MUSCLE ACTIVITY IN PAINFUL TEMPOROMANDIBULAR DISORDERS: THEORIES AND CONCEPTUAL MODELS

MARCIN BERGER[1], JOLANTA SZYMAŃSKA[2]

[1] *Department of Functional Masticatory Disorders, Medical University of Lublin*
[2] *Department of Integrated Paediatric Denstitry, Medical University of Lublin*

Abstract

Introduction. Temporomandibular disorders (TMD) are defined by the American Association for Dental Research as a group of musculoskeletal and neuromuscular conditions that involve the temporomandibular joints (TMJ), the masticatory muscles, and all the associated tissues.

Painful TMJ disorders, excluding cancer-related pain, is the third, after tension headaches and spinal pain, most frequent cause of chronic pain. Analyzing the mechanisms that may explain the connection of psychological factors with pain chronification, it is also necessary to consider their role in producing behavioral responses to pain that may lead to its maintenance.

Aim. The aim of the study was to present theories and conceptual models addressing motor responses in painful TMJ disorders.

Material and methods. The literature published in the 10 years prior to the study from PubMed database has been reviewed using keywords: temporomandibular disorders; pain; masticatory muscles; muscle activity, risk factors, psychological factors.

Results. An analysis of the existing conceptualizations of motor responses to pain shows that their evolution is similar to that of the theories of pain experience – from the biomedical model to the biopsychosocial one. Mechanistic theories, based merely on changes in neurons excitability, could not fully account for the variability in the observed motor response to pain. Those theories were thus replaced by more complex models that included the role of psychosocial factors contributing to the individual character of pain experience.

Conclusions. A thorough knowledge of the connection between psychological factors and a change in muscle activity in response to pain may significantly help to understand the course of musculoskeletal disorders associated with chronic pain. This is due above all to the individual, dependent on psychological factors, character of reaction to pain and to a postulated possibility of activity change that may support pain or lead to the development of new disorders.

Keywords: temporomandibular disorders (TMD), pain, masticatory muscles, muscle activity, risk factors, psychological factors.

Introduction

Temporomandibular disorders (TMD) are defined by the American Association for Dental Research as a group of musculoskeletal and neuromuscular conditions that involve the temporomandibular joints (TMJ), the masticatory muscles, and all the associated tissues [1]. It should be noted that the definition, so formulated, refers mainly to the common location of complaints, and thus the otherwise unrelated diseases are often included under the heading of TMD. Among them there are, first of all, congenital TMJ conditions (e.g. agenesis, aplasia of the head of mandible), acquired TMJ conditions (e.g. joint disc disorders, TMJ inflammations related to systemic diseases, neoplasms), masticatory muscles disorders (e.g. myofascial pain, myositis, myospasm), and movement disorders (e.g. oromandibular dystonia) [2].

Apart from the common location, TMD are also connected by similar clinical symptoms, such as acoustic symptoms in TMJ, orofacial pain, and jaw mobility disorders [3]. Therefore, despite a considerable heterogeneity, it is practically justified to consider this group of complaints jointly.

TMD are diagnosed by clinical examination, supplemented, if necessary, by suitable imaging examinations [1,4,5]. In scientific research, as well as in clinical practice, the Research Diagnostic Criteria for Temporomanidbular Disorders (RDC/TMD), considered as the 'golden standard', are most frequently used [6]. RDC/TMD allow to diagnose three most frequent groups of temporomandibular complaints: myofascial disorders, joint disc displacement, and arthralgias, joint inflammations and degenerations.

The classification of functional disorders according to RDC/TMD includes the following groups of disorders: Group I – Muscle Disorders: (Ia) myofascial pain; (Ib) myofascial pain with limited opening. Group II – Disc Displacements: (IIa) disc displacement with reduction; (IIb) disc displacement without reduction with limited opening; (IIc) disc displacement without reduction without limited opening. Group III – Arthralgia, Arthritis, Arthrosis: (IIIa) arthralgia; (IIIb) osteoarthritis; (IIIc) osteoarthrosis.

In 2014, the Diagnostic Criteria for Temporomandibular Disorders (DC/TMD), which are to replace RDC/TMD, were published [2,5]. DC/TMD modified and extended, among others, diagnostics of muscle pain, taking into account its various clinical manifestations and adding disorders not included in RDC/TMD. The latter criteria were based mainly on the opinions of TMD experts, while DC/TMD were developed using research results, according to the principles of evidence-based medicine. Also, in contradistinction to RDC/TMD, DC/TMD were designed for use both in research and in clinical practice [5]. The extended TMD classification, according to DC/TMD, is presented in Table 1.

Symptoms of the temporomandibular disorders

Most frequently, TMD symptoms occur between 20 and 40 years of age, less often in people of other age groups [7]. It should be noted, however, that the peak morbidity

Table 1. The extended TMD classification according to DC/TMD

I. TEMPOROMANDIBULAR JOINT DISORDERS
1. Joint pain
A. Arthralgia
B. Arthritis
2. Joint disorders
A. Disc disorders
1. Disc displacement with reduction
2. Disc displacement with reduction with intermittent locking
3. Disc displacement without reduction with limited opening
4. Disc displacement without reduction without limited opening
B. Other hypomobility disorders
1. Adhesions / adherence
2. Ankylosis
a. Fibrous
b. Osseous
C. Hypermobility disorders
1. Dislocations
a. Subluxation
b. Luxation
3. Joint diseases
A. Degenerative joint disease
1. Osteoarthrosis
2. Osteoarthritis
B. Systemic arthritides
C. Condylysis/idiopathic condylar resorption
D. Osteochondritis dissecans
E. Ostronecrosis
F. Neoplasm
G. Synovial chondromatosis
4. Fractures
5. Congenital/developmental disorders
A. Aplasia
B. Hypoplasia
C. Hyperplasia
II. MASTICATORY MUSCLE DISORDERS
1. Muscle pain
A. Myalgia
1. Local myalgia
2. Myofascial pain
3. Myofascial pain with referral
B. Tendonitis
C. Myositis
D. Spasm
2. Contracture
3. Hypertrophy
4. Neoplasm
5. Movement disorders
A. Orofacial dyskinesia
B. Oromandibular dystonia
6. Masticatory muscle pain attributed to systemic/central pain disorders
A. Fibromyalgia/ widespread pain
III. HEADACHE
1. Headache attributed to TMD
IV. ASSOCIATED STRUCTURES
1. Coronoid hyperplasia

differs for different complaints. For instance, joint disc dislocation is most frequently diagnosed in ca. 30-year old patients, while arthralgia, joint inflammation and joint degeneration – in patients at the age of ca. 50 years [8,9]. Acoustic symptoms in TMJ, as a sign of disc displacement with reduction, are the most frequent manifestation of TMD in general population and occur in 11% of subjects [7]. As TMJ disc displacements with reduction are usually not particularly troublesome, they remain unnoticed by patients and do not require treatment. It is estimated that between 4% and 7% of general population needs therapy due to TMD [10]. The most common reason for seeking treatment for TMD is pain [8]. Painful TMD, excluding cancer-related pain, is the third, after tension headaches and back pain, most frequent cause of chronic pain [11], it is also the second, after dental pain, most common source of all facial pain complaints [3].

The main type of pain in TMD is myofacial pain. Masseter muscle pain is observed in ca. 10% of general population. It is noteworthy that TMJ pain is much less frequent, and its prevalence is estimated at ca. 3%. At the same time, myofacial pain in patients seeking treatment for TMD is diagnosed in 45% of cases and TMJ pain in 34% [7].

Painful TMD considerably decreases the quality of life and involves high economic and social costs [5]. It is estimated that for every 100 million working adults in the United States of America, there are 18 million of sick leave days per year due to TMD. It is also worth noting, that although chronic painful TMD occurs in a relatively small group of patients, the means allocated for treatment of this complaint constitute 85% of the total TMD treatment costs [12].

Etiology of painful TMD

Over the years, the theories of TMD etiology have been subject to controversy and significant change. Initially, there were attempts to explain the origin of TMD using a mechanistic biomedical model, based exclusively on the influence of biological factors, mostly structural ones, and especially occlusal disorders. At present, much less importance is attributed to those factors in the etiology of TMD than in the past [10]. Moreover, researchers believe that it is impossible to identify only one cause of TMD, as it results from an interaction of numerous factors that lead to a decrease in the adaptive capacity of the organism. The factors that may reduce the temporomandibular adaptive capacity include anatomical determinants, as well as systemic, pathophysiological and psychological ones [10]. Psychological factors, whose role in the etiology of painful TMD was recognized only after the biopsychosocial model had been introduced, are particularly worthy of note. The biopsychosocial model was proposed as an alternative to the biomedical model which did not include the role of psychosocial factors in the origin of diseases [13]. The biomedical model assumed that pain is a direct result of noxious impulses transmitted from the peripheral structures to the central nervous system (nociceptions). Although this model became the basis for development of numerous methods of chronic pain treatment, it did not sufficiently address the complexity of pain experience. The biomedical model also failed to explain such phenomena as:

the presence of pain without a clearly identified pathology, pain greater (or smaller) than expected on the basis of medical tests results, or varying individual responses to pain and treatment. The clinical image of pain becomes easier to understand when analyzed using the biopsychosocial model. The concept of pain as a subjective experience that includes affective, cognitive, and behavioral components, conditioned by biological factors but also dependent on the ontogenetic development of individuals and their interaction with the environment in which they live, enables a comprehensive assessment of pain and its appropriate treatment. In this connection, an assessment of pain should include intensity, disability (functional limitations), emotional distress, emotional factors, cognitive factors, coping with pain and quality of life [14]. The biopsychosocial model was applied to the study of TMD in 1992 together with the publication of RDC/TMD and it is currently used in the examination of all orofacial pain complaints [15].

Numerous studies conducted in the recent years, aiming at identification of risks related to painful TMD [16-21]. Multicenter research, which started in 2006 (Orofacial Pain Prospective Evaluation and Risk Assessment Study) led to a discovery of numerous risk factors associated with the origin and chronification of painful TMD [20]. It is estimated that about 4% of general population develops painful TMD [19]. The morbidity is affected by many variables: sociodemographic and psychological factors, factors related to the general health status (e.g. tobacco smoking, sleep disorders), sensitivity to pain, parafunctional mandibular activity, painless TMD symptoms, autonomic nervous system activity, and genetic factors [20]. Acute TMD-related pain is usually short-term and disappears spontaneously, without treatment, within about two months [22]. However, in almost 50% of subjects suffering from acute pain, the complaint did not subside in 6 months [20], while in ca. 30% of subjects with myofacial pain (diagnosed according to RDC/TMD criteria), the complaint remitted in 5 years; the remaining patients continued to suffer from constant or recurrent pain [23].

It is also noteworthy that painful TMD occurs three times more often in women than in men [9]. It should be observed, however, that it is primarily chronic TMD that occurs more often in women than in men. In the case of acute TMD-related pain, the differences between genders are much smaller [20]. The phenomenon of chronification of TMD-related pain is more often found in people with other concurrent chronic pain disorders, such as headaches, endometriosis, fibromyalgia, bladder pain syndrome, lower back pain, chronic fatigue syndrome, and vulvodynia [24]. It should also be noted that TMD and the listed disorders often occur together and are therefore believed to be connected by a common etiology, i.e. they result from alterations in central nervous system processes that lead to pain hypersensitivity [12,25]. The neuropysiological processes related to increased nociceptive transmission in the central nervous system are described as central sensitization [25,26].

Chronification of TMD-related pain

Acute and chronic pain are distinguished mainly based on the criterion of time. Pain is described as chronic when it persists longer than the typical healing period. Due to the difficulty to precisely define the typical healing time, the time threshold as the criterion to classify pain as chronic is determined arbitrarily; most often it is 3 or 6 months [27]. In scientific studies on TMD, pain disorders persisting over 6 months are considered as chronic [28].

A great majority of scientific studies suggests that chronic pain develops and persists as a result of a complex interaction of psychological, cognitive, environmental, and neurophysiological factors [29]. Research results also confirm a multifactorial etiology of chronic painful TMD. Chronification of TMD-related pain results from an influence of numerous factors, which may be most generally divided into environmental, psychological, genetic, and related to amplified nociceptive transmission [12].

It is worth noting that the studies conducted as part of the OPPERA project identified the gene polymorphisms related to chronic painful TMD. The polymorphisms are relevant for the function of autonomic system, nociceptive transmission and the processes underlying affective states [22].

Psychological factors associated with chronification of pain

Psychological factors play probably one of key roles in the etiology of chronic pain. Numerous studies found a connection between chronic pain and intensity of psychological factors related to negative affect and general anxiety – fear, depression and catstrophizing. In addition, patients suffering from chronic pain-related disorders more often report non-specific somatic complaints of psychogenic origin, which is described as somatization [30]. Anxiety symptoms are observed in over 80% of patients who suffer from persistent pain more than 4-6 months [14].

Anxiety is defined as a negative emotional state related to anticipation of danger coming from outside of the organism or originating inside it. In contradistinction to fear, anxiety is not related to immediate danger, but to imaginings connected to the experience of danger [31].

Anxiety is an essential component of depression, understood as mood disorder characterized by loss of interests and inability to feel positive emotions. Additionally, depression is manifested by energy decrease, which leads to fatigability, limits usual activities, weakens attention and concentration, causes low self-esteem, self-worth, and pessimism, as well as disturbed sleep and appetite [32].

The term "catastrophizing" was coined to describe a maladaptive cognitive style related to an irrational negative forecast of events [33], while pain catastrophizing may be characterized as a negative cognitive-emotional response to anticipated or actually experienced pain [34].

Catastrophizing often occurs in connection with other psychological factors, showing a correlation with the intensity of depression, anxiety, and fear of pain, as well as a negative correlation between the sense of self-efficacy, optimism, and

other positive factors. Pain catastrophizing appears as a cognitive element of anxiety, besides physiological reactivity and behavioral responses of the organism, and refers to the process during which pain is interpreted as extremely threatening [35].

In patients with chronic painful TMD, like in other musculoskeletal disorders with chronic pain, a greater intensity of such psychological factors as anxiety, depression, and catastrophizing, is observed more frequently than in healthy subjects [16].

It is worth noting that, according to some authors, depression plays a more important role in painful TMD than anxiety [36], while catastrophizing may increase the risk of pain chronification both in TMD patients and in people suffering from other musculoskeletal disorders [17,30]. Despite considerable progress made in recent years in the field of knowledge on factors leading to chronification of painful TMD, the precise role of those factors is not sufficiently known.

The influence of emotional states on the development of painful TMD is probably multidirectional. Various mechanisms are taken into account, among others: intensification of parafunctional activity, influence on the endocrine system, activation of the sympathetic nervous system, and influence of neurophysiological processes related to the experience of pain [30,37]. Catastrophizing, anxiety, and other negative affects are connected to a decreased effectiveness in descending pain-inhibitory (antinociceptive) system [30]. In addition, a connection between catastrophizing and the experienced pain intensity was demonstrated. The connection may be reciprocal: on the one hand, intensification of pain causes increased catastrophizing, on the other hand, intensified catastrophizing leads to an increase in the experienced pain intensity. Kjøgs *et al*. [38] showed that experimental increasing or decreasing of pain catastrophizing affects the intensity of experienced pain. It is interesting that intensification of depression, contrary to catastrophizing, is associated with an increased pain perception threshold [39]. In this connection it should be noted that catastrophizing shows a unique, independent from other factors, influence on pain-related phenomena [30].

Analyzing the mechanisms that may explain the connection between psychological factors and pain chronification, it is necessary to consider also role of such factors in behavioral responses that maintain pain and lead to motor dysfunctions in chronic patients. The fear of pain may bring short-term benefits associated with a decrease in the anxiety caused by noxious stimuli. However, if pain-related fear persists for a long time, it may cause non-adaptive responses: increased anxiety, limited activity, and other physical and psychological consequences [29]. An excessive, irrational and debilitating fear of physical movement and activity resulting from a feeling of vulnerability due to painful injury or reinjury is defined as kinesiophobia [40].

Fear-avoidance model

One of the main concepts related to the connection between fear of pain and the onset of chronic musculoskeletal pain is the fear-avoidance model. It was proposed to explain the mechanism of transformation of acute lower back pain into chronic pain and has become the main theory explaining the development of functional

disabilities related to musculoskeletal disorders [41,42]. The fear-avoidance model posits that fear of pain recurrence or intensification leads to behaviors helping to avoid movement and activity. Such behaviors are usually beneficial in the case of disorders associated with acute pain – unloading of the injured body part allows healing. Long-term avoidance of movement, however, may have negative consequences that result from, among others, the effect of limited activity on daily functioning. Being incapable of normal functioning brings negative psychological effects, such as decreased mood or even depression due to a feeling of handicap and inability to fulfill social roles. Those psychological responses are often conducive to an increase in pain-related fear, maintaining and aggravating the problem [42]. It is worth noting that catastrophizing may precede fear of movement [35]. Therefore, co-occurrence of catastrophizing and the related psychological factors, especially anxiety and depression, plays an important role in the fear-avoidance model. Those factors contribute above all to an increase in the intensity of pain-related fear.

The fear-avoidance model gained a considerable popularity, unlike any other psychological models conceptualizing pain. This is probably due to its simplicity, conceptual clarity, and clinical significance [41]. An unquestionable advantage of the fear-avoidance model is combining psychological factors with biological ones. Thanks to the explanation the model provided of how psychological factors affect physiological or pathophysiological processes that maintain pain disorders, it is possible to eliminate a still (unfortunately) frequent interpretation of psychological factors as associated with imaginary pain. In recent years a lot of attention was given to the role of fear in the onset, development and maintenance of pain. Numerous studies investigated these problems, especially in the context of lower back pain [42]. It is worth observing that patients with chronic painful TMD and chronic lower back pain show numerous similarities concerning the psychological and behavioral factors. Despite those similarities between lower back pain and painful TMD (in particular myofascial pain), the role of fear of movement and pain in the onset of TMD has not aroused an equal interest.

The connection between pain and muscular activity

Conceptualizations concerning the connection between musculoskeletal pain and motor activity are not limited to the fear-avoidance model. It is believed that reorganization of motor control in response to pain, manifested in a change in muscle activity, may play a significant role in musculoskeletal disorders, including the transition from acute to chronic pain [43-46]. The role of muscular activity in this area has interested researchers for a long time and the theories of an interaction between pain and muscular activity have considerably changed over recent years.

The vicious cycle theory

One of the first conceptualizations designed to explain the effect of pain on muscular activity was the vicious cycle theory (also known as the pain-spasm-pain theory). The theory attempted to explain the mechanism of the onset and persistence

of chronic muscular pain, basing on the assumption that pain initiated by a factor of any kind causes a reflexive increase in muscular activity. When the muscular activity increase is sufficiently high and lasts for a sufficiently long time, it may lead to muscle fatigue and to the development of a new source of pain that maintains the cycle [3].

The vicious cycle theory was readily accepted by the medical community, including dental specialists, and adopted as a basis for treatment. In dentistry, irreversible occlusal corrections were performed, as malocclusion was believed the main structural disorder leading to muscle hyperactivity that initiates myalgia [3]. The theory was believed valid as the therapies based on its premises proved highly effective. Such evidence, however, cannot be considered in terms of scientific proof unequivocally confirming the truth of the cited theory [47].

The vicious cycle theory was eventually undermined by results of numerous studies [48]. Despite the lack of confirmation by scientific research, the theory remains popular among clinical practitioners, probably owing to its simplicity. Also, some symptoms that accompany musculoskeletal disorders may be falsely interpreted, suggesting validity of the vicious circle theory. In clinical examination painful muscles often present higher tonus which is interpreted as an increase in activity. This interpretation is erroneous, as muscle tonus is primarily affected by, apart from contractility, the viscoelastic characteristics of muscle tissue [46].

Pain adaptation model

Lund *et al*. [48] proposed a model alternative to the vicious cycle theory, suggesting that pain is not associated with muscle hyperactivity, but, on the contrary, to a decrease in activity levels. The authors supported their claim with the results of studies on, among others, painful TMD, lower back pain, postexercise muscle soreness, and fibromyalgia. Rejecting the premises of the vicious cycle theory, they proposed a new conceptualization in which pain leads to changes in muscle activity that aim at unloading and protection of the injured body part to support healing. This theory was named pain adaptation model. The findings of Lund's team show that motor response to pain is based mainly on an increase in antagonist activity and a decrease in agonist activity. Its mechanism is that of inhibition and facilitation of motor neuron transmission at the level of spinal cord or brainstem. They called the muscles lengthened during movement the antagonists, while the muscles that were shortened – the agonists. Also, in contradistinction to previous assumptions, the authors assumed that muscle activity may be affected not only by pain originating from the structures of the motor system, but also from the skin, mucous membranes, and teeth [48].

The researchers also proposed that the activity change should be influenced by nociception from structures other than muscles, which was a novelty compared to the vicious cycle theory.

The pain adaptation model was quickly accepted by the scientific community as a considerable part of findings, especially those concerning the activity at the submaximum and maximum level, was consistent with the premises of the model [44]. Some results, however, e.g. those related to a change in muscle activity in patients

with lower back pain, did not agree with the premises of the pain adaptation model [49]. It was also noted that the described model does not explain all the observed changes that occur at the individual level [44,49].

Integrated pain adaptation model

Because of discrepancies between the presuppositions of the pain adaptation model and research results, Murray and Peck [44] proposed to expand the model developed by Lund *et al*. [48]. The new model designed to explain the interaction between orofacial pain and jaw muscle activity was named the integrated pain adaptation model. The main assumption of this model is the unique, individual character of motor response to pain. The uniqueness of the response is due to, among others, the fact that, in order to minimize provoking further pain, a new motor strategy of muscle activation is generated to enable performance of a given motor task. Therefore, a change in activity depends on a clinical problem, a motor task to be performed and an organization of the sensorimotor system of a given patient. In addition, as the way pain is experienced is individual, owing to the interaction of biological, psychological and social factors, the new strategy also depends on the mentioned factors [44].

It should be observed that reorganization of the motor control in response to pain involves various levels of the nervous system: the brainstem and the cortex. The change in motor activity controlled by the brainstem is mainly of reflexive character, whereas, thanks to the involvement of higher structures of the nervous system, such as the cortex, an influence of psychological factors on the change in muscle activity in response to pain is also possible [44,48,50].

Hodges' and Tucker's theory

The theory proposed by Hodges and Tucker [45] is also an extension of the model developed by Lund *et al.* [48] and assumes that a change in muscle activity induced by pain aims to protect from pain and further injury. The authors also suggest that functional reorganization of the motor system may lead to short-term positive results, but prove harmful in the long term. Hodges and Tucker [45], like Murray and Peck [44], allow for a possibility of change in a pathological activity that does not lead to healing but supports existing problems or causes new ones. For instance, a change in muscle recruitment may favor the use of muscle groups not adapted to a given motor task. This, in turn, may lead to muscle overload, initiating pain [44]. This is, in a way, a confirmation of the existence of a vicious cycle in musculoskeletal pain disorders, but the mechanism of the cycle is different that it was originally postulated.

It should be noted that an analysis of the existing conceptualizations of motor responses to pain shows that their evolution is similar to that of the theories of pain experience – from the biomedical model to the biopsychosocial one. Mechanistic theories, based merely on changes in neurons excitability, could not fully account for the variability in the observed motor response to pain. Those theories were thus

replaced by more complex models that included the role of psychosocial factors contributing to the individual character of pain experience.

Conclusion

A thorough knowledge of the connection between psychological factors and a change in muscle activity in response to pain may significantly help to understand the course of musculoskeletal disorders associated with chronic pain. This is due to, above all, the individual, dependent on psychological factors, character of reaction to pain and to a postulated possibility of activity change that may support pain or lead to the development of new disorders.

References

1. Greene CS. Managing the care of patients with temporomandibular disorders. *J Am Dent Assoc*. 2010;141(9):1086-8.
2. Peck CC, Goulet J-P, Lobbezoo F, et al. Expanding the taxonomy of the diagnostic criteria for temporomandibular disorders (DC/TMD). *J Oral Rehabil*. 2014;41(1):2-23.
3. Sessle BJ, Lavigne GJ, Lund JP, Dubner R (eds). *Orofacial Pain: From Basic science to clinical management*, 2nd ed. 2009. Available from: http://www.quintpub.com/display_detail.php3?psku=B4580#.V98POKPURE8
4. Dworkin SF, LeResche L. Research diagnostic criteria for temporomandibular disorders: review, criteria, examinations and specifications, critique. *J Craniomandib Disord Facial Oral Pain*. 1992;6(4):301-55.
5. 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 Group. *J Oral Facial Pain Headache*. 2014;28(1):6-27.
6. Durham J, Wassell R. Recent advancements in temporomandibular disorders (TMDs). *Rev Pain*. 2011;5(1):18-25.
7. Manfredini D, Guarda-Nardini L, Winocur E, et al. Research diagnostic criteria for temporomandibular disorders: a systematic review of axis I epidemiologic findings. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2011;112(4):453-62.
8. Guarda-Nardini L, Piccotti F, Mogno G, et al. Age-related differences in temporomandibular disorder diagnoses. *Cranio J Craniomandib Pract.* 2012;30(2):103-9.
9. Manfredini D, Piccotti F, Ferronato G, Guarda-Nardini L. Age peaks of different RDC/TMD diagnoses in a patient population. *J Dent.* 2010;38(5):392-9.
10. de Leeuw R, Klasser GD. *Orofacial Pain: Guidelines for Assessment, Diagnosis, and Management*, 5th ed. 2013. Available from: http://www.quintpub.com/display_detail.php3?psku=B6102#.VpGgpTYnofA
11. Dworkin SF. Temporomandibular disorder (TMD) pain-related disability found related to depression, nonspecific physical symptoms, and pain duration at 3 international sites. *J Evid Based Dent Pract*. 2011;11(3):143-4.

12. Maixner W, Diatchenko L, Dubner R, et al. Orofacial pain prospective evaluation and risk assessment study – The OPPERA study. *J Pain Off J Am Pain Soc*. 2011;12(11 suppl): T4-T11.e2.
13. Adler RH. Engel's biopsychosocial model is still relevant today. *J Psychosom Res*. 2009; 67(6):607-11.
14. Mehta NR. *Head, face, and neck pain: science, evaluation, and management*. Hoboken, NJ: Wiley-Blackwell; 2009. p. 722.
15. Sharav Y, Benoliel R. *Orofacial Pain and Headache*, 2nd ed. Chicago: Quintessence Pub Co; 2015. p. 664.
16. Fillingim RB, Ohrbach R, Greenspan JD, et al. Potential psychosocial risk factors for chronic TMD: descriptive data and empirically identified domains from the OPPERA case control study. *J Pain Off J Am Pain Soc*. 2011;12(11 Suppl):T46-T60.
17. Fillingim RB, Ohrbach R, Greenspan JD, et al. Psychological factors associated with development of TMD: the OPPERA prospective cohort study. *J Pain Off J Am Pain Soc*. 2013;14(12 suppl):T75-T90.
18. Greenspan JD, Slade GD, Bair E, et al. Pain sensitivity risk factors for chronic TMD: Descriptive data and empirically identified domains from the OPPERA case control study. *J Pain Off J Am Pain Soc*. 2011;12(11 suppl):T61-T74.
19. Slade GD, Bair E, Greenspan JD, et al. Signs and symptoms of first-onset TMD and sociodemographic predictors of its development: The OPPERA prospective cohort study. *J Pain Off J Am Pain Soc*. 2013;14(12 suppl):T20-T32.e3.
20. Slade GD, Fillingim RB, Sanders AE, et al. Summary of findings from the OPPERA prospective cohort study of incidence of first-onset temporomandibular disorders: Implications and future directions. *J Pain Off J Am Pain Soc*. 2013;14 (12 suppl):T116-T124.
21. Smith SB, Mir E, Bair E, et al. Genetic variants associated with development of TMD and its intermediate phenotypes: the genetic architecture of TMD in the OPPERA prospective cohort study. *J Pain Off J Am Pain Soc*. 2013;14(12 suppl):T91-T101.e3.
22. Slade GD, Ohrbach R, Greenspan JD, et al. Painful temporomandibular disorder decade of discovery from OPPERA studies. *J Dent Res*. 2016;95(10):1084-92.
23. 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. *J Orofac Pain*. 2003;17(1):9-20.
24. Munzenmaier DH, Wilentz J, Cowley AW. Genetic, epigenetic, and mechanistic studies of temporomandibular disorders and overlapping pain conditions. *Mol Pain*. 2014;10:72.
25. Woolf CJ. Central sensitization: Implications for the diagnosis and treatment of pain. *Pain*. 2011;152(3 suppl):S2-S15.
26. Woolf CJ. Central sensitization: Uncovering the relation between pain and plasticity. *Anesthesiology*. 2007;106(4):864-7.
27. 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 Off J Am Pain Soc*. 2011;12 (11 suppl):T27-T45.
28. Okeson JP. *Orofacial Pain: Guidelines for Assessment, Diagnosis, and Management*. 1st ed. Chicago: Quintessence Pub Co; 1996. p. 285.
29. Turk DC, Wilson HD. Fear of pain as a prognostic factor in chronic pain: Conceptual models, assessment, and treatment implications. *Curr Pain Headache Rep*. 2010;14(2): 88-95.
30. Edwards RR, Dworkin RH, Sullivan MD, et al. The role of psychosocial processes in the development and maintenance of chronic pain. *J Pain*. 2016;17(9):T70-T92.
31. Mielimąka M, Rutkowski K, Cyranka K, et al. Trait and state anxiety in patients treated with intensive short-term group psychotherapy for neurotic and personality disorders. *Psychiatr Pol*. 2015;36. Available from: http://www.psychiatriapolska.pl/online–first–nr36.html.
32. *International Statistical Classification of Diseases and Related Health Problems*. World Health Organization. 2004. p. 824.
33. Leung L. Pain Catastrophizing: An updated review. *Indian J Psychol Med*. 2012;34(3): 204-17.
34. Quartana PJ, Campbell CM, Edwards RR. Pain catastrophizing: a critical review. *Expert Rev Neurother.* 2009;9(5):745-58.
35. Leeuw M, Goossens MEJB, Linton SJ, et al. The fear-avoidance model of musculoskeletal pain: Current state of scientific evidence. *J Behav Med.* 2006;30(1):77-94.
36. Reiter S, Emodi-Perlman A, Goldsmith C, et al. Comorbidity between depression and anxiety in patients with temporomandibular disorders according to the research diagnostic criteria for temporomandibular disorders. *J Oral Facial Pain Headache.* 2015;29(2):135-43.
37. Cairns BE. Pathophysiology of TMD pain – basic mechanisms and their implications for pharmacotherapy. *J Oral Rehabil.* 2010;37(6):391-410.
38. Kjøgx H, Kasch H, Zachariae R, et al. Experimental manipulations of pain catastrophizing influence pain levels in patients with chronic pain and healthy volunteers. *Pain*. 2016;157(6):1287-96.
39. Dickens C, McGowan L, Dale S. Impact of depression on experimental pain perception: a systematic review of the literature with meta-analysis. *Psychosom Med*. 2003; 65(3):369-75.
40. Larsson C, Ekvall Hansson E, Sundquist K, Jakobsson U. Kinesiophobia and its relation to pain characteristics and cognitive affective variables in older adults with chronic pain. *BMC Geriatr*. 2016;16. Available from: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC4936054/
41. Crombez G, Eccleston C, Van Damme S, et al. Fear-avoidance model of chronic pain: The next generation. *Clin J Pain*. 2012;28(6):475-83.
42. Zale EL, Ditre JW. Pain-related fear, disability, and the fear-avoidance model of chronic pain. *Curr Opin Psychol*. 2015;5:24-30.
43. Graven-Nielsen T, Arendt-Nielsen L. Impact of clinical and experimental pain on muscle strength and activity. *Curr Rheumatol Rep*. 2008;10(6):475-81.

44. Murray GM, Peck CC. Orofacial pain and jaw muscle activity: a new model. *J Orofac Pain*. 2007;21(4):263-78; discussion 279-88.
45. Hodges PW, Tucker K. Moving differently in pain: A new theory to explain the adaptation to pain. *Pain*. 2011;152(suppl):S90-S98.
46. Mense S, Gerwin RD. *Muscle pain: Understanding the mechanisms* [Internet]. Berlin, Heidelberg: Springer Berlin Heidelberg. 2010. Available from: http://link.springer.com/10.1007/ 978-3-540-85021-2
47. Stohler CS. Craniofacial pain and motor function: Pathogenesis, clinical correlates, and implications. *Crit Rev Oral Biol Med*. 1999;10(4):504-18.
48. Lund JP, Donga R, Widmer CG, Stohler CS. The pain-adaptation model: a discussion of the relationship between chronic musculoskeletal pain and motor activity. *Can J Physiol Pharmacol.* 1991;69(5):683-94.
49. van Dieën JH, Selen LPJ, Cholewicki J. Trunk muscle activation in low-back pain patients, an analysis of the literature. *J Electromyogr Kinesiol*. 2003;13(4):333-51
50. Hodges PW, Ervilha UF, Graven-Nielsen T. Changes in motor unit firing rate in synergist muscles cannot explain the maintenance of force during constant force painful contractions. *J Pain*. 2008;9(12):1169-74.

Corresponding author:
Jolanta Szymańska
Department of Integrated Paediatric Dentistry
Medical University of Lublin
Lubartowska St. 58, 20-094 Lublin
e-mail: szymanska.lublin@gmail.coml