

## Current Diagnosis of Temporomandibular Pathologies

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**ABSTRACT:** The current scientific knowledge of TMJ pathologies points to the importance of etiological research and the need for differential diagnosis using the most modern technological resources. Those include MRI, computed tomography, serologic studies, genetic mapping, and bioelectronic instruments which allow clinicians to study, understand, and measure respectively, the structural changes of soft and hard tissues, infections, genetic susceptibility for autoimmune diseases, and stomatognathic function. The purpose of this article is an overview of the current knowledge and related tools for the diagnosis of TMJ pathologies.

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For many years, occlusive problems were considered the only etiological factor for temporomandibular disorders. Current knowledge indicates that this may not always be the case.

Hippocrates<sup>1</sup> was the first to report a link between the loss of dental pieces and headache, **Figure 1**. Fortunately, although occlusive disorders may play a role in temporomandibular joint (TMJ) pathologies, there is no direct link between the disorders, otherwise dentistry would be deemed guilty for millions of people affected by this problem worldwide.

However, the current scientific literature contains references to other factors or noxious conditions that can produce temporomandibular joint (TMJ) pathologies,<sup>2-21</sup> a more adequate term for the disorders than the commonly used *temporomandibular dysfunction*—a term that should not be used because of its inaccurate and generic nature. Inaccurate because it does not denote the presence of a specific pathology, but rather describes the existence of an altered functioning, and generic because it includes a vast number of pathologies that may take place in the temporal fossa. A clear example of the inadequacy of this term is the inclusion within this syndrome of facial pain, an undoubtedly neurological entity. In the scientific literature, traumatic, bacterial, autoimmune, and occlusal causes are mentioned as etiological factors of this pathology.<sup>2</sup>



**Figure 1**  
Maneuver proposed by Hypocrates to reduce mandibular luxations. (Florence, Medicea-Laurenziana Library).

**Traumatic Causes**

The literature contains hundreds of references to lesions that can occur in the TMJ as a consequence of trauma.<sup>2-8,10</sup> Such traumatic events can be direct, when applied on the TMJ itself or on different parts of the mandible. Alternatively, traumatic events can be indirect, when they produce a sudden displacement of the mandible, resulting in articular lesions. A classical example of this type of displacement is that produced by automobile accidents, i.e., backing into a parked vehicle or being in a car that rolls over. Such traumas can result in damage or lesion to hard tissues, soft tissues, or both.<sup>9</sup> Soft tissue lesions can involve the ligaments, the disk or both.

*Articular Disk Lesions*

As a consequence of trauma, the articular disk can suffer a displacement, a perforation or a burst. Here, the reference to a displaced disk is when the lesion results in a temporary or permanent displacement, which can be associated or not to a ligament lesion (Figures 2, 3).

The term *perforated disk* is used for trauma resulting in a disk lesion that allows a connection between the glenoid and condyle compartments. Such connection can be due to distension or rupture of collagen fibers that constitute the articular disk. Finally, the term *disk burst* is used to denote that a disk has been broken into two or more pieces.



**Figure 2**  
Temporomandibular joint showing a perforation in the articular disk.



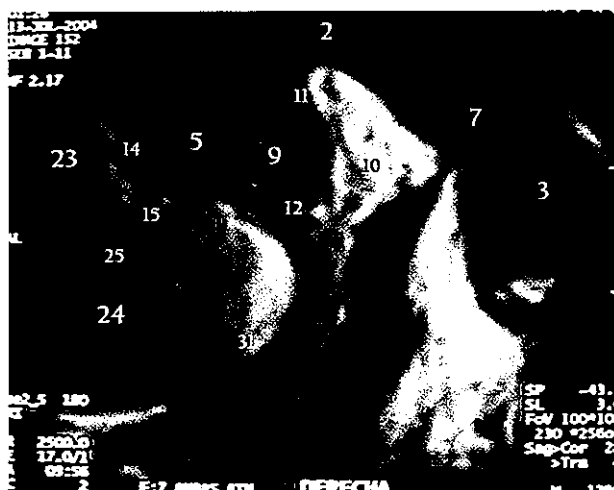
**Figure 3**  
MRI of a temporomandibular joint showing a burst articular disk. Note that the mandible head presents an abnormal morphology.

*Ligament Lesions*

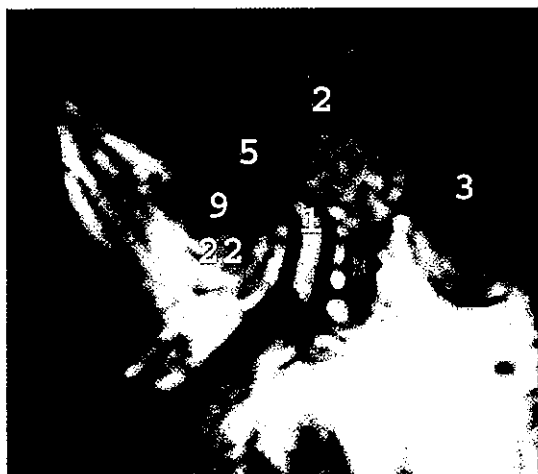
Ligaments are responsible for the displacements and the repositioning of the articular disk. While the anterior luxation of the articular disk has been considered for years, diskal displacements are produced, as in other joints, by ligament disorders and constitute the only cause of its repositioning. These ligaments can suffer distension and partial or total amputation as a consequence of trauma. As aforementioned, this lesion will lead to a secondary displacement of the articular disk.

*Hard Tissue Lesions*

Lesions that involve the mandible head, the mandibular neck, or both (Figures 4, 5).<sup>7-8</sup>



**Figure 4**  
MRI of a TMJ with post-traumatic deformation of the mandible head (a deflection of the vertical axis) with a healthy disk and ligaments.



**Figure 5**  
MRI of a TMJ in maximal opening and showing a post-traumatic sequel in the mandible head and ligament lesion with irreducible luxation of the disk. The following are the numerical references to the anatomic labels for Figures 4 and 5: 1. mandible head; 2. roof of the mandible fossa; 3. external acoustic meatus; 5. zygomatic tubercle; 7. petrotympanic fissure; 9. articular disk; 10. retrodiskal zone; 11. bilaminar ligament (upper fascia); 12. bilaminar ligament (lower fascia); 14. anterior diskal ligament (upper fascia); 15. anterior diskal ligament (lower fascia); 23. external pterygoid muscle (upper fascia); 24. external pterygoid muscle (lower fascia).

### Lesions to the Mandible Head

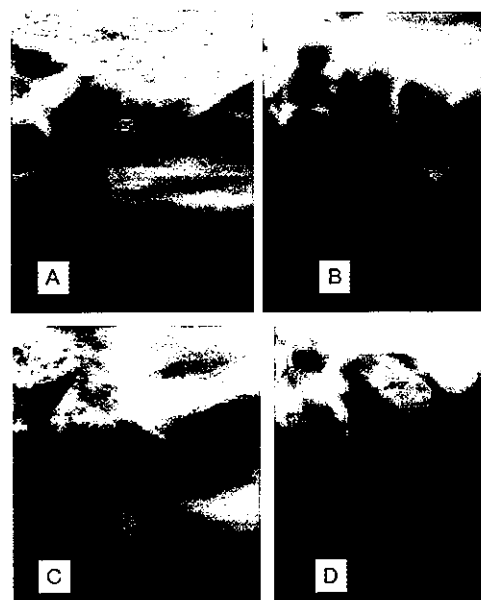
Lesions to the mandible head can be found at a high, medium, or low level and can be vertical or horizontal.<sup>2,7-10</sup> These fractures can be present even in children, leading to morphological alterations in joint structures, which in turn produce functional disorders.

### Lesions to the Mandibular Neck

Such lesions are frequent and can be homolateral or contralateral to the trauma region. Occasionally, lesions similar to greenstick fractures have been observed in children.<sup>9</sup>

### Diagnostic Procedures

The method of choice for the study of these pathologies are imaging techniques. Methods used to image hard tissue lesions differ from those used for soft tissue lesions. In the first case, current radiological techniques include different technologies ranging from simple laminography to volumetric images. The first consists of images obtained using the same panoramic instruments, which by virtue of their tomographic potential yield accurate and reliable images of structures from the temporomandibular and craniomandibular joints (as named by modern anatomy experts). Such images are very similar to those obtained years ago using linear tomography, precursor to the current computed tomography (Figure 6).<sup>10,13-15</sup> However, helical tomography (e.g., Cone beam)<sup>11,12,16</sup> and volumetric tomography (i.e., i-CAT) allow a higher resolution and a minimal image distortion, together with a lower level of radiation for the patient.<sup>16,17</sup>



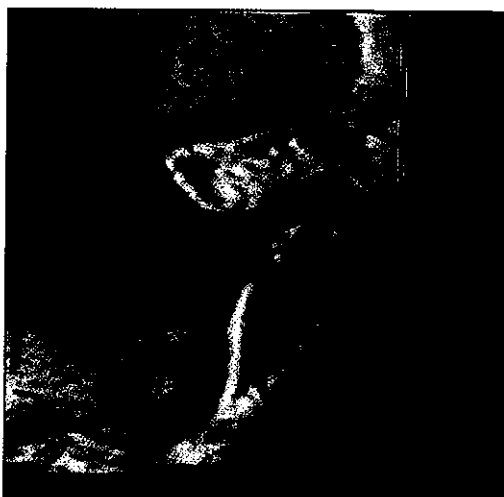
**Figure 6**  
TMJ laminographies showing different lesion types: A. healthy joint, note the spherical form of the mandible head; B. post-traumatic lesion caused by a high fracture of the condyle in early childhood; C. characteristic lesion as a sequel of beta-hemolytic streptococci infection in the anterior contour of the condyle; D. lesion caused by rheumatoid arthritis.

**Bacterial Causes**

Bacterial lesions are widely described as initial causes of arthritis and/or arthrosis in joints from every part of the body, and the TMJ is not an exception. Laskin<sup>20</sup> described a great variety of bacteria capable of producing pathology on the TMJ. In 1999, the treatment of a patient presenting with an infection by beta-hemolytic streptococci was described.<sup>20</sup> The same year, Henry<sup>21</sup> reported the presence of *Chlamydia trachomatis* in the TMJ. Currently, there are many articles describing TMJ pathologies of bacterial origin (Figures 7-9).<sup>22-23</sup> In 2003, Kim<sup>24</sup> described the presence of staphylococcus aureus,



**Figure 7**  
CT scan image with 3-D reconstruction of the TMJ in basal view. Photo courtesy of Dr. Yavich.



**Figure 8**  
Image obtained using Cone Beam CT, showing a lesion produced by beta-hemolytic streptococcus in the anterior contour of the condyle. Photo courtesy of Dr. Bedoya.



**Figure 9**  
Densitometry of the TMJ obtained using a technique from One Shot Software (wareseeker.com), showing a lesion in the posterior contour of the condyle and close to the petrotympanic fissure due to a bacterial infection from past otitis.

*mycoplasma genitalium*, *mycoplasma fermentans*, and *streptococcus mitis*, among others. All these microbes produce monoarticular or biarticular atropathies, which in many cases affect only the TMJ,<sup>25,26</sup> but in other cases represent the local expression of a systemic infection.

*Diagnostic Procedures*

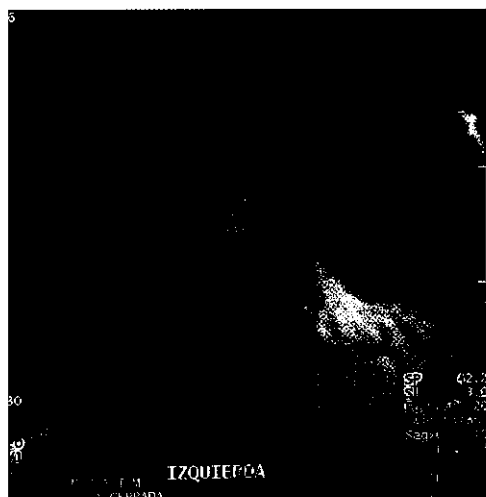
While images can provide clues to the existing pathology in these cases, laboratory analyses will confirm or rectify the presumptive diagnosis. The most common techniques are those aimed at detecting infections caused by beta-hemolytic streptococci and *Chlamydia trachomatis*.<sup>27</sup>

The most commonly used test for the detection of beta-hemolytic streptococci are ASTO and streptozyne, which in view of differing opinions regarding so-called normal values, must be performed quantitatively.

The most important tests to detect *Chlamydia trachomatis* are those based in direct immunofluorescence, (Figure 10) although some authors consider it possible to use indirect immunofluorescence on serum samples or even ELISA on urine samples.<sup>27</sup> Others prefer to use colposcopy with sampling in the cervix, although a positive result in the later two studies does not always indicate the presence of the microbe in blood.

**Autoimmune Etiology**

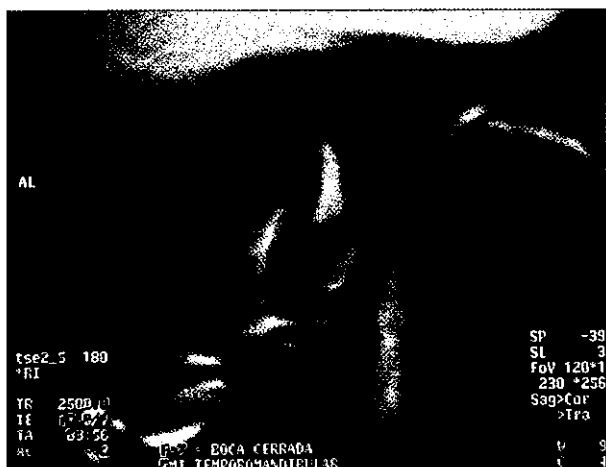
Immunology is one of the newest tools for the diagnosis of etiologic factors in TMJ pathologies or craniomandibular joint pathologies.<sup>28-34</sup>



**Figure 10**  
Pathognomonic image of chlamydia trachomatis of the mandible head.

Although genetic factors are not direct causes of these pathologies, they create unfavorable conditions in patients for clinical recovery and health restoration. As proposed by Okeson,<sup>28</sup> once the pathology has begun, it cannot be stopped if perpetrating factors are present.<sup>35</sup>

Among genetic characteristics potentially involved as perpetuating factors, the most widely known are HLA-B27<sup>37-38</sup> (also associated with ankylosing spondylitis) and those of the so-called Series II, including HLA-DR1,<sup>39-40</sup> HLA-DR4, and HLA-DR8, among others (Figures 11, 12).<sup>41</sup> Within Series II, the first factor is related to rheumatic diseases and the last two with Hashimoto's thyroiditis, with HLA-DR8 only present in patients of Asian ancestry.



**Figure 11**  
Characteristic MRI image of an autoimmune lesion.

*Diagnostic Procedures*

The diagnostic test of choice for determining the existence of autoimmune pathologies is genetic mapping analysis, known as HLA Genetic Mapping (HLA stands for human leukocyte antigen). These studies, which have allowed researchers to identify the etiological factors of several medical conditions, also have a place in dentistry.<sup>42</sup>

**Dental Causes**

Interferences have been widely described in the scientific literature, but advances in the past five years have changed certain concepts. Some of the concepts that have been dogmatic for several years are being reversed as a consequence of technological improvements.<sup>9</sup>

The most important change is perhaps the change in the concept of occlusion as a stable point. From an anatomical, histological, and physiological point of view, dental occlusion is a relation constituted by an important number of variables, biologically adaptable and pathologically modifiable. It is widely known that dental occlusion is related to the TMJ. It is also known that teeth are attached to maxillary bones by a dental-osteal joint, called the periodontium, and that pieces articulate with their antagonists through a dentodental joint known as occlusion. Therefore, the later joint results from the physiology and the pathophysiology of the TMJ, the bone, and

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**TIPIFICACION DE ANTIGENOS DE HISTOCOMPATIBILIDAD**

FECHA DEL ESTUDIO: 3/8/06

PACIENTE:

TECNICA UTILIZADA: Tipificación molecular. Amplificación enzimática, hibridación con oligonucleótidos (PCR-SSOP).

**RESULTADOS:**

APELLIDO Y NOMBRE	Parentesco	DRB1
PACIENTE	14	-

**Observaciones:**

- se solicita muestra de los padres para confirmar la homocigosis de DR 14.
- La paciente es HLA-B27 positiva.
- La muestra fue extraída por Susana Solzón

*C. Solzón*  
FIRMA  
CINTIA YANINA MARCOS  
BIOQUIMICA  
M.N. 6699

**Figure 12**  
Genetic mapping using HLA testing to show alterations in Series I (B27) and Series II (DRI-14).

the dental pieces, making it impossible to consider dental occlusion a stable entity. The elements are activated by the neuromuscular system, which has its own physiology and pathophysiology.

The usual occlusion position is the terminal point of mandibular movements, i.e., the maximal opening. Consequently, the ideal position for studying mandibular movements is the resting position. With a subject in resting position, it is possible to study occlusion in a different way and to detect the presence of pathologies that have never been observed before.<sup>43</sup>

**Incisor Guide:** A large body of literature exists regarding incisor angle guide, which is important for mandibular protrusion movements. However, the incisor angle guide can be incorrectly placed in the antero-posterior relationship, constituting an interference that leads to a distal mandibular displacement<sup>43</sup> that produces a retro-diskal compression and anterior luxation of the articular disk. Studies by Isberg<sup>10,44</sup> showed that 55% of patients with anterior luxation of the disk presented a distal position of the mandible head.

**Canine Guide:** The alteration of the canine guide produces a mandibular lateralization that can lead to either a mesial or a distal displacement of the mandible.<sup>43</sup> In the latter case, a distal displacement of the mandible can also occur (Figure 13).

**Alteration of the Freeway Space (FWS):** Freeway space (FWS) has always been considered healthy, but current knowledge indicates that this space is variable.<sup>44-45</sup> Excessive FWS can produce a distal condyle displacement, resulting in a mandibular overclosing. Therefore, the alteration of FWS can be recognized as a lack of occlusion, which is itself an occlusion disorder.

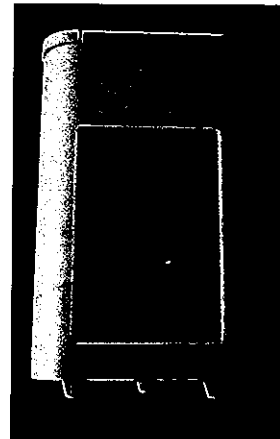
*Diagnostic Procedures*

The most modern techniques for studying occlusion are electronic mandibular deprogramming, the magnetograph, and the T-Scan.

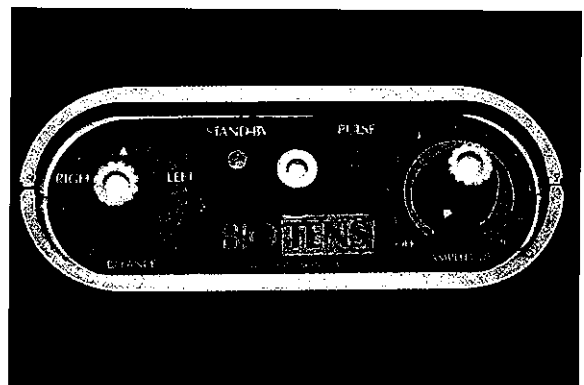


**Figure 13**  
Diagram of rest position and distal closing trajectory that in this case is necessary to reach the usual occlusion position.

**Electronic Mandibular Deprogramming (TENS):** This technique consists of determining mandibular position in a spatial position corresponding to no occlusion, which is determined by genetic muscular length (Figures 14, 15).<sup>44</sup> From this position, the mandible can perform all the movements, including occlusion.<sup>2,46</sup> Such position can be manually registered using a caliper or it can be



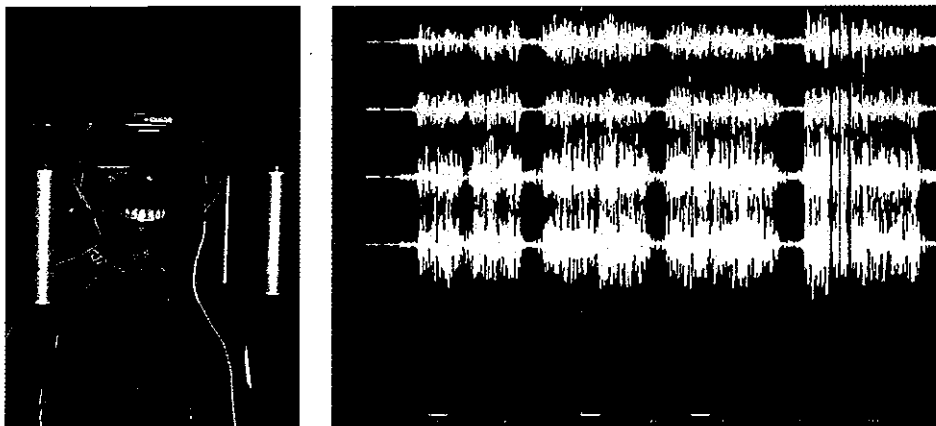
**Figure 14**  
Electronic mandibular deprogramming device (BioTENS, BioResearch Associates, Inc., Brown Deer, WI).



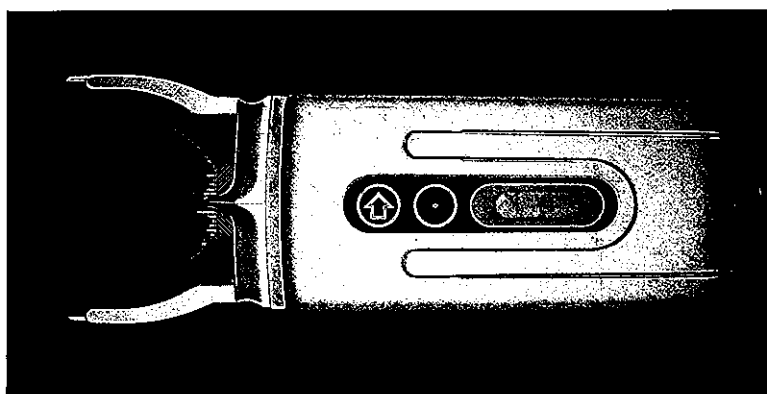
**Figure 15**  
Control panel. From right to left: pulse, balance, wait mode button, pulse intensity. (BioTENS, BioResearch Associates, Inc., Brown Deer, WI).

computationally determined using a magnetograph.<sup>47-49</sup>

**Mandibular Kinesiology:** This device consists of applying magnetic sensors to the subject's head to monitor the displacement of a magnet attached to the lower incisor area (Figure 16).<sup>42,47-49</sup> This simple device allows the registration of not only resting mandibular position, but also displacements. Masticatory movements or cycles can be analyzed which is useful for early detection of TMJ pathologies.<sup>50-70</sup>



**Figure 16**  
Patient with jaw tracker (JT-3D) and electromyography (BioEMG II) positioned. On the right, the EMG record of the patient's muscle activity during dental closing test with interposed cotton rolls. (Both products are trademarks of BioResearch, Inc., Brown Deer, WI.)



T-Scan II and dental load record in 3-D view (Tekscan, Inc., South Boston, MA).



**Figure 17**

T-Scan: The T-Scan device consists of a sensor optimized to register dental contacts with unprecedented accuracy (**Figure 17**).<sup>71-77</sup> Such devices can be synchronized with electromyographs, allowing compatible readings of dental contacts and muscular activity.<sup>74-77</sup> The use of T-Scan allows visualization and registration of the functional activity of an occlusion.

### Conclusion

Current knowledge allows a higher specificity in the diagnosis of craniomandibular pathologies. Such specificity results from scientific and technological improvements, which make a better and more accurate differential diagnosis possible, leading to more efficacious treatments.

The differential diagnosis allows for comparison of results, definition of the origin of errors, and firm covenant with our patients with regard to their results. The specificity of a diagnosis should be supported by improved

technologies, allowing a better outcome with treatments.

The inclusion of craniomandibular pathologies in the diagnosis will result in greater stability of treatments.

### References

1. Ring, ME: *Dentistry, an illustrated history*. Japan: Harry N Abrams, Inc., 1985.
2. Learreta J: *Compendio sobre patologias de la ATM*. Sao Paulo: Artes Médicas, 2003.
3. Bradley P: Injuries of the condylar region and coronoid process. In: Rowe NL, Williams JL, eds. *Maxillofacial injuries*. London: Churchill Livingstone, 1985.
4. Antoniadis K, Karakasis D, Elephtheriades J: Bifid mandibular condyle resulting from a sagittal fracture of the condylar head. *Br J Oral Maxillofac Surg* 1993; 31(2):124-126.
5. Gotte P, Fraccari F: Unilateral sagittal fracture of the head of the mandibular condyle. *Minerva Stomatol* 1980; 29(1):51-54.
6. Yamaoka M, Furusawa K, Iguchi K, Tanaka M, Okuda D: The assessment of fracture of the mandibular condyle by use of computerized tomography. Incidence of sagittal split fracture. *Br J Oral Maxillofac Surg* 1994; 32(2):77-79.
7. Wu XG, Hong M, Sun KH: Severe osteoarthritis after fracture of the mandibular condyle: a clinical and histologic study of seven patients. *J Oral Maxillofac Surg* 1994; 52(2):138-142.
8. Schimming R, Eckelt U, Kittner T: The value of coronal computer tomograms in fractures of the mandibular condylar process. *Oral Surg Oral Med*

- Oral Pathol Oral Radiol Endod* 1999; 87(5):632-639.
9. Cascone P, Leonardi R, Marino S, Carmemolla ME: Intracapsular fractures of mandibular condyle: diagnosis, treatment, and anatomical and pathological evaluations. *J Craniofac Surg* 2003; 14(2):184-191.
  10. Raustia AM, et al.: Conventional radiographic and computed tomographic findings in cases of fracture of the mandibular condylar process. *J Oral Maxillofac Surg* 1990; 48(12):1258-1262; discussion 1263-1264.
  11. Wang MH, Fang YM, Li JL, et al.: Application of MRI in indirect temporomandibular joint injury without condylar fracture. *Clin J Traumatol* 2007; 10(2):116-119.
  12. Isberg, A: *Temporomandibular joint dysfunction. A practitioner's guide*. London: Martin Dunitz, 2001.
  13. Tsiklakis K, Syriopoulos K, Stamatakis HC: Radiographic examination of the temporomandibular joint using cone beam computed tomography. *Dentomaxillofac Radiol* 2004; 33(3):196-201.
  14. Hilgers ML, Scarfe WC, Scheetz JP, Farman AG: Accuracy of linear temporomandibular joint measurements with cone beam computed tomography and digital cephalometric radiography. *Am J Orthod Dentofacial Orthop* 2005; 128(6):803-811.
  15. Doyon, D, Pajoni, D, et al.: *Cahiers de radiologie 11. Imagerie dento maxillaire*. Paris: Masson, 1995.
  16. De Mot B, Casselman J, Widelec J: Imaging of the temporomandibular joint. *Rev Belge Med Dent* 1997; 52(1):283-303.
  17. Loubele M, et al.: Assessment of bone segmentation quality of cone-beam CT versus multislice spiral CT: a pilot study. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2006; 102(2):225-234. E-pub 2006; Apr 21.
  18. Ludlow JB, et al.: Dosimetry of three CBCT devices for oral and maxillofacial radiology: CB Mercuray, NewTom 3G and i-CAT. *Dentomaxillofac Radiol* 2006; 35(4):219-226. Erratum in: *Dentomaxillofac Radiol* 2006; 35(5):392.
  19. Sarnat BG, Laskin DM: *The Temporomandibular joint: a biological basis for clinical practice*. 4th ed. WB Saunders Co: Philadelphia, 1992.
  20. Learreta JA: Regeneration ad integrum of the condyle head in a patient with temporomandibular disorders. *J Craniomandib Pract* 1999; 17(3):221-227.
  21. Henry CH, et al.: Identification of chlamydia trachomatis in the human temporomandibular joint. *J Oral Maxillofac Surg* 1999; 57(6):683-688.
  22. Henry CH, et al.: Reactive arthritis: preliminary microbiologic analysis of the human temporomandibular joint. *J Oral Maxillofac Surg* 2000; 58(10):1137-1142.
  23. Henry CH, Pitta MC, Wolford LM: Frequency of chlamydial antibodies in patients with internal derangement of the temporomandibular joint. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2001; 91(3):287-292.
  24. Kim SJ, et al.: The presence of bacteria in the synovial fluid of the temporomandibular joint and clinical significance: preliminary study. *J Oral Maxillofac Surg* 2003; 61(10):1156-1161.
  25. Paegle DI, et al.: The occurrence of antibodies against chlamydia species in patients with monoarthritis and chronic closed lock of the temporomandibular joint. *J Oral Maxillofac Surg* 2004; 62(4):435-439.
  26. Jeon HS, Hong SP, et al.: Hematogenous infection of the human temporomandibular joint. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2005; 99(2):E11-7.
  27. Wollenhaupt J, et al.: Evaluation of ELISA to detect chlamydia trachomatis antigen in urine samples from arthritis patients. *Clin Exp Rheumatol* 1997; 15(2):169-174.
  28. Chang H, Israel H: Analysis of inflammatory mediators in temporomandibular joint synovial fluid lavage samples of symptomatic patients and asymptomatic controls. *J Oral Maxillofac Surg* 2005; 63(6):761-765.
  29. Henry CH, Nikaen A, Wolford LM: Analysis of human leukocyte antigens in patients with internal derangement of the temporomandibular joint. *J Oral Maxillofac Surg* 2002; 60(7):778-783.
  30. Fu, K: Interleukin-6 in synovial fluid and HLA-DR expression in synovium from patients with temporomandibular disorders. *J Orofacial Pain* 1995; 9(2):131-137.
  31. Nordahl S, Alstergren P, Eliasson S, Kopp S: Interleukin-1 beta in plasma and synovial fluid in relation to radiographic changes in arthritic temporomandibular joints. *Eur J Oral Sci* 1998; 106(1):559-563.
  32. Shinoda C, Takaku S: Interleukin-1 beta, interleukin-6, and tissue inhibitor of metalloproteinase-1 in the synovial fluid of the temporomandibular joint with respect to cartilage destruction. *Oral Dis* 2000; 6(6):383-390.
  33. Sato J, Segami N, Nishimura M, Demura N, Yoshimura H, Yoshitake Y, Nishikawa K: Expression of interleukin-6 in synovial tissues in patients with internal derangement of the temporomandibular joint. *Br J Oral Maxillofac Surg* 2003; 41(2):95-101.
  34. Kaneyama K, Segami N, Sato J, Nishimura M, Yoshimura H: Interleukin-6 family of cytokines as biochemical markers of osseous changes in the temporomandibular joint disorders. *Br J Oral Maxillofac Surg* 2004; 42(3):246-250.
  35. Sato J, Segami N, Nishimura M, et al.: Expression of interleukin-8 in synovial tissues in patients with internal derangement of the temporomandibular joint and its relationship with clinical variables. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2007; 103(4):467-474. E-pub 2006, Oct 27.
  36. Okeson JP: *Management of temporomandibular disorders and occlusion*. 4th ed. Mosby-Year Book, Inc. 1998.
  37. Helenius LM, et al.: Clinical, radiographic and MRI findings of the temporomandibular joint in patients with different rheumatic diseases. *Int J Oral Maxillofac Surg* 2006; 35(11):983-989. E-pub 2006, Oct 18.
  38. Ramos-Remus C, et al.: Magnetic resonance changes in the temporomandibular joint in ankylosing spondylitis. *J Rheumatol* 1997; 24(1):123-127.
  39. Helenius LM, et al.: HLA-DRB1\* alleles and temporomandibular joint erosion in patients with various rheumatic diseases. *Scand J Rheumatol* 2004; 33(1):24-29.
  40. Helenius LM, et al.: Clinical and radiographic findings of the temporomandibular joint in patients with various rheumatic diseases. A case-control study. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2005; 99(4):455-463.
  41. Manfredini D, et al.: Ultrasonography of the temporomandibular joint: comparison of findings in patients with rheumatic diseases and temporomandibular disorders. A preliminary report. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2005; 100(4):481-485.
  42. Rakosi T, Jonas I: Published: *Color atlas of dental medicine: orthodontic diagnosis*. Thieme Medical Publishers: Stuttgart, 1993.
  43. Ren YF, Isberg A, Westesson P-L: Condyle position in the temporomandibular joint. Comparison between asymptomatic volunteers with normal disk position and patients with disk displacement. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1995; 80:101-107.
  44. Jankelson B: Electronic control of muscle contraction—a new clinical era in occlusion and prosthodontics. *Sci Educ Bull* 1969; 2(1):29-31.
  45. Jankelson B, Swain CW: Physiological aspects of masticatory muscle stimulation: the myomonitor. *Quintessence Int Dent* 1972; 3(12):57-62.
  46. Jankelson B, Swain CW, Crane PF, Radke JC: Kinesiometric instrumentation: a new technology. *J Am Dent Assoc* 1975; 90(4):834-840.
  47. Learreta JA, Bono AE: A importância da desprogramação mandibular no diagnóstico ortodôntico. *J Bras Ortodon Ortop Facial* 1998; 3(18):72-77.
  48. Learreta JA; Bono, AE, Azolin M: A importância da desprogramação mandibular no diagnóstico ortodôntico, Parte II. *J Bras Ortodon Ortop Facial* 4(21):215-222.
  49. Learreta JA, Moses AJ: Cephalometric variation in patients with and without intraoral neuromuscular repositioning appliance. *J Gen Orthod* 1999; 10(2):14-21.
  50. Gibbs CH, Messerman T, Reswick JB, Derda HJ: Functional movements of mandible. *J Prosthet Dent* 1971; 26(6):604-620.
  51. Gibbs CH, Mahan PE, Lundeen HC, Brehnan K, Walsh EK, Sinkewicz SL, Ginsberg SB: Occlusal forces during chewing: influence on biting strength and food consistency. *J Prosthet Dent* 1981; 46(5):561-567.
  52. Ow RKK, Carlsson GE, Jemt T: Craniomandibular disorders and masticatory mandibular movements. *J of Craniomandib Disord Fac Oral Pain* 1988; 2(2):96-100.
  53. Mongini F, Tempia-Valenta G, Conserva E: Habitual mastication in dysfunction: a computer-based analysis. *J Prosthet Dent* 1989; 61(4):484-494.
  54. Horio T, Kawamura Y: Effects of texture of food on chewing patterns in human subjects. *J Oral Rehabil* 1989; 16(2):177-183.
  55. Kuwahara T, Miyauchi S, Murayama T: Condylar movements during mastication [Summary]. *J Oskia Univ Dent Sch* 1989; 29:87-102.
  56. Kuwahara T: Clinical study on the relationship between chewing movements and temporomandibular joint abnormalities. *J Osaka Univ Dent Soc* 1989; 34:1:64-105.
  57. Kuwahara T, Miyauchi S, Murayama T: Condylar movements during mastication [Summary]. *J Oskia Univ Dent Sch* 1989; 29:87-102.
  58. Kuwahara T, Miyauchi S, Maruyama T: Characteristics of condylar movements during mastication in stomatognathic dysfunction. *Int J Prosthodont* 1990; 3(6):555-566.
  59. Nielsen IL, Marcel T, Chun D, Miller AJ: Patterns of mandibular movements in subjects with craniomandibular disorders. *J Prosthet Dent* 1990; 63(2):202-217.
  60. Kuwahara T, Miyauchi S, Maruyama T: Clinical classification of the patterns of mandibular movements during mastication in subjects with TMJ disorders. *Int J Prosthodont* 1992; 5(2):122-129.
  61. Ferrario VF, Sforza C: Coordinated electromyographic activity of the human masseter and temporalis anterior muscles during mastication. *Eur J Oral Sci* 1996; 104(5-6):511-517.
  62. Sato S, Ohta M, Goto S, Kawamura H, Morigi K: Electromyography during chewing movement in patients with anterior disc displacement of the temporomandibular joint. *Int J Oral Maxillofac Surg* 1998; 27(4):274-277.