RJ, Coppieters MW. Reappraising entrapment neuropathies--mechanisms, diagnosis and management. Man Ther 2013;18:449–457.
39. Backonja MM, Attal N, Baron R, et al. Value of quantitative sensory testing in neurological and pain disorders: NeuPSIG consensus. Pain 2013;154:1807–1819.

CHAPTER 3

ORIGINAL RESEARCH MANUSCRIPTS

STUDY II

Risk factors for temporomandibular disorders: evidence from a Portuguese population-based survey

Submitted for publication in

Journal of Oral & Facial Pain and Headache

Risk factors for temporomandibular disorders: evidence from a Portuguese population-based survey

Paço, M[1]; Simões, D[1,2]; Chaves, P[1]; Almeida, V[1]; Rocha, J.C[1]; Duarte, J.A.[3]; Pinho, T*[1,4];

[1]CESPU, Instituto de Investigacão e Formação Avançada em Ciências e Tecnologias da Saúde, Rua Central de Gandra, 1317, 4585-116 Gandra PRD, Portugal

[2]Escola Superior de Saúde de Santa Maria, Travessa Antero de Quental, 173, 4049-024 Porto, Portugal.

[3]CIAFEL, Faculdade de Desporto da Universidade do Porto, Rua Dr. Plácido Costa, 91 - 4200.450 Porto, Portugal.

[4]IBMC - Inst. Biologia Molecular e Celular., i3S - Inst. Inovação e Investigação em Saúde, Universidade do Porto.

* Corresponding author: Pinho, T; Rua Central de Gandra, 1317, 4585-116 Gandra PRD, Portugal; +351224157151; teresa.pinho@iucs.cespu.pt

ABSTRACT

Background: Temporomandibular disorders represent the most common chronic orofacial pain and has a multifactorial etiology. The literature is not conclusive regarding risk factors and it is important to ascertain what factors enhance the risk of developing this disorder. **Objectives:** (a) To quantify the severity and prevalence of temporomandibular disorders in the general population, (b) to assess the association between temporomandibular disorders' severity and demographic, medical and oral risk factors. **Methods:** Analytical cross-sectional survey design. The sample consisted of 2164 participants selected through a snow-ball sampling method. The participants completed an online questionnaire regarding social and demographic characteristics, medical history, oral habits, Fonseca's Anamnestic Index and a scale about difficulties on impulsiveness control. Chi-square and Kruskal-Wallis tests were used to compare proportions and continuous variables, respectively. A multinomial logistic regression was used to assess the association of putative determinants with the outcome. Crude and adjusted odds ratios and 95% confidence intervals were computed. The p-value set was 0.05. **Results:** 65.3% of the participants were identified has having TMDs (39.7% mild TMDs, 18.1% moderate TMDs and 7.5% severe TMDs). Multivariate-adjusted odds showed that female gender, diagnosis of tension-type headache, migraine, anxiety, impulsiveness, facial trauma and parafunctional habits increased the risk of developing TMDs (adjusted Odds Ratios from 1,84 to 49,38). **Conclusion:** A high prevalence of TMDs among the Portuguese population was found. The risk factors associated with TMDs were: female gender, impulsiveness, tension-type headache, migraine, anxiety, facial trauma and parafunctional habits.

Keywords: epidemiology, temporomandibular joint disorders, prevalence, chronic pain, cross-sectional study, psychological factors

INTRODUCTION

The American Academy of Orofacial Pain defines temporomandibular disorders (TMDs) as a group of musculoskeletal and neuromuscular conditions that involve the temporomandibular joints, the masticatory muscles and all associated structures.[1] It is the most common chronic orofacial pain condition with prevalence studies demonstrating that TMDs can affect from 10% to 25% of the population.[2,3] Notwithstanding its etiology is not yet well known and is considered to be multifactorial.[1] Several contributing factors have been described, such as morphological, neuromuscular, occlusal, psychological, genetic and parafunctional habits among others.[4-8] However, the different etiologies described in the literature needs further highlighting and remain as a subject of disagreement among researchers, conditioning the establishment of effective treatment plans.[9] Considering this, the aims of this study were (a) to quantify the severity and prevalence of TMDs in the general population and (b) to assess the association between TMDs severity and demographic, medical and oral risk factors.

MATERIALS AND METHODS

Study design

This is an analytical cross-sectional, survey design, carried out from September 2015 to March 2016. A snow-ball sampling was applied and intended to recruit participants not only from health related professions but also from the general population. Exclusion criteria were: age lower than 18 years old and not Portuguese citizens. Ethical approval was guaranteed by the Ethics Committee from Instituto Universitário de Ciências da Saúde, CESPU.

Procedures

A questionnaire was developed and the participants were asked about social and demographic characteristics, medical history, oral habits (bruxism, nails biting, gum chewing and any other parafunctional habit), Fonseca's Anamnestic Index and an Impulsiveness control scale. The questionnaire was built in an online survey software (Qualtrics©) and the link to the survey was sent to e-mail lists and through online social networks, asking every participant to invite others to participate in the study (by sharing the link to the questionnaire). This study was performed following the STROBE Statement guidelines.

Fonseca's Anamnestic Index (FAI)

FAI is a low cost and easy to apply instrument proposed in the Portuguese language, consisting of 10 questions whose answers are arranged in a three-point scale format ("No", "Sometimes", "Yes"). It is used to classify individuals according to TMDs severity (score 0-15: "TMDs Free", score 20-40: "Mild TMDs", score 45-60: "Moderate TMDs" and score 70-100: "Severe TMDs"), and also to screen patients for further developments in diagnosing TMDs.[10] Its main advantages are the simplicity of its application, and the fact that it does not need a physical examination of the patient, which makes it suitable for fast epidemiological screening by telephone, mail or internet surveys,[11] as in our study. It has a good correlation coefficient (r=0,6169) with the Helkimo Index.[10]

Difficulties on impulsiveness control scale

This scale evaluates the difficulties in controlling impulsiveness and a subscale from Difficulties in Emotion Regulation Scale (DERS)[12] composed by six items. The DERS is a measure of the difficulties of emotional regulation in a fully covering and comprehensive way, with 36 items, evaluating the frequency of feelings on a scale from 1 (almost never) to 5 (almost always). The scale items were organised in six factors: strategies, non-acceptance, awareness, impulse, goals and clarity. The final result of DERS reflects the flexibility of the individual on emotional regulation strategies use, effective and appropriate to the situation.[13] In the original version of the DERS the internal consistency was α = .93 and on the Portuguese validation study was α = .92.[14] The subscale "impulse" score ranges between 6 and 30, where a highest score represents a higher difficulty in controlling impulsiveness.

Statistical Analysis

Statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS), version 24 (IBM® company, Chicago, USA). Sample characteristics are presented as counts and proportions for categorical variables and median and interquartile range (IQR) for age and impulsiveness scale, since normal distribution was not confirmed. Chi-square and Kruskal-Wallis tests were used to compare proportions and continuous variables, respectively. A multinomial logistic regression was used to assess the association of putative determinants with the outcome. Crude and adjusted odds ratios and 95% confidence intervals were computed, taking participants without TMDs as the reference category of the outcome. From a list of a priori potential determinants, a final model was conducted comprising only variables associated with the outcome, even after adjustment. The critical value for significance in all the analysis was p-value < 0.05.

RESULTS

A sample of 2165 Portuguese citizens aged over 18 years old retrieved the online questionnaire complete, and formed the sample of our study (Table 1). All the Portuguese districts were represented in our sample, with the lowest rate response being from Évora (n=5) and the highest from Porto (n=1052). Overall, 1413 participants (65.3%) were identified has having TMDs according to FAI (score ≥ 20). Considering TMDs severity, 39.7% participants reported having mild TMDs (FAI score 20-40), 18.1% had moderate TMDs (FAI score 45-65), and 7.5% had severe TMDs (FAI score ≥ 70). In this sample, participants without TMDs were the oldest, with a median (IQR) age of 29.0 (16.0) years, while participants with severe TMDs were more impulsive, with a median (IQR) on the impulsiveness scale of 10.0 (6.0). Compared to those without TMDs, participants with TMDs were more often female and had more diagnosis of tension-type headache, migraine, depression, anxiety and obsessive-compulsive disorder (OCD). The prevalence of these diagnosis seems to be increasing along TMDs severity. Regarding dental history, the presence of facial trauma, parafunctional habits and orthodontic treatments were higher among participants with TMDs.

Molar extraction was higher among those with severe TMDs but not in those with mild or moderate TMDs (Table 1).

Study II - Table 1: Participants' characteristics, according to FAI (n=2165, Portugal, 2016).

	Temporomandibular Disorders Severity (FAI)				
	TMDs Free [a] n (%)	Mild TMDs [a] n (%)	Moderate TMDs [a] n (%)	Severe TMDs [a] n (%)	p
Overall	752 (34.7)	859 (39.7)	391 (18.1)	163 (7.5)	
Age					
Median (IQR)	29.0 (16.0)	26.0 (13.0)	26.0 (11.0)	27.0 (10.0)	<0.001
Sex					
Male	339 (45.1)	262 (30.5)	83 (21.2)	22 (13.5)	
Female	413 (54.9)	597 (69.5)	308 (78.8)	141 (86.5)	<0.001
Education (years)					
≤ 9	17 (2.3)	20 (2.3)	6 (1.5)	3 (1.9)	
10 – 12	216 (29.0)	266 (31.0)	115 (29.4)	44 (27.2)	
> 12	512 (68.7)	572 (66.7)	270 (69.1)	115 (71.0)	0.871
Impulsiveness score					
Median (IQR)	7.0 (4.0)	8.0 (4.0)	9.0 (5.0)	10.0 (6.0)	<0.001
Tension-type headache diagnosis					
No	738 (98.1)	802 (93.4)	331 (84.7)	118 (72.4)	
Yes	14 (1.9)	57 (6.6)	60 (15.3)	45 (27.6)	<0.001
Migraine diagnosis					
No	694 (92.5)	698 (81.4)	279 (71.5)	103 (63.2)	
Yes	56 (7.5)	160 (18.6)	111 (28.5)	60 (36.8)	<0.001
Depression diagnosis					
No	693 (92.3)	732 (85.3)	316 (81.0)	123 (75.9)	
Yes	58 (7.7)	126 (14.7)	74 (19.0)	39 (24.1)	<0.001
Anxiety diagnosis					
No	627 (83.5)	590 (68.8)	213 (54.6)	81 (49.7)	
Yes	124 (16.5)	268 (31.2)	177 (45.4)	82 (50.3)	<0.001
OCD diagnosis					
No	744 (99.2)	845 (98.5)	383 (98.0)	156 (95.7)	
Yes	6 (0.8)	13 (1.5)	8 (2.0)	7 (4.3)	0.010
Rheumatic diseases diagnosis					
No	723 (96.4)	810 (94.5)	365 (93.6)	150 (92.0)	
Yes	27 (3.6)	47 (5.5)	25 (6.4)	13 (8.0)	0.051
Non-invasive ventilation utilization					
No	747 (99.3)	855 (99.5)	390 (99.7)	162 (99.4)	
Yes	5 (0.7)	4 (0.5)	1 (0.3)	1 (0.6)	0.779
Facial Trauma					
No	625 (83.2)	644 (75.1)	299 (76.5)	124 (76.5)	
Yes	126 (16.8)	214 (24.9)	92 (23.5)	38 (23.5)	0.001
Parafunctional habits					
No	581 (77.3)	266 (31.0)	56 (14.3)	11 (6.7)	
Yes	171 (22.7)	593 (69.0)	335 (85.7)	152 (93.3)	<0.001
Orthodontic treatment					
No	468 (62.3)	515 (60.0)	210 (53.7)	85 (52.1)	
Yes	283 (37.7)	344 (40.0)	181 (46.3)	78 (47.9)	0.010
Molar removal					
No	583 (77.8)	691 (80.4)	315 (80.6)	114 (69.9)	
Yes	166 (22.2)	168 (19.6)	76 (19.4)	49 (30.1)	0.017
Dental prosthesis					
No	719 (95.9)	814 (94.8)	376 (96.2)	156 (96.3)	
Yes	31 (4.1)	45 (5.2)	15 (3.8)	6 (3.7)	0.579
Dental implant					
No	699 (93.1)	795 (92.5)	366 (93.6)	155 (95.1)	
Yes	52 (6.9)	64 (7.5)	25 (6.4)	8 (4.9)	0.666

In the crude analysis, and compared to participants without TMDs, lower age and higher impulsiveness were associated with a higher likelihood of having TMDs, regardless of severity. Female gender and a diagnosis of tension-type headache, migraine, depression, or anxiety was associated with a higher probability of having TMDs, with this association being stronger with increased severity. OCD diagnosis was only associated with severe TMDs. Regarding oral characteristics, we observed that participants with history of facial trauma had more chances of having TMDs, regardless of severity. Also, the presence of parafunctional habits was associated with a higher likelihood of TMDs severity. The existence of an orthodontic treatment increased the odds of having moderate or severe TMDs, while molar removal only increased the odds of having severe TMDs (Table 2).

Study II - Table 2: Crude odds ratio for the association between demographic, medical and oral characteristics, according to TMDs severity (Portugal, 2016).

	Temporomandibular Disorders Severity (FAI)		
	Mild TMDs	Moderate TMDs	Severe TMDs
		OR (95% CI) [a]	
Age	0.98 (0.97-0.99)	0.98 (0.96-0.99)	0.97 (0.96-0.99)
Sex			
Male	1	1	1
Female	1.87 (1.53-2.29)	3.05 (2.30-4.04)	5.26 (3.28-8.43)
Impulsiveness score	1.10 (1.06-1.13)	1.15 (1.11-1.19)	1.19 (1.14-1.24)
Tension-type headache diagnosis			
No	1	1	1
Yes	3.75 (2.07-6.78)	9.56 (5.27-17.34)	20.10 (10.70-37.76)
Migraine diagnosis			
No	1	1	1
Yes	2.84 (2.06-3.92)	4.93 (3.47-7.00)	7.22 (4.75-10.98)
Depression diagnosis			
No	1	1	1
Yes	2.06 (1.48-2.86)	2.80 (1.94-4.05)	3.79 (2.42-5.94)
Anxiety diagnosis			
No	1	1	1
Yes	2.30 (1.81-2.92)	4.20 (3.19-5.54)	5.12 (3.56-7.36)
OCD diagnosis			
No	1	1	1
Yes	1.91 (0.72-5.04)	2.59 (0.89-7.52)	5.56 (1.85-16.78)
Facial Trauma			
No	1	1	1
Yes	1.65 (1.29-2.11)	1.53 (1.13-2.06)	1.52 (1.01-2.29)
Parafunctional habits			
No	1	1	1
Yes	7.57 (6.06-9.47)	20.33 (14.61-28.28)	46.95 (24.88-88.62)
Orthodontic treatment			
No	1	1	1
Yes	1.11 (0.90-1.35)	1.43 (1.11-1.83)	1.52 (1.08-2.13)
Molar removal			
No	1	1	1
Yes	0.85 (0.67-1.09)	0.85 (0.63-1.15)	1.51 (1.04-2.20)

Table 3 presents the results of the multivariate analysis. Female sex, impulsiveness, and the presence of tension-type headache, migraine, depression, anxiety diagnosis, facial trauma or parafunctional habits were associated with a higher probability of having mild, moderate or severe TMDs, even after adjustment to each other. While impulsiveness and the presence of facial trauma seem to be associated with TMDs presence, regardless of severity, all the other characteristics seems to have an association with TMDs severity, since we found a stronger association in higher TMDs severity categories, independently of confounders. Parafunctional habits and tension-type headache diagnosis have the strongest associations with TMDs severity.

Study II - Table 3: Multivariate-adjusted odds ratio for the association between demographic, medical and oral characteristics, according to TMDs severity (Portugal, 2016).

	Temporomandibular Disorders Severity (FAI)		
	Mild TMDs	Moderate TMDs	Severe TMDs
	Adjusted OR (95% CI) [a b]		
Sex			
Male	1	1	1
Female	1.84 (1.44-2.35)	2.49 (2.27-9.13)	4.12 (2.43-7.00)
Impulsiveness score	1.07 (1.03-1.11)	1.10 (1.06-1.15)	1.14 (1.08-1.20)
Tension-type headache diagnosis			
No	1	1	1
Yes	2.22 (1.16-4.28)	4.55 (2.27-9.13)	9.11 (4.31-19.24)
Migraine diagnosis			
No	1	1	1
Yes	2.37 (1.63-3.44)	3.29 (2.13-5.07)	3.80 (2.26-6.40)
Anxiety diagnosis			
No	1	1	1
Yes	2.07 (1.56-2.73)	3.40 (2.42-4.76)	3.45 (2.24-5.33)
Facial Trauma			
No	1	1	1
Yes	1.86 (1.39-2.50)	1.89 (1.30-2.74)	2.12 (1.31-3.46)
Parafunctional habits			
No	1	1	1
Yes	7.58 (5.99-9.60)	22.70 (15.79-32.54)	49.38 (25.51-95.60)

95% CI, 95% confidence interval; OR, odds ratio.
Note: performed only with subjects with information for all the variables considered (n = 2113).
[a] TMDs free is the reference category.
[b] Each factor in the table is adjusted for every other factor in the table.

DISCUSSION

The present study provides data regarding the prevalence and severity of TMDs, based on the FAI in the Portuguese population. Our results showed that 65,3%

of the participants were classified as having TMDs, which is according with the literature. Similar prevalence rates have been found among students[15-17] which may be related with the stressful demands on this population related to study, responsibility and expectations.[18] Notwithstanding, these results should be interpreted with caution as the non-TMDs population may also present some signs and/or symptoms of TMDs.

Our results in the Portuguese population, regarding TMDs risk factors are consistent with findings of previous studies in other populations[3,19-21] and support the multifactorial aetiology proposed and accepted by the scientific community. Furthermore, this study has looked for factors not previously described as impulsiveness and has also retrieved results regarding factors with conflicting evidence in the literature, contributing to a better understanding of the risk factors to develop TMDs.

Female gender, facial trauma, parafunctional habits and psychological factors are frequently reported risk factors.[5,6,8,9,19,22,23] Other factor that has been reported in the literature is molar removal which is according with the results we have found.[24]

Our results show a higher prevalence and a higher risk for women to develop TMDs, which is according with other studies and may have biological, social and behavioural causes, consistent with the biopsychosocial model.[3,16,21] This may suggest a possible link between TMDs' pathogenesis and the estrogen.[25-27] Literature has shown that women present more sensitivity to most of pain modalities, suggesting a possible link between TMDs and the mechanisms of pain modulation.[27-29]

When analyzed the risk of developing severe TMDs, the presence of psychosocial factors like impulsiveness, anxiety, depression and OCD represent a high risk to develop it. Depression is sought to cause an increase in muscular tension that may spread to the pericranium muscles and might act as a cause for TMDs symptoms.[30] Several studies have shown that psychological-psychiatric problems seems to be associated with TMDs, with patients with psychological-psychiatric being more prone to TMDs than individuals without these problems[17,30-32] and that anxiety is intrinsically associated with an increase of the odds for pain-related TMDs by the factor 1.04.[20] Moreover, emotion regulation related disorders as anxiety-depressive disorders, somatisation and catastrophizing seems to contribute to chronic TMDs, mainly in the form of myofascial pain.[33] Considering our results, the specific underlying psychosomatic factor is associated with the ability to regulate high emotional activation. Similarly with other studies, our results have shown that the presence of parafunctional habits increased the risk of developing TMDs. Parafunctional habits are sought to contribute to TMDs because they can be considered a form of repetitive microtrauma that results in pain.[34] Furthermore this can be related with psychological issues, once disorders related to stress, anxiety and depression seems to intensify parafunctional activity that may lead to the onset or exacerbation of TMDs.[33]

Our study showed that molar extraction increased the odds of having severe TMDs, which has been shown in other studies.[19] One possible explanation may be the procedure involved in the removal of the molar, once it implies a wide opening of the mouth, for a considerable long period and associated with forces applied over the mandible, beyond its normal range of motion. This may constitute a trauma to the temporomandibular joint or even to the mastication muscles, and, because it is performed under anaesthesia, may be accompanied by a reduction in the protective mechanisms of the person under treatment.

The presence of facial trauma also increased the odds of developing TMDs, independently of its severity. It is well known that significant forces transmitted to the soft tissues of the TMJ and supporting structures can result in severe dysfunction.[35]

The presence of tension-type headache was found to be strongly associated with TMDs. Headache is one of the most common symptoms of TMDs patients,[16,30,36] while 55% of chronic headache patients referred to a neurologist had signs or symptoms of TMDs.[37] Tension-type headache is the most common type of primary headache and seems to have a neurobiological basis. The exact mechanisms of tension-type headache are not known, however peripheral and central pain mechanisms seems to play an important role in the different types of tension-type headache [38]. The presence of myofascial trigger points can generate nociceptive impulses, which, when sustained over time, may lead to a state of central sensitization. Moreover myofascial pain has been purported to play a key role in the establishment of tension-type headaches.[39] Furthermore, the temporomandibular joint has muscular, ligamentar and neural connections to the cervical region, creating a functional complex, with the potential to influence reciprocally.[34] The trigeminocervical nucleus is responsible for the input from the trigeminal nerve and craniocervical region, and seems to be one of the reasons why pain from any of the above inputs may be referred to cervical, face, head or mandibular region.[34,40]

All these contributing factors for TMDs makes difficult the correct assessment of the disease, which demonstrates the need to understand the physical and psychological characteristics of an individual patient. Hence, the degree of contribution of the different factors to TMDs may be related to individual differences among people and should be further studied.

All data analysed in our study were collected from a self-administered questionnaire, that relied on memory and self-reporting of the participants. Having this in mind, the authors recognize that there might have been incorrect answers to the questions, but due to the high rate of response as well as the fact that there were redundant questions, the impact of this possible bias is very low. Another limitation of this study is the absence of a clinical examination and laboratory findings for TMDs diagnosis' of the participants. Instead we have used a valid and reliable questionnaire (FAI) often used for epidemiological studies on TMDs, that allowed to characterize the signs and symptoms of TMDs and to get a score about TMDs severity.[10] Once this was a cross-sectional study, no etiological conclusions can be drawn and the reader should have in mind that no clinical confirmation of the data retrieved by the participants was available.

Although our study provided information regarding the prevalence and severity of TMDs in the general Portuguese population, long-term clinical studies should be performed in order to complement and confirm our data.

A significant point to be learned is the need to be aware of the several risk factors for TMDs, that translate the need for a thorough and early diagnosis as well as a preventive action of future complications associated with TMDs. These may play a key role in the success of TMDs treatment.

CONCLUSION

The results from our study showed a high prevalence of TMDs among the Portuguese population. The risk factors found to be associated with TMDs were: female gender, impulsiveness, tension-type headache, migraine, anxiety, facial trauma and parafunctional habits.

ACKNOWLEDGEMENTS:

CIAFEL – Research Centre in Physical Activity, Health and Leisure

IINFACTS - Institute of Research and Advanced Training in Health Sciences and Technologies.

REFERENCES

1 Leeuw R, Klasser GD. Orofacial Pain: Guidelines for Assessment, Diagnosis, and Management. 5th ed.: Quintessence Books, 2013.

2 Gremillion H. The prevalence and etiology of temporomandibular disorders and orofacial pain. Tex Dent J 2000;117:30-39.

3 LeResche L. Epidemiology of Temporomandibular Disorders: Implications for the Investigation of Etiologic Factors. Crit Rev Oral Biol Med 1997;8:291-305.

4 Melis M, Di Giosia M. The role of genetic factors in the etiology of temporomandibular disorders: a review. Cranio 2016;34:43-51.

5 Michelotti A, Cioffi I, Festa P, Scala G, Farella M. Oral parafunctions as risk factors for diagnostic TMD subgroups. J Oral Rehabil 2010;37:157-162.

6 Fillingim R, Ohrbach R, Greenspan J, et al. Potential psychosocial risk factors for chronic TMD: descriptive data and empirically identified domains from the OPPERA case-control study. J Pain 2011;12:T46-T60.

7 Cruz CL, Lee KC, Park JH, Zavras AI. Malocclusion Characteristics as Risk Factors for Temporomandibular Disorders: Lessons Learned from a Meta-Analysis. J Oral Dis 2015;2015.

8 Dıraçoğlu D, Yıldırım NK, Saral İ, et al. Temporomandibular dysfunction and risk factors for anxiety and depression. J Back Musculoskelet Rehabil 2016;29:487-491.

9 Magalhães BG, de-Sousa ST, de Mello VV, et al. Risk factors for temporomandibular disorder: binary logistic regression analysis. Med Oral Patol Oral Cir Bucal 2014;19:e232-236.

10 Fonseca D, Bonfante G, Valle A, Freitas S. Diagnóstico pela anamnese da disfunção craniomandibular. RGO (Porto Alegre) 1994;42:23-28.

11 Campos JC, A.C, Bonafé F, Maroco J. Severity of temporomandibular disorders in women: validity and reliability of the Fonseca Anamnestic Index. Braz Oral Res 2014;28:1-6.

12 Gratz K, Roemer L. Multidimensional Assessment of Emotion Regulation and Dysregulation: Development, Factor Structure, and Initial Validation

of the Difficulties in Emotion Regulation Scale. J Psychopathol Behav Assess 2004;26:41-54.

13 Gratz KL, Roemer L. Multidimensional Assessment of Emotion Regulation and Dysregulation: Development, Factor Structure, and Initial Validation of the Difficulties in Emotion Regulation Scale. J Psychopathol Behav Assess 2004;26:41-54.

14 Coutinho J, Ribeiro E, Ferreirinha R, Dias P. The Portuguese version of the difficulties in emotion regulation scale and its relationship with psychopathological symptoms. Rev Psiq Clín 2010;37:145-151.

15 Pedroni CR, De Oliveira AS, Guaratini MI. Prevalence study of signs and symptoms of temporomandibular disorders in university students. J Oral Rehabil 2003;30:283-289.

16 Kitsoulis P, Marini A, Iliou K, et al. Signs and Symptoms of Temporomandibular Joint Disorders Related to the Degree of Mouth Opening and Hearing Loss. BMC Ear Nose Throat Disord 2011;11:1-8.

17 Modi P, Shaikh SS, Munde A. A cross sectional study of prevalence of temporomandibular disorders in university students. International Journal of Scientific and Research Publications 2012;2:1-3.

18 Habib SR, Al Rifaiy MQ, Awan KH, Alsaif A, Alshalan A, Altokais Y. Prevalence and severity of temporomandibular disorders among university students in Riyadh. Saudi Dent J 2015;27:125-130.

19 Huang GJ, LeResche L, Critchlow CW, Martin MD, Drangsholt MT. Risk factors for diagnostic subgroups of painful temporomandibular disorders (TMD). J Dent Res 2002;81:284-288.

20 Reissmann DR, John MT, Seedorf H, Doering S, Schierz O. Temporomandibular disorder pain is related to the general disposition to be anxious. J Oral Facial Pain Headache 2014;28:322-330.

21 Suvinen TI, Reade PC, Kemppainen P, Könönen M, Dworkin SF. Review of aetiological concepts of temporomandibular pain disorders: towards a biopsychosocial model for integration of physical disorder factors with psychological and psychosocial illness impact factors. Eur J Pain 2005;9:613-633.

22 Ohrbach R, Fillingim RB, Mulkey F, et al. Clinical Findings and Pain Symptoms as Potential Risk Factors for Chronic TMD: Descriptive Data and Empirically Identified Domains from the OPPERA Case-Control Study. J Pain 2011;12:T27-T45.

23 Poveda Roda R, Bagan JV, Díaz Fernández JM, Hernández Bazán S, Jiménez Soriano Y. Review of temporomandibular joint pathology. Part I: classification, epidemiology and risk factors. Med Oral Patol Oral Cir Bucal 2007;12:E292-298.

24 Akhter R, Hassan NMM, Ohkubo R, Tsukazaki T, Aida J, Morita M. The relationship between jaw injury, third molar removal, and orthodontic treatment and TMD symptoms in university students in Japan. J Orofac Pain 2008;22:50-56.

25 Warren MP, Fried JL. Temporomandibular disorders and hormones in women. Cells Tissues Organs 2001;169:187-192.

26 Cairns BE. Pathophysiology of TMD pain--basic mechanisms and their implications for pharmacotherapy. J Oral Rehabil 2010;37:391-410.

27 LeResche L, Mancl L, Sherman J, Gandara B, Dworkin S. Changes in temporomandibular pain and other symptoms across the menstrual cycle. Pain 2003;106:253-261.

28 Fillingim R, King C, Ribeiro-Dasilva M, Rahim-Williams B, Riley J. Sex, gender, and pain: a review of recent clinical and experimental findings. J Pain 2009;10:447-485.

29 Bereiter D, Okamoto K. Neurobiology of estrogen status in deep craniofacial pain. In: Kobayashi M, Koshikawa N, Iwata K, Waddington J, editors. Translating Mechanisms of Orofacial Neurological Disorder. Academic Press, 2011:251-284.

30 Akhter R, Morita M, Ekuni D, et al. Self-reported aural symptoms, headache and temporomandibular disorders in Japanese young adults. BMC Musculoskelet Disord 2013;14:58.

31 Buljan D. Psychological and psychiatric factors of temporomandibular disorders. Rad Hrvatske Akademije Znanosti i Umjetnosti Medicinske Znanosti 2010;507:119-133.

32 Wright EF, Clark EG, Paunovich ED, Hart RG. Headache Improvement Through TMD Stabilization Appliance and Self-management Therapies. Cranio 2006;24:104-111.

33 Berger M, Oleszek-Listopad J, Marczak M, Szymanska J. Psychological aspects of temporomandibular disorders – literature review. Curr Issues Pharm Med Sci 2015;28:55-59.

34 Okeson J. Management of temporomandibular disorders and occlusion. 7th ed.: Elsevier Health Sciences, 2013.

35 Smith WS, Kracher CM. Sports-related dental injuries and sports dentistry. Dent Assist 1998;67:12-16.

36 Graff-Radford SB, Abbott JJ. Temporomandibular Disorders and Headache. Oral Maxillofac Surg Clin North Am 2016;28:335-349.

37 Schokker RP, Hansson TL, Ansink BJJ. The result of treatment of the masticatory system of chronic headache patients. J Craniomandib Disord 1990;4:126-130.

38 Headache Classification Committee of the International Headache Society (IHS). The international classification of headache disorders, (beta version). Cephalalgia 2013;33:629-808.

39 Bendtsen L. Central Sensitization in Tension-Type Headache—Possible Pathophysiological Mechanisms. Cephalalgia 2000;20:486-508.

40 Bogduk N, Govind J. Cervicogenic headache: an assessment of the evidence on clinical diagnosis, invasive tests, and treatment. Lancet Neurol 2009;8:959-968.

STUDY III

Knowledge about temporomandibular disorders: evidence from a Portuguese population-based survey

Submitted for publication in

Journal of Oral & Facial Pain and Headache

Knowledge about temporomandibular disorders: evidence from a Portuguese population-based survey

Paço, M.[1]; Chaves, P.[1]; Simões, D.[1,2]; Almeida, V.[1]; Rocha, J.C.[1]; Moreira, L.[1]; Duarte, J.A.[3]; Pinho, T.*[1,4];

[1]CESPU, Instituto de Investigacão e Formação Avançada em Ciências e Tecnologias da Saúde, Rua Central de Gandra, 1317, 4585-116 Gandra PRD, Portugal

[2]Escola Superior de Saúde de Santa Maria, Travessa Antero de Quental, 173, 4049-024 Porto, Portugal.

[3]CIAFEL, Faculdade de Desporto da Universidade do Porto, Rua Dr. Plácido Costa, 91 - 4200.450 Porto, Portugal.

[4]IBMC - Inst. Biologia Molecular e Celular., i3S - Inst. Inovação e Investigação em Saúde, Universidade do Porto, Portugal.

* Corresponding author: Pinho, T; Rua Central de Gandra, 1317, 4585-116 Gandra PRD, Portugal; +351224157151; teresa.pinho@iucs.cespu.pt

Abstract

Background and Aims: Knowledge about temporomandibular disorders (TMDs) may represent a health determinant being an important factor that influences health behaviors and attitudes. The present study aims primarily to develop and validate a scale to assess common knowledge about TMDs in the general population. The second aim is to evaluate the status of TMDs knowledge in the Portuguese population. **Methods:** Cross-sectional, descriptive survey design. The TMDs knowledge scale was developed in two phases: concept analysis and construction (Item development and identification of domains and pilot testing on a small number of participants) and testing the psychometric properties (n=210). For the second objective, 2165 participants selected through a snow ball sampling method, and the participants were asked to answer an online questionnaire. **Results:** Concerning psychometric properties all items showed a moderate-to-strong positive association with the loading factor. The Cronbach's Alpha was 0.956, showing good reliability. The median score of TMDs knowledge in the Portuguese population was 13.0 points (interquartile range: 10.0). Three hundred and thirty-four participants (15.4%) had 0 correct answers, while eighteen participants (0.8%) had 21 correct answers. TMDs knowledge was positive (equal or above 11 correct answers) in 1295 participants (59.8%). **Conclusion:** The results from our study showed that the TMDs knowledge scale developed is psychometrically valid and reliable. It also demonstrated that the participants had an overall positive knowledge about TMDs.

Key-words: Temporomandibular joint disorders, health literacy; Health education; Scale development; Scale validation

INTRODUCTION

Temporomandibular disorders (TMDs) comprises a group of varied conditions involving the temporomandibular joint, the masticatory muscles and surrounding structures.[1] Prevalence rates ranges from 10% to 25% of the population[2,3] being the most common, non-dental chronic orofacial pain condition. It is considered to have a multifactorial etiology, with several factors, such as morphological, neuromuscular, occlusal, psychological and parafunctional habits among others[4-8] that may contribute to the onset or aggravation of this problem.

There is a growing concern about the identification of health determinants, not only by the different political and social entities, but mainly by the medical and scientific community. Thus, it is important to enhance the practical and theoretical knowledge related with the source, orientation and changes of behaviors that promote health and quality of life of the individual and the community. This raising concern about the identification of health determinants, has shown that the level of knowledge and literacy is a factor proportionally related with the health level.[9] This determinant was defined by the World Health Organization as the cognitive and social skills of the individual, that determine the motivation and ability to access, understand and effectively use the information, as a strategy of health promotion and maintenance.[10] Health knowledge is an important trigger to change behaviors and attitudes[11] and contributes to the improvement of individuals' and populations' health.[12] Furthermore, concerning the ability of developing strategies to health maintenance and self-management, knowledge acts as an empowerment tool.[9,13] The biopsychosocial model reinforces the importance of empowering the patient through knowledge about diagnosis, prognosis and the nature of the problem, in order to involve the patient in the disease management, namely in therapeutic decision, especially regarding chronic conditions.[14] Adequate information has shown to provide self-management skills and better coping strategies in rheumatoid arthritis patients as well as compliance to therapy and treatment success.[15,16]

Thus, it becomes fundamental to ascertain the levels of individual and collective knowledge about potential health pathologies known to represent a health problem, in order to define strategic actions and organize literacy/education health programs, intended to influence individual and/or collective lifestyle decisions, allowing to explore the influence of this health determinant and modify its impact.

Having this, since there are no validated instruments to determine the level of knowledge about TMDs, to our knowledge, this study was performed and the main objective was to develop and validate a scale to assess common knowledge about TMDs in the general population. The second aim of this study was to evaluate the status of TMDs knowledge in the Portuguese population.

MATERIALS AND METHODS

Study design

This is a cross-sectional, descriptive survey design, carried out from September 2015 to March 2016, and followed the STROBE Statement guidelines. A snow-ball sampling was applied and intended to recruit participants not only from health related professions but also from the general population. Exclusion criteria were: age lower than 18 years old and not Portuguese citizens. This study was approved by the Ethics Committee from Instituto Universitário de Ciências da Saúde, CESPU.

This study was divided in two moments. The first moment encompassed the development and validation of the TMDs knowledge scale (1) and the second moment the assessment of the level of knowledge about TMDs (2).

1. TMDs knowledge scale

In order to develop the scale, an experts' committee was created that included one lay person and eight health professionals as follows: one doctor, one orthodontist, one dentist, four physiotherapists, and one psychologist. The TMDs knowledge scale was developed in two phases: (a) concept analysis and construction (Item development and identification of domains and pilot testing on a small number of participants) and (b) testing the psychometric properties.

a. Concept analysis and construction (item development)

Items were generated through a multi-step process: i) Literature review; ii) Experts meeting; iii) Item selection and writing; iv) Pilot testing of the questionnaire; v) Examination by the experts' committee; vi) Final item selection and writing.

The relevant scientific literature research was conducted using electronic databases (Pubmed/Medline, Embase), and information regarding prevalence data, diagnosis, pathophysiology, comorbidities and psychosocial factors associated with TMDs was acquired. Concerning any other questionnaire or scale regarding the assessment of TMDs knowledge for the general population, we have not found any instrument described in the literature. After this, the experts' committee met together and selected the items to be included in the questionnaire and more specifically the construct concept of the TMDs knowledge scale. After items generation, a consensus about the items to include was reached between the experts' committee. The scale was comprised by 21 items (Figure 1) that were assessed through a 3-level Likert scale ("correct answer", "wrong answer" and "I do not know") whether each item is related with TMDs. After this a pilot study was conducted with a total of 10 participants that fulfilled the self-administered questionnaire and were asked to identify any problems regarding questions interpretation, clarity and objectivity

After gathering all the information, the experts' committee reached a consensus regarding the final version of the scale. This scale was part of a wider questionnaire (encompassing questions regarding social and demographic characteristics as well as Fonseca's Anamnestic Index) that was then built in an online survey software (Qualtrics©) and the link to the survey was sent to e-mail lists and through online social networks.

	Correto	Errado	Não Sei
1. Pode ser considerado um problema músculo-esquelético.	X		
2. Apresenta elevados custos socio-económicos.	X		
3. Parece acontecer mais frequentemente no sexo feminino.	X		
4. Afeta essencialmente adultos entre os 20 e os 40 anos.	X		
5. Afeta muito crianças e idosos.		X	
6. O principal critério de diagnóstico é a sintomatologia associada.	X		
7. Pode afetar os músculos da mastigação e do pescoço.	X		
8. Pode ter como sintoma formigueiros na região do queixo.	X		
9. Pode ter como sintoma dor na região da face e pescoço.	X		
10. Os ruídos articulares quando abre e fecha a boca não são um sintoma.		X	
11. Pode ter dores de cabeça associadas.	X		
12. Mascar pastilha elástica, roer as unhas ou ranger os dentes pode agravar a sintomatologia.	X		
13. A ansiedade e/ou depressão são fatores de risco.	X		
14. Pode ser agravada pelo stress.	X		
15. Alimentos ricos em açúcar levam ao desenvolvimento do problema.		X	
16. Uma boa higiene oral previne o aparecimento do problema.		X	
17. Deverá ter um tratamento multidisciplinar.	X		
18. Pode ser tratada e diagnosticada pelo médico dentista e/ou estomatologista (nas suas diferentes especialidades).	X		
19. Tem como tratamento mais eficaz o tratamento cirúrgico.		X	
20. A fisioterapia é uma das opções de tratamento.	X		
21. A psicologia pode contribuir para o tratamento.	X		

Study III - Figure 1: TMDs knowledge scale (in Portuguese) with correct answers

b. Psychometric validation of TMDs knowledge scale

In order to validate the TMDs knowledge scale, a sample comprising 210 individuals was used. The participants were recruited according with the criteria listed above, and the answers to the online questionnaire were analyzed.

2. TMD knowledge assessment

As described above, the complete questionnaire (encompassing questions regarding social and demographic characteristics, TMDs knowledge scale as well as Fonseca's Anamnestic Index) was built in an online survey software (Qualtrics©) and the link to the survey was sent to e-mail lists and through online social networks.

<u>Fonseca's Anamnestic Index</u>

Fonseca's Anamnestic Index is a low cost and easy to apply instrument proposed in the Portuguese language, consisting of 10 questions whose answers are arranged in a three-point scale format (“No”, “Sometimes”, “Yes”). It is used to classify individuals according to TMDs severity (score 0-15: “TMDs Free”, score 20-40: “Mild TMDs”, score 45-60: “Moderate TMDs” and score 70-100: “Severe TMDs”), and also to screen patients for further developments in diagnosing TMDs.[17] Its main advantages are the simplicity of its application, and the fact that it does not need a physical examination of the patient, which makes it suitable for fast epidemiological screening by telephone, mail or internet survey,[18] as in our study. It has a correlation coefficient of (r=0,6169) with the Helkimo Index.[19]

STATISTICAL ANALYSIS

The factor structure was investigated using an exploratory factor analysis (ie, principal component analysis [PCA]) with orthogonal rotation, by use of the Varimax method. The number of factors for extraction was based on Kaiser’s eigenvalue criterion (eigenvalue ≥1) and evaluation of the scree plot. After selecting the number of factors to be retained, a factorial matrix was generated, in which the relationships between the items and the factors were observed via factor loadings. Factor loading over 0.3 were considered as appropriate. Sampling adequacy was assessed by using the Keiser-Meyer Olkin test (KMO) and the Bartlett’s test of sphericity.

The standardized Cronbach’s alpha was estimated to evaluate the internal consistency of the group of items.

Descriptive statistics comprised the following: counts and proportions, mean and standard deviation (SD) or median and interquartile range (IQR). The final score was obtained by assigning one point for each correct answer, with a possible maximum score of 21 points. To assess the construct validity, results were compared by education level, TMDs global awareness (have already heard about TMDs), TMDs professional awareness (have a profession where TMDs knowledge is expected), and TMDs severity (evaluated by Fonseca's Anamnestic Index score). Mann-Whitney test and Kruskal Wallis test were used to compare the score between two or three independent groups, respectively.

Crude and adjusted odds ratios (OR and adj OR) with 95% confidence intervals

(95% CI) were computed to test the association between positive TMDs knowledge and the potential predictive factors that can be used to predict TMDs knowledge in the general population.

All statistical analyses were performed using the Statistical Package for the Social Sciences (SPSS), version 24 (IBM® company, Chicago, USA). The critical value for significance was p-value < 0.05.

RESULTS

Development of the TMDs knowledge scale

As mentioned previously, the development of the scale comprised initially a comprehensive literature review of the relevant publications on the topic. Twenty-one items were developed by the main researchers in accordance to the main areas of importance for the patient's education. The items were divided according to the content proposed as follows: TMDs epidemiology, risk factors, signs and symptoms, and treatment. Each question was presented with three multiple-choice alternatives, as follows: "correct answer", "wrong answer", and 'I do not know'.

The scale was presented in the experts' committee, and each health professional provided their opinion about the content and clarity of each item. Some items generated comments regarding content and semantics. The suggested changes were implemented. The second version of the scale was individually validated by each experts' committee member. Additionally, ten individuals completed the questionnaire as described, and referred to each set of question/answer regarding understanding and clarity. No relevant questions were addressed.

TMDs knowledge scale psychometric characteristics

This scale was pilot-tested in a sample of 210 participants (21 items x 10 participants per item). As no item was missing and no outlier existed, no cases were deleted from the data set. The mean age was 30.3 ± 8.44 years, 119

(56.7%) were women, 123 (58.6%) had more than 12 years of education, 136 (64.8%) have already heard about TMDs, and 58 (27.6%) have a profession where TMDs knowledge is expected, such as dentists, stomatologists, physiotherapists, and speech therapists, among others. Eighty-seven participants (41.4%) reported having no TMDs (Fonseca's Anamnestic Index score ≤ 15), 73 (34.8%) had mild TMDs (Fonseca's Anamnestic Index score 20-40), 38 (18.1%) had moderate TMDs (Fonseca's Anamnestic Index score 45-65), and 12 (5.7%) had severe TMDs (Fonseca's Anamnestic Index score ≥ 70).

The proportion of individuals that correctly answered each item ranged from 24.8% (items 5 and 15) to 70% (item 7). As no prior information on the number of factors to be held was available, exploratory factor analysis was performed. In total four factor analyses were conducted. During the first three analyses, several items did not fulfill the criteria of loading significantly and exclusively on an appropriate factor and due to this fact a one factor solution emerged. This one factor solution was globally interpreted as TMDs knowledge. The KMO test and the Bartlett's test of sphericity showed that the data were adequate for factorial analysis (KMO = 0.952 and Bartlett had a $p < 0.001$). This factor accounted for 56.9% of the total variance of the items (initial eigenvalues 11.94). All items showed a moderate-to-strong positive association with the loading factor. The Cronbach's Alpha was 0.956, showing good reliability, and did not improve if items deleted (Table 1).

Study III - Table 1:Proportion of correct answers, Factor Loadings and Cronbach's Alpha (n=210)

Item	**Correct Answer** n (%)	**Factor Loadings** Factor I	**Cronbach's Alpha if Item Deleted**
1	140 (66.7%)	0.663	0.955
2	86 (41.0%)	0.680	0.955
3	56 (26.7%)	0.606	0.956
4	61 (29.0%)	0.638	0.955
5	52 (24.8%)	0.714	0.954
6	108 (51.4%)	0.791	0.954
7	147 (70.0%)	0.784	0.954
8	63 (30.0%)	0.675	0.955
9	126 (60.0%)	0.845	0.953
10	101 (48.1%)	0.838	0.953
11	122 (58.1%)	0.855	0.953
12	121 (57.6%)	0.812	0.954
13	104 (49.5%)	0.790	0.954
14	122 (58.1%)	0.822	0.954
15	52 (24.8%)	0.677	0.955
16	65 (31.0%)	0.752	0.954
17	121 (57.6%)	0.772	0.954
18	111 (52.9%)	0.785	0.954
19	67 (31.9%)	0.788	0.954
20	125 (59.5%)	0.746	0.954
21	90 (42.9%)	0.733	0.954
Cronbach's Alpha			0.956

The median score of TMDs knowledge was 11.0 points (interquartile range: 15.0). Thirty-eight participants (18.1%) had the lowest score, while 2 participants (1.0%) had the highest score. Table 2 shows that TMDs knowledge was significantly higher in individuals with higher education, higher TMDs global and professional awareness, and higher TMDs severity.

Study III - Table 2: Score of the questionnaire by education level, TMDs global and professional awareness and history of TMDs (n=210)

	TMDs knowledge Scale Median (IQR)	**Test Statistics**	***p***
Education level (years)			
≤ 9 years	2 (13.0)		
10 – 12 years	4.5 (12.0)	37.958*	<0.001
> 12 years	14.0 (9.0)		
TMDs global awareness			
No	1.0 (7.0)	842.000**	<0.001
Yes	15.0 (5.0)		
Professional TMDs awareness			
TMDs knowledge not expected	6.5 (13.0)	595.500**	<0.001
TMDs knowledge expected	17.0 (4.0)		
TMDs			
TMDs free	9.0 (14.0)		
Mild TMDs	8.0 (15.0)	13.644*	0.003
Moderate TMDs	14.0 (7.0)		
Severe TMDs	16.0 (4.0)		

IQR – Interquartile range; TMDs – Temporomandibular disorders
* Kruskal Wallis Test; ** Mann-Whitney Test

TMDs knowledge in the Portuguese population

Overall, 2165 participants (mean age: 30.1 ± 10.58 years, females: 67.4%) participated in this part of the study. Sample characteristics are presented in table 3.

The median score of TMDs knowledge in the Portuguese population was 13.0 points (interquartile range: 10.0). Three hundred and thirty-four participants (15.4%) had 0 correct answers, while 18 participants (0.8%) had 21 correct answers. TMDs knowledge was positive (equal or above 11 correct answers) in 1295 participants (59.8%).

Study III - Table 3: Association between positive TMDs knowledge and sample characteristics (n=2165)

	Final Sample n (%)	**Positive TMDs Knowledge** [a] OR (95% CI)	**Positive TMDs Knowledge** [a] adj [b] OR (95% CI)
Sex			
Male	706 (32.6%)	1.0	1.0
Female	1456 (67.4%)	1.67 (1.39; 2.01)	1.35 (1.06; 1.73)
Education level (years)			
≤ 9 years	46 (7.1%)	1.0	1.0
10 – 12 years	641 (29.7%)	2.05 (1.04; 4.03)	1.01 (0.47; 2.15)
> 12 years	1469 (68.1%)	6.24 (3.20; 12.15)	1.42 (0.67; 3.01)
TMDs global awareness			
No	860 (39.7%)	1.0	1.0
Yes	1305 (60.3%)	17.21 (13.86; 21.36)	8.68 (6.85; 10.99)
Professional TMDs awareness			
TMDs knowledge not expected	1557 (72.9%)	1.0	1.0
TMDs knowledge expected	588 (27.4%)	23.78 (15.96; 35.44)	7.59 (4.94; 11.65)
TMDs			
TMDs free	752 (34.7%)	1.0	1.0
Mild TMDs	859 (39.7%)	0.95 (0.78; 1.16)	1.01 (0.78; 1.31)
Moderate TMDs	391 (18.1%)	2.07 (1.59; 2.69)	1.55 (1.10; 2.18)
Severe TMDs	163 (7.5%)	2.92 (1.96; 4.36)	1.74 (1.07; 2.81)

TMDs – Temporomandibular disorders; OR – Odds Ratio; 95% CI – 95% Confidence Interval
[a] Negative Knowledge (score <11) was used as the reference category.
[b] OR adjusted to the other predictive factors.

A logistic regression model was used to identify the potential predictive factors that can be used to predict TMDs knowledge in the general population. Results showed that females, have already heard about TMDs, have a profession where TMDs knowledge is expected, and have moderate or severe TMDs had significantly higher TMDs knowledge, even after adjustment to the other predictive factors (Table 3).

DISCUSSION

The present study describes a methodological approach to the development and validation of a new self-administered scale to measure the knowledge about TMDs. Our results demonstrate that the scale developed is psychometrically valid and reliable. The TMDs knowledge scale was developed in Portugal, notwithstanding, since it does not contain items that are specifically related to

Portuguese culture, it could be translated and used abroad. Moreover, the scale has shown to be easy to understand, complete and requires a relatively short time to answer.

Since the first step in the development of educational actions is to identify the need for information on what patients really know about their own disease, this study demonstrated an overall positive knowledge of the sample studied (59,8% had 11 or above correct answers). As expected, the individuals with higher professional awareness, had higher knowledge about TMDs. This may be justified by the fact that the knowledge scale developed intended to evaluate the general population's knowledge about TMDs, which means that the statements of the scale were not too technical nor requiring advanced knowledge about the topic. This could explain the difference between our results and the results from studies assessing the knowledge of specific health professionals, that reveal lack of knowledge from the professionals.[19,20]

Our results also showed that most of the participants (60%) had global awareness about TMDs, meaning that they have already heard about this dysfunction, and that this factor is, consequently associated with TMDs knowledge. The participants with self-reported higher TMDs severity had also higher knowledge about the condition. This can be explained by the fact that the impact of TMDs in these participants is higher, which may lead to the search of more information about the condition and its management.

Concerning predictive factors, our study showed that females, have already heard about TMDs, have a profession where TMDs knowledge is expected, and have moderate or severe TMDs had significantly higher TMDs knowledge.

One of the limitations of the present study is the lack of comparison with a clinical gold standard. Another limitation is the self-reported severity of TMDs, through Fonseca Anamnestic Index, because despite this is a validated and reliable tool for this purpose, we were not able to perform the clinical evaluation of the individuals. Future studies should perform the clinical assessment of the participants, in order to perform the diagnose of TMDs.

CONCLUSION

The results from our study showed that the TMDs knowledge scale developed is psychometrically valid and reliable. It also demonstrated that the participants had an overall positive knowledge about TMDs and that females, which have already heard about TMDs, have a profession where TMDs knowledge is expected, and have moderate or severe TMDs had significantly higher TMDs knowledge.

ACKNOWLEDGEMENTS:

CIAFEL – Research Centre in Physical Activity, Health and Leisure

IINFACTS - Institute of Research and Advanced Training in Health Sciences and Technologies.

REFERENCES

1 Leeuw R, Klasser GD. Orofacial Pain: Guidelines for Assessment, Diagnosis, and Management. 5th ed.: Quintessence Books, 2013.

2 Gremillion H. The prevalence and etiology of temporomandibular disorders and orofacial pain. Tex Dent J 2000;117:30-39.

3 LeResche L. Epidemiology of Temporomandibular Disorders: Implications for the Investigation of Etiologic Factors. Crit Rev Oral Biol Med 1997;8:291-305.

4 Cruz CL, Lee KC, Park JH, Zavras AI. Malocclusion Characteristics as Risk Factors for Temporomandibular Disorders: Lessons Learned from a Meta-Analysis. J Oral Dis 2015;2015.

5 Dıraçoğlu D, Yıldırım NK, Saral İ, et al. Temporomandibular dysfunction and risk factors for anxiety and depression. Journal of Back and Musculoskeletal Rehabilitation 2015:1-5.

6 Fillingim R, Ohrbach R, Greenspan J, et al. Potential psychosocial risk factors for chronic TMD: descriptive data and empirically identified domains from the OPPERA case-control study. J Pain 2011;12:T46-60.

7 Melis M, Di Giosia M. The role of genetic factors in the etiology of temporomandibular disorders: a review. Cranio 2016;34:43-51.

8 Michelotti A, Cioffi I, Festa P, Scala G, Farella M. Oral parafunctions as risk factors for diagnostic TMD subgroups. J Oral Rehabil 2010;37:157-162.

9 Kickbusch IS. Health literacy: addressing the health and education divide. Health Promot Int 2001;16:289-297.

10 World Health Organization: http://www.who.int/healthpromotion/conferences/7gchp/track2/en/ Accessed [6 October 2016].

11 Brekke M, Hjortdahl P, Kvien TK. Involvement and satisfaction: A Norwegian study of health care among 1,024 patients with rheumatoid arthritis and 1,509 patients with chronic noninflammatory musculoskeletal pain. Arthritis Care Res 2001;45:8-15.

12 Pang T, Sadana R, Hanney S, Bhutta ZA, Hyder AA, Simon J. Knowledge for better health: a conceptual framework and foundation for health research systems. Bull World Health Organ 2003;81:815-820.

13 Sabbahi DA, Lawrence HP, Limeback H, Rootman I. Development and evaluation of an oral health literacy instrument for adults. Community Dent Oral Epidemiol 2009;37:451-462.

14 Charles C, Whelan T, Gafni A. What do we mean by partnership in making decisions about treatment? BMJ 1999;319:780-782.

15 Taal E, Rasker JJ, Wiegman O. Goup education for Rheumatoid Arthritis Patients. Semin Arthritis Rheum 1997;26:805-816.

16 Lubrano E, Helliwell P, Moreno P, Griffiths B, Emery P, Veale D. The assessment of knowledge in ankylosing spondylitis patients by a self-administered questionnaire. Br J Rheumatol 1998;37:437-441.

17 Fonseca D, Bonfante G, Valle A, Freitas S. Diagnóstico pela anamnese da disfunção craniomandibular. Rev Gaucha Odontol (RGO) 1994;42:23-28.

18 Campos JC, A.C, Bonafé F, Maroco J. Severity of temporomandibular disorders in women: validity and reliability of the Fonseca Anamnestic Index. Braz Oral Res 2014;28:1-6.

19 Baharvand M, Sedaghat Monfared M, Hamian M, Jalali Moghaddam E, Sadat Hosseini F, Alavi KA. Temporomandibular Disorders: Knowledge, Attitude and Practice among Dentists in Tehran, Iran. J Dent Res Dent Clin Dent Prospects 2010;4:90-94.

20 Lee W-Y, Choi J-W, Lee JW. A study of dentists' knowledge and beliefs regarding temporomandibular disorders in Korea. Cranio 2000;18:142-146.

STUDY IV

Orthodontic treatment produces changes in the craniocervical posture of patients with temporomandibular disorders: a quasi-experimental retrospective study.

Submitted for publication in

Physical Therapy

Orthodontic treatment produces changes in the craniocervical posture of patients with temporomandibular disorders: a quasi-experimental retrospective study.

Paço, M[1]; Duarte, J.A.[2]; Pinho, T*[1,3]

[1]CESPU, Instituto de Investigacão e Formação Avançada em Ciências e Tecnologias da Saúde, Gandra-Paredes, Portugal.

[3]CIAFEL, Faculdade de Desporto da Universidade do Porto, Portugal.

[4]IBMC - Inst. Biologia Molecular e Celular., i3S - Inst. Inovação e Investigação em Saúde, Universidade do Porto.

* Corresponding author: Pinho, T; Rua Central de Gandra, 1317, 4585-116 Gandra PRD, Portugal; teresa.pinho@iucs.cespu.pt

ABSTRACT

Background: Orthodontic treatment acts through the application of forces and/or by stimulating and redirecting the functional forces within the craniofacial complex. Considering the interrelationship between craniomandibular and craniocervical systems, this intervention may alter craniocervical posture. **Objectives:** (a) To compare craniocervical posture, hyoid bone position and craniofacial morphology before, after and also in the contention phase of orthodontic treatment in patients with temporomandibular disorders, (b) To verify if the presence of condylar displacement, the skeletal Class or the facial biotype interfere with the above mentioned outcomes. **Methods:** Quasi-experimental, longitudinal and retrospective design. A non-probabilistic convenience sampling method was applied. The sample consisted of clinical records of patients to compare pre orthodontic treatment with post orthodontic treatment (n=42) and contention phase data (n=26). A cephalometric analysis of the variables CV angle, C0-C1, C1-C2, C3-H, C3-Rgn, H-H1, H-Rgn, AA-PNS, CVT/Ver, NSL/CVT, NSL/OPT, NSL/Ver, OPT/CVT, OPT/Ver, facial biotype, skeletal Class and facial proportion was performed. The p-value was set as 0.05. **Results:** When analysed pre and post orthodontic treatment data: CV angle, C0-C1, AA-PNS and C3-Rgn had significant changes. When analysed pre, post orthodontic treatment and contention phase data: C0-C1, CVT/Ver, NSL/OPT, NSL/CVT, NSL/Ver; OPT/CVT, OPT/Ver and facial biotype had significant changes. **Conclusion:** In the sample studied there were significant differences regarding hyoid bone position (pre *versus* post orthodontic treatment) and craniocervical posture (pre *versus* post orthodontic *versus* contention), with the craniocervical posture being prone to return to basal values. The presence of condylar displacement was found to significantly increase the distance H-H1 in the three moments of evaluation. Facial biotype was found to significantly increase the angle NSL/Ver on hypodivergent compared with hyperdivergent, in the contention phase.

Keywords: temporomandibular joint disorders, posture, cervical spine, cephalometry

INTRODUCTION

Concerning dentistry, orthodontics is usually one of the most used treatment approaches when targeting malocclusion or temporomandibular disorders (TMDs).[1-3] Orthodontic treatment is thought to address those alterations by improving the dental occlusion as well as occlusion stability, not only by changing the position but also the morphology of temporomandibular joint (TMJ).[1,2,4]

The literature regarding orthodontic treatment effects is somewhat controversial, with several studies reporting good results on the TMDs resolution or, at least, on reducing the risk of the patient to develop it, while other studies suggest that orthodontic treatment increases the risk of onset of signs and symptoms of TMDs[2,4-10] or it is TMDs neutral.[11]

One of the possible explanations to these controversial results is the heterogeneity of TMDs, a multifactorial entity without a well-defined etiopathogenesis[12,13] that encompasses several conditions, as TMJ pain, masticatory muscles pain or a combination of both.[14-17] The attention to signs and symptoms associated with TMDs have modified the clinical management before and during orthodontic treatment.[9] It is described in the literature that an altered position of the condyle (deviation from the centric relation) may potentially increase the risk of developing TMDs.[18,19] It is hypothesized that condylar displacement in relation to the articular eminence may influence negatively the articular stability, since that displacement causes the loss of relation between the condyle, the articular disc and glenoid cavity,[20] increasing the liability of TMDs.[21,22]

It has been described the close relationship between the craniomandibular and craniocervical systems, showing its functional, biomechanical, neurodynamic and physiological interrelationship, having both the potential to influence each other reciprocally.[23-29] The head and neck posture has been studied in order to highlight the relation between these structures and TMDs, dentofacial structures and maxillofacial morphology.[30-33] A possible explanation for this relation is the differential growth of the muscles and fascia that are attached to the mandible and pass to the cranium above and to the hyoid bone and shoulder girdle below

(Houston, 1988 cit in Motoyoshi et al., 2002[32]). Having this in mind, it is expected that alterations on the head position may influence the stomatognathic system by changing occlusal stress distributions and affecting craniofacial morphology.[32-35]

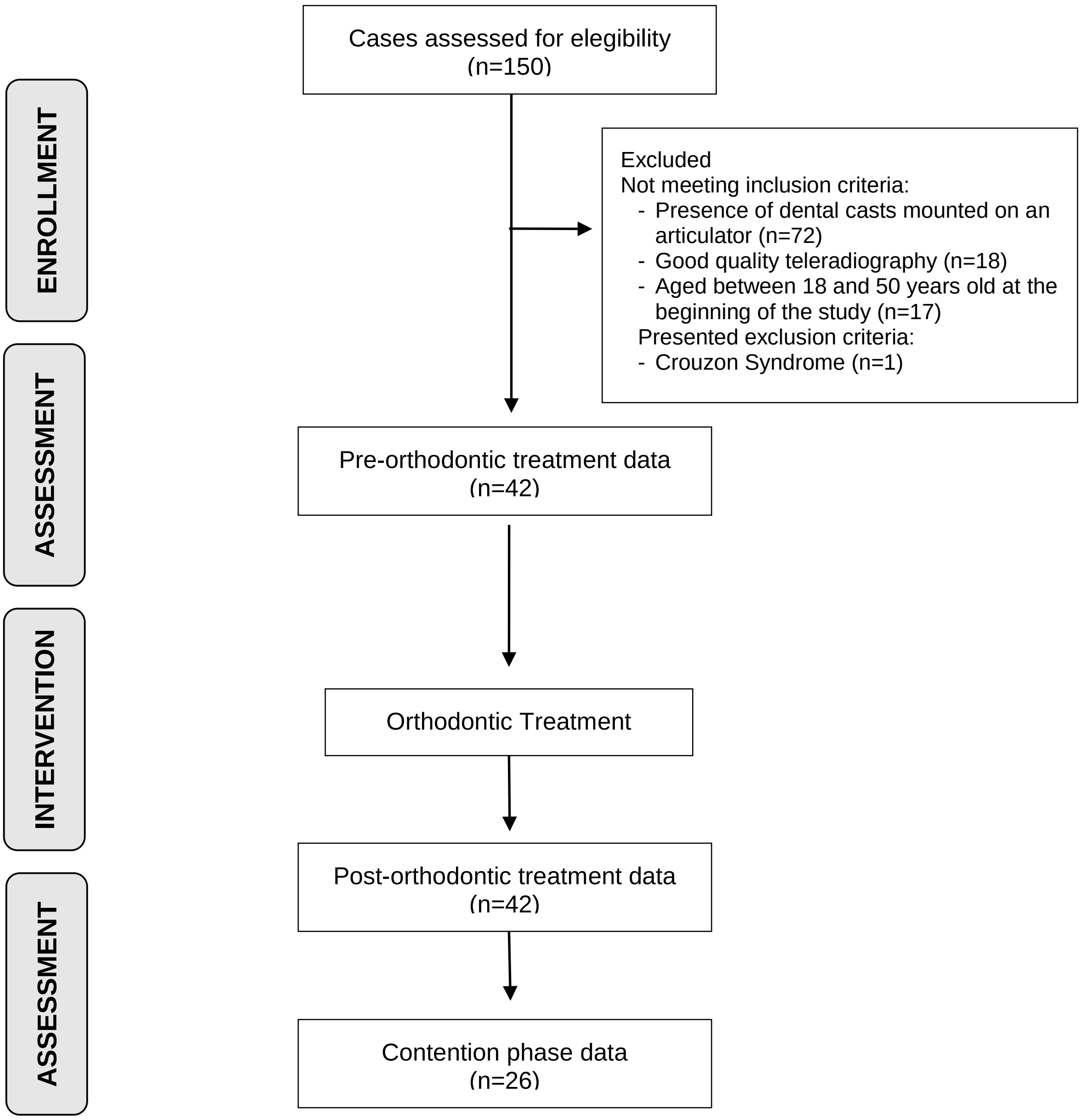
The literature has shown that a craniocervical dysfunction may lead to, or perpetuate the TMDs,[36-39] showing the necessity of addressing these impairments in order to achieve the greatest results for the patients. On the other hand, the mechanical effects from orthodontics may lead to muscular and articular adaptations that, with time, may lead to craniocervical dysfunction.

Thus, since the relationship between orthodontic treatment and craniocervical posture have not been fully addressed so far, the main objective of this work was to compare craniocervical posture, hyoid bone position and craniofacial morphology before and after orthodontic treatment and also in the contention phase in patients with TMDs. A secondary objective was to verify if the presence of condylar displacement, the skeletal Class or the facial biotype interfere with the above mentioned outcomes.

METHODS

Study design

This is a quasi-experimental, longitudinal, analytical and retrospective design. A non-probabilistic convenience sampling method was applied, accessing clinical documentation (clinical cases) from patients that have been submitted to orthodontic treatment and had a clinical diagnose of TMDs. The sample consisted of clinical records of 42 patients from two orthodontic clinics in the district of Porto (Portugal), to compare pre orthodontic treatment with post orthodontic treatment. From this initial sample a sub-group of 26 clinical records (that contained a 1 year after orthodontic treatment teleradiography) was analysed in order to compare pre, post orthodontic treatment and contention phase data (Fig 1).



Study IV - Figure 1: Flow diagram, according with CONSORT statement.

To be included in the study the patients had to be examined by an Orthodontist regarding clinical history (clinical diagnosis of TMDs according with signs and/or symptoms), lateral and anterior photographs (in natural head position), have good quality teleradiography (also in natural head position and should include head and cervical column, with at least the fourth cervical vertebra completely visible), have dental casts mounted on an articulator in centric relation and be aged at the beginning of the study, between 18 and 50 years old. The existence of dental casts mounted on an articulator in centric relation was also an inclusion criterion since this procedure was performed in the cases where there was a clinical diagnosis of TMDs. Another inclusion criterion was the achievement of a canine Class I relation and normalized overjet and overbite values after orthodontic treatment.

Cases were excluded if they presented history of traumatic injuries, fibromyalgia syndrome, diagnosis of systemic disease or presence of neurological disorders.

Ethical approval was guaranteed by the Ethics Committee from Instituto Universitário de Ciências da Saúde, CESPU.

Procedures

After checking the eligibility of the cases, the assessment of craniocervical posture, hyoid bone position, craniofacial morphology and occlusal factors was performed.

The occlusal parameters studied were the presence of malocclusions and condylar displacement. This was performed using intra-oral photographs as well as dental casts. Furthermore, it was adopted the mounting models in centric relation on a semi-adjustable articulator SAM 3® (Präzisionstechnik, Taxisstr. 41, D-80637 München, mGermany) and the register of the condyle position and consequently the amount of condylar displacement, was registered with a mandible position indicator (MPI 120®, Präzisionstechnik, Taxisstr. 41, D-80637 München, Germany). These procedures have been previously described and considered reliable.[22,40,41]

When analysed the condylar displacement, it was considered that a $\Delta \geq 2$mm, was consistent with a higher risk to develop TMDs, and the participants were classified as “condylar displacement present”.[19]

Regarding craniocervical posture, hyoid bone position and craniofacial morphology analysis, these were performed by teleradiography cephalometric analysis’ with lateral photograph sobreposition (also in natural head position) through Nemoceph® software (Nemoceph 6—Dental Studio NX, version 6.0, Spain)®. The cephalometric points used were marked as previously described[42-47] and are defined in Table 1.

Study IV - Table 1: Cephalometric landmarks, angles and reference measures

Measure	Definition
Cranio-vertebral angle (CV angle)	The angle resultant from the intersection between a horizontal line that goes from the Bolton point (Bo) (the intersection of the outline of the occipital condyle and the forâmen magnum at the highest point on the notch posterior to the occipital condyle) to the posterior nasal spine and the vértice of the odontoid process and the anteroinferior point of the odontoid process.
C0-C1	The distance between the horizontal line that goes from the posterior nasal spine and the most anterior point of the first cervical vertebra.
C1-C2	The distance between the most anterior aspect of the first cervical vertebra and the second cervical vertebra.
C3-H	The distance between the most anterior aspect of the third cervical vertebra and the most anterior point of the hyoid bone.
C3-Rgn	The distance between the most anterior aspect of the third cervical vertebra and the most dorsal and inferior point of mandibular symphysis (*retrognation*).
H-H1	The distance from the most anterior point of the hyoid bone and the horizontal line that goes from the most anterior aspect of the third cervical vertebra and *retrognation*.
H-Rgn	The distance from the most anterior point of the hyoid bone and the *retrognation*.
AA-PNS	The distance from the most anterior point of atlas vertebra (AA) to posterior nasal spine .
CVT/Ver	The angle resultant from the intersection between a line that goes from OT point to the most posterior and inferior aspect of the fourth vertebral body and the vertical line that correponds to the true vertical.
NSL/CVT	The angle resultant from the intersection between a line that goes from *sela turcica* to *nasion* and the line that goes from OT point to the most posterior and inferior aspect of the fourth vertebral body.
NSL/OPT	The angle resultant from the intersection between a line that goes from *sela turcica* to *nasion* and the line that goes from OT point to the most posterior and inferior aspect of the odontoid process.
NSL/Ver	The angle resultant from the intersection between a line that goes from *sela turcica* to *nasion* and the vertical line that corresponds to the true vertical.
OPT/CVT	The angle resultant from the intersection between a line that goes from OT point to the most posterior and inferior aspect of the odontoid process and the line that goes from OT point to the most posterior and inferior aspect of the fourth vertebral body.
OPT/Ver	The angle resultant from the intersection between a line that goes from OT point to the most posterior and inferior aspect of the odontoid process and the vertical line that correponds to the true vertical.
Facial biotype	Through the measurement of FMA where a score less than 22 means hypodivergent, between 22 and 28 means normodivergent and higher than 28 means hyperdivergent.
Skeletal Class	Through the measurement of ANB, where a score inferior to 0 represents a Class III, between 0-5 represents a Class I and a score superior to 5 represents a Class II.
Facial proportion	Calculated by the intersection ratio of the Sn-Gnc line with the Gnc-C line.

Lateral cephalograms of 10 randomly selected subjects were measured twice, with one week interval between measurements, to assess the magnitude of measurement errors (Intraclass Correlation Coefficient $(ICC)_{(2,1)}$). $ICC_{(2,1)}$ for the reliability of landmark identification was 0,98, demonstrating an excellent reliability.[48]

Statistical Analysis

Statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS)®, version 24 (IBM® company, Chicago, USA). To assess the normal distribution of the variables, the Shapiro-Wilk test was applied. Sample characteristics are presented as absolute frequencies in categorical variables and mean and standard deviation (SD) in quantitative variables. The presence of potential differences between pre and post-intervention results were analysed through paired samples *t*-test or Wilcoxon test, whether the outcomes had a normal distribution or not, respectively. A repeated-measures ANOVA was used to evaluate the presence of potential differences between the three assessment moments (pre-intervention, post-intervention and contention phase). The assumptions to perform this test were normal distribution of the variables (Shapiro-Wilk test) and esphericity (Mauchly's test). When the esphericity assumption was not fulfilled, the F-value was corrected, accordingly with previously described methods.[49] Multiple comparisons between the three assessment moments were performed through Bonferroni *post-hoc* test. When the assumptions for parametric tests were not fulfilled, the Friedman test was used, and multiple comparisons were performed through Wilcoxon tests. To compare the outcome variables, according with the presence or absence of condylar displacement, independent samples *t*-test or Mann-Whitney tests were used, as parametric and non-parametric tests, respectively. To compare the outcome variables, according with the skeletal Class and facial biotype, One-Way ANOVA (with Bonferroni *post-hoc* test) and Kruskal-Wallis tests were used, as parametric and non-parametric tests, respectively. The critical value for significance in all the analysis was p-value < 0.05.

RESULTS

The sample regarding pre and post orthodontic treatment results consisted in 42 individuals (6 men, 36 women), age of 28,14±11,36 years in the beginning of the treatment and the duration of orthodontic treatment was 2,87±1,45 years.

Table 2 presents data regarding facial and skeletal characteristics of the participants, pre-orthodontic treatment.

Study IV - Table 2: Sample characterization regarding skeletal Class, facial biotype and condylar displacement, before orthodontic treatment (n=42)

Characteristics		**Frequency (%)**
Skeletal Class	Skeletal Class I	45,2
	Skeletal Class II	50
	Skeletal Class III	4,8
Facial Biotype	Hypodivergent	16,7
	Normodivergent	23,8
	Hyperdivergent	59,5
Condylar Displacement	Present	23,8
	Absent	76,2

Table 3 presents the variables that had statistically significant changes, when compared the values pre-orthodontic treatment with the values post-orthodontic treatment.

When the cephalometric variables were adjusted to the presence or absence of condylar displacement, to the skeletal Class and also to the facial biotype, there were no significant differences among the different groups in any of the assessment moments, except for the variable H-Rgn, with differences between skeletal Class I (43,69±4,33) and Class II (39,72±5,55) after orthodontic treatment (p=0,009).

Study IV - Table 3: Cephalometric variables in the 2 moments: pre orthodontic treatment and post orthodontic treatment (n=42).

	Cephalometric Variable	**Pre OT Mean (SD)**	**Post OT Mean (SD)**	***p* value (Paired samples *t*-test)**
Craniocervical Posture	CV angle	99,90 (11,65)*	98,10 (13,00)*	0,036†
	C0-C1	6,75 (4,01)	7,84 (3,96)	0,017
	C1-C2	20,15 (2,18)	20,80 (2,35)	NS
	CVT/Ver	7,42 (7,32)	7,38 (8,07)	NS
	NSL/OPT	78,50 (15,25)*	78,30 (9,30)*	NS†
	NSL/CVT	92,94 (7,45)	95,34 (8,22)	NS
	NSL/Ver	79,67 (4,30)	77,26 (4,49)	NS
	OPT/CVT	15,72 (4,80)	15,10 (4,54)	NS
	OPT/Ver	23,14 (9,21)	22,48 (10,64)	NS
	AA-PNS	36,53 (4,35)	35,61 (4,41)	0,009
Hyioid Bone Position	C3-H	36,60 (3,92)	36,98 (4,36)	NS
	C3-Rgn	74,70 (8,49)	76,80 (7,84)	0,018
	H-H1	5,11 (6,14)	4,31 (6,04)	NS
	H-Rgn	40,15 (6,46)	41,26 (5,42)	NS
Craniofacial Morphology	Facial biotype	28,68 (7,10)	29,02 (7,12)	NS
	Skeletal Class	4,88 (3,03)	5,11 (3,02)	NS
	Facial proportion	1,50 (0,30)	1,46 (0,28)	NS

* Median (Interquartile Range); † Wilcoxon Test; SD – Standard deviation; OT - Orthodontic treatment; NS – Non-significant

When analysed the subgroup of participants with data regarding contention phase, the total of participants were 26 (4 men, 22 women), age of 27,77±8,49 years old in the beginning of the treatment.

Table 4 presents the variables that had statistically significant changes, when compared pre-orthodontic treatment with post-orthodontic treatment and with the contention phase.

Study IV - Table 4: Cephalometric variables in the 3 moments: pre orthodontic treatment, post orthodontic treatment and contention (n=26)

	Cephalometric Variable	**Pre OT Mean (SD)**	**Post OT Mean (SD)**	**Contention Mean (SD)**	***p* value (ANOVA repeated measures)**	**Multiple Comparisons *p* value (Bonferroni)**
Craniocervical Posture	CV angle	98,99 (8,92)	97,72 (9,60)	96,87 (8,99)	NS	-
	C0-C1	8,50 (6,00) *	9,40 (5,50) *	9,60 (4,45) *	0,028†	0,002‡ (PreOT/Contention)
	C1-C2	19,96 (2,35)	20,64 (2,39)	21,14 (2,79)	NS	0,033 (PreOT/Contention) <0,001 (PostOT/Contention)
	CVT/Ver	7,20 (12,05) *	9,00 (13,20) *	13,90 (12,05) *	<0,001†	<0,001‡ (PreOT/Contention) <0,001‡ (PostOT/Contention)
	NSL/OPT	75,00 (18,70) *	77,90 (11,60) *	68,40 (16,45) *	<0,001†	0,033‡ (PreOT/Contention) <0,001‡ (PostOT/Contention)
	NSL/CVT	93,33 (7,84)	95,44 (9,88)	88,71 (9,70)	<0,001	0,008 (PreOT/Contention) <0,001 (PostOT/Contention)
	NSL/Ver	79,18 (3,81)	76,90 (4,10)	75,90 (4,38)	<0,001	0,008 (PreOT/Contention) <0,001 (PostOT/Contention)
	OPT/CVT	15,24 (6,44)	14,86 (5,11)	17,97 (4,90)	0,011	0,027 (PostOT/Contention)
	OPT/Ver	22,74 (10,51)	22,52 (11,98)	33,37 (9,51)	<0,001	0,001 (PreOT/Contention) <0,001 (PostOT/Contention)
	AA-PNS	37,88 (4,22)	37,17 (4,09)	37,55 (4,20)	NS	-
Hyioid Bone Position	C3-H	36,70 (4,07)	37,31 (4,67)	37,50 (4,27)	NS	-
	C3-Rgn	75,33 (8,38)	77,36 (7,85)	76,70 (6,55)	NS	-
	H-H1	3,99 (6,25)	3,32 (6,64)	2,80 (6,93)	NS	-
	H-Rgn	40,11 (6,67)	41,40 (5,47)	40,66 (5,23)	NS	-
Craniofacial Morphology	Facial biotype	29,54 (7,34)	29,75 (6,11)	29,10 (7,81)	<0,001	0,008 (PreOT/Contention) <0,001 (PostOT/Contention)
	Skeletal Class	5,10 (3,95) *	5,20 (2,95) *	5,30 (3,90) *	NS†	-
	Facial proportion	1,46 (0,31) *	1,48 (0,37) *	1,49 (0,36) *	NS†	-

* Median (Interquartile Range); † Friedman Test; ‡ Wilcoxon Test; SD – Standard deviation; NS – Non-significant.

When the cephalometric variables were adjusted to the presence or absence of condylar displacement, to the skeletal Class, and also to the facial biotype, there were no differences among the different groups in any of the assessment moments, except for the variables H_H1, facial proportion and NSL/Ver. H_H1 was found to have statistically significant changes between the participants with condylar displacement and those without it before orthodontic treatment (“condylar displacement present” 8,41±3,80; “condylar displacement absent” 2,62±6,24; p=0,031), after orthodontic treatment (“condylar displacement present” 7,63±2,97; “condylar displacement absent” 2,14±7,10; p=0,11) and in the contention phase (“condylar displacement present” 8,16±5,57;“condylar displacement absent” 1,28±6,66; p=0,023).

Facial proportion was found to have statistically significant changes between hypo and hyperdivergent facial type participants' before orthodontic treatment (hyperdivergent 1,60±0,31; hypodivergent 1,21±0,14; p=0,027), after orthodontic treatment (hyperdivergent 1,59±0,29; hypodivergent 1,17±0,13; p=0,014) and in the contention phase (hyperdivergent 1,62±0,28; hypodivergent 1,15±0,28; p=0,032).

NSL/Ver was found to have statistically significant changes between hyperdivergent (74,81±3,59) and hypodivergent facial type participants' (82,00±2,72) only in the contention phase (p=0,005).

DISCUSSION

Subjects presented an increase in CV angle concomitantly with an increase in C0-C1 distance and in C3-Rgn distance, as well as a decrease in AA-PNS distance. The increase in CV angle is associated with an anterior rotation of the head.[43,50] This rotation of the head is also corroborated by the decrease in AA-PNS distance that is usually associated with a flexed craniocervical posture. This finding is also confirmed by the results of the distance C0-C1, whose increase reflects the rectification of the cervical column. The increase in the distance C3-Rgn is also compatible with a loss of cervical lordosis. In spite of the variables NSL/OPT and NSL/CVT do not present statistically significant changes, they also present relevant mean increases, which is also compatible with an anterior rotation of the head. This anterior rotation of the head and rectification of the cervical column is thought to increase the sub-occipital space favouring a progressive tension over posterior soft tissues, which in turn may be responsible for peripheral neuropathies with craniocervical pain.[43]

After adjustment of the cephalometric variables, the only variable that presented significant changes was skeletal Class. According with skeletal Class, H-Rgn distance was lower in Class II compared with Class I participants after orthodontic treatment. This finding was as expected since Class II individuals may present with a retrognathic mandible, thus decreasing the distance between hyoid bone and the mandible.

This study also intended to assess the stability of the results obtained, and did this by evaluating the presence of TMDs signs and/or symptoms, the craniocervical posture, hyoid bone position and craniofacial morphology (including dental Class and overbite and overjet values) in the contention phase (1 year after finishing orthodontic treatment) and comparing with pre orthodontic treatment and post orthodontic treatment data. This comparison was performed in a subgroup of the initial sample. When analysed the results obtained, all the patients remained TMDs signs and symptoms' free, had no relapse on dental Class and overbite and overjet values remained within normal values. On the other hand, significant changes were found mainly in the craniocervical posture variables and also in the facial biotype that demonstrated a tendency to normodivergency. The craniocervical posture variables that had statistically significant changes (C0-C1, CVT/Ver, NSL/OPT, NSL/CVT, NSL/Ver, OPT/CVT, OPT/Ver) had differences compatible with a posterior rotation of the head and an extended cervical column that highlights the increase in the cervical lordosis. This posterior rotation of the head and increase of the cervical lordosis is thought to decrease the sub-occipital space and produce a progressive mechanical compression over posterior soft tissues, which in turn may be responsible for peripheral neuropathies with craniocervical pain.[43] It has also been described that these features may impose an excessive tension over the supra and infrahyoid muscles in a dorsal and caudal direction, affecting the growth and development of the mandibular bone, lingual rest and also deglutition.[43]

These differences had a particular impact when analysed "pre orthodontic treatment" *versus* "contention phase" and "post orthodontic treatment" *versus* "contention phase". It is interesting to observe that in the majority of the measures that had significant changes (NSL/OPT, NSL/CVT, OPT/CVT, OPT/Ver), when compared "pre orthodontic treatment" *versus* "post orthodontic treatment" the tendency shown was the opposite (anterior rotation of the head and rectification of the cervical column, although without statistically significant differences). We hypothesize that, despite the sample studied did not have occlusal nor symptoms relapse, this inversion of the results achieved during orthodontic treatment (by analysing "post orthodontic treatment" *versus* "contention phase") may be a possible explanation in cases where there is occlusal and/or TMDs symptoms' relapse. This may happen because, during "contention phase" the patient uses a fixed lower contention apparatus and a removable upper contention apparatus during the night, which helps to maintain the occlusal stability. Nonetheless, and considering the changes found in the craniocervical posture, after the contention phase, the maintenance of the results' stability (occlusal and symptomatic) may be questioned. Having in mind the results found, that are supported by the interrelationship between both systems and considering the fact that the literature has shown that a craniocervical dysfunction may lead to, or perpetuate the TMDs,[36-39] it is conceivable that the craniocervical changes have the potential to contribute to occlusal and/or TMDs' symptoms relapse seen in clinical practice and described in the literature.[51]

After adjustment of the cephalometric variables according with the presence or absence of condylar displacement, the skeletal Class and also to the facial biotype, the results showed that the presence of condylar displacement was found to significantly increase the distance H-H1 in the three moments of evaluation when compared with the participants without condylar displacement. This distance increase is associated with a downward position of the hyoid bone[43,50] and may reflect muscular asymmetry between supra and infra-hyoid muscles. Facial biotype was found to significantly increase the angle NSL/Ver on hypodivergent compared with hyperdivergent participants, in the contention phase. This result is according with the literature, since a decreased NSL/Ver angle is associated with a posterior rotation of the head and a forward inclination of the cervical column, which is related with hyperdivergency morphology and retrognathic profile.[39,47]

The relatively reduced sample size is the result of our inclusion criterion regarding the presence of TMDs signs and/or symptoms. This fact allowed us to be more specific regarding TMDs sufferers, however it narrowed the sample that we could had access to, because it was restricted to the cases with dental casts mounted in the articulator in centric relation. However, despite the sample size, the effect sizes are important.

Because there are no standardized values for most of the variables studied, we did not intend to classify the final result as normal or abnormal alterations, but mostly to characterize and verify if there were changes after orthodontic treatment and in the contention phase. The presence of changes was interpreted as a signal of the interrelationship between craniomandibular and craniocervical systems, alerting the clinician for the necessity of addressing these alterations during the treatment and contention phase, since they may contribute to the development/aggravation of TMDs'. Thus, it seems important to conduct well-designed longitudinal and randomized controlled trials, comparing craniocervical posture, hyoid bone position and TMDs' signs and symptoms, in individuals diagnosed with TMDs, before and after the orthodontic treatment and a follow-up period superior to the contention phase (one year).

ACKNOWLEDGEMENTS

IINFACTS - Institute of Research and Advanced Training in Health Sciences and Technologies.

CONCLUSIONS

Our results demonstrated that in the sample studied there were statistically differences regarding hyoid bone position (pre orthodontic treatment *versus* post orthodontic treatment) and craniocervical posture (between the three moments of evaluation: pre orthodontic treatment, post orthodontic treatment and contention phase), with the craniocervical posture being prone to return to basal values.

The presence of condylar displacement was found to significantly increase the distance H-H1 in the three moments of evaluation. Facial biotype was found to significantly increase the angle NSL/Ver on hypodivergent compared with hyperdivergent participants, in the contention phase.

REFERENCES

1. Henrikson T, Nilner M. Temporomandibular disorders and the need for stomatognathic treatment in orthodontically treated and untreated girls. *European Journal of Orthodontics.* 2000;22:283-292.

2. Imai T, Okamoto T, Kaneko T, Umeda K, Yamamoto T, Nakamura S. Long-term follow-up of clinical symptoms in TMD patients who underwent occlusal reconstruction by orthodontic treatment. *European Journal of Orthodontics.* 2000;22:61-67.

3. Luther F, Layton S, McDonald F. Orthodontics for treating temporomandibular joint (TMJ) disorders. *Cochrane Database of Systematic Reviews (Online).* 2010;7(7).

4. Egermark I, Carlsson GE, Magnusson T. A prospective long-term study of signs and symptoms of temporomandibular disorders in patients who received orthodontic treatment in childhood. *The Angle Orthodontist.* 2005;75(4):645-650.

5. Luther F. TMD and occlusion part I. Damned if we do? Occlusion: the interface of dentistry and orthodontics. *Brazilian Dental Journal.* 2007;202(E3):1-7.

6. Nielsen L, Melsen B, Terp S. TMJ function and the effects on the masticatory system on 14-16 year-old Danish children in relation to orthodontic treatment. *European Journal of Orthodontics.* 1990;12:254-262.

7. Ohlsson M, Linquist B. Mandibular function before and after orthodontic treatment. *European Journal of Orthodontics.* 1995;17:205-214.

8. Henrikson T, Nilner M, Kurol J. Symptoms and signs of temporomandibular disorders before, during and after orthodontic treatment. *Swedish Dental Journal.* 1999;23:107-117.

9. Leite RA, Rodrigues JF, Sakima MT, Sakima T. Relationship between temporomandibular disorders and orthodontic treatment: A literature review. *Dental Press Journal of Orthodontics.* 2013;18(1):150-157.

10. Egermark I, Blomqvist JE, Cromvik U, Isaksson S. Temporomandibular dysfunction in patients treated with orthodontics in combination with orthognathic surgery. *European Journal of Orthodontics.* 2000;22(5):537-544.

11. Manfredini D, Stellini E, Gracco A, Lombardo L, Nardini LG, Siciliani G. Orthodontics is temporomandibular disorder–neutral. *The Angle Orthodontist.* 2016;86(4):649-654.

12. Reneker J, Paz J, Petrosino C, Cook C. Diagnostic accuracy of clinical tests and signs of temporomandibular joint disorders: a systematic review of the literature. *Journal of Orthopaedic & Sports Physical Therapy.* 2011;41(6):408-416.

13. Stegenga B. Nomenclature and classification of temporomandibular joint disorders. *Journal of Oral Rehabilitation.* 2010;37(10):760-765.

14. Okeson J. The classification of orofacial pains. *Oral & Maxillofacial Surgery Clinics of North America.* 2008;20(2):133–144.

15. Manfredini D, Bucci MB, Montagna F, Guarda-Nardini L. Temporomandibular disorders assessment: medicolegal considerations in the evidence-based era. *Journal of Oral Rehabilitation.* 2011;38(2):101-119.

16. Leeuw R, Klasser GD. *Orofacial Pain: Guidelines for Assessment, Diagnosis, and Management.* 5th ed: Quintessence Books; 2013.

17. Cairns BE. Pathophysiology of TMD pain--basic mechanisms and their implications for pharmacotherapy. *Journal of Oral Rehabilitation.* 2010;37(6):391-410.

18. Williamson EH. Dr. Eugene H. Williamson on occlusion and TMJ dysfunction. Interview by S. Brandt. *Journal of Clinical Orthodontics.* 1981;15(5):333-350.

19. Chisnoiu AM, Picos AM, Popa S, et al. Factors involved in the etiology of temporomandibular disorders - a literature review. *Clujul Medical.* 2015;88(4):473-478.

20. Roth RH. Temporomandibular Pain-Dysfunction and Occlusal Relationships. *The Angle Orthodontist.* 1973;43(2):136-153.

21. Okeson J. *Management of temporomandibular disorders and occlusion.* 7th ed: Elsevier Health Sciences; 2013.

22. Crawford SD. Condylar axis position, as determined by the occlusion and measured by the CPI instrument, and signs and symptoms of temporomandibular dysfunction. *The Angle Orthodontist.* 1999;69(2):103-115.

23. Ballenberger N, von Piekartz H, Paris-Alemany A, La Touche R, Angulo-Diaz-Parreño S. Influence of Different Upper Cervical Positions on Electromyography Activity of the Masticatory Muscles. *Journal of Manipulative and Physiological Therapeutics.* 2012;35(4):308-318.

24. Armijo-Olivo S, Fuentes JP, da Costa BR, et al. Reduced endurance of the cervical flexor muscles in patients with concurrent temporomandibular disorders and neck disability. *Manual Therapy.* 2010;15(6):586-592.

25. Armijo-Olivo S, Silvestre R, Fuentes J, et al. Electromyographic Activity of the Cervical Flexor Muscles in Patients With Temporomandibular Disorders While Performing the Craniocervical Flexion Test: A Cross-Sectional Study. *Physical Therapy.* 2011;91(8):1184-1197.

26. Armijo-Olivo S, Silvestre RA, Fuentes JP, et al. Patients with temporomandibular disorders have increased fatigability of the cervical extensor muscles. *Clinical Journal of Pain.* 2012;28(1):55-64.

27. De Laat A, Meuleman H, Stevens A, Verbeke G. Correlation between cervical spine and temporomandibular disorders. *Clinical Oral Investigations.* 1998;2(2):54-57.

28. La Touche R, Fernandez-de-las-Penas C, Fernandez-Carnero J, et al. The effects of manual therapy and exercise directed at the cervical spine on pain and pressure pain sensitivity in patients with myofascial temporomandibular disorders. *Journal of Oral Rehabilitation.* 2009;36(9):644-652.

29. Olivo SA, Bravo J, Magee DJ, Thie NM, Major PW, Flores-Mir C. The association between head and cervical posture and temporomandibular disorders: a systematic review. *Journal of Orofacial Pain.* 2006;20(1):9-23.

30. D'Attilio M, Filippi MR, Femminella B, Festa F, Tecco S. The influence of an experimentally-induced malocclusion on vertebral alignement in rats: a controlled pilot study. *Cranio.* 2005;23(2):119-129.

31. McGuinness NJ, McDonald JP. Changes in natural head position observed immediately and one year after rapid maxillary expansion. *European Journal of Orthodontics.* 2006;28(2):126-134.

32. Motoyoshi M, Shimazaki T, Sugai T, Namura S. Biomechanical influences of head posture on occlusion: an experimental study using finite element analysis. *European Journal of Orthodontics.* 2002;24(4):319-326.

33. Michelotti A, Manzo P, Farella M, Martina R. Occlusion and posture: is there evidence of correlation? *Minerva Stomatologica.* 1999;48:525-534.

34. Rocabado M, Johnston B, Blakney M. Physical therapy and dentistry: an overview. *Journal of Craniomandibular Practice.* 1982;1(1):46-49.

35. Solow B, Sonnesen L. Head posture and malocclusions. *European Journal of Orthodontics.* 1998;20:685-693.

36. Michelotti A, Buonocore G, Manzo P, Pellegrino G, Farella M. Dental occlusion and posture: an overview. *Progress in Orthodontics.* 2011;12:53-58.

37. Mew J. The postural basis of malocclusion: A philosophical overview. *American Journal of Orthodontics and Dentofacial Orthopedics.* 2004;126(6):729-738.

38. Aldana PA, Báez RJ, Sandoval CC, Vergara NC, Cauvi LD, Fernández de la Reguera A. Asociación entre Maloclusiones y Posición de la Cabeza y Cuello. *International Journal of Odontostomatology.* 2011;5(2):119-125.

39. Gomes L, Horta K, Gonçalves J, Santos-Pinto A. Systematic review: craniocervical posture and craniofacial morphology. *European Journal of Orthodontics.* 2014;36(1):55-66.

40. Wood DP, Elliott RW. Reproducibility of the centric relation bite registration technique. *The Angle Orthodontist.* 1994;64(3):211-220.

41. Alexander SR, R.N M, DuBois LM. Mandibular condyle position: comparison of articulator mountings and magnetic resonance imaging. *American Journal of Orthodontics and Dentofacial Orthopedics.* 1993;10(4):230-239.

42. Vion PE. *Anatomia Cefalométrica.* São Paulo: Livraria Santos Editora; 1994.

43. Rocabado M. Analisis biomecánico cráneocervical a través de una teleradiografia lateral. *Revista Chilena de Ortodoncia.* 1984;1:42-52.

44. Gregoret J, Tuber E, Escobar H, Matos da Fonseca A. *Ortodoncia y cirugía ortognática: diagnóstico y planificación*. 2nd ed. Madrid: NM ediciones, 2007.

45. Graber L, Vanarsdall Jr R, Vig K. *Orthodontics: current principles and techniques.* 5th ed. Philadelphia: Elsevier Health Sciences; 2011.

46. Sonnesen L, Bakke M, Solow B. Temporomandibular disorders in relation to craniofacial dimensions, head posture and bite force in children selected for orthodontic treatment. *European Journal of Orthodontics.* 2001;23(2):179-192.

47. Solow B, Tallgren A. Head posture and craniofacial morphology. *American Journal of Physical Anthropology.* 1976;44(3):417-435.

48. Portney LG, Watkins MP. *Foundations of clinical research: applications to practice.* 3rd ed: Prentice Hall; 2009.

49. Field A. *Discovering statistics using SPSS: and sex and drugs and rock 'n' roll* 4th ed. London: Sage; 2013.

50. von Piekartz H. *Craniofacial Pain.* Elsevier; 2007.

51. Rammelsberg P, LeResche L, Dworkin S, Mancl L. Longitudinal outcome of temporomandibular disorders: a 5-year epidemiologic study of muscle disorders defined by research diagnostic criteria for temporomandibular disorders. *Journal of Orofacial Pain.* 2003;17(1):9-20.

STUDY V

Common symptoms of temporomandibular disorders do not mean same treatment plans: A case series

Submitted for publication in

Australian Journal of Orthodontics

Common symptoms of temporomandibular disorders do not mean same treatment plans: A case series

Paço, M[1]; Chaves, P[1]; Pinho, F[1,2,3]; Lemos, C[4]; Costa, R[2]; Duarte, JA[5]; Pinho, T*[1,4]

[1]CESPU, Instituto de Investigacão e Formação Avançada em Ciências e Tecnologias da Saúde, Gandra - Paredes, Portugal.

[2]Universidade de Aveiro – Escola Superior de Saúde, Aveiro, Portugal; [3]Algoritmi - Universidade do Minho, Guimarães, Portugal.

[4]IBMC - Inst. Biologia Molecular e Celular; i3S - Inst. Inovação e Investigação em Saúde, Universidade do Porto

[5]CIAFEL, Faculdade de Desporto da Universidade do Porto, Porto, Portugal.

* Corresponding author: Teresa Pinho; PhD; +351224157151 teresa.pinho@iucs.cespu.pt

ABSTRACT

Introduction: Temporomandibular disorders (TMDs) present several risks and different contributing factors with consequently diverse treatment approaches. It is important to recognize what patients' characteristics may benefit from orthodontics, from physiotherapy, from other treatment modalities or even from a combined approach.

Cases presentation: We present three cases of patients with common TMDs signs and/or symptoms and different treatment approaches and outcomes, and our aim is to understand what might explain the different outcomes observed and also provide a rationale about the skeletal, muscular, facial and occlusal characteristics that may be indicative of a particular intervention benefit.

Conclusion: It has been shown that orthodontics plays an important role solving occlusal problems as well as changes in the vertical dimension. On the other hand, physiotherapy was effective in pain management and range improvement, when musculoskeletal changes were clearly found. Finally, it has also been shown that a multidisciplinary approach may be crucial, and the clinician should be aware of a comprehensive assessment, valuing all the contributing factors, namely the psychological ones.

INTRODUCTION

Temporomandibular disorders (TMDs) are defined as a group of musculoskeletal and neuromuscular conditions that involve the temporomandibular joints (TMJs), the masticatory muscles and all associated structures,[1] whose etiology is not well known.[2,3] TMDs can affect from 10% to 25% of the population[4,5] and it is the most common chronic orofacial pain condition. TMDs have several contributing factors as structural, neuromuscular, occlusal, psychological, genetic and parafunctional habits, among others.[6-10] Orthodontic treatment is considered to be one of the first options regarding malocclusions[11,12] and when necessary it is accompanied by orthognathic surgery.[11] The main therapeutic objective is to obtain a normal occlusion and a function improvement, when TMDs are present. Orthodontics has been historically associated with the development of TMDs, however several studies have demonstrated that there is no relationship between these two variables.[13-16] These contradictory data may be the result of specific characteristics of the patients submitted to orthodontic treatment. Considering all the contributing and risk factors to develop TMDs, a thorough assessment is important as well as a multidisciplinary approach to address all the impairments presented by the patients. Physiotherapy seems to be an effective treatment modality to address pain, range of movement and motor control issues in TMDs patients.[17-19]

It is important to acknowledge what patients' characteristics may benefit from orthodontics, physiotherapy, other treatment modalities or even from a combined approach therapy. For these reasons, we present three cases of TMDs patients, with common symptoms and different treatment approaches and outcomes, and our aim is to understand what might justify the different outcomes observed and also provide a rationale about the skeletal, muscular, facial and occlusal characteristics that may be indicative of a particular intervention benefit.

MATERIALS AND INSTRUMENTS

Fonseca's Anamnestic Index (FAI) is a low cost and easy to apply instrument proposed in the Portuguese language, consisting of 10 questions whose answers are arranged in a three-point scale format ("No", "Sometimes", "Yes"). It is used to classify individuals according to TMDs' severity (score 0-15: "TMDs Free", score 20-40: "Mild TMDs", score 45-60: "Moderate TMDs" and score 70-100: "Severe TMDs"), and also to screen patients in diagnosing TMDs.[20] The main advantages are the simplicity of its application, and the fact that it does not need a physical examination of the patient, which makes it suitable for fast epidemiological screening.[21] It has a good correlation coefficient (r=0,6169) with the Helkimo Index.[20]

Non-neural muscle tone and stiffness of anterior temporalis and masseter muscles were determined at rest using a hand-held myometer (MyotonPRO®; Myoton Ltd, Estonia). This method measures the viscoelastic response of the muscle due to a brief (15 milliseconds) mechanical impulse (force 0.4 N) on the skin surface above the muscle (http://www.myoton.com/en/Technology/Technical-specification). The device was used in multiscan mode, where one measurement corresponded to the mean of six mechanical taps. If a measurement failed to fulfill the parameters (variation coefficient lower than 3%), an error message was displayed and the trial was repeated. These procedures have already been reported in the literature.[22-25] These studies have demonstrated the validity and reliability of Myoton® measures in limb, trunk, and orofacial musculature. From the oscillation acceleration signal, we investigated two parameters computed in real time by MyotonPRO® software: dynamic stiffness and oscillation frequency. Dynamic stiffness characterizes the resistance of the muscle to the force that changes its shape. Oscillation frequency characterizes the muscle tone or the mechanical tension in a relaxed muscle.[26]

Electromyographic (EMG) activity was recorded during rest and mandibular movements (mouth opening and closing). Video recording was performed in parallel to signals acquisition to posterior analysis of the different movements correspondent signals. To synchronize, it was used a light emitting diode placed on the field view of the camcorder, that changed state according to electromyograph device status. The electrodes were positioned on the muscular bellies parallel to muscular fibres as already described previously.[27] A disposable reference electrode was applied to on the clavicle. Before electrode placement, the skin was cleaned with ethanol to reduce its impedance, according to SENIAN guidelines.[28]

EMG activity was recorded using the Biopac® MP 150 platform with TSD150® 20mm active electrodes at a sampling frequency of 1000 samples per second.

Using the AcqKnowledege® 4.1.0 software (Biopac® Systems Inc), the signals were IIR digital filtered in a bandpass of 25-450Hz and the root-mean-square variable was calculated over a 25ms window. Three measurements of EMG activity were performed in each movement (rest, mouth opening and mouth closing). In order to verify if there were statistically significant differences between EMG results immediately after and before physiotherapy intervention, in Case 2 and Case 3, a paired samples *t*-test was applied. Statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS), version 24 (IBM® company, Chicago, USA). The level of significance was set at α=0,05.

Cases presentation

Case 1 (PV)

The patient, a businesswoman born in 1975, was referred with urgency to the orthodontic specialist in April 2013, presenting very severe pain in both TMJ, headaches, limited mouth opening by pain (range of movement (ROM): 20mm). The patient had been subject to a previous orthodontic treatment in 2010, and referred that pain started 3 years after this treatment. The patient felt very fatigued and had lost about 15 Kg, because she was not able to eat. The pain was constant and motivated several visits to the hospital where she was medicated with nonsteroidal anti-inflammatory drug and cortisone (in the last visit the cortisone dosage was doubled). The medication produced no relief and the pain was only relieved when she positioned in lying position or performed hyperextension of the head. The patient scored the pain through visual analogue scale (VAS) with 97 mm and mouth limitation was caused by pain. Despite having a very limited mouth opening (by pain) in the emergency consultation, the patient referred the history of articular noises during mouth opening and closing, that were predominantly in the left TMJ (the patient referred that sometimes was associated with tinnitus). The patient had a history of facial traumatism (when she was 10 years old) and parafunctional habits (gum chewing). FAI score was 90, representing a severe TMDs.

Based on clinical findings and according to manual functional analysis[29] an anterior disc displacement (DD) with reduction in both TMJ's was confirmed.

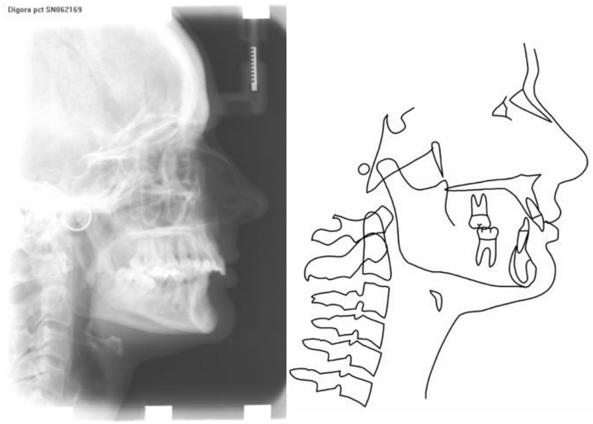
Intra oral photos showed a total Class II canine relationship bilaterally. The mandibular dental midline was deviated to the left related to the maxillary dental midline, this one centered with the facial midline (Fig 1). Crowding was not present neither in the maxilla nor mandible but there was a high curve of Spee.

Study V - Figure 1: Extra and intra-oral photos before orthodontic treatment (Case 1)

The panoramic radiograph showed the extruded and mesially inclined second and third lower left molars, which had tipped into the spaces created by the missing second premolar and first molar. Also the second lower right premolar and the first upper molar were missing by extraction. Asymmetrically positioned condyles were also evident (Fig 2).

In maximum intercuspidation there was an apparent block of the mandible due to the incisors and canines high deep bite and high overjet. As well as a Frankfort-mandibular plane angle (FMA) measure consistent with a hypodivergent facial pattern, contributing to a reduction in the vertical dimension of occlusion. The angle between A point, *nasion*, and B point (ANB) was consistent with a skeletal Class I (Fig 3 and Table 1).

Study V - Figure 2: Panoramic x-ray before orthodontic treatment (Case 1)



Study V - Figure 3: Lateral cephalometric radiograph and tracing before orthodontic treatment (Case 1)

It is important to remember that this was an emergency situation, and for that reason dental casts were not mounted in centric relation.

Treatment objectives:

- Reduce pain;
- Increase vertical dimension;
- Recuperate disc displacement (bilaterally);
- Reconstruct the occlusal plane;
- Improve the Spee curvature (with posterior extrusion associated with intrusion and pro-inclination of the inferior incisors);
- Improve mandibular position.

<u>Intervention performed</u>

As a first intervention measure a temporary occlusal composite bite was applied on upper canines and on posterior occlusal face of second molars in order to increase vertical dimension, improve the apparent block of the mandible and reduce pain (Fig 4).

Once the pain was relieved, the orthodontic treatment was planned and began in May 2013 and ended in May 2015. During orthodontic treatment, the posterior temporary occlusal composite bites were progressively removed as the mandible was positioned in a more stable occlusal position. The canine temporary occlusal composite bites were maintained in order to enable the extrusion of posterior teeth allowing the increase on posterior vertical dimension and consequently reposition the mandible anteriorly, improving overjet and overbite.

Study V - Figure 4: First intervention, temporary occlusal composite bite (Case 1)

During orthodontic treatment, pain was completely controlled, with the patient reporting its presence only sporadically, less intense and completely controllable. At the end of the treatment the patient reported no pain.

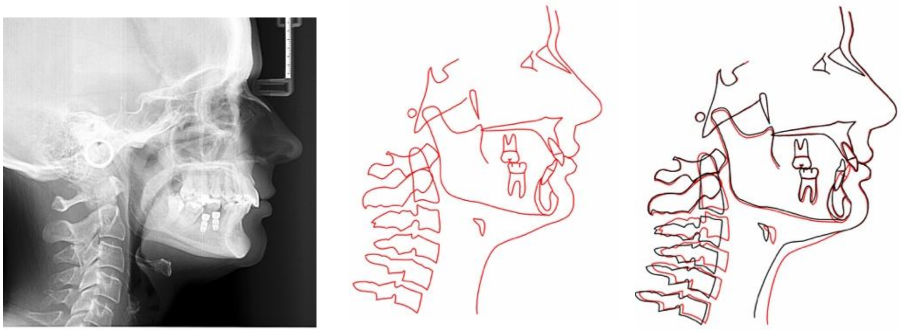
<u>Treatment results</u>

The major aim of attaining a stable dental occlusion and TMJ harmony was accomplished. A bilateral Class I canine relation, upper and lower midlines co-incident with the facial midline, and the establishment of a normal overjet and overbite relationships were obtained (Fig 5).

Study V - Figure 5: Extra and intra-oral photos after orthodontic treatment (Case 1)

Cephalometric measurements and superimpositions (Table 1 and Fig 6, respectively) document the changes produced by treatment, highlighting the occlusal vertical dimension increased and the pro inclination in the lower incisor.

On the contention phase the implant and crowns were placed, and the panoramic radiograph confirmed good root positioning.



Study V - Figure 6: Lateral cephalometric radiograph, tracing after orthodontic treatment and general overlap before and after orthodontic treatment (Case 1)

<u>Clinical evolution assessment in contention phase</u>

One year after orthodontic treatment the patient was observed by a physical therapist regarding clinical signs and symptoms, FAI as well as muscular properties (non-neural muscle tone and stiffness) and EMG activity from anterior temporalis and masseter muscles.

The patient presented a canine Class I bilaterally, with the maxillary dental midline centered with the mandibular dental midline, overbite and overjet were corrected (Fig 7).

Study V - Figure 7: Extra and intra-oral photos, one year after the end of orthodontic treatment (Case 1)

The cephalometric cranio-facial-cervical analysis showed an improvement of the outcomes (Table 1). The patient had no pain complaints regarding headaches and reported that only occasionally feels pain on the TMJ (VAS= 5 mm). Regarding mouth ROM, it was completely restored, and despite some articular noises that remained on the left side during mouth opening and closing movements, their frequency and intensity decreased. FAI final score was 10 that corresponds to a TMDs free score. On muscular palpation, the patient presented tenderness on masseter muscles (bilaterally), posterior temporal (on the left) as well as on the upper trapezius and levator scapulae (bilaterally), however these were not valued by the patient.

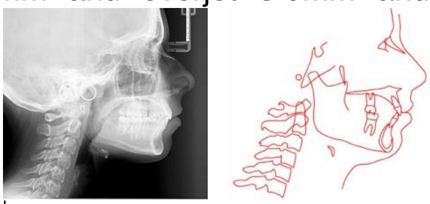
Tables 2 and 3 show the results regarding EMG and muscular properties, respectively.

Case 2 (ML)

The patient, a woman born in 1995, student that was referred to the orthodontic specialist in December 2015 with history of previous orthodontic treatment only in upper arch, from March 2014 until March 2015, and history of pain in the face (bilaterally, although predominant in the left side), limited mouth opening (due to pain) and articular noises. There was agenesis of both upper second premolars and a molar and canine Class I relationship bilaterally (Fig 8). The orthodontic treatment performed aimed to provide dental alignment and leveling and also to get the balance of spaces corresponding to agenesis of the upper second pre-molars agenesis in order to prepare to prosthetic rehabilitation. This intervention was effective regarding occlusal alterations; notwithstanding it did not provide symptoms relief to the patient.

Study V - Figure 8: Extra and intra-oral photos after orthodontic treatment (Case 2)

After orthodontic treatment overbite was 3.5 mm and overjet 3.0mm and maintained an ANB measure consistent with a skeletal Class II. The FMA measure was improved, though maintaining consistent with a hypodivergent facial pattern (Fig 9; Table 1). Dental casts showed no difference between centric relation and maximal intercuspidation.



Study V - Figure 9: Lateral cephalometric radiograph and tracing after orthodontic treatment (Case 2)

On December 2015, after the orthodontic treatment and before the prosthetic rehabilitation the patient was assessed by a physical therapist regarding clinical signs and symptoms, FAI as well as muscular properties and EMG activity from the temporalis and masseter muscles.

The patient had pain complaints on the left hemiface including the left TMJ (VAS=8,3mm). The pain was worse during chewing and mouth opening movements (no pain at rest). Regarding mouth ROM, there was a limitation (ROM=20mm) by pain and the articular noises remained during mouth opening (on the left). FAI final score was 40, that correspond to a light TMDS score. On muscular palpation, the patient presented pain on masseter muscle (on the left), anterior and posterior temporal muscle (on the left) as well as tenderness on the upper trapezius and levator scapulae (bilaterally). When asked about the pain on palpation, the patient recognized it as her "usual pain".

Physiotherapy intervention performed

In order to address the impairments found, the physiotherapy session included the following procedures: Patient education (explaining the diagnosis, intervention and empowering the patient through teaching home exercises); cranio-cervical mobilization; TMJ mobilization (distraction and lateral movements); trigger points therapy on the left masseter muscle (massage, manual therapy, stretching), therapeutic exercise (condylar rotation, opening reeducation).

Immediately after physiotherapy intervention, the patient was asked about pain intensity, which had decreased (VAS=54 mm) and had mouth opening improved (ROM= 33 mm).

Tables 2 and 3 presents the results regarding EMG and muscular properties (non-neural muscle tone and stiffness), respectively, immediately before and after physiotherapy intervention.

Since physiotherapy improved the patient's symptoms, the treatment was continued beyond the scope of this study. Clinically, the patient kept improving symptoms, having restored full ROM. The patient reports that very sporadically feels pain (less intense) and has articular noises (less intense), which she is able to manage with home exercises. Moreover, she also refers that when she is more stressed there is a tendency to symptoms' increase.

Case 3 (CF)

The patient, a woman born in 1983, nurse, was referred to the orthodontic specialist in April 2009, complaining of pain in both TMJ and articular clicking (during mouth opening) in the right side with limited mouth opening. The pain was intensified upon chewing and was worse at the end of the day. It was localized at the TMJ, face, mandibular and maxilla (bilaterally but more aggravated on the left side). The patient graduated the pain through VAS (60mm – generally; 90mm - during popping of TMJ). Regarding limited mouth opening the active ROM was 36mm and the passive ROM was 41mm and during the movement there was a deviation to the left until half the available ROM and then centered on maximum opening. The patient also complained about daily locking of the TMJ, though reducible with the maneuver. Protrusion was done only with right incisor contacts. On the left and also right laterotrusal movement, the pain appeared only in the left TMJ.

The symptoms did not seem to be related, notwithstanding accordingly with the patient "when I was more nervous I did more pressure in the mouth". There was no medication intake at that time. The patient considered to be a stressed person and had history of scoliosis and lumbar hiperlordosis. FAI score was 90, representing a severe TMDs.

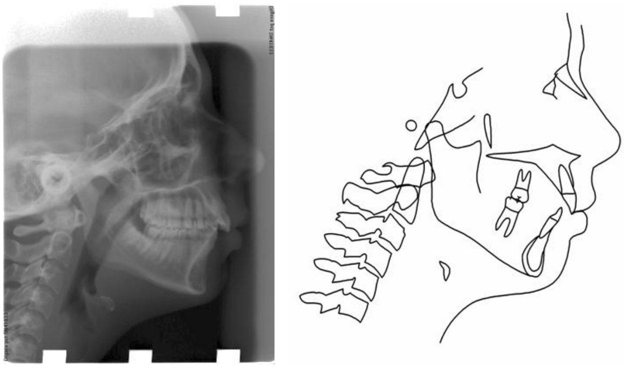
Extra-oral photos showed asymmetry of the lower third of the face and a shift of the mandible to the right. Noticeable differential gingival display reflected an occlusal plane canted upward on the left side, with a high-compensated occlusion. There was also a scissor bite on the left first premolars that collapsed more this malocclusion (Fig 10).

Intra oral photos showed a molar and canine full Class II relationship bilaterally. The maxillary dental midline was centered with the mandibular dental midline. However, the upper dental midline was inclined to the left in relation to the facial midline, the same side that the occlusal plane was canted upward (Fig 10).

Crowding was not present neither in the maxilla or mandible. There was labial tipping of the anterior maxillary teeth with normal overbite and high overjet in maximum intercuspidation.

Study V - Figure 10: Extra and intra-oral photos before orthodontic treatment (Case 3)

Lateral cephalometric analysis showed normal mandibular angle, skeletal Class II with a normal maxillary position, and a retrusive mandible. The FMA measure was consistent with an hyperdivergent facial type (Fig 11, Table 1).



Study V - Figure 11: Cephalometric radiograph and tracing before orthodontic treatment (Case 3)

Pre-treatment dental casts mounted in articulator SAM 3® and mandibular position indicator (MPI), showed a difference between centric relation and maximal intercuspidation position [right condyle: (X = -1,5; Z = +2.5); left condyle: (X = -1; Z = +3)].

Based on clinical findings and according to manual functional analysis [29] (painful left TMJ under active and passive compressions) an anterior DD without reduction in the left TMJ was confirmed as well as a diagnose of anterior DD with reduction on the right TMJ (the patient stated that had experienced reciprocal clicking). Concerning radiological findings, panoramic x-ray showed a non-symmetrical relationship between the left and the right condyle (the left condylar head was pointed with a deplaned anterior surface) (Fig 12). The bilateral anterior DD was confirmed by MRI. Anterior DD without reduction was confirmed in the left side and anterior DD with reduction was determined in the right TMJ, which explains the symptom of clicking during mouth opening. In closed mouth position, the disc was placed anteriorly but recovers his correct position in open mouth position; however, the mobility of the right condyle is more pronounced than in the left joint. The condylar head was slightly pointed with a hint of osteoarthritic changes appearing as a thickened tip of the cortical bone. Subchondral structures had an adequate signal and there was no articular effusion.

Study V - Figure 12: Panoramic x-ray before orthodontic treatment (Case 3).

Despite the difference in height of the occlusal plane a symmetric mandibular aspect is noted during the clinical testing of midline coincidences. This proves that the lower dental midline is centered within the mandible. The chin was not shifted to the same side of the occlusal plane that was canted upward probably because of the scissor bite condition on the left premolars that contra balanced this tendency and centered the chin with mandibular dentoalveolar compensations.

Treatment objectives

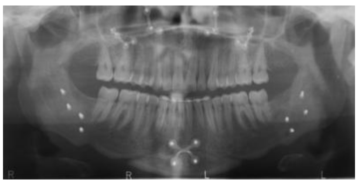
- Reduce pain;
- Decrease muscular activity;
- Correct the articular position;
- Reconstruct the occlusal plane, with asymmetrical maxillary impaction (more at the right – transversal plane);
- Decompensate sagittal and transversal dental arches;
- Recuperate disc displacement (bilaterally);
- Improve smile asymmetry;
- Correct mandibular position.

Intervention performed

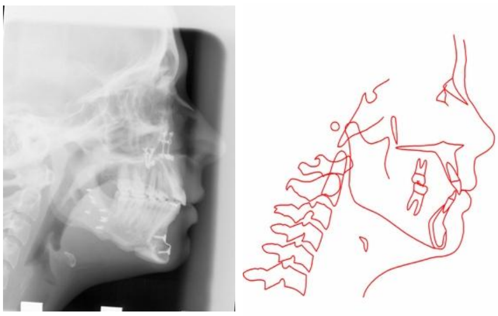
A stabilization splint in centric relation position was the first treatment option in order to reduce pain, decrease muscular activity and improve the articular position. To improve occlusion stability and smile asymmetry a bi-maxillary surgery was performed with maxilla impaction at the right. The 3rd molars were extracted so that the posterior discrepancy was corrected and to facilitate the sagittal mandibular surgery. After this a pre-surgical orthodontic treatment (October 2010) was performed with a multi-bracket fixed treatment (.022 x .025'' slot) in order to align, level and correct the compensation from the mandibular and maxilla arcades. During the subsequent 12 months the symptoms were aggravated, with more intense pain on the left, opening mouth difficulties and pain in all mandibular movements. In order to reduce pain and muscular activity a splint was given to the patient to use during the night. Since the conservative treatment (splint and orthodontic treatment) was not effective regarding TMDs' symptoms, it was decided that the patient would benefit from arthroscopic treatment, prior to maxillofacial surgery, to reduce pain, muscular activity, improve the disc position and mandibular range of movement. So, in March 2012, the patient was subject to arthroscopic treatment, which confirmed internal derangement of both TMJs, with intra-articular adhesions. The pre-surgical orthodontic study confirmed the need for a bilateral maxillary impaction (right: 3mm, left: 1mm) and mandibular advancement (7mm). On June 2013, the surgical treatment was performed, with a LeFort I osteotomy, a nasal inferior turbinectomy, a remodelation of the piriformis fossa and osteotomy of the sagittal ramus of the mandible. There was a rigid fixation with titanium plates and screws, and the mentoplasty performed aimed to advance and deviate the menton to the left. After surgery, the analysis from the facial profile showed a significant improvement in all the parameters (Table 1) with a normalization of the cervicofacial proportion (Fig 13).

Study V - Figure 13: Extra and intra-oral photos after orthodontic treatment (Case 3).

After orthodontic-surgical treatment (Fig 14-15) the patient referred that pain was a rare event, that occurred on the mandible and maxilla bone (bilaterally) with a pain intensity of 20 mm according with VAS. There was only a limited mouth opening in the end-range without blocking of the joints. Regarding joint noises, the patient referred that the intensity was lower but the noises were still present. The patient ended the treatment with a final score of 50 (according to FAI), which indicates a moderate TMDs. It should be noted that during the treatment procedures (2011) the patient was medicated with anti-depressives.



Study V - Figure 14: Panoramic x-ray after orthodontic treatment (Case 3).



Study V - Figure 15: Lateral cephalometric radiograph and tracing after orthodontic treatment (Case 3)

The relapse

One and a half month after ending orthodontic treatment, the relapse began, showing a tendency to open bite, with lingual interposition (a “tongue pricker” was applied on lingual face of the lower incisors). The patient was advised to use the contention only when sleeping and to seek for physiotherapy assistance. Three months after ending orthodontic treatment the sagittal and vertical relapse was confirmed with guides loss and dental contacts only on the first pre-molars. Despite the relapse (the inferior dental midline was deviated and there was a molar and canine class II on the right), the patient showed no symptoms and no complaints. In an attempt to control the relapse, on 18th June 2013 it was applied triangular elastics (2oz, 3/16’’: 13-43-44 and 23-43-44), for daily use (1 month), which aggravated the symptoms. On December 2013 it was applied an elastic (2oz 5/16’’) anteriorly to use during the night for a month. During these procedures the symptoms kept persisting, so the elastics were removed on January 2014, which coincided with the symptoms relieve. In June 2014, the symptoms got worsen, and an occlusal splint was given to the patient, which slightly improved the symptoms (that were bilateral, but worse at the right).

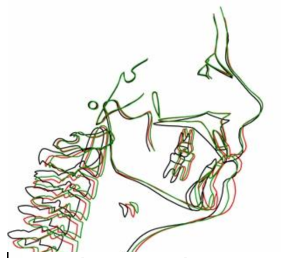
Reassessment (2015)

On February 2015, one year and a half month after the end of orthodontic treatment (Fig 16), the patient presented with a molar and canine Class II on the right, a deviation to the right of the inferior dental midline as well as the menton and anterior inocclusion.

Study V - Figure 16:Extra and intra-oral photos one year after the end of orthodontic treatment (Case 3)

The cephalometric analysis shows an aggravation of the outcomes due to the proportion between the posterior and anterior facial height, with a posterior rotation of the mandible.

The lower 1/3 of the face showed a vertical increase with a menton posterior rotation (aggravating the cervicofacial proportion) (Fig 17). The patient has no pain complaints on the TMJ and no range of movement limitations, however feels muscular pain during chewing, pain on the occipital and cervical area, and complaints about occasional headaches and TMJ noises. FAI score was 40, showing that there is the presence of light TMDs.



Study V - Figure 17: General lateral cephalometric overlap before, after and orthodontic treatment (Case 3).

Lateral cephalometric analysis showed a skeletal Class II and a hyperdivergent facial type. Overbite measure was lower and overjet remained the same (Table 1).

In May 2015, the patient had anxiety crisis and was diagnosed by psychiatry as obsessive-compulsive syndrome, depression and sleep disturbances. The patient re-started to be medicated with anti-depressive (which had happened on 2011) and started to be followed by psychiatry consultation.

In October 2015, the patient was assessed by a physical therapist regarding clinical signs and symptoms, FAI as well as the EMG activity and muscular properties from the temporalis and masseter muscles. After the assessment, a physiotherapy intervention and reassessment were performed. The results from EMG and muscular properties are presented on table 2 and 3 respectively, with the results immediately before and again after physiotherapy intervention.

The anamnesis revealed the presence of pain scored as 43 mm (VAS), localized at the TMJ (bilaterally) that was worse at the end of the day. The patient also complained about pain in the face and cervical as well as recurrent headaches (twice a week). The patient had no limitation on mouth opening, however she referred weakness while eating and chewing. Concerning articular noises, they were present on maximal mouth opening, and there was a click on the left in maximal mouth opening. FAI score revealed the presence of severe TMDs (score 70/100).

In order to address the impairments found, the physiotherapy session included the following procedures: Patient education (explaining the diagnosis, intervention and empowering the patient through teaching home exercises); craniocervical mobilization; TMJ mobilization (distraction and lateral movements); trigger points therapy on the left masseter muscle (massage, manual therapy, stretching), therapeutic exercise (condylar rotation, opening reeducation).

Immediately after physiotherapy intervention, the patient was asked about pain intensity, which had slightly decreased (VAS=38mm).

It should be noted that between February and September 2015, after the assessment, and because musculoskeletal impairments were found, the patient performed physiotherapy treatments. Physiotherapy has shown inconclusive results, with the patient reporting some symptoms relief; this relief was not maintained through the sessions. For that reason, the patient was subject to a further analysis through muscular properties analysis and EMG, performed immediately before and after physiotherapy intervention, and those are the results reported in this study.

Study V - Table 1: Cephalometric records of the three cases

	Cephalometric Variables	Case 1		Case 2		Case 3		
		Pre OT	Post OT	Pre OT	Post OT	Pre OT	Post OT	Contention
Skeletal and facial variables	**FMA**	17.5	18.8	19.8	20.9	31.1	29.6	31.8
	IMPA	91.7	109.1	104.0	101.7	102.2	95.7	96.1
	SNA	79.4	78.9	83.1	83.8	78.7	77.0	79.2
	SNB	75.1	74.6	77.3	77.8	70.9	73.0	72.7
	UI to NA (angle)	19.7	21.0	17.5	14.7	13.6	21.7	19.3
	ANB	4.3	4.3	5.8	6.1	7.8	5.0	6.5
	AO-BO	4.6	4.2	3.9	3.9	2.3	0.8	2.2
	Posterior Facial Height	52.5	52.6	49.2	53.1	42.0	46.1	45.2
	Posterior/Anterior Index	63.2	64.8	0.81	0.83	69.7	77.3	77.3
	Overjet (B1-A1 Horz.)	9.5	4.2	3.7	3.0	7.5	3.2	3.2
	Overbite (B1-A1 Vert.)	8.3	4.4	3.5	3.5	1.6	1.8	0.6
	Interincisal Angle (A1-B1)	143.9	124.9	128.8	132.6	123.6	124.4	125.2
Cranio-cervical variables	**CV Angle**	98.1	104.7	93.9	89.1	81	75.4	78.1
	AO	14.6	12.7	5.9	4.7	5.3	8.1	8.3
	C1-C2	19	16.8	16.4	16.6	16.6	22.3	21.9
	C0-C2	12.3	12.3	4.6	6.3	10.2	10.9	11
	C3-Rgn	77.5	69.5	68.6	82.2	89.3	90.6	85.1
	C3-H	35.4	32	31.7	35.7	37.3	36.9	26.3
	H-Rgn	42.1	37.6	40.8	46.7	48.5	56.3	50.7
	H-H1	-0.6	2.7	11.7	2.4	8.9	10.6	8.9
	OPT/CVT	13.7	17.3	19.1	29.8	29	27.4	26.2
	CVT/EVT	8.7	13.7	16.3	13.6	11	7.3	8.8

OT – Orthodontic Treatment

Study V - Table 2: EMG activity records of the 3 cases, and p value of paired samples t-test

EMG Muscle (volts)	Case 1	Case 2			Case 3		
	Mean (SD)	**Pre Intervention Mean (SD)**	**Post Intervention Mean (SD)**	**T' Test (*p*)**	**Pre Intervention Mean (SD)**	**Post Intervention Mean (SD)**	**T' Test (p)**
Left Temporalis (rest)	0,002 (0,001)	0,006	0,047	-	0,999	0,001	-
Right Temporalis (rest)	0,004 (0,001)	0,062	0,014	-	0,996	0,003	-
Left Temporalis (mouth opening)	0,002 (0,001)	0,007 (0,001)	0,061 (0,012)	0,018	2,883 (0,144)	0,0,92 (0,155)	<0,0001
Right Temporalis (mouth opening)	0,004 (0,001)	0,114 (0,244)	0,012 (0,001)	0,019	1,237 (0,085)	0,005 (0,001)	0,002
Left Temporalis (mouth closing)	0,002 (0,0003)	0,006 (0,001)	0,059 (0,012)	0,018	2,45 (0,364)	0,007 (0,010)	0,007
Right Temporalis (mouth closing)	0,004 (0,0004)	0,063 (0,014)	0,012 (0,001)	0,026	1,60 (0,18)	0,004 (0,001)	0,004
Left Masseter (rest)	0,002 (0,0002)	0,002	0,001	-	0,996	0,003	-
Right Masseter (rest)	0,007 (0,0003)	0,456	0,001	-	0,999	0,001	-
Left Masseter (mouth opening)	0,129 (0,016)	0,007 (0,009)	0,001 (0,00001)	0,357	22,723 (2,72)	0,251 (0,146)	0,005
Right Masseter (mouth opening)	0,01 (0,005)	0,246 (0,121)	0,002 (0,001)	0,073	4,58 (0,272)	0,006 (0,002)	0,001
Left Masseter (mouth closing)	0,041 (0,002)	0,002 (0,001)	0,002 (0,001)	1	32,443 (1,223)	0,288 (0,283)	0,001
Right Masseter (mouth closing)	0,018 (0,001)	0,31 (0,175)	0,002 (0,001)	0,093	2,467 (0,578)	0,004 (0,002)	0,018

SD: Standard Deviation

Study V - Table 3: Muscular properties records of the 3 cases

Muscle	Muscular properties	Case 1	Case 2		Case 3	
		Mean (SD)	Pre-Intervention Mean (VC)	Post-Intervention Mean (VC)	Pre-Intervention Mean (VC)	Post-Intervention Mean (VC)
Right Masseter	Non-Neural Muscle Tone (Hz)	15,7(1,7)	12,4(0,8)	12.1(1,0)	15,8 (0,9)	15,9 (1,4)
	Stiffness (N/m)	353 (1,6)	256(2,1)	241(2,2)	350 (0,7)	355 (1,5)
Left Masseter	Non-Neural Muscle Tone (Hz)	15 (1,3)	12(0,8)	10,9(0,7)	14,4 (1,3)	15,0 (2,4)
	Stiffness (N/m)	323(1,4)	205(2.6)	173(1,2)	304 (1,2)	310 (2,3)
Right Temporalis	Non-Neural Muscle Tone (Hz)	45,7(1,9)	44,5(3,0)	48,6(1,3)	34,6 (2,2)	35,2 (2,7)
	Stiffness (N/m)	1142(3,0)	1127(1,1)	1799(2,9)	757 (1,9)	793 (0,7)
Left Temporalis	Non-Neural Muscle Tone (Hz)	39(1,3)	44,4(2,7)	39,5(2,5)	34,9 (2,2)	31,3 (2,4)
	Stiffness (N/m)	789(0,5)	1585(2,5)	1101(2,4)	776 (0,8)	782 (1,2)

VC - variation coefficient

DISCUSSION

This case series highlights the different approaches and effects, observed in three different patients submitted to orthodontic treatment, presenting with similar symptoms (pain in the TMJ region, mouth opening limitation, articular noises) but with different dental, skeletal, muscular and psychological characteristics, thus demanding different treatment interventions.

In Case 1 there were signs and symptoms of TMDs and occlusal changes and the orthodontic treatment was effective in the symptoms resolution. This resolution is indicative that the strategy used targeted the causes of the problem. The rationale behind the strategy used lies in the fact that, the patient presented with a high deep bite and high overjet, associated with an hypodivergent facial type and a decreased vertical dimension. This decrease implies more blockage of the mandible which, combined with a decreased mandibular plane angle and with the fact that the lower incisors were blocked in the upper incisors cingulum, resulted in a forced posteriorized mandible position. This posteriorized position of the mandible may result in the compression of the retrodiscal tissues, which is known to produce severe symptoms.[30,31] Knowing this, the main objective was to increase vertical dimension and unlock the mandible. As the vertical dimension was improved and the mandible was repositioned anteriorly, allowing its stability by the improvement of the dental occlusion, curve of Spee correction by lateral extrusion and pro-inclination of the lower incisors in order to reduce also the initial overjet. This pro-inclination was allowed due to a good gingival and a hypodivergent facial biotype. This mandible repositioning eliminated the compressive forces acting over retrodiscal tissues leading to symptoms relief. When compared the cephalometric values pre and post orthodontics, regarding craniovertebral measurements, it shows that there was a slight increase in cervical lordosis associated with an anteriorization of the hyoid bone. When the patient was assessed regarding musculoskeletal parameters there were no clinically significant impairments and EMG analysis retrieved no changes and no significant asymmetry between left and right masticatory muscles. Also FAI score was consistent with TMDs free.

In Case 2, there were signs and symptoms of TMDs, the occlusion was stable with a normal sagittal relationship, normal overbite and overjet, although there was bilateral upper second premolar agenesis. In this case, the orthodontic treatment only in the upper arch intended to prepare to prosthetic rehabilitation, and was successful regarding occlusal factors, however the TMDs' symptoms remained the same. When compared the cephalometric values pre and post orthodontic treatment, regarding craniovertebral measurements, it shows that there was a decrease in the craniovertebral angle, which associated with the variables related to cervical lordosis (OPT/CVT, CVT/EVT) is consistent with a rectification of the lordosis. Cephalometric data also showed that there was a downward position of the hyoid bone. When the patient was assessed regarding musculoskeletal parameters there were clinically significant impairments, with the patient presenting pain on muscular palpation and EMG and muscular properties analysis' retrieved changes demonstrating asymmetry between left and right masticatory muscles. Considering these alterations in the musculoskeletal system, a physiotherapy intervention was performed, and immediately after the intervention the patient reported less pain, an improvement in the mandibular opening and was able to perform mandibular movements without articular noises, as long as she was aware about the correct movement pattern. Regarding EMG results, these showed a significant change between pre and post physiotherapy intervention, narrowing the asymmetry between left and right muscles. Muscular properties results' showed a decrease in non-neural muscle tone as well as on dynamic stiffness, more evident on the left muscles. However, the right temporalis did not follow this tendency and revealed an increase in both outcomes. The tone is the intrinsic tension, on the cellular level, of a muscle in its resting state. A high tone causes reduced blood supply and consequently slower muscle recovery and quicker muscle fatigue. The stiffness characterizes the resistance of the muscle to the force that deforms its shape and a higher stiffness leads to an inefficient economy of movement.[32] The increase found on non-neural muscle tone and stiffness of the right temporalis, was not consistent with clinical findings (pain decrease and ROM improvement) nor EMG findings. When analyzed EMG findings from the right temporalis, a significant decrease is found in all the moments assessed (rest, mouth opening, mouth closing). Notwithstanding, the decrease in muscle tone and stiffness more evident on the left, may be explained by the fact that the left hemi-face was the pain side, and the muscles tend to have a protective contraction from pain. So, being the left muscles the most affected by pain, these muscles were also the ones having a greater response to manual therapy. Manual therapy is sought to produce a significant reduction on resting pain, that may be explained by peripheral, spinal, supraspinal and neurophysiological mechanisms.[33] In response to injury, the peripheral nociceptors and inflammatory mediators act together, and manual therapy may directly interfere with this process.[33] In addition, manual therapy has proven to trigger mechanical hypoalgesia and other changes related to lessening of temporal summation and the activation of the sympathetic nervous system, suggesting a mechanism mediated by the periaqueductal gray and the dorsal horn of the spinal cord.[33,34]

Case 3, also presented signs and symptoms of TMDs as well as skeletal, occlusal and psychological factors. In this case, the orthodontic-surgical treatment intended to unblock and increase mandibular mobility, by improving and stabilizing occlusion. Immediately after orthodontic treatment, the objectives seemed to be accomplished with the patient reporting a decrease in all the symptoms (lower pain intensity, less frequent headaches, ROM restored, articular noises less frequent, lower FAI score) and cephalometric measures showing an improvement in all the variables. However, a month and a half after ending treatment the occlusal relapse began, in a first stance without symptoms' increase, and then with symptoms increase. When compared the cephalometric values pre and post orthodontics-surgery treatment regarding craniovertebral measurements, it shows that there was a decrease in the craniovertebral angle as well as a decrease in the variables related to cervical lordosis, which is consistent with a rectification of the cervical lordosis. Cephalometric data also showed that the hyoid bone was positioned more anteriorly, following the mandibular advancement. On reassessment, one year after ending orthodontic treatment, symptoms were aggravated (pain increase, headaches frequency increased, FAI score increased), the mandible retruded, the patient was diagnosed with depression and sleep disturbances and had generalized poliarticular pain. Cephalometric measures related to craniovertebral measurements showed a tendency to keep increasing the rectification of cervical lordosis as well as the anteriorization of the hyioid bone.

Considering the case described, and despite following all the guidelines concerning treatment procedures (such as splint therapy, orthodontic correction, arthroscopy, surgical correction and occlusal stabilization), the final result of the treatment, did not provide to the patient the resolution sought. The relapse presented in this case is not only concerning symptoms, but also concerning occlusion. However, when analyzed the patient through the biopsychosocial model, there are several factors that may contribute to the relapse, and were not considered during the orthodontic-surgical-orthodontic treatment. For instance, the treatment performed initially comprised, essentially the mandibular and occlusal correction, without encompassing the muscular and articular changes that would occur simultaneously with the orthodontic and surgical treatment. Being this patient a skeletal Class II, with a very retruded mandible and a cervico-facial proportion increased, it may be hypothesized that a previous preparation of the infra-mandibular musculature would improve its stretching ability, possibly decreasing the grade of relapse. This hypothesis grounds its foundation in the findings regarding craniovertebral outcomes as well as the hyoid bone position outcomes. These results raise the possibility that muscular length probably remained the same throughout the treatment, so, the muscle was stretched but gradually shortened towards its initial length, contributing to the skeletal relapse. It is important to note that during the orthodontic-surgical-orthodontic treatment the patient was advised to perform myofunctional and physical therapy, however, the interventions performed and its results are not possible to describe nor quantify, because these interventions were performed outside the clinical settings. When asked about these procedures, the patient reported that no symptoms improvement was felt.

The final reassessment, encompassed musculoskeletal parameters, with the patient presenting clinically significant impairments (pain in muscular palpation) as well as EMG differences among left and right sides, being this difference more pronounced on the masseter muscles and higher on the left masseter. Muscular properties results' showed differences, also on the masseter muscles. After physiotherapy intervention the EMG signals were lower at rest and during mandibular movements, in both masseter and temporalis muscles. Left masseter kept having higher EMG values compared with the right one, but the asymmetry was not as pronounced as pre-physiotherapy. Muscular properties' results showed an increase in both muscle tone and stiffness, except for the left temporalis, where muscle tone decreased. Despite EMG findings, the patient reported only a slight improvement in pain after physiotherapy.

Moreover, the patient presented and developed through the years emotional disturbances, such as depression, anxiety, sleep disturbances and obsessive-compulsive syndrome, which is known to contribute to the chronicity of TMDs.[35]

This case represents a very complex TMDs problem, with several contributing factors (occlusal, musculoskeletal, psychological), that was accompanied by several professionals but without consistent results.

CONCLUSION

This case series shows how similar symptoms may have different causes that should be addressed specifically. It has shown that orthodontics played an important role solving occlusal problems as well as changes in the vertical dimension. On the other hand, physiotherapy was effective in pain management and range improvement, when musculoskeletal changes were clearly found. Finally, it has also shown that a multidisciplinary approach may be crucial, and the clinician should be aware of a comprehensive assessment, valuing all the contributing factors, namely the psychological ones.

This study highlights the importance of etiological factors and the need to have further studies regarding TMDs subgroups, so that the clinician may confidently provide the best treatment to his patient.

REFERENCES

1 Leeuw R, Klasser GD. Orofacial Pain: Guidelines for Assessment, Diagnosis, and Management. 5th ed.: Quintessence Books, 2013.

2 Greenberg MS, Glick M, Ship JA. Burket's Oral Medicine. BC Decker Inc, 2008:223-255.

3 Oral K, Bal Küçük B, Ebeoğlu B, Dinçer S. Etiology of temporomandibular disorder pain. Ağrı 2009;21:89-94.

4 Gremillion H. The prevalence and etiology of temporomandibular disorders and orofacial pain. Tex Dent J 2000;117:30-39.

5 LeResche L. Epidemiology of Temporomandibular Disorders: Implications for the Investigation of Etiologic Factors. Crit Rev Oral Biol Med 1997;8:291-305.

6 Melis M, Di Giosia M. The role of genetic factors in the etiology of temporomandibular disorders: a review. Cranio 2016;34:43-51.

7 Cairns B, List T, Michelotti A, Ohrbach R, Svensson P. JOR-CORE recommendations on rehabilitation of temporomandibular disorders. Journal of oral rehabilitation 2010;37:481-489.

8 Fillingim R, Ohrbach R, Greenspan J, et al. Potential psychosocial risk factors for chronic TMD: descriptive data and empirically identified domains from the OPPERA case-control study. J Pain 2011;12:T46-60.

9 Cruz CL, Lee KC, Park JH, Zavras AI. Malocclusion Characteristics as Risk Factors for Temporomandibular Disorders: Lessons Learned from a Meta-Analysis. Journal of Oral Diseases 2015;2015.

10 Dıraçoğlu D, Yıldırım NK, Saral İ, et al. Temporomandibular dysfunction and risk factors for anxiety and depression. J Back Musculoskelet Rehabil 2016;29:487-491.

11 Wahl N. Orthodontics in 3 millennia. Chapter 13: the temporomandibular joint and orthognathic surgery. American journal of orthodontics and dentofacial orthopedics : official publication of the American Association of Orthodontists, its constituent societies, and the American Board of Orthodontics 2007;131:263-267.

12 Wahl N. Orthodontics in 3 millennia. Chapter 12: Two controversies: early treatment and occlusion. American journal of orthodontics and dentofacial orthopedics : official publication of the American Association of Orthodontists, its constituent societies, and the American Board of Orthodontics 2006;130:799-804.

13 Kim M-R, Graber TM, Viana MA. Orthodontics and temporomandibular disorder: A meta-analysis. Am J Orthod Dentofacial Orthop 2002;121:438-446.

14 Manfredini D, Stellini E, Gracco A, Lombardo L, Nardini LG, Siciliani G. Orthodontics is temporomandibular disorder–neutral. Angle Orthod 2016;86:649-654.

15 Luther F, Layton S, McDonald F. Orthodontics for treating temporomandibular joint (TMJ) disorders. Cochrane Database Syst Rev 2010;7.

16 Luther F. TMD and occlusion part I. Damned if we do? Occlusion: the interface of dentistry and orthodontics. Braz Dent J 2007;202:1-7.

17 Calixtre LB, Moreira RF, Franchini GH, Alburquerque-Sendin F, Oliveira AB. Manual therapy for the management of pain and limited range of motion in subjects with signs and symptoms of temporomandibular disorder: a systematic review of randomised controlled trials. Journal of oral rehabilitation 2015;42:847-861.

18 Paço M, Peleteiro B, Duarte J, Pinho T. The Effectiveness of Physiotherapy in the Management of Temporomandibular Disorders: A Systematic Review and Meta-analysis. J Oral Facial Pain Headache 2016;30:210-220.

19 Martins WR, Blasczyk JC, Oliveira MA, et al. Efficacy of musculoskeletal manual approach in the treatment of temporomandibular joint disorder: A systematic review with meta-analysis. Man Ther 2016;21:10-17.

20 Fonseca D, Bonfante G, Valle A, Freitas S. Diagnóstico pela anamnese da disfunção craniomandibular. RGO (Porto Alegre) 1994;42:23-28.

21 Campos JC, A.C, Bonafé F, Maroco J. Severity of temporomandibular disorders in women: validity and reliability of the Fonseca Anamnestic Index. Braz Oral Res 2014;28:1-6.

22 Dietsch AM, Solomon NP, Sharkey LA, Duffy JR, Strand EA, Clark HM. Perceptual and instrumental assessments of orofacial muscle tone in dysarthric and normal speakers. J Rehabil Res Dev 2014;51:1127-1142.

23 Aird L, Samuel D, Stokes M. Quadriceps muscle tone, elasticity and stiffness in older males: reliability and symmetry using the MyotonPRO. Arch Gerontol Geriatr 2012;55:e31-39.

24 Chuang LL, Wu CY, Lin KC. Reliability, validity, and responsiveness of myotonometric measurement of muscle tone, elasticity, and stiffness in patients with stroke. Arch Phys Med Rehabil 2012;93:532-540.

25 Pruyn EC, Watsford ML, Murphy AJ. Validity and reliability of three methods of stiffness assessment. J Sport Health Sci 2015;*In Press*.

26 Bailey L, Samuel D, Warner M, Stokes M. Parameters Representing Muscle Tone, Elasticity and Stiffness of Biceps Brachii in Healthy Older Males: Symmetry and Within-Session Reliability Using the Myotonpro. J Neurol Disord 2013;1.

27 Burnett C, Fartash L, Murray B, Lamey PJ. Masseter and temporalis muscle EMG levels and bite force in migraineurs. Headache: The Journal of Head and Face Pain 2000;40:813-817.

28 Hermens H, Freriks B, Merletti R, et al. European recommendations for surface electromyography - results of the SENIAN project. Enschede: Roessingh Research and Development, 1999:13-58.

29 Bumann A, Lotzmann U. TMJ disorders and orofacial pain: The role of dentistry in a multidisciplinary diagnostic approach. . Stuttgart: Thieme, 2002.

30 Sano T, P.L W, Larheim TA, Takagi R. The association of temporomandibular joint pain with abnormal bone marrow in the mandibular condyle. Journal of oral and maxillofacial surgery : official journal of the American Association of Oral and Maxillofacial Surgeons 2000;58:254-257.

31 Wilkes CH. Internal derangements of the temporomandibular joint. Pathological variations. Arch Otolaryngol Head Neck Surg 1989;115:469-477.

32 Treffel L, Dmitrieva L, Gauquelin-Koch G, et al. Craniomandibular System and Postural Balance after 3-Day Dry Immersion. PLoS ONE 2016;11.

33 Bialosky JE, Bishop MD, Price DD, Robinson ME, George SZ. The mechanisms of manual therapy in the treatment of musculoskeletal pain: A comprehensive model. Man Ther 2009;14:531-538.

34 Schmid A, Brunner F, Wright A, Bachmann LM. Paradigm shift in manual therapy? Evidence for a central nervous system component in the response to passive cervical joint mobilisation. Man Ther 2008;13:387-396.

35 Berger M, Oleszek-Listopad J, Marczak M, Szymanska J. Psychological aspects of temporomandibular disorders – literature review. Curr Issues Pharm Med Sci 2015;28:55-59.

CHAPTER 4

DISCUSSION

The results found in the several studies performed demonstrate the global burden of TMDs and also the areas where physiotherapy may play a role, namely in health promotion and function improvement. Our results showed that, considering the defined criteria in study I, physiotherapy interventions are more effective than other treatment modalities and shams in the management of TMDs concerning pain reduction, and a tendency towards improved active range of movement exists. Our results have also shown that the main risk factors associated with TMDs were: female gender, impulsiveness, tension-type headache, migraine, anxiety, facial trauma and parafunctional habits. Considering the importance of physiotherapy in health promotion, our study reported that Portuguese population have an overall positive knowledge about TMDs. Having in mind that orthodontics is one of the first treatment approaches sought by patients, our results have shown that orthodontic treatment produced statistically significant differences regarding hyoid bone position (pre orthodontic treatment *versus* post orthodontic treatment) and craniocervical posture (pre orthodontic treatment *versus* post orthodontic treatment *versus* contention phase), with the craniocervical posture being prone to return to basal values. The case series highlighted the importance of a thorough assessment and that similar symptoms may have different causes that should be addressed specifically.

When treating patients with TMDs, physiotherapy aims to decrease musculoskeletal pain, to get muscular relaxation, to reduce muscular hyperactivity, to improve muscular function and control and also to enhance articular mobility (Fonseca, Paço, & Oliveira, 2016) The results from the systematic review and meta-analysis (study I), produced evidence that physiotherapy interventions are more effective than the other treatment modalities and shams with which it was compared, in TMDs pain reduction, and that a tendency towards improved active range of movement exists. Our results are accordingly with the results of other systematic reviews (Calixtre et al., 2015) and meta-analysis (Martins et al., 2015) recently published, although in our study there were no significant differences in range of movement. Nonetheless, another recent systematic review and meta-analysis (Armijo-Olivo et al., 2016) concluded that although physiotherapy showed promising effects, there was great uncertainty about the effectiveness of physiotherapy in the treatment of TMDs (both pain and range of movement). The contradictory results between these studies and our results may be explained by different reasons. The fact that the aims of each study were not exactly the same implies that the included studies were different. For instance, our study aimed to evaluate the effectiveness of physiotherapy in subjects diagnosed with TMDs, however the operational definition of physiotherapy was very specific, targeting only Interventions performed by therapists and within the scope of physiotherapy practice, excluding studies whose intervention was acupuncture, solely home-physical therapy or electrical modalities, interventions involving passive range of movement devices along with studies with mixed treatments (physiotherapy combined with other forms of treatment). In the study from Calixtre et al. (2015) the objective was to synthetize evidence regarding the isolated effect of manual therapy in improving TMJ function (maximal mouth opening and pain), excluding studies where manual therapy could be combined with other modalities. The study from Martins et al. (2015) aimed to assess the effectiveness of musculoskeletal manual approach in TMDs patients, where there were only included studies performing any manipulations of body, muscles and bones by hands to improve healing of the craniocervical mandibular system. Studies where manual therapy could be combined with other modalities were also excluded. In the study from Armijo-Olivo et al. (2016) the objective was to summarize the evidence and evaluate the methodological quality of randomized controlled trials that examined the effectiveness of manual therapy and therapeutic exercise interventions in the management of TMDs and determine the magnitude of the effects of those interventions. Armijo-Olivo et al. (2016) included studies comparing any type of manual therapy intervention (e.g. mobilization, manipulation, soft tissue mobilization) or exercise therapy alone or in combination with other therapies compared to a placebo intervention, controlled comparison intervention, or standard care. The outcomes were pain, range of motion and oral function (measured through questionnaires). As a reflex of their different aims, the number of included studies was also very different when compared the four works. While three of them had a lower number of included studies, n=8 (Calixtre et al., 2015; Martins et al., 2015) and n=7 (our study I), the study from Armijo-Olivo et al. (2016) had 48 articles included.

Having these differences in mind and considering the fact that, for example, there are trials published that compares physiotherapy interventions combined with other interventions (Ismail, Demling, Heßling, Fink, & Stiesch-Scholz, 2007), that uses electrotherapy as a unique intervention (Taube, Ylipaavalneimi, Kononen, & Sunden, 1988) and that refer to "controlled gum chewing" as controlled masticatory exercises (Gavish, Winocur, Astandzelov-Nachmias, & Gazit, 2006), it seems plausible to assume that these kind of characteristics may mask the effects of "real physiotherapy intervention", acting as potential bias when performing an analysis about the role of physiotherapy by itself.

The positive results found in our study regarding physiotherapy effectiveness on TMDs may be explained by the pathophysiology of this condition, that is often associated with hypoxia, ischaemia and an insufficient synthesis of adenosine triphosphate that lead to an accumulation of calcium and consequently to sarcomeres shortening (Ribeiro, Paço, & Oliveira, 2016). These alterations are in line with the aims of physiotherapy intervention and its ability to directly interfere with the process of nociception by peripheral, neurophysiological, spinal, and supraspinal mechanisms (Bialosky et al., 2009a; Schmid et al., 2008b) as described previously in the first chapter (introduction).

Despite the conclusion that physiotherapy is effective in TMDs management, but considering the conflicting evidence found, it is important to account with all the factors with assumed importance in TMDs management that may enhance the physiotherapist approach to these patients. One of those factors is the ability to correctly identify TMDs risk factors, and with the growing rhythm of scientific knowledge about orofacial pain, an up-to-date knowledge about TMDs risk factors is crucial to provide the patient the best practice considering the best available evidence. Our results (study II) have shown that female gender, impulsiveness, the presence of tension-type headache, migraine, anxiety, history of facial trauma, and the presence of parafunctional habits were the main risk factors associated with TMDs in the Portuguese population. This was the first study in the Portuguese population, to our knowledge, and these results support the multifactorial aetiology currently accepted by the scientific community (Greenberg et al., 2008; Greene, 1995; Gremillion, 2000b; Liu & Steinkeler, 2013; Melis & Di Giosia, 2016a; Oral et al., 2009; Suvinen et al., 2005a). Our results were also consistent with findings of previous studies in other populations (Dıraçoğlu et al., 2016b; Fillingim et al., 2011b; Huang et al., 2002; Magalhães et al., 2014a; Michelotti et al., 2010b; Ohrbach et al., 2011a; Poveda Roda et al., 2007). One risk factor not often reported in the literature is third molar removal, that our study concluded to be a risk factor as described previously (Akhter et al., 2008a). One possible explanation may be the procedure involved in the removal of the third molar, that may constitute a trauma to the temporomandibular joint or even to the mastication muscles and may be associated with a reduction in the protective mechanisms of the person under the surgical intervention. Regarding psychosocial factors, it has already been described that they represent a high risk to develop TMDs (Akhter et al., 2013a; Buljan, 2010; Modi et al., 2012; Wright, Clark, Paunovich, & Hart, 2006a), but one psychological factor that we have found to be associated with TMDs, and not yet described elsewhere, is impulsiveness. This is an emotion regulation related disorder, as anxiety-depressive disorders, somatisation and catastrophizing, and these forms of disorders seem to contribute to chronic TMDs, mainly in the form of myofascial pain (Berger, Oleszek-Listopad, Marczak, & Szymanska, 2015a). The specific underlying psychosomatic factor seems to be associated with the individual ability to regulate high emotional activation. These disorders also seem to intensify parafunctional habits, which will exacerbate or lead to the onset of TMDs (Berger et al., 2015a). All the risk factors found and that may contribute to TMDs reinforces the need to understand the physical and psychological characteristics of an individual patient, in order to outline the best intervention for the person that is suffering.

Another aspect that is a key-factor to provide the best practice, based on the best available evidence, is literacy, an health determinant highly advocated by the WHO (World Health Organization, 2016a). Literacy may be considered a risk factor for a lower demand of healthcare and conditioning the attitude towards health choices. Taking into account that the physiotherapist is also an educator and an important element in the field of health promotion, with capabilities to influence the health of the individual (European Region - World Confederation for Physical Therapy, 2016), it is fundamental that this professional is aware of the importance of knowledge and educating the patient about his condition (diagnosis, prognosis, natural course, self-management). To evaluate the knowledge about TMDs, we have developed a tool (TMDs Knowledge scale) that is psychometrically valid and reliable (study III). When applied to the Portuguese population we found that the participants had an overall positive knowledge about TMDs, and that females and the participants with self-reported moderate or severe TMDs had significantly higher knowledge. This is the only study, as far as we know, that assesses the level of TMDs' knowledge in the general population. The fact that the participants with self-reported moderate or severe TMDs had significantly higher knowledge, may be indicative that a higher impact of the disorder leads to more search of information, which in turn allows the patient to play an active role in the decisions regarding his/her recovery. Furthermore, knowledge about the disorder seems to provide self-management skills and better coping strategies as well as compliance to therapy and treatment success (Lubrano et al., 1998b; Taal, Rasker, & Wiegman, 1997b). This empowerment of the patient is a key-element on the biopsychosocial model and allows the patient involvement in the decision-making process granting an informed decision.

Considering the importance that the identification of the risk factors and the correct diagnosis have in the definition of the best intervention, and once orthodontic treatment is one of the most sought treatments by TMDs patients (Luther et al., 2010b; Macfarlane et al., 2009), as found in the study II, the clinician must be alert to the fact that patients submitted to orthodontic treatment present craniocervical changes as well as alterations on hyoid bone position (study IV). Those were the results from study IV, which concluded that there were statistically differences regarding hyoid bone position (pre orthodontic treatment versus post orthodontic treatment) and craniocervical posture (pre orthodontic treatment vs. post orthodontic treatment vs. contention phase), with the craniocervical posture being prone to return to basal pre orthodontic values. It is important to bear in mind that since there are no normative values described, to our knowledge, in relation to the variables studied, we can not conclude whether the differences found were beneficial or not to the patient. Our analysis is only regarding the alterations found, and the clinical implications those changes may have if the individual person's adaptive capacity is exceeded. Having this, when compared the craniocervical variables before orthodontic treatment with those variables after orthodontic treatment, the changes found were consistent with an anterior rotation of the head and a rectification of the cervical column (Rocabado, 1984; von Piekartz, 2007). The hyoid bone position variable that also presented a significant change, is also compatible with a loss of cervical lordosis (Rocabado, 1984; von Piekartz, 2007). This anterior rotation of the head and rectification of the cervical column is thought to increase the sub-occipital space favouring a progressive tension over posterior soft tissues, which in turn may be responsible for peripheral neuropathies with craniocervical pain (Rocabado, 1984). When adjusted the variables accordingly with skeletal Class, it was shown that Class II patients had a lower distance between the most anterior-superior point of the body of the hyoid bone and the most posterior-inferior point of the mandibular symphysis (*retrognation*) when compared with Class I patients. This was an expected result, since Class II individuals may present a retrognathic mandible, thus decreasing the distance between hyoid bone and the mandible. The skeletal Class is an important variable that physiotherapists should take into account when assessing TMDs because depending on the skeletal Class the patient presents, the liability to develop determined TMDs' symptoms will be higher (Pullinger, Hollender, & Solberg, 1988a; Pullinger, Seligman, & Solberg, 1988b; Riolo, Seligman, & Solberg, 1987). For instance, Class I individuals have the mandible positioned normally. Class II individuals, due to retrognathism, have a posterior rotation of the mandible that produces a higher compression of the posterior structures and is quite often associated with supra-hyoid muscles' hyperactivity. These seem the individuals more susceptible to develop TMDs (Pullinger et al., 1988a; Pullinger et al., 1988b; Riolo et al., 1987). In the opposite direction, Class III individuals, due to a prognathism, have an anterior rotation of the mandible, producing higher compressive forces on the anterior structures. The skeletal Class is obtained through cephalometry, which is a tool that is part of the daily practice of the orthodontist. Notwithstanding, for the physiotherapist this is not the reality. Taking this into consideration, an alternative to clinical verify the skeletal Class is through the analysis of the cervico-facial proportion. It is known that the height of the lower face must be 20% higher than the submental, with the standard value of this proportion being 1.2 (Gregoret et al., 2007). Values greater than 1.2 indicate that there is an increased lower cervico-facial ratio, which is associated with mandibular retrognathism, consistent with a Class II patient, while values below 1.2 indicate a tendency to Class III with an anterior rotation of the mandible (Gregoret et al., 2007). Another important feature is the facial biotype. Each facial biotype as specific characteristics, namely muscular tone, which may be useful for the physiotherapist when performing physical assessment and planning the intervention in TMDs patients. There are described three different facial types: hypodivergent, normodivergent and hyperdivergent. Hypodivergency is characterized by a square and wide face, an increase in muscular tone, a lower vertical development, with a high projection of the muscles in the goniac angle and a diminished lower face height (Capelozza, Cardoso, & Cardoso, 2004; Cardoso, Bertoz, & Filho, 2005; Gregoret et al., 2007). Hyperdivergency is characterized by an excessive vertical development of the lower third of the face, a decreased muscular tone, is often associated with an anterior open bite and a mandibular and maxilar retrognathism are also characteristic (Cardoso et al., 2005; Farella, Iodice, Michelotti, & Leonardi, 2005; Gregoret et al., 2007). Normodivergency describes facial characteristics that are between hypo and hyperdivergent types with a normal cranial width as well as a normal muscular tone (Gregoret et al., 2007).

The orthodontic treatment aims not only to obtain a normal occlusion, but also to get occlusal stability (American Association of Orthodontists, 1997) and the best facial aesthetic possible. When we analysed the stability of the results obtained with orthodontic treatment (one year after ending orthodontic treatment), the patients remained TMDs' signs and symptoms free, had no relapse on dental Class and overbite and overjet values remained within normal values. Thus, the occlusal stability was achieved. However, when analysed the variables regarding craniocervical posture, there were statistically significant changes in several variables whose values showed a tendency to return to pre orthodontic treatment values. This means that the alterations found in the contention phase were the opposite of those found in post orthodontic treatment. Thus, the changes found were compatible with a posterior rotation of the head and an increase of the cervical lordosis. These changes are thought to decrease the sub-occipital space and produce a progressive mechanical compression over posterior soft tissues, which in turn may be responsible for peripheral neuropathies with craniocervical pain (Rocabado, 1984). It has also been described that these features may impose an excessive tension over the supra and infrahyoid muscles in a dorsal and caudal direction, affecting the growth and development of the mandibular bone, lingual rest and also deglutition (Rocabado, 1984). If we analyse the possible clinical repercussions of the changes described, the symptoms would be the same (pain in the craniofacial region by neuropathies) whether there was an anterior rotation or a posterior rotation of the head. However, the cause of the symptoms would be different: in cases with an anterior rotation of the head, this would cause a progressive tension of the tissues, while in cases with a posterior rotation of the head, this would cause compression of the posterior tissues. These symptoms occur because neural structures react to injury both to tension (elongation) and compression, requiring opposite treatment approaches depending of the causal factor (Shacklock, 2005). It is also important to note that during contention phase (one year) the patient uses a fixed lower contention apparatus and upper removable during the night, which helps to maintain the occlusal stability, which according to our results were in fact maintained within the normal values. Nonetheless, and considering the changes found in the craniocervical posture, after the contention phase, will the results (occlusal and symptomatic) be maintained? All the above mentioned changes in the craniocervical system, that occurred during and one year after of the orthodontic treatment may be the consequence of the close relationship between the craniomandibular and craniocervical systems. Several studies had studied the head and neck posture in order to highlight the relation between these structures and TMDs, dentofacial structures and maxillofacial morphology (D'Attilio et al., 2005; McGuinness & McDonald, 2006; Michelotti et al., 1999; Motoyoshi et al., 2002). The literature seems conclusive in describing the close relationship between craniomandibular and craniocervical systems, and attributes this close relationship to muscular, ligamentar and neural connections between TMJ and the cervical region, creating a functional complex, with the potential of both to influence reciprocally (Armijo-Olivo et al., 2010; Armijo-Olivo et al., 2011; Armijo-Olivo et al., 2012; Ballenberger et al., 2012; De Laat et al., 1998; Gomes et al., 2014; La Touche et al., 2009; Okeson, 2013; Olivo et al., 2006; Rosa, 2012). One of the structures that explains this mutuality is the trigeminocervical nucleus. This structure is responsible for the input from the trigeminal nerve and craniocervical region, and seems to be one of the reasons why pain from any of the above inputs may be referred to cervical, face, head or mandibular region (Bogduk & Govind, 2009b; Okeson, 2013).

Having in mind the results found, that are supported by the interrelationship between both systems and considering the fact that the literature has shown that a craniocervical dysfunction may lead to, or perpetuate the TMDs (Aldana et al., 2011; Gomes et al., 2014; Mew, 2004; Michelotti et al., 2011), the clinician should be aware of these changes and address them in the intervention outlined.

So, it can be hypothesized that the craniocervical changes have the potential to contribute to occlusal and/or TMDs' symptoms relapse seen in clinical practice and described in the literature (Rammelsberg et al., 2003). Naturally, the relapse does not happen in all the cases, but if it happens it is important to identify what characteristics are present in those cases and what intervention is best suited to address those impairments. Study V presents three cases with common signs and symptoms of TMDs that sought orthodontic treatment and had different outcomes. This case series concluded that orthodontics played an important role solving occlusal problems as well as changes in the vertical dimension. On the other hand, physiotherapy was effective in pain management and range improvement, when musculoskeletal changes were clearly found. Finally, it has also shown that a multidisciplinary approach may be crucial, and the clinician should be aware of a comprehensive assessment, valuing all the contributing factors, namely the psychological ones. Despite being a case series, the results are supported by the multifactorial aetiology and are also consistent with the findings from study IV, because where musculoskeletal changes were found the intervention suited to those impairments (physiotherapy) produced improvements. It was also consistent with the risk factors found on study II, where psychosocial factors highly increased the risk of developing TMDs.

Concerning the limitations of the studies performed, on study II and III, all data analysed was collected through online survey. It is recognized that the data relied on self-reporting, and for that reason, there might have been possible incorrect answers. In a try to overtake this, there were some redundant questions, whose answers were checked for consistency. Another limitation in these studies is the absence of a clinical diagnosis of TMDs in the participants. In the impossibility of performing one, we have used an easy to apply, reliable and validated instrument that is suitable for fast epidemiological screening, that enables to classify individuals according to TMDs' severity (Fonseca Anamnestic Index). Moreover, different studies support the use and validity of questionnaires for epidemiological studies on TMDs' symptoms (Akhter et al., 2008a; Matsuka, Yatani, Kuboki, & Yamashita, 1996). The main limitation of study IV, is the lack of a control group, without intervention, but for ethical reasons it would not be possible.

CLINICAL IMPLICATIONS

In light of the results found in the different studies presented, it was found that there are several risk factors that the clinician should be looking for, when assessing a patient with orofacial pain, and the main ones are: female gender, impulsiveness, tension-type headache, migraine, anxiety, facial trauma, third molar removal and parafunctional habits. It was also highlighted the importance of the patients' knowledge about TMDs, in order to empower the patient and improve the treatment compliance, enhancing the treatment outcomes, and the scale developed is valid, reliable and may be easily used in the clinical setting. Since TMDs patients often seek for orthodontic treatment, it is important to be aware that concomitantly with orthodontic treatment craniocervical posture changes seems to occur, and these alterations should be taken into account once they may contribute to TMDs. Considering TMDs is a multifactorial entity, the different contributing factors should be taken into account during assessment and when musculoskeletal changes are present, physiotherapy is an effective intervention addressing pain in TMDs patients and also seems to improve mouth range of motion.

CHAPTER 5

CONCLUSIONS

CONCLUSIONS

The studies described intended to explore the global burden of TMDs and how physiotherapy's contribution may be enhanced in the management of this very complex condition. Each study had different and specific aims, from risk factors and literacy about the condition to postural changes associated with a common treatment option on TMDs (orthodontics) and to state of the art regarding physiotherapy effectiveness on TMDs. It was established that physiotherapy interventions are more effective than the other treatment modalities and shams to which physiotherapy was compared with, in the management of TMDs concerning pain reduction, and a tendency towards improved active range of movement exists. Our results have also shown that there is a high prevalence of TMDs among the Portuguese population and the main risk factors associated with TMDs were: female gender, impulsiveness, tension-type headache, migraine, anxiety, facial trauma and parafunctional habits. Taking into account that literacy is a health determinant, and that low levels of knowledge may represent a potential risk factor for chronicity, worst coping strategies and lower compliance to the treatment, the TMDs knowledge scale developed is psychometrically valid and reliable, and can be used to assess the patient's knowledge about TMDs. Furthermore, our results have also shown that the Portuguese population have an overall positive knowledge about TMDs. Considering the multidisciplinary team that may contribute to TMDs management, our results have shown that orthodontic treatment produced statistically significant differences regarding hyoid bone position (pre orthodontic treatment *versus* post orthodontic treatment) and craniocervical posture (pre orthodontic treatment *versus* post orthodontic treatment *versus* contention phase), with the craniocervical posture being prone to return to basal values. Thus, a thorough assessment should be performed, since similar symptoms may have different causes that should be addressed specifically. The clinician should value all the contributing factors, namely the psychological ones, and be aware of the importance of a multidisciplinary approach

As a final consideration, being the physiotherapist a specialist in health promotion as well as in the restoration of function, physiotherapy plays an important role on the global burden of TMDs and the physiotherapist seems to be an important element within the multidisciplinary health team.

FUTURE PERSPECTIVES

During the process of performing the systematic reviews, it was clear that there were several methodological issues in many of the studies available in the literature. It is common to find several diagnostic criteria, or worst, without defined criteria, interventions without considering the TMDs' subgroup, several instruments to assess outcomes, among others. Taking into consideration that this heterogeneity may contribute to the conflicting results described in the literature, large-scale, high quality, experimental studies with a standardized physiotherapy protocol, for a specific TMDs subgroup accurately diagnosed are needed to establish whether physiotherapy modalities are effective and has real therapeutic value in the management of different TMDs subgroups. Considering that the presence of headaches is not only a risk factor for TMDs but also a comorbidity, it is important to conduct well-designed, longitudinal, observational and analytical studies encompassing a thorough assessment (including physical examination) to verify the common signs and symptoms and also the relationship between these two conditions. Furthermore, and since both may have an hereditary component, it would also be interesting to perform a genetic study, in order to ascertain the relationship between these two conditions. Being orthodontics one of the most sought treatment by patients with TMDs, and since our results found significant changes in the craniocervical posture concomitantly with orthodontics, it seems important to conduct longitudinal and randomized controlled trials, comparing craniocervical posture, hyoid bone position and TMDs' signs and symptoms, in individuals diagnosed with TMDs not only before and after orthodontic treatment but also during a follow-up period longer than one year.

CHAPTER 6

REFERENCES

REFERENCES

Akhter, R., Hassan, N. M., Ohkubo, R., Tsukazaki, T., Aida, J., & Morita, M. (2008). The relationship between jaw injury, third molar removal, and orthodontic treatment and TMD symptoms in university students in Japan. *Journal or Orofacial Pain*, *22*(1), 50-56.

Akhter, R., Morita, M., Ekuni, D., Hassan, N. M., Furuta, M., Yamanaka, R., . . . Wilson, D. (2013). Self-reported aural symptoms, headache and temporomandibular disorders in Japanese young adults. *BMC Musculoskeletal Disorders*, *14*, 58.

Al-Riyami, S., Moles, D. R., & Cunningham, S. J. (2009). Orthognathic treatment and temporomandibular disorders: a systematic review. Part 1. A new quality-assessment technique and analysis of study characteristics and classifications. *American Journal of Orthodontics and Dentofacial Orthopedics*, *136*(5), 624 e621-624 e615.

Aldana, P. A., Báez, R. J., Sandoval, C. C., Vergara, N. C., Cauvi, L. D., & Fernández de la Reguera, A. (2011). Asociación entre Maloclusiones y Posición de la Cabeza y Cuello. I*nternational Journal of Odontostomatology, 5*(2), 119-125.

Ambalavanar, R., Moritani, M., Moutanni, A., Gangula, P., Yallampalli, C., & Dessem, D. (2006). Deep tissue inflammation upregulates neuropeptides and evokes nociceptive behaviors which are modulated by a neuropeptide antagonist. *Pain*, *120*(1-2), 53-68.

American Association of Orthodontists. (1997). Excerpts from American association of orthodontists clinical practice guidelines for orthodontics and dentofacial orthopedics 1996. *American Journal of Orthodontics and Dentofacial Orthopedics*, *11*1(4), 443-449.

Armijo Olivo, S., Magee, D., Parfitt, M., Major, P., & Thie, N. (2006). The association between the cervical spine, the stomatognathic system, and craniofacial pain: a critical review. *Journal of Orofacial Pain*, *20*(4), 271-287.

Armijo-Olivo, S., Fuentes, J. P., da Costa, B. R., Major, P. W., Warren, S., Thie, N. M., & Magee, D. J. (2010). Reduced endurance of the cervical flexor muscles in patients with concurrent temporomandibular disorders and neck disability. *Manual Therapy*, *15*(6), 586-592.

Armijo-Olivo, S., Pitance, L., Singh, V., Neto, F., Thie, N., & Michelotti, A. (2016). Effectiveness of Manual Therapy and Therapeutic Exercise for Temporomandibular Disorders: Systematic Review and Meta-Analysis. *Physical Therapy*, *96*(1), 9-25.

Armijo-Olivo, S., Silvestre, R., Fuentes, J., da Costa, B. R., Gadotti, I. C., Warren, S., . . . Magee, D. J. (2011). Electromyographic Activity of the Cervical Flexor Muscles in Patients With Temporomandibular Disorders While Performing the Craniocervical Flexion Test: A Cross-Sectional Study. *Physical Therapy*, *91*(8), 1184-1197.

Armijo-Olivo, S., Silvestre, R. A., Fuentes, J. P., da Costa, B. R., Major, P. W., Warren, S., . . . Magee, D. J. (2012). Patients with temporomandibular disorders have increased fatigability of the cervical extensor muscles. *Clinical Journal of Pain*, *28*(1), 55-64.

Asaki, S., Sekikawa, M., & Kim, Y. (2006). Sensory innervation of temporomandibular joint disk. *Journal of Orthopaedic Surgery*, *14*(1), 3-8.

Ballenberger, N., von Piekartz, H., Paris-Alemany, A., La Touche, R., & Angulo-Diaz-Parreño, S. (2012). Influence of Different Upper Cervical Positions on Electromyography Activity of the Masticatory Muscles. *Journal of Manipulative an Physiological Therapeutics*, *35*(4), 308-318.

Berger, M., Oleszek-Listopad, J., Marczak, M., & Szymanska, J. (2015). Psychological aspects of temporomandibular disorders – literature review. *Current Issues in Pharmacy and Medical Sciences*, *28*(1), 55-59.

Bialosky, J. E., Bishop, M. D., Price, D. D., Robinson, M. E., & George, S. Z. (2009). The mechanisms of manual therapy in the treatment of musculoskeletal pain: A comprehensive model. *Manual Therapy*, *14*(5), 531-538.

Bogduk, N., & Govind, J. (2009). Cervicogenic headache: an assessment of the evidence on clinical diagnosis, invasive tests, and treatment. *The Lancet Neurology, 8*(10), 959-968.

Bourzgui, F., Sebbar, M., Nadour, A., & Hamza, M. (2010). Prevalence of temporomandibular dysfunction in orthodontic treatment. *International Orthodontics*, *8*(4), 386-398.

Buljan, D. (2010). Psychological and psychiatric factors of temporomandibular disorders. *Rad Hrvatske Akademije Znanosti i Umjetnosti. Medicinske Znanosti, 507*(34), 119-133.

Cairns, B. E. (2010). Pathophysiology of TMD pain--basic mechanisms and their implications for pharmacotherapy. *Journal of Oral Rehabilitation, 37*(6), 391-410.

Calixtre, L. B., Moreira, R. F., Franchini, G. H., Alburquerque-Sendin, F., & Oliveira, A. B. (2015). Manual therapy for the management of pain and limited range of motion in subjects with signs and symptoms of temporomandibular disorder: a systematic review of randomised controlled trials. *Journal of Oral Rehabiitationl, 42*, 847-861.

Campos, J., Carrascosa, A. C., Bonafé, F., & Maroco, J. (2014). Severity of temporomandibular disorders in women: validity and reliability of the Fonseca Anamnestic Index. *Brazilian Oral Research, 28*(1), 1-6.

Capelozza, F., Cardoso, M., & Cardoso, N. (2004). Placa de Reposicionamento Mandibular para Incremento do Crescimento Vertical Posterior da Maxila - Técnica de Construção. *Revista Clínica Ortodontia Dental Press, 3*, 41-53.

Cardoso, M., Bertoz, F., & Filho, L. (2005). Características cefalométricas do padrão face longa. Revista Dental Press de Ortodontia e Ortopedia Facial, 10(2), 29-43.

Chisnoiu, A. M., Chisnoiu, R., Moldovan, M., Lascu, L. M., & Picos, A. M. (2016). Etiological factors associated with temporomandibular joint disorder - study on animal model. *Romanian Journal of Morphology and Embryology, 57*(1), 185-189.

Chisnoiu, A. M., Picos, A. M., Popa, S., Chisnoiu, P. D., Lascu, L., Picos, A., & Chisnoiu, R. (2015). Factors involved in the etiology of temporomandibular disorders - a literature review. *Clujul Medical, 88*(4), 473-478.

Clark, G. T. (1991). Etiologic Theory and the Prevention of Temporomandibular Disorders. *Advances in Dental Research, 5*(1), 60-66.

Costen, J. B. (1934). A syndrome of ear and sinus symptoms dependent upon disturbed function of the temporomandibular joint. *Annals of Otology, Rhinology & Laryngology, 43*(1), 1-15.

D'Attilio, M., Filippi, M. R., Femminella, B., Festa, F., & Tecco, S. (2005). The influence of an experimentally-induced malocclusion on vertebral alignement in rats: a controlled pilot study. *CRANIO®: The Journal of Craniomandibular & Sleep Practice, 23*(2), 119-129.

De Laat, A., Meuleman, H., Stevens, A., & Verbeke, G. (1998). Correlation between cervical spine and temporomandibular disorders. *Clinical Oral Investigations, 2*(2), 54-57.

de Toledo, E. G., Silva, D. P., de Toledo, J. A., & Salgado, I. O. (2012). The interrelationship between dentistry and physiotherapy in the treatment of temporomandibular disorders. *The Journal of Contemporary Dental Practice, 13*(5), 579-583.

Dıraçoğlu, D., Yıldırım, N. K., Saral, İ., Özkan, M., Karan, A., Özkan, S., & Aksoy, C. (2016). Temporomandibular dysfunction and risk factors for anxiety and depression. *Journal of Back and Musculoskeletal Rehabilitation., 29*(3), 487-491.

Durham, J. (2013). Oral surgery: part 3. Temporomandibular disorders. *British Dental Journal, 215*(7), 331-337.

Egermark, I., Blomqvist, J. E., Cromvik, U., & Isaksson, S. (2000). Temporomandibular dysfunction in patients treated with orthodontics in combination with orthognatic surgery. *European Journal of Orthodontics, 22*, 537-544.

Egermark, I., Carlsson, G. E., & Magnusson, T. (2005). A prospective long-term study of signs and symptoms of temporomandibular disorders in patients who received orthodontic treatment in childhood. *The Angle Orthodontist, 75*(4), 645-650.

Ernberg, M., Hedenberg-Magnusson, B., Kurita, H., & Kopp, S. (2006). Effects of local serotonin administration on pain and microcirculation in the human masseter muscle. *Journal of Orofacial Pain, 20*(3), 241-248.

European Region - World Confederation for Physical Therapy. (2016). Health Promotion & Disease Prevention. Retrieved from http://www.erwcpt.eu/physiotherapy_and_practice/health_promotion_and_disease_prevention

Farella, M., Iodice, G., Michelotti, A., & Leonardi, R. (2005). The relationship between vertical craniofacial morphology and the sagittal path of mandibular movements. *Journal of Oral Rehabilitation, 32*(12), 857-862.

Fernández-Carnero, J., La Touche, R., Ortega-Santiago, R., Galan-del-Rio, F., Pesquera, J., Ge, H.-Y., & Fernández-de-Las-Peñas, C. (2010). Short-term effects of dry needling of active myofascial trigger points in the masseter muscle in patients with temporomandibular disorders. *Journal of Orofacial Pain, 24*(1), 106-112.

Ferreira, C. L. P., Silva, M. A. M. R. d., & Felício, C. M. d. (2015). Signs and symptoms of temporomandibular disorders in women and men. *Brazilian Oral Research, 28*(1), 17-21.

Fillingim, R., Ohrbach, R., Greenspan, J., Knott, C., Dubner, R., Bair, E., . . . Maixner, W. (2011). Potential psychosocial risk factors for chronic TMD: descriptive data and empirically identified domains from the

OPPERA case-control study. *Journal of Pain, 12*(11 Suppl), T46-T60.

Fonseca, D., Bonfante, G., Valle, A., & Freitas, S. (1994). Diagnóstico pela anamnese da disfunção craniomandibular. *Revista Gaúcha de Odontologia (RGO), 42*(1), 23-28.

Fonseca, J., Paço, M., & Oliveira, T. (2016). DTM: Subgrupo dos distúrbios musculares. In Sociedade Portuguesa De Disfunção Temporomandibular e Dor Orofacial (Ed.), *Dor orofacial e disfunções temporomandibulares: tratamento farmacológico.* (pp. 39-42).

Gallo, L. M. (2005). Modeling of temporomandibular joint function using MRI and jaw-tracking technologies--mechanics. *Cells Tissues Organs, 180*(1), 54-68.

Gavish, A., Winocur, E., Astandzelov-Nachmias, T., & Gazit, E. (2006). Effect of Controlled Masticatory Exercise on Pain and Muscle Performance in Myofascial Pain Patients: A Pilot Study. *CRANIO®: The Journal of Craniomandibular & Sleep Practice, 24*(3), 184-190.

Gomes, L., Horta, K., Gonçalves, J., & Santos-Pinto, A. (2014). Systematic review: craniocervical posture and craniofacial morphology. *European Journal of Orthodontics, 36*(1), 55-66.

Greenberg, M. S., Glick, M., & Ship, J. A. (2008). *Burket's Oral Medicine* (11th ed., pp. 223-255): BC Decker Inc.

Greene, C. S. (1995). Etiology of temporomandibular disorders. *Seminars in Orthodontics,* 1(4), 222-228.

Greene, C. S. (2010). Managing patients with temporomandibular disorders: a new "standard of care". *American Journal of Orthodontics and Dentofacial Orthopedics, 138*(1), 3-4.

Gregoret, J., Tuber, E., Escobar, H., & Matos da Fonseca, A. (2007). *Ortodoncia y cirugía ortognática: diagnóstico y planificación* (2nd ed.). Madrid: NM ediciones.

Gremillion, H. (2000). The prevalence and etiology of temporomandibular disorders and orofacial pain. *Texas Dental Journal, 117*(7), 30-39.

Helkimo, M. (1974). Studies on Function of the Mastigatory System. Index for Anamnestic and Clinical Dysfunction and Occlusal state. *Swedish Dental Journal, 67*, 101–121.

Henrikson, T., Nilner, M., & Kurol, J. (1999). Symptoms and signs of temporomandibular disorders before, during and after orthodontic treatment. *Swedish Dental Journal, 23*, 107-117.

Huang, G. J., LeResche, L., Critchlow, C. W., Martin, M. D., & Drangsholt, M. T. (2002). Risk factors for diagnostic subgroups of painful temporomandibular disorders (TMD). J*ournal of Dental Research, 81*(4), 284-288.

Imai, T., Okamoto, T., Kaneko, T., Umeda, K., Yamamoto, T., & Nakamura, S. (2000). Long-term follow-up of clinical symptoms in TMD patients who underwent occlusal reconstruction by orthodontic treatment. *European Journal of Orthodontics, 22*, 61-67.

Ismail, F., Demling, A., HeßLing, K., Fink, M., & Stiesch-Scholz, M. (2007). Short-term efficacy of physical therapy compared to splint therapy in treatment of arthrogenous TMD. *Journal of Oral Rehabilitation, 34*(11), 807-813.

Kim, M.-R., Graber, T. M., & Viana, M. A. (2002). Orthodontics and temporomandibular disorder: A meta-analysis. *American Journal of Orthodontics and Dentofacial Orthopedics, 121*(5), 438-446.

King, C. D., Wong, F., Currie, T., Mauderli, A. P., Fillingim, R. B., & Riley 3rd, J. L. (2009). Deficiency in endogenous modulation of prolonged heat pain in patients with Irritable Bowel Syndrome and Temporomandibular Disorder. *Pain, 143*(3), 172-178.

La Touche, R., Fernandez-de-las-Penas, C., Fernandez-Carnero, J., Escalante, K., Angulo-Diaz-Parreno, S., Paris-Alemany, A., & Cleland, J. A. (2009). The effects of manual therapy and exercise directed at the cervical spine on pain and pressure pain sensitivity in patients with myofascial temporomandibular disorders. *Journal of Oral Rehabilitation, 36*(9), 644-652.

Langdon, J. D. (1994). Physical therapy in craniomandibular disorders. *Journal of Dentistry, 22*(5), 299-299.

Leeuw, R., & Klasser, G. D. (2013). *Orofacial Pain: Guidelines for Assessment, Diagnosis, and Management* (R. de Leeuw & G. D. Klasser Eds. 5th ed.): Quintessence Books.

Leite, R. A., Rodrigues, J. F., Sakima, M. T., & Sakima, T. (2013). Relationship between temporomandibular disorders and orthodontic treatment: A literature review. *Dental Press Journal of Orthodontics, 18*(1), 150-157.

LeResche, L. (1997). Epidemiology of Temporomandibular Disorders: Implications for the Investigation of Etiologic Factors. *Critical Reviews in Oral Biology & Medicine, 8*(3), 291-305.

List, T., & Axelsson, S. (2010). Management of TMD: evidence from systematic reviews and meta-analyses. *Journal of Oral Rehabilitation, 37*(6), 430-451.

Liu, F., & Steinkeler, A. (2013). Epidemiology, Diagnosis, and Treatment of Temporomandibular Disorders. *Dental Clinics of North America, 57*, 465-479.

Lubrano, E., Helliwell, P., Moreno, P., Griffiths, B., Emery, P., & Veale, D. (1998). The assessment of knowledge in ankylosing spondylitis patients by a self-administered questionnaire. *British Journal of Rheumatology, 37*, 437-441.

Lund, J. P., Donga, R., Widmer, C. G., & Stohler, C. S. (1991). The pain-adaptation model: a discussion of the relationship between chronic musculoskeletal pain and motor activity. *Canadian Journal of Physiology and Pharmacology, 69*, 683-694.

Luther, F. (2007). TMD and occlusion part I. Damned if we do? Occlusion: the interface of dentistry and orthodontics. *Brazilian Dental Journal, 202*(E3), 1-7.

Luther, F., Layton, S., & McDonald, F. (2010). Orthodontics for treating temporomandibular joint (TMJ) disorders. *The Cochrane Database of Systematic Reviews, 7*(7).

Macfarlane, T. V., Kenealy, P., Kingdon, H. A., Mohlin, B. O., Pilley, J. R., Richmond, S., & Shaw, W. C. (2009). Twenty-year cohort study of health gain from orthodontic treatment: temporomandibular disorders. *American Journal of Orthodontics and Dentofacial Orthopedics, 135*(6), 692.e691-e698

Magalhães, B. G., de-Sousa, S. T., de Mello, V. V., da-Silva-Barbosa, A. C., de-Assis-Morais, M. P., Barbosa-Vasconcelos, M. M., & Caldas-Júnior, A. D. (2014). Risk factors for temporomandibular disorder: binary logistic regression analysis. *Medicina oral, patologia oral y cirugia bucal, 19*(3), e232-e236.

Magnusson, T., Egermark, I., & Carlsson, G. E. (2005). A prospective investigation over two decades on signs and symptoms of temporomandibular disorders and associated variables. A final summary. *Acta Odontologica Scandinavica, 63*(2), 99-109.

Maixner, W., Diatchenko, L., Dubner, R., Fillingim, R. B., Greenspan, J. D., Knott, C., . . . Slade, G. D. (2011). Orofacial Pain Prospective Evaluation and Risk Assessment Study – The OPPERA Study. J*ournal of Pain, 12*(11 Suppl), T4-T11.e12.

Manfredini, D., Guarda-Nardini, L., Winocur, E., Piccotti, F., Ahlberg, J., & Lobbezoo, F. (2011). Research diagnostic criteria for temporomandibular disorders: a systematic review of axis I epidemiologic findings. *Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology and Endodontics, 112*(4), 453-462.

Manfredini, D., Stellini, E., Gracco, A., Lombardo, L., Nardini, L. G., & Siciliani, G. (2016). Orthodontics is temporomandibular disorder–neutral. *The Angle Orthodontist, 86*(4), 649-654.

Martins, W. R., Blasczyk, J. C., Furlan de Oliveira, M. A., Lagoa Goncalves, K. F., Bonini-Rocha, A. C., Dugailly, P. M., & de Oliveira, R. J. (2015). Efficacy of musculoskeletal manual approach in the treatment of temporomandibular joint disorder: A systematic review with meta-analysis. *Manual Therapy, 21*, 10-17.

Matsuka, Y., Yatani, H., Kuboki, T., & Yamashita, A. (1996). Temporomandibular disorders in the adult population of Okayama City, Japan. *CRANIO®: The Journal of Craniomandibular & Sleep Practice, 14*(2), 158-162.

Maydana, A. V., Tesch, R. d. S., Denardin, O. V. P., Ursi, W. J. d. S., & Dworkin, S. F. (2010). Possible etiological factors in temporomandibular disorders of articular origin with implications for diagnosis and treatment. *Dental Press Journal of Orthodontics, 15*(3), 78-86.

McGuinness, N. J., & McDonald, J. P. (2006). Changes in natural head position observed immediately and one year after rapid maxillary expansion. *European Journal of Orthodontics, 28*(2), 126-134.

McNeely, M. L., Olivo, S. A., & Magee, D. J. (2006). A systematic review of the effectiveness of physical therapy interventions for temporomandibular disorders. *Physical Therapy, 86*(5), 710-725.

Medlicott, M. S., & Harris, S. R. (2006). A Systematic Review of the Effectiveness of Exercise, Manual Therapy, Electrotherapy, Relaxation Training, and Biofeedback in the Management of Temporomandibular Disorder. *Physical Therapy, 86*(7), 955-973.

Melis, M., & Di Giosia, M. (2016). The role of genetic factors in the etiology of temporomandibular disorders: a review. *CRANIO®: The Journal of Craniomandibular & Sleep Practice, 34*(1), 43-51.

Mew, J. (2004). The postural basis of malocclusion: A philosophical overview. *American Journal of Orthodontics and Dentofacial Orthopedics, 126*(6), 729-738.

Michelotti, A., Buonocore, G., Manzo, P., Pellegrino, G., & Farella, M. (2011). Dental occlusion and posture: an overview. *Progress in Orthodontics, 12*, 53-58.

Michelotti, A., Cioffi, I., Festa, P., Scala, G., & Farella, M. (2010). Oral parafunctions as risk factors for diagnostic TMD subgroups. *Journal of Oral Rehabilitation, 37*(3), 157-162.

Michelotti, A., & Iodice, G. (2010). The role of orthodontics in temporomandibular disorders. *Journal of Oral Rehabilitation, 37*(6), 411-429.

Michelotti, A., Manzo, P., Farella, M., & Martina, R. (1999). Occlusion and posture: is there evidence of correlation? *Minerva Stomatologica, 48*, 525-534.

Modi, P., Shaikh, S. S., & Munde, A. (2012). A cross sectional study of prevalence of temporomandibular disorders in university students. *International Journal of Scientific and Research Publications, 2*(9), 1-3.

Motoyoshi, M., Shimazaki, T., Sugai, T., & Namura, S. (2002). Biomechanical influences of head posture on occlusion: an experimental study using finite element analysis. *European Journal of Orthodontics, 24*(4), 319-326.

Murray, G. M., & Peck, C. C. (2007). Orofacial pain and jaw muscle activity: a new model. *Journal of Orofacial Pain, 21*(4), 263-278.

Nascimento, M., Vasconcelos, B., Porto, G., Ferdinanda, G., Nogueira, C., & Raimundo, R. (2013). Physical therapy and anesthetic blockage for treating temporomandibular disorders: A clinical trial. *Medicina oral, patologia oral y cirugia bucal, 18*(1), e81-e85.

National Institute of Dental and Craniofacial Research. (2016). Facial Pain. Retrieved from http://www.nidcr.nih.gov/DataStatistics/FindDataByTopic/FacialPain/

Nicolakis, P., Erdogmus, B., Kopf, A., Djaber-Ansari, A., Piehslinger, E., & Fialka-Moser, V. (2000). Exercise therapy for craniomandibular disorders. *Archives of Physical Medicine and Rehabilitation, 81*(9), 1137-1142.

Nielsen, L., Melsen, B., & Terp, S. (1990). TMJ function and the effects on the masticatory system on 14-16 year-old Danish children in relation to orthodontic treatment. *European Journal of Orthodontics, 12*, 254-262.

Ohlsson, M., & Linquist, B. (1995). Mandibular function before and after orthodontic treatment. *European Journal of Orthodontics, 17*, 205-214.

Ohrbach, R., Fillingim, R. B., Mulkey, F., Gonzalez, Y., Gordon, S., Gremillion, H., . . . Slade, G. (2011). Clinical findings and pain symptoms as potential risk factors for chronic TMD: descriptive data and empirically identified domains from the OPPERA case-control study. *Journal of Pain, 12*(11), T27-T45.

Okeson, J. (1996). *Orofacial Pain: Guidelines for assessment, diagnosis and management* (4th ed.): Quintessence.

Okeson, J. (2013). *Management of temporomandibular disorders and occlusion* (7th ed.): Elsevier Health Sciences.

Okeson, J., & de Leeuw, R. (2011). Differential diagnosis of temporomandibular disorders and other orofacial pain disorders. *Dental Clinics of North America, 55*(1), 105-120.

Olivo, S. A., Bravo, J., Magee, D. J., Thie, N. M., Major, P. W., & Flores-Mir, C. (2006). The association between head and cervical posture and temporomandibular disorders: a systematic review. *Journal of Orofacial Pain, 20*(1), 9-23.

Oral, K., Bal Küçük, B., Ebeoğlu, B., & Dinçer, S. (2009). Etiology of temporomandibular disorder pain. *Ağrı, 21*(3), 89-94.

Peck, C. C., Goulet, J. P., Lobbezoo, F., Schiffman, E. L., Alstergren, P., Anderson, G. C., . . . List, T. (2014). Expanding the taxonomy of the diagnostic criteria for temporomandibular disorders. *Journal of Oral Rehabilitation*, 41(1), 2-23.

Piekartz, H. v. (2009). *Headache, Orofacial Pain and Bruxism*: Elsevier.

Poveda Roda, R., Bagan, J. V., Díaz Fernández, J. M., Hernández Bazán, S., & Jiménez Soriano, Y. (2007). Review of temporomandibular joint pathology. Part I: classification, epidemiology and risk factors. *Medicina oral, patologia oral y cirugia bucal, 12*(4), E292-298.

Pullinger, A., Hollender, L., & Solberg, W. (1988a). Temporomandibular disorders. Parte I: Functional Status, Dentomorphologic Features, and Sex Differences in a Nonpatient Population. *Journal of Prosthetic Dentistry, 59*, 228–235.

Pullinger, A., Seligman, D., & Solberg, W. (1988b). Temporomandibular Disorders. Part II: Occlusal Factors Associated with Temporomandibular Tenderness and Dysfunction. *Journal of Prosthetic Dentistry, 59*, 363–367.

Rammelsberg, P., LeResche, L., Dworkin, S., & Mancl, L. (2003). Longitudinal outcome of temporomandibular disorders: a 5-year epidemiologic study of muscle disorders defined by research diagnostic criteria for temporomandibular disorders. *Journal of Orofacial Pain, 17*(1), 9-20.

Randolph, C. S., Greene, C. S., Moretti, R., Forbes, D., & Perry, H. T. (1990). Conservative management of temporomandibular disorders: a posttreatment comparison between patients from a university clinic and from private practice. *American Journal of Orthodontics and Dentofacial Orthopedics, 98*(1), 77-82.

Ribeiro, J. J., Paço, M., & Oliveira, T. (2016). Dor miofascial. In Sociedade Portuguesa De Disfunção Temporomandibular e Dor Orofacial (Ed.), *Dor orofacial e disfunções temporomandibulares: tratamento farmacológico* (pp. 43-47).

Riolo, M., Seligman, D., & Solberg, W. (1987). Associations between occlusal characteristics and signs and symptoms of TMJ dysfunction in children and young adults. . *American Journal of Orthodontics & Dentofacial Orthopedics, 92*, 467–477.

Rocabado, M. (1984). Analisis biomecánico cráneocervical a través de una teleradiografia lateral. *Revista Chilena de Ortodoncia, 1*, 42-52.

Rosa, L. P. (2012). Relation between the craniocervical cephalometric analysis and class I , II and III skeletic patterns in young subjects. *Brazilian Dental Science, 15*(1), 53-60.

Sarlani, E., Grace, E. G., Reynolds, M. A., & Greenspan, J. D. (2004). Evidence for up-regulated central nociceptive processing in patients with masticatory myofascial pain. *Journal of Orofacial Pain, 18*(1), 41-55.

Sarlani, E., & Greenspan, J. D. (2003). Evidence for generalized hyperalgesia in temporomandibular disorders patients. *Pain, 102*(3), 221-226.

Schiffman, E., Ohrbach, R., Truelove, E., Look, J., Anderson, G., J.P., G., . . . Dworkin, S. F. (2014). Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) for Clinical and Research Applications: recommendations of the International RDC/TMD Consortium Network* and Orofacial Pain Special Interest Group. *Journal of Oral & Facial Pain and Headache, 28*(1), 6-27.

Schmid, A., Brunner, F., Wright, A., & Bachmann, L. M. (2008). Paradigm shift in manual therapy? Evidence for a central nervous system component in the response to passive cervical joint mobilisation. *Manual Therapy, 13*(5), 387-396.

Shacklock, M. (2005). Clinical neurodynamics: a new system of musculoskeletal treatment: Elsevier Health Sciences.

Slade, G. D., L, D., Bhalang, K., Sigurdsson, A., Fillingim, R. B., Belfer, I., . . . Maixner, W. (2007). Influence of psychological factors on risk of temporomandibular disorders. *Journal of Dental Research, 86*(11), 1120-1125.

Slade, G. D., Ohrbach, R., Greenspan, J. D., Fillingim, R. B., Bair, E., Sanders, A. E., . . . Maixner, W. (2016). Painful Temporomandibular Disorder: Decade of Discovery from OPPERA Studies. *Journal of Dental Research, 95*(10), 1084-1092.

Suvinen, T. I., Reade, P. C., Kemppainen, P., Könönen, M., & Dworkin, S. F. (2005). Review of aetiological concepts of temporomandibular pain disorders: towards a biopsychosocial model for integration of physical disorder factors with psychological and psychosocial illness impact factors. *European Journal of Pain, 9*(6), 613-633.

Taal, E., Rasker, J. J., & Wiegman, O. (1997). Goup education for Rheumatoid Arthritis Patients. *Seminars in Arthritis and Rheumatism, 26*(6), 805-816.

Tallents, R. H. (1991). Etiologic Theory and Prevention of Temporomandibular Joint Disorders: Reaction Paper. *Advances in Dental Research, 5*(1), 67-68.

Tanaka, E., Detamore, M. S., & Mercuri, L. G. (2008). Degenerative disorders of the temporomandibular joint: etiology, diagnosis, and treatment. *Journal of Dental Research, 87*(4), 296-307.

Taube, S., Ylipaavalneimi, P., Kononen, M., & Sunden, B. (1988). The effect of pulsed ultrasound on myofascial pain: a placebo controlled study. 84:241–246. *Proceedings of the Finnish Dental Society, 84*, 241–246.

Tuncer, A. B., Ergun, N., Tuncer, A. H., & Karahan, S. (2013). Effectiveness of manual therapy and home physical therapy in patients with temporomandibular disorders: A randomized controlled trial. *Journal of Bodywork and Movement Therapies, 17*(3), 302-308.

Van Der Weele, L. T., & Dibbets, J. M. H. (1987). Helkimo's index: a scale or just a set of symptoms? Journal of Oral Rehabilitation, 14(3), 229-237.

Von Korff, M., Le Resche, L., & Dworkin, S. F. (1993). First onset of common pain symptoms: a prospective study of depression as a risk factor. *Pain, 55*(2), 251-258.

von Piekartz, H. (2007). *Craniofacial Pain*: Elsevier.

World Health Organization. (2016a). The determinants of health. Retrieved from http://www.who.int/hia/evidence/doh/en/

World Health Organization. (2016b). Health literacy and health behaviour. Retrieved from http://www.who.int/healthpromotion/conferences/7gchp/track2/en/

Wright, E. F., Clark, E. G., Paunovich, E. D., & Hart, R. G. (2006). Headache Improvement Through TMD Stabilization Appliance and Self-management Therapies. *CRANIO®: The Journal of Craniomandibular & Sleep Practice*, *24*(2), 104-111.

Zarb, G. A., & Carlsson, G. E. (1999). Temporomandibular disorders: osteoarthritis. *Journal of Orofacial Pain*, *13*(4), 295-306.

APPENDIX

BOOK CHAPTERS

APPENDIX I

BOOK CHAPTER - DTM: Subgrupo dos distúrbios musculares

Fonseca, J., Paço, M., & Oliveira, T. (2016). DTM: Subgrupo dos distúrbios musculares. In Sociedade Portuguesa De Disfunção Temporomandibular e Dor Orofacial (Ed.), Dor orofacial e disfunções temporomandibulares: tratamento farmacológico (pp. 39-42).

ISBN: 978-989-20-6409-3

// DTM: SUBGRUPO DOS DISTÚRBIOS MUSCULARES

Júlio Fonseca | Maria Paço | Tiago Oliveira

Introdução

Os distúrbios dos músculos mastigatórios que direta ou indiretamente atuam sobre o sistema estomatognático e a esfera orofacial podem surgir por inúmeras causas e fatores. A sobrecarga associada a hábitos parafuncionais ou ao bruxismo; fatores locais (infeciosos, inflamatórios ou outros) e sistémicos (como a mialgia mediada centralmente ou a fibromialgia), ou co-contração protetora secundária a um problema articular da ATM ou outro estímulo, são apenas algumas das situações que podem levar a patologia muscular afetando o sistema estomatognático e causadores de DTM e dor orofacial[1]

Estabelecer um diagnóstico correto para pacientes com dor orofacial, entre os quais os pacientes com DTM, é particularmente difícil e complexo devido à complexidade de fatores físicos e psíquicos envolvidos. Muitos dos distúrbios existentes têm sinais e sintomas semelhantes, e a estrutura específica que causa a dor é incerta.[2] Efetivamente a presença de uma única estrutura afetada isoladamente tem uma prevalência muito baixa, sendo mais frequente a combinação de diagnósticos,[3] o que muitas vezes dificulta a abordagem e o planeamento de uma intervenção, caso esta não seja realizada no âmbito de uma equipa multidisciplinar.

Epidemiologia

Os valores de prevalência de DTM na população em geral variam bastante de estudo para estudo, sobretudo devido à grande variabilidade dos critérios de diagnóstico adotados pelos diferentes grupos de investigação. Tendo isto em conta, a literatura revela uma prevalência de sintomas que varia de 6% a 93% e de sinais clínicos que varia de 0% a 93%.[4] Segundo a American Academy of Orofacial Pain (AAOP) cerca de 4 a 7% da população apresenta DTM com sintomatologia suficiente para procurar tratamento.2 Sendo consensual para a comunidade cientifica que não existe uma causa única para as DTM, a prevalência de dor da musculatura mastigatória, na população em geral é de 13%.[2] No que diz respeito às diferentes classificações da DTM, estudos têm demonstrado uma prevalência de disfunção muscular que varia entre os 45% e os 49,7% em indivíduos com DTM.[3,5]

Etiologia e características clínicas

As causas e sintomatologia das DTM são multifatoriais e multissistémicas, não existindo um único fator etiológico nem um único modelo teórico que seja capaz de explicar o inicio das DTM. Assim, uma perda da integridade estrutural, uma função alterada ou a imposição de stresses biomecânicos no sistema cranio-mandibular poderão afetar a capacidade adaptativa dos tecidos, aumentando a probabilidade de desenvolver DTM. Para além disso, os neurónios responsáveis pela mediação da dor da musculatura esquelética estão sujeitos a uma forte influência modulatória, em que as substâncias endógenas podem sensibilizar as terminações dos nocicetores com muita facilidade.[6]

Desta forma um dos possíveis fatores etiológicos descrito é o traumatismo, podendo ser direto, indireto (exemplo: golpe de chicote da cabeça) ou microtraumatismos repetidos (exemplo: hábitos parafuncionais). Do ponto de vista anatómico, as relações esqueléticas desproporcionais e os fatores oclusais constituem-se como fatores de risco para o aparecimento de sintomatologia.[2]

Os fatores psicossociais também têm demonstrado influenciar a capacidade adaptativa do sistema mastigatório, pela sua capacidade de alterar o tónus muscular, constituindo assim importantes mecanismos predisponentes e perpetuantes da DTM, nomeadamente os elevados níveis de ansiedade e stresse.[2]

Diagnóstico e classificação

Como já foi referido anteriormente existem diferentes sistemas de diagnóstico e classificação das DTM musculares. Nos capítulos apresentados neste livro será seguida a classificação mais consensual e validada atualmente, comum à AAOP e DC-TMD, prevendo a integração biopsicossocial do paciente considerando a avaliação da sua vertente física, mas também psicossocial respondendo desta forma aquilo que é a perspetiva ontológica atual da DTM e dor orofacial.[7, 8] As propostas de expansão desta classificação propostas por Deck et al. (2014) e consideradas válidas pela comissão científica da SPDOF e pelos seus membros, serão consideradas pela sua mais valia clínica e de intervenção. Para a componente física (muscular e articular) será então seguida a classificação apresentada no Quadro 3 do capítulo anterior. Disfunções Temporomandibulares: Introdução e Classificação. Para a caracterização da dor e avaliação psicoemocional será considerada a classificação já prevista pelo Eixo II do RDC-TDM.

Dentro da categoria de "Distúrbios dos Músculos Mastigatórios" várias sub-categorias estão incluídas, entre as quais várias patologias, cujos termos estaremos habituados a ouvir por vezes indiscriminadamente, são adequadamente categorizadas, tais como: mialgia, tendinite, miosite, espasmo muscular, contractura, hipertrofia e neoplasia.[2]

Assim, o correto diagnóstico e classificação do problema do paciente assume extrema importância, uma vez que cada subcategoria tem características muito distintas das restantes, o que implica que o tratamento que será adequado numa situação, poderá estar contra-indicado noutra. Por este motivo a identificação clara da disfunção presente é fundamental para que o tratamento adequado seja iniciado, assim como para excluir patologias intra e extra-cranianas sérias, que possam colocar a vida do paciente em risco.[2]

De todos estes distúrbios relacionados com a musculatura mastigatória é fundamental fazer o diagnóstico diferencial entre estruturas locais e estruturais mais distantes. Por exemplo um trigger-point do masséter pode referir dor para estruturas próximas como a região ocular. No entanto outras estruturas músculo-esqueléticas mais distantes podem também referir dor para a região orofacial como seja um trigger-point do trapézio superior.9 Da mesma forma, estruturas musculares podem referir dor para as peças dentárias, como por exemplo um trigger-point do músculo masséter,[2, 9] e as peças dentárias também podem ser responsáveis por dor referida a outras estruturas orofaciais, simulando por vezes um problema muscular.[2, 9] Este fenómeno pode ser explicado do ponto de vista neurofisiológico pela presença do núcleo trigeminocervical, que recebe e envia informação cumulativamente da região cervical e da região orofacial.10

A relação entre a coluna cervical e a DTM parece comprovada[11-14] e mais uma vez o conhecimento sobre dor referida é muito importante. Os distúrbios da coluna cervical podem contribuir para a dor orofacial, isoladamente ou em conjunto com as DTM,[2] logo o diagnóstico diferencial assim como uma intervenção direcionada para as estruturas que se encontram alteradas é muito importante. Também neste aspeto uma equipa multi e transdisciplinar é muito importante na identificação e tratamento destas disfunções. Em caso de diagnóstico incorreto ou ausência de tratamento adequado, os pacientes com DTM podem desenvolver uma condição dolorosa crónica, que potencialmente resultará em perda de dias laborais, distúrbios na vida diária, aumento dos custos para o sistema nacional de saúde assim como ao desenvolvimento de fenómenos de sensibilização central e de catastrofização, do ponto de vista psicossocial.[15]

No caso dos distúrbios dos músculos mastigatórios o diagnóstico é essencialmente clínico.

Este é obtido pela anamnese e pela provocação/comprovação da dor familiar ao paciente através da palpação criteriosa das estruturas locais e á distância (que poderão irradiar para o sítio onde a dor é referida).

Tratamento

O tratamento das DTM musculares deverá ter uma abordagem multimodal e multidisciplinar. A abordagem terapêutica deverá incluir, primariamente, técnicas de abordagem multidisciplinar reversíveis e pouco invasivas, em detrimento das irreversíveis e invasivas. As primeiras, para a maioria dos pacientes têm demonstrado eficácia clínica e uma relação custo/benefício clinico favorável. Incluem, de acordo com a avaliação e plano de abordagem, a terapia psico-comportamental, a terapia com goteira oclusal, a terapia física e a terapia medicamentosa. As técnicas mais invasivas (infiltrações anestésicas ou com botox) devem limitar-se aos casos que não respondem às técnicas mais conservadoras.

O objetivo comum passa assim por, primariamente e em trabalho multidisciplinar integrado, garantir o controlo da dor e o reequilíbrio funcional, emocional e ortopédico do paciente. Além da avaliação e intervenção física necessária, passa também pela terapia de autocontrolo emocional e comportamental. É fundamental a restituição da capacidade funcional, aliada à melhoria da qualidade de vida e bem-estar geral dos pacientes.16-18 Os clínicos devem focalizar a sua atenção, primariamente, na estabilização e auxílio aos quadros clínicos que apresentem sinais e sintomas efetivos e limitantes. Isto é, situações de limitação de abertura bucal e/ou presença de dor muscular e/ou articular concomitantes. A avaliação, interpretação e valorização da capacidade funcional, extensão/desvio dos movimentos mandibulares, assim como dos fenómenos e processos de dor associados (localização, intensidade, frequência, incapacidade, etc.) devem ser o principal objetivo e motivo de atuação para os clínicos envolvidos, numa primeira instância. De notar a influência etiológica e as alterações psicoemocionais associadas a estas patologias, que levam à necessidade de avaliação e valorização deste componente, determinante não só na evolução, mas também no controlo da patologia. [19-24]

A intervenção de Fisioterapia tem como objetivos diminuir a dor músculo-esquelética, promover o relaxamento muscular, reduzir a hiperatividade muscular, melhorar o controlo e função muscular e maximizar a mobilidade articular. O Fisioterapeuta deverá basear a sua intervenção numa avaliação cuidada e na utilização de diversas estratégias e metodologias de intervenção como a utilização de meios electro-físicos (ultra-som, laser), eletro-analgésicos (TENS, correntes interferenciais, biofeedback), terapia manual (técnicas articulares, neurodinâmicas e musculares, por exemplo), a punção seca, e o exercício terapêutico para a correção postural, educação e auto-regulação do paciente. A terapia manual juntamente com a punção seca apresenta um papel especialmente preponderante na resolução dos trigger points, e são procedimentos frequentemente utilizados que têm sido objeto de vários estudos científicos, tendo demonstrado uma melhoria na sintomatologia dolorosa. [25-29]

O tratamento Farmacológico

No caso específico do tratamento farmacológico dos distúrbios dos músculos mastigatórios, por não diferir na sua essência do tratamento farmacológico da Dor Miofascial tratada no próximo capítulo, e para não ser redundante nesta publicação, remete-se aqui para a leitura do tratamento farmacológico no capítulo seguinte intitulado. Dor Miofascial".

Referências Bibliográficas:

1. Harrison AL, Thorp JN, Ritzline PD. A Proposed Diagnostic Classification of Patients With Temporomandibular Disorders: Implications for Physical Therapists. J Orthop Sports Phys Ther 2014;44(3):182-97.

2. Leeuw Rd, Klasser GD. Orofacial Pain : guidelines for assessment, diagnosis, and management. Fifth edition. ed: American Academy of Orofacial Pain - Quintessence Books; 2013.

3. Manfredini D, Piccotti F, Ferronato G, Guarda-Nardini L. Age peaks of different RDC/TMD diagnoses in a patient population. Journal of Dentistry 2010;38(5):392-99.

4. Roda RP, Bagan J.V, Fernandez JMD, Bazan SH, Soriano YJ. Review of temporomandibular joint pathology. Part I: classification, epidemiology and risk factors. Med Oral Patol Oral Cir Bucal 2007;12:E292-8.

5. Manfredini D, Guarda-Nardini L, Winocur E, et al. Research diagnostic criteria for temporomandibular disorders: a systematic review of axis I epidemiologic findings. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology 2011;112(4):453-62.

6. Mense S. The pathogenesis of muscle pain. Current Pain and Headache Reports 2003;7(6):419-25.

7. Schiffman E, Ohrbach R, Truelove E, et al. Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) for Clinical and Research Applications: recommendations of the International RDC/TMD Consortium Network* and Orofacial Pain Special Interest Groupdagger. J Oral Facial Pain Headache 2014;28(1):6-27.

8. Peck CC, Goulet JP, Lobbezoo F, et al. Expanding the taxonomy of the diagnostic criteria for temporomandibular disorders. J Oral Rehabil 2014;41(1):2-23.

9. Simons DG, Travell JG, Simons LS. Travell & Simons' Myofascial pain and dysfunction: The trigger point manual. . 2nd ed: Williams & Wilkins; 1999.

10. Bogduk N, Govind J. Cervicogenic headache: an assessment of the evidence on clinical diagnosis, invasive tests, and treatment. Lancet Neurol 2009;8:959-68.

11. La Touche R, Paris-Alemany A, von Piekartz H, et al. The influence of cranio-cervical posture on maximal mouth opening and pressure pain threshold in patients with myofascial temporomandibular pain disorders. Clin J Pain 2011;27(1):48-55.

12. Silveira A, Gadotti IC, Armijo-Olivo S, Biasotto-Gonzalez DA, Magee D. Jaw dysfunction is associated with neck disability and muscle tenderness in subjects with and without chronic temporomandibular disorders. (2314-6141 (Electronic)).

13. Armijo-Olivo S, Magee D. Cervical musculoskeletal impairments and temporomandibular disorders. J Oral Maxillofac Res 2013;3(4):e4-e4.

14. Armijo Olivo S, Magee Dj Fau - Parfitt M, Parfitt M Fau - Major P, Major P Fau - Thie NMR, Thie NM. The association between the cervical spine, the stomatognathic system, and craniofacial pain: a critical review. (1064-6655 (Print)).

15. Kraus S. Characteristics of 511 TMD Patients Referred for Physical Therapy: Elsevier Ltd; 2014.

16. Roda RP, Bagán JV, J.M.D. F, Bazán SH, Soriano YJ. Review of temporomandibular joint pathology. Part I: classification, epidemiology and risk factors. Med Oral Patol Oral Cir Bucal 2007;12:292-8.

17. Cooper BC, International College of Cranio-Mandibular O. Temporomandibular disorders: A position paper of the International College of Cranio-Mandibular Orthopedics (ICCMO). Cranio 2011;29(3):237-44.

18. de Felicio CM, Melchior Mde O, Da Silva MA. Clinical validity of the protocol for multi-professional centers for the determination of signs and symptoms of temporomandibular disorders. Part II. Cranio 2009;27(1):62-7.

19. Manfredini D, Bucci MB, Nardini LG. The diagnostic process for temporomandibular disorders. Stomatologija 2007;9(2):35-9.

20 Manfredini D. Current Concepts on Temporomandibular Disorders. Quintessence Publishing 2010.

21. Magalhaes BG, de-Sousa ST, de Mello VV, et al. Risk factors for temporomandibular disorder: binary logistic regression analysis. Med Oral Patol Oral Cir Bucal 2014;19(3):e232-6.

22. Manfredini D, Ahlberg J, Winocur E, Guarda-Nardini L, Lobbezoo F. Correlation of RDC/TMD axis I diagnoses and axis II pain-related disability. A multicenter study. Clin Oral Investig 2011;15(5):749-56.

23. Miettinen O, Lahti S, Sipila K. Psychosocial aspects of temporomandibular disorders and oral health-related quality-of-life. Acta Odontol Scand 2012;70(4):331-6.

24. Molina OF, dos Santos Junior J, Nelson SJ, Nowlin T. Profile of TMD and Bruxer compared to TMD and nonbruxer patients regarding chief complaint, previous consultations, modes of therapy, and chronicity. Cranio 2000;18(3):205-19.

25. Dommerholt J. Dry needling — peripheral and central considerations. J Man Manip Ther 2011;19(4):223-27.

26. Dunning J, Butts R, Mourad F, et al. Dry needling: a literature review with implications for clinical practice guidelines. Phys Ther Rev 2014;19(4):252-65.

27. Dommerholt J, Mayoral-Del-Moral O, Gröbli C. Trigger point dry needling. J Man Manip Ther 2006;14:E70-E87.

28. Gonzalez-Perez LM, Infante-Cossio P, Granados-Nuñez M, Urresti-Lopez FJ. Treatment of temporomandibular myofascial pain with deep dry needling. Med Oral Patol Oral Cir Bucal 2012;17(5):781-5.

29. Kalamir A, Graham PL, Vitiello AL, Bonello R, Pollard H. Intra-oral myofascial therapy versus education and self-care in the treatment of chronic, myogenous temporomandibular disorder: a randomised, clinical trial. Chiropractic & Manual Therapies 2013;21:17-17.

APPENDIX II

BOOK CHAPTER - Dor miofascial

Ribeiro, J.J., Paço, M., & Oliveira, T. (2016). Dor miofascial. In Sociedade Portuguesa De Disfunção Temporomandibular e Dor Orofacial (Ed.), *Dor orofacial e disfunções temporomandibulares: tratamento farmacológico* (pp. 43-47). ISBN: 978-989-20-6409-3

// DOR MIOFASCIAL

Joana Jesus Ribeiro | Maria Paço | Tiago Oliveira

Definição:

O Síndrome de Dor Miofascial (SDM) é uma disfunção muscular dolorosa associada normalmente à presença de trigger points ou pontos gatilho.[1, 2] Os trigger points são caracterizados como nódulos focais e hiperirritáveis localizados numa banda tensa de um músculo-esquelético (Figura 2). Estes nódulos são dolorosos à palpação e podem produzir dor ou sensibilidade referida, disfunção motora e fenómenos autonómicos.[3, 4] A existência de dor referida (um sub-grupo da ´´Dor Secundária ou Heterotópica´´ ver capitulo posterior na zona azul) é uma característica importante de um trigger point, caracterizando-se como uma dor que é sentida não apenas no local de origem mas também à distância, sendo clinicamente descrita pelo paciente como uma dor irradiada. É a presença deste padrão que permite ao clínico distinguir um trigger point de um ponto sensível à palpação ou de uma simples contractura. A etiologia do SDM é complexa e ainda não totalmente compreendida, podendo referir-se como factores predisponentes não só condições locais (trauma, tensão muscular, hábitos posturais), como também sistémicas (alterações vitamínicas, infeções, hiperuricémia, deficiência de estrogénio, anemia, deficiência de ferro, entre outras).[5]

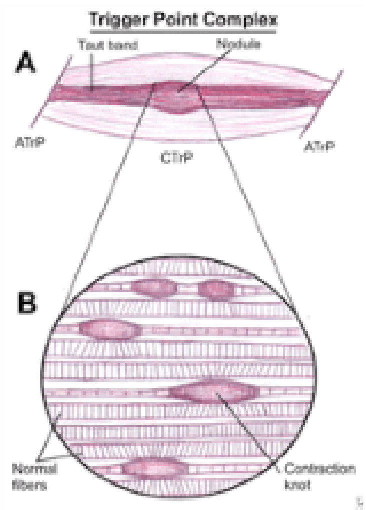


Figura 2: Complexo do trigger point: visão microscópica que ilustra vários pontos de contração de fibras musculares individuais. Adaptado de: Simons DG, Travell JG, Simons LS. Travell & Simons' Myofascial pain and dysfunction: The trigger point manual. 2nd ed: Williams & Wilkins; 1999. pg 70

Epidemiologia:

A dor miofascial é uma das principais causas de dor não dentária na região orofacial e corresponde a cerca de 30% dos pacientes em consulta para tratamento da disfunção temporomandibular (DTM).6 Foi demonstrada uma variação substancial no curso da dor miofascial associada a DTM, com 31% dos indivíduos a referir a manutenção da DTM por um período de 5 anos, 33% a referirem que houve remissão da sintomatologia e 36% dos pacientes a reportarem recidiva.[7]

Caraterísticas clínicas:

É frequente o doente com SDM apresentar queixas de dor regional e persistente que poderá resultar em perda de amplitude de movimento dos músculos afetados. Embora a dor esteja normalmente relacionada com a atividade muscular, o doente poderá descrevê-la como constante. É uma dor reprodutível e que não segue o trajeto de uma raiz nervosa. [1,2] Na região cefálica e cervical, os trigger points podem manifestar-se como cefaleia tipo tensão, tinnitus, dor na ATM, sintomas visuais e torcicolo.[8] A dor miofascial associada aos músculos da mastigação pode provocar dor facial ou cervical, podendo apresentar-se como parte do espectro da DTM, sendo importante pesquisar a presença de bruxismo, som articular, avaliar a amplitude da abertura bucal e o seu encerramento adequado.9 Em geral, o curso natural do SDM não é progressivo e tem remissão após um período de tempo. No entanto, podem verificar-se recorrências, sendo importante excluir fatores predisponentes.[5]

Patofisiologia:

Diferentes mecanismos patofisiológicos têm sido descritos no SDM. A isquémia, a hipóxia e uma síntese insuficiente de ATP nas fibras das unidades motoras tipo I podem ser responsáveis por um aumento na acidez, acumulação de cálcio e consequente encurtamento de sarcómeros. Por sua vez, este estado mantido de encurtamento dos sarcómeros poderá levar a uma diminuição da perfusão muscular com consequente hipóxia e isquémia, tornando-se assim um ciclo vicioso que propicia o desenvolvimento de trigger points. Como resultado, várias substâncias sensibilizantes poderão ser libertadas levando ao desenvolvimento de dor local e referida, para além de sensibilidade muscular, que são caraterísticas clínicas do SDM. [2]

A dor referida apresenta determinadas caraterísticas: é mais comum em diferentes ramos do mesmo nervo e ocorre num padrão laminado (por exemplo: dor no ramo mandibular do V par referida ao ramo maxilar do V par). No entanto, a dor pode ser referida para territórios de outro nervo (por exemplo: dor de ponto gatilho cervical que irradia para a face). Neste caso move-se mais frequentemente em direção cefálica e geralmente não atravessa a linha média.[10,11] Existem várias teorias que suportam este fenómeno. Uma das causas é a complexidade dos nervos encarregados de recolher a sensibilidade oro-facial. Os nociceptores somáticos da face enviam sinais ao tronco cerebral através das fibras dos V, VII, IX e X pares cranianos. [10-12] O trigémio (V par) assegura através das suas fibras sensitivas a inervação da totalidade da face e da metade anterior da cabeça, das mucosas ocular, nasal, sinusal e bucal, dos dentes e de uma grande superfície da dura-máter craniana. Através das suas fibras motoras inerva os músculos mastigatórios.[12-14] No entanto, outros pares cranianos também participam na condução da nocicepção da cavidade oral, uma vez que a integração aferente dolorosa é também transportada através de fibras do VII, IX, X e raízes cervicais superiores.[12-16] Esta convergência de neurónios aferentes que fazem sinapse num único interneurónio ao nível da ponte pode confundir o córtex resultando em dor referida.[10-12] Outro fator que concorre para a existência de dor referida é a presença de estímulos dolorosos aferentes constantes que levam à acumulação de neurotransmissores nas sinapses, com passagem para o interneurónio adjacente (efeito de excitação central) e consequente despolarização de outra via nociceptiva ascendente. Assim o córtex recebe dois impulsos, um é da fonte da dor, o outro será dor heterotópica.[10-12] Por último, é ainda importante referir que representação cortical da face e boca é extensa devido à grande densidade de inervação por unidade de superfície que existe nas estruturas oro-faciais.[13]

Diagnóstico:

O diagnóstico de dor miofascial é clínico.[5] Existem três critérios para o diagnóstico de trigger points: a existência de uma banda tensa palpável, a existência de um nódulo palpável e a reprodução da dor do paciente ao realizar pressão no nódulo sensível. [2,4] Os trigger points podem ser classificados, de acordo com as suas caraterísticas clínicas em ativos (dor em repouso, sensível à palpação e com um padrão de dor referida semelhante à queixa dolorosa do paciente) ou latentes (não causa dor espontânea nem em repouso, mas pode restringir o movimento ou causar fraqueza muscular e reproduz os sintomas se o nódulo na banda densa for pressionado).[1, 3, 4]

São várias as patologias que devem ser consideradas no diagnóstico diferencial com o SDM, nomeadamente patologia articular (osteoartrite) ou dos tecidos moles (tendinite), patologia inflamatória (polimialgia reumática), patologia neurológica (radiculopatias) ou patologia sistémica (hipotiroidismo).[17] Assim, torna-se importante não só a realização de um controlo analítico de base, bem como em casos específicos a utilização do apoio imagiológico para exclusão de outras patologias subjacentes.[17] No caso de dor orofacial é importante uma avaliação completa do sistema musculoesquelético local e da ATM, assim como a exclusão de patologia dentária.[5]

Tratamento:

O tratamento do SDM deverá apresentar-se como uma abordagem multidisciplinar, com cariz bio-psico-social que engloba a farmacoterapia, a fisioterapia e a psicoterapia, entre outros. [1, 4, 8, 18-20] O tratamento farmacológico utilizado nos distúrbios dos músculos mastigatórios em geral e no SDM, em particular, é heterogéneo, tendo como alvos quer o componente muscular local, quer o sistema nervoso central.[17]

Os anti-inflamatórios não esteróides, apesar da limitada evidência quanto à sua eficácia, são frequentemente integrados no tratamento do SDM, preferencialmente associados a outras terapêuticas.[17, 21] Tanto a via de administração oral como a tópica foram já reportadas no SDM, destacando-se o melhor perfil de segurança desta última.[17]

A dor miofascial associa-se a um aumento da tensão muscular, com consequente espasmo e formação de trigger points[5] Assim, os miorrelaxantes desempenham um papel preponderante no alívio da dor.[5] A Tizanidina, agonista 2-adrenérgico, é um dos relaxantes musculares com ação central mais frequentemente usados.[5, 17] A ciclobenzaprina é frequentemente utilizada na dor músculo-esquelética, sendo em geral bem tolerada.[17] Como efeitos secundários é importante destacar o efeito sedativo destes fármacos, devendo ser usados preferencialmente numa toma noturna.[17]

O uso de analgésicos é outra vertente importante no tratamento do SDM. O uso de opióides tradicionais é controverso e geralmente não recomendado, principalmente na dor miofascial associada aos músculos da mastigação.[21] O tramadol é um fraco agonista opióide usado frequentemente no tratamento da dor crónica generalizada, não existindo porém estudos publicados que suportem o seu uso na dor miofascial.[17, 21] O selo transdérmico de lidocaína apresenta penetração local com absorção sistémica limitada, tendo mostrado eficácia no alívio da dor no SDM num estudo randomizado. [17]

O clonazepam e diazepam são benzodiazepinas muitas vezes integradas no tratamento do SDM, não só pelo seu efeito como relaxante muscular, mas também pela sua ação em sintomas frequentemente associados a este síndrome como ansiedade e alteração do padrão de sono.[17]

O stress e a patologia psiquiátrica são fatores que predispõem ao desenvolvimento de dor miofascial. De fato, a utilização de antidepressivos no controlo da dor tem mostrado um efeito benéfico através da ação nas vias serotoninérgicas e noradenérgicas.[5] Os antidepressivos tricíclicos, com destaque para amitriptilina, provaram ser eficazes no SDM, ainda que de forma modesta.[5, 17, 21] Relativamente aos inibidores seletivos da recaptação da serotonina, a sua eficácia nos síndromes de dor regional ainda não esta comprovada.[17, 21] Os inibidores da recaptação da serotonina e noraradrenalina, como a duloxetina, demonstraram benefício no tratamento da dor miofascial.[5, 17, 21]

Antiepiléticos, como a gabapentina ou pregabalina, apresentam um papel preponderante no alívio da dor, principalmente de cariz neuropático.[5, 17, 21]

Nos casos refractários ou não responsivos ao tratamento farmacológico pode recorrer-se à injeção de fármacos a nível dos trigger points, nomeadamente corticóides, anestésicos locais ou toxina botulínica.[5, 17] A injeção do trigger point provoca não só uma ação química pela substância injetada como também uma disrupção mecânica, promovendo uma vasodilatação da área com diluição das substância patogénicas acumuladas no músculo e relaxamento da fibra muscular.[5] Apesar da inflamação desempenhar um papel importante no SDM, o benefício dos corticóides é limitado.[17]

Sempre que está indicado um bloqueio anestésico com fins diagnósticos ou terapêuticos devem estar preenchidos alguns requisitos básicos. O clínico deve conhecer muito bem a anatomia local, deve injetar uma solução de baixa toxicidade muscular[22] e sempre sem vasoconstritor, usar uma técnica asséptica e limpeza adequada da pele, aspiração prévia à injeção para evitar injeções intravasculares.[22, 23] Os anestésicos mais comummente usados são a lidocaína e a bupivacaína sem vasoconstritor (são ambos amidas e menos tóxicas do que os anestésicos do grupo éster). [22]

A toxina botulínica é uma neurotoxina produzida pela bactéria Clostridium botulinum que apresenta uma ação periférica e central no alívio da dor.[17] Por um lado, bloqueia a libertação de acetilcolina na junção neuromuscular, inibindo a contração/espasmo muscular de forma seletiva com consequente interrupção do ciclo de dor.[9, 24, 25] Por outro lado, reduz a transmissão nociceptiva no sistema nervoso periférico e central.[17, 25] A duração do efeito é cerca de 3-4 meses.[24] Atualmente, apresenta-se como um tratamento emergente na área da dor miofascial, embora ainda com grau de evidência discutível.[24, 25] No entanto, no SDM dos músculos da mastigação a injeção de toxina botulínica apesar de não ter garantido a resolução completa dos sintomas, demonstrou um efeito benéfico em 79% dos pacientes com melhoria dos sintomas9, devendo ser considerada como um tratamento alternativo nos pacientes nos quais a terapêutica convencional falhou.[9, 26] Os efeitos adversos são incomuns e geralmente ligeiros, podendo provocar fraqueza muscular transitória e estando contra-indicada na gravidez e em algumas doenças neuromusculares.[26]

Estão contra-indicadas as injeções de pontos gatilho quando o paciente toma anticoagulantes ou apresenta distúrbios da coagulação, na presença de infecções locais ou sistémicas, alergia aos agentes injetados ou fobia de agulhas.[27] Com os adequados cuidados para evitar injeções intravasculares, estes procedimentos são na generalidade seguros, sendo os efeitos adversos graves raros.[22, 23, 27] As possíveis complicações são a dor no local da injeção, hemorragia/hematomas, infeção cutânea, atrofia muscular, síncope vasovagal e a quebra da agulha.[22, 23, 27, 28]

Dentro da intervenção da fisioterapia destacam-se procedimentos como a terapia manual, o exercício terapêutico (alongamento, biofeedback), a termoterapia, a electroterapia (estimulação elétrica nervosa transcutânea, estimulação eléctrica muscular), a aplicação de ultra-sons e a punção seca.[24] A terapia manual juntamente com a junção seca apresenta um papel especialmente preponderante na resolução dos trigger points. São procedimentos frequentemente utilizados que têm sido objeto de vários estudos científicos, tendo demonstrado contribuir para uma melhoria da sintomatologia dolorosa. [18, 29-32]

Referências Bibliográficas:

1. Alvarez D, Rockwell PG. Trigger Points: Diagnosis and Management. Am Fam Physician 2002;65(4):653-60.
2. Shah JP, Thaker N, Heimur J, et al. Myofascial Trigger Points Then and Now: A Historical and Scientific Perspective. PM&R 2015;7(7):746-61.
3. Simons DG. Review of enigmatic MTrPs as a common cause of enigmatic musculoskeletal pain and dysfunction. J Electromyogr Kinesiol 2004;14:95-107.
4. Simons DG, Travell JG, Simons LS. Travell & Simons' Myofascial pain and dysfunction: The trigger point manual... 2nd ed: Williams & Wilkins; 1999.
5. Pal US, Kumar L, Mehta G, et al. Trends in management of myofacial pain. Natl J Maxillofac Surg 2014;5(2):109-16.
6. McNeill C. Temporomandibular Disorders: Guidelines for Classification, Assessment and Management. Chicago: Quintessence; 1990.
7. Rammelsberg P, LeResche L, Dworkin S, Mancl L. Longitudinal Outcome of Temporomandibular Disorders: A 5-year Epidemiologic Study of Muscle Disorders Defined by RDC/TMD. J Orofac Pain 2003;17(1):9-20.
8. Fernández-Carnero J, LaTouche R, Ortega-Santiago R, et al. Short-term effects of dry needling of active myofascial trigger points in the masseter muscle in patients with temporomandibular disorders. Journal of orofacial pain 2010;24(1):106-12.
9. Sidebottom AJ, Patel AA, Amin J. Botulinum injection for the management of myofascial pain in the masticatory muscles. A prospective outcome study. Br J Oral Maxillofac Surg 2013;51(3):199-205.
10. Oliveira Wd. Disfunçoes Temporomandibulares 6. Artes Médicas 2002;Ediçao 1:480.
11. Okeson JP. Management of temporomandibular disorders and occlusion. 3rd ed. St. Louis: Mosby-Year Book; 1993.
12. Bell W. Orofacial pains: Classification, Diagnosis, Management. Year Book Medical Publishers, Chicago 1985.
13. Noback CR DR. Sistema Nervioso Humano. Fundamentos de Neurobiología. McGraw-Hill Book Co, México 1975.pg: 352-3.
14. Lazorthes G. Nervio trigémino. Em "Sistema nervioso periférico" Toray-Masson 1976;Barcelona:61-9.
15. Penarrocha M, Alfaro A, Bagan JV, Milian A. [Evaluation of the patient with facial pain]. Rev Actual Estomatol Esp 1987;47(369):25-8, 21-2, 25-6.
16. Penarrocha M, Bagan JV, Oltra MJ, Milian MA. [Determining the cause of facial pain]. Rev Actual Estomatol Esp 1987;47(365):27-30.
17. Borg-Stein J, Iaccarino MA. Myofascial pain syndrome treatments. Phys Med Rehabil Clin N Am 2014;25(2):357-74.
18. Kalamir A, Graham PL, Vitiello AL, Bonello R, Pollard H. Intra-oral myofascial therapy versus education and self-care in the treatment of chronic, myogenous temporomandibular disorder: a randomised, clinical trial. Chiropractic & Manual Therapies 2013;21:17-17.
19. Kindelan-Calvo P, Gil-Martínez A, Paris-Alemany A, et al. Effectiveness of Therapeutic Patient Education for Adults with Migraine. A Systematic Review and Meta-Analysis of Randomized Controlled Trials. Pain Med 2014;15(9):1619-36.
20. de Leeuw R, Klasser GD. Orofacial Pain: Guidelines for Assessment, Diagnosis, and Management. . 5th ed: Quintessence Books.; 2013.
21. Clark GT. Classification, causation and treatment of masticatory myogenous pain and dysfunction. Oral Maxillofac Surg Clin North Am 2008;20(2):145-57, v.
22. Ashkenazi A, Blumenfeld A, Napchan U, et al. Peripheral nerve blocks and trigger point injections in headache management - a systematic review and suggestions for future research. Headache 2010;50(6):943-52.
23. Bernstein RM. Injections and surgical therapy in chronic pain. Clin J Pain 2001;17(4 Suppl):S94-104.
24. Soares A, Andriolo RB, Atallah AN, da Silva EM. Botulinum toxin for myofascial pain syndromes in adults. Cochrane Database Syst Rev 2014;7:CD007533.
25. Zhou JY, Wang D. An update on botulinum toxin A injections of trigger points for myofascial pain. Curr Pain Headache Rep 2014;18(1):386.
26. Mor N, Tang C, Blitzer A. Temporomandibular Myofacial Pain Treated with Botulinum Toxin Injection. Toxins (Basel) 2015;7(8):2791-800.
27. Scott NA, Guo B, Barton PM, Gerwin RD. Trigger point injections for chronic non-malignant musculoskeletal pain: a systematic review. Pain Med 2009;10(1):54-69.
28. Blumenfeld A, Ashkenazi A, Grosberg B, et al. Patterns of use of peripheral nerve blocks and trigger point injections among headache practitioners in the USA: Results of the American Headache Society Interventional Procedure Survey (AHS-IPS). Headache 2010;50(6):937-42.
29. Dommerholt J. Dry needling — peripheral and central considerations. J Man Manip Ther 2011;19(4):223-27.
30. Dunning J, Butts R, Mourad F, et al. Dry needling: a literature review with implications for clinical practice guidelines. Phys Ther Rev 2014;19(4):252-65.
31. Dommerholt J, Mayoral-Del-Moral O, Grobli C. Trigger point dry needling. J Man Manip Ther 2006;14:E70-E87.
32. Gonzalez-Perez LM, Infante-Cossio P, Granados-Nuñez M, Urresti-Lopez FJ. Treatment of temporomandibular myofascial pain with deep dry needling. Med Oral Patol Oral Cir Bucal 2012;17(5):781-5.