Recent Reviews on the Influence of Orthodontic Treatment on the Appearance of Temporo-Mandibular Joints Dysfunction

Aktualne poglądy na temat wpływu leczenia ortodontycznego na występowanie dysfunkcji stawów skroniowo-żuchwowych

Abstract

The article is a reply to the increasingly frequent attempts to blame orthodontists for causing dysfunction of the temporomandibular joints (TMJ) as an iatrogenic consequence of orthodontic treatment. The presented review of literature is not only a careful analysis of orthodontic treatment as a potential risk factor favoring dysfunction. Articles available in the literature also allowed authors to draw conclusions about specific functional appliances and their effect on the abnormal function of the temporomandibular joints. As is clear from the papers published in the last decade, there is no scientific evidence supporting any association of either the correction of malocclusions itself, as well as the specific orthodontic technique used in this treatment approach with impaired TMJ function. The thesis quoted after Seligman and Okeson allow orthodontists to solidify the view that the careful treatment of malocclusion, even if it does not lead to an ideal occlusion (in accordance with the gnathologist’s concept), helps to meet the aesthetic requirements of patients. Exposure of the TMJ to dysfunctions – in the absence of the evidence such as meta-analyses – cannot support the speculation on this very controversial subject, especially in the claim cases (Dent. Med. Probl. 2012, 49, 3, 427–432).

Key words: orthodontics, temporo-mandibular joints dysfunction, dysfunctions.

Streszczenie


Słowa kluczowe: leczenie ortodontyczne, stawy skroniowo-żuchwowe, dysfunkcje.

Exact pathophysiology and etiology of craniofacial pain are so unknown that causal treatment is not possible [1]. In the etiology of the temporo-mandibular joint dysfunction (TMD), genetics, stress and psychological factors, as well as the patient’s sex, the growth stage, untreated malocclu-
sion or unstable occlusion after orthodontic treatment, are considered [2].

Slade et al. [3], according to the Stohler’s conception, did not focus on malocclusion, but rather concentrated on the weakness of the genes. In this way they were able to isolate patients who underwent orthodontic treatment in the past and showed disturbed pain reply. In those patients, the researchers identified a genetic code, namely the allele responsible for the synthesis of the catechol-O-methyl transference, in this way finding the group of patients liable to TMD. The authors concluded with an interesting finding: orthodontic treatment as the major factor of the TMD was excluded. They found, in turn, that the unusual pain appearing during orthodontic treatment is a way of identifying the gene and thus is helpful in revealing TMD susceptibility. It should not be forgotten that, despite Slade’s et al. studies published in 2002, already in 1980s Wigdorowicz-Makowerowa [4] reported increased catecholamines and 17-hydroxy steroids in the urine of patients with TMD.

Ramfjord [5] emphasizes the value of nervous and psychiatric disorders correlated to TMD; they define bruxism (or teeth grinding) as protection during psychological tension or stress. According to Klasser et al. [6] this theory is outdated, since in the last 20 years it was proved that those malfunctions appear usually at night and are nothing else but a kind of para-insomnia.

As for gender and growth stages, it has been proven that the risk of TMD is more common in women and increases with age. It is often correlated with puberty. This is a period of dramatic physical and psychological changes and a time when orthodontic treatment is often undertaken. Considering the duration of orthodontic treatment is approximately 2 years, TMD disorder may appear during this period or immediately after. It is no wonder that speculation regarding the influence of orthodontic treatment on TMD prevalence occurred. Forasmuch the disorder has a wide and incompletely discovered etiology, it encourages court cases: considering orthodontic treatment to cause these problems spurring patients to seek compensation.

To provide the evidence supporting or refuting the hypothesis of a relation between orthodontic treatment and TMD, scientific research had been started. It has been found that TMD appears to be related to patients with increased vertical facial dimension and skeletal class II malocclusion [7]. Forward displacement of the articular disc in these patients was demonstrated using magnetic resonance – however, there was no relation between the position of the disc and the occurrence of TMD. Wyatt [8] believes that a similar mechanism accompanies camouflaging treatment of skeletal class II. After upper premolar extractions the mandible can be locked in its distal position. Subsequently, the overloaded disc displaces mesially, whereas the condylar process moves distally. However, since Wyatt brings no scientific evidence supporting this concept, Luther [7] considers it to be merely private speculations.

Kim et al. [9] presented the results of 31 studies in a systematic review, however no firm conclusion regarding positive or negative influences of orthodontic treatment to temporomandibular joint (TMJ) was found. The authors emphasized that their meta-analysis was limited by the shortage of coherent and reliable diagnosis criteria. The longitudinal and comparative studies of two groups – treated and control ones – meeting the criteria of epidemiological research and permitting for the evaluation of the existing feedback relations, were found only in 8 articles. Unfortunately, different symptoms to identify TMD were used in 6 of them. The remaining 2 were based on dysfunction indicators instead of the descriptive analysis indispensable for TMD risk estimate. No unequivocal influence of the occlusion and orthodontic treatment to TMD prevalence was proven, allowing the conclusion that if such correlations do exist they would become obvious due to coincidence.

It has been, however, emphasized that a lack of evidence should not be understood as a lack of the relation of TMD to orthodontic treatment [10]. Despite the fact that the role of the bite or its pathology has never been conceded as the direct factor responsible for TMD development [7], there is a dispute in literature regarding the influence of malocclusion and orthodontic intervention to TMD.

Mohlin et al. [2] reviewed literature from 1966 to 2003 looking for relations between a particular type of the malocclusion and TMD prevalence. The authors found no significant differences as for changes in TMJs of patients with and without malocclusion. However, individuals with untreated cross-bite, crowding or large overjet showed a higher prevalence of signs and symptoms of TMD [11–14]. Woźniak [15] observed similar relationships in the scissors bite as well. Ergo, no consensus had been found regarding the influence of deep bite to TMD etiology in literature. According to Japanese researchers [16], the disorder could be triggered as a result of posterior position of a condyle of the mandible; it had not, however, been proven by the others [12, 17–19].

Controversy, regarding TMD development arouses also canine guidance, in particular – op-
posed teeth contacts on balancing side that are according to Solberg [20] responsible for clenching and grinding. Those premature contacts may appear spontaneously, but also as a result of orthodontic treatment. For this reason, according to Roth the occlusion should be checked precisely at the end of the treatment using a positioner [21]. He et al. [22] attempted at finding any correlation between the discrepancy of the centric relation (CR) and maximal intercuspidation (MI) and TMD. They proved more frequent occurrence of TMD in patients who have the discrepancy between CR and MI: mesio-distal and lateral exceeding 1 mm and 0.5 mm respectively. The authors also proved that increase in this discrepancy significantly influences the symptoms intensity. Nevertheless, they rejected the influence of psychical and traumatic factors as the etiological factors, since no intensified susceptibility to depression, stress, joints inflammation or trauma was found in patients showing symptoms of TMD. Roth [21], Williamson [23] and Cordray [24] confirmed that orthodontic treatment should lead to a functional intercuspidation achieved after the closing the mandible, with no shift of a condyle on the way from CR to MI. This means that no premature contacts are allowed during mandible closure. If there is any, disharmony between CR and MI develops; therefore, the lateral pterygoid muscle is being provoked to contract, and the balance between adductors and abductors muscles is disturbed. It further provokes the contraction of the masseters and pain. A prolongation of muscular hypertonia may theoretically be the reason for TMD. Roth [21], Pullinger [25] and Seligman [26] proved no correlation between an occlusion and TMD; moreover, they affirmed that occlusal factors seemed to be a rarity and only in isolated patients presenting symptoms of TMD. Additionally, they proved that a discrepancy between a CR and MI might be the causal factor of TMD only when it exceeds 5 mm (the norm is 1 mm) due to large possibilities of the joints adaptation.

Hirsch [10], while studying a group of children and teenagers, not only determined an increased risk of dysfunction after orthodontic treatment, but almost noticed a significant reduction in bruxism after treatment of malocclusion.

In the absence of clear evidence supporting any influence of orthodontic treatment generally to development or increase of TMD symptoms, orthodontic appliances were being studied in more detail. Findings turned out to be confusing again. Researching an influence of treatment method to TMJs, significant changes had been observed after a Herbst appliance and facemask therapy. According to Pancherz [27] there are 3 mechanisms responsible for forward movement of a mandible after use of a Herbst appliance: 1) increased condyle growth due to a remodeling of the joint, 2) movement of the fossa downward and forward, 3) forward movement of a condyle in the fossa. All mechanisms come into being during adolescence, when growth potency exists. They are possible due to the 24-hour activity of the Herbst appliance forcing the mandible forward. However, according to some authors, this can be the reason for inflammation of tissue lying distal to the condyle due to tension forces. The inflammation causes a decrease in synovial fluid viscosity leading to a decreased slide in the upper parts of the joints – which can be the reason for TMD in some patients [28, 29]. Moreover, a functional treatment – especially class II division 1 is associated with the constrained position of the disc, and is a threat for patients that have had TMD diagnosed [30–32]. Naturally, Pancherz and Ruf [33] argue with this claim and consider the forward allocation of the disc as a temporary phenomena. They proved it examining 15 patients and finding, in some cases, a more backward position of the disc than initially. The reason is still unknown, although a remodeling of condyle and glenoid fossa has been considered. What is, therefore, important, although distal displacement of the disc after orthodontic treatment is minor and, according to the authors, within physiological range; however, application of the Herbst appliance in patients with unidentified posterior allocation of a disc can increase or engender TMD symptoms. In long term retrospective studies Pancherz et al. [27] observed moderate to severe TMD symptoms in 25% patients treated with the Herbst appliance. The severity of symptoms was dependant on a range of allocation of the disc. Taking into account that the TMJ condition had not been evaluated before the Herbst appliance was used, it is reliable that an asymptomatic TMD could exist before the treatment was started, and the appearance of symptoms was not correlated to the presence of the appliance. This information acknowledges the importance of a detailed TMJ examination before functional treatment is started – clinically – using a magnetic resonance or an articulator.

Harrison [34] suggested that not only functional treatment of class II affects TMD development. He deems all the mechanisms, leading to the retropositioning of the mandible in class III treatment, also allocate the disc, thus possibly leading to TMD development. It seems to be quite reasonable due to the fact that forces used in facemask therapy are of great magnitude – 700–800 grams. 70–75% of applied forces load the joints indirect-
ly, via the chin. Overloaded TMJ are thus exposed to the risk of dysfunction. El and Cigerb [35] compared effects of Delaire and Grummons facemasks where the latter sets on the zygomatic arches instead of the chin. They proved that the usage of the Grummons facemask permits proper mandibular growth and does not create any pressure to the TMJ disc. Moreover, authors observed a greater discrepancy between CR and MI after the treatment with Delaire facemask compared to a Grummons one. It may be concluded that – as in developing mandibular growth – patients with class III liable to TMD development should rather be treated with a Grummons facemask.

Greene [36] as contrasted with Slavicek [37] analyzed an occlusion after orthodontic treatment and proved that the TMJ is remarkably resilient and capable of putting up with orthodontic forces. Greene, however, recommends to relinquish functional treatment in adults; he also believes that most condylar positions, obtained after good orthodontic treatment concluding with biological balance, will not favor TMD development, unless the case is finished in a mandibular protrusion. What is determined nonspecifically as the “biological balance” by Green, is namely specified by Okeson [38] listing the following conditions: 1) In patients sitting upright the condyles should be in their most superior anterior positions, with the discs properly interposed; all lateral teeth should have even and simultaneous contact, heavier than the front teeth; 2) During the lateral movement of the mandible, adequate canine guidance must exist, no contacts of the opposing teeth on the balancing side; 3) In protrusive mandibular position, the opposing lateral teeth must have no contacts.

Presented consideration on possible relation between TMD development and orthodontic treatment can be perhaps best summed up by quoting Seligman and Okeson arguments [26, 38]: 1) signs and symptoms of TMD occur in healthy individuals, 2) signs and symptoms of TMD increase with age, particularly during adolescence; therefore, orthodontic treatment during pubertal spurt may not be related to TMD, 3) orthodontic treatment performed during adolescence generally does not increase or decrease the chances of developing TMD later in life, 4) the extraction of teeth as part of orthodontic treatment plan does not increase the risk of developing TMD, 5) there is no higher risk for TMD associated with any particular type of orthodontic mechanics, 6) although a stable occlusion is a reasonable orthodontic treatment goal, achieving no ideal – from the perspective of gnathologists – occlusion does not result in TMD signs and symptoms, 7) no method of TMD prevention has been demonstrated, 8) when more severe TMD signs and symptoms are present, simple treatments can alleviate them in most patients.

Conclusion

In the face of increasingly frequent attempts to accuse orthodontists of TMD development following the treatment of malocclusion, evidence based arguments seem to be essential not only for dentists, but for their patients as well. The presented review of the recent literature apparently proves that the discussed issue is still controversial. Nevertheless, what is of utmost importance is that so far there is no meta-analysis supporting the concept of any direct relationship between orthodontic treatment and TMD occurrence. Thus, any attempt to support, by the medical experts, the legal consequences of the controversial cases “with TMD in the background” is both premature as well as scientifically unfounded.

References:


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