MRI of the Temporomandibular Joint

Correlation Between Imaging and Pathology Tiziana Robba Carlotta Tanteri

Giulia Tanteri *Editors*



EXTRAS ONLINE

MRI of the Temporomandibular Joint

Tiziana Robba • Carlotta Tanteri Giulia Tanteri Editors

MRI of the Temporomandibular Joint

Correlation Between Imaging and Pathology



Editors Tiziana Robba Department of Diagnostical Imaging CTO – Città della Salute e della Scienza Turin Italy

Carlotta Tanteri Private Practice, Studio Tanteri Turin Italy

Giulia Tanteri Private Practice, Studio Tanteri Turin Italy

ISBN 978-3-030-25420-9 ISBN 978-3-030-25421-6 (eBook) https://doi.org/10.1007/978-3-030-25421-6

© Springer Nature Switzerland AG 2020

This work is subject to copyright. All rights are reserved by the Publisher, whether the whole or part of the material is concerned, specifically the rights of translation, reprinting, reuse of illustrations, recitation, broadcasting, reproduction on microfilms or in any other physical way, and transmission or information storage and retrieval, electronic adaptation, computer software, or by similar or dissimilar methodology now known or hereafter developed.

The use of general descriptive names, registered names, trademarks, service marks, etc. in this publication does not imply, even in the absence of a specific statement, that such names are exempt from the relevant protective laws and regulations and therefore free for general use.

The publisher, the authors, and the editors are safe to assume that the advice and information in this book are believed to be true and accurate at the date of publication. Neither the publisher nor the authors or the editors give a warranty, expressed or implied, with respect to the material contained herein or for any errors or omissions that may have been made. The publisher remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

This Springer imprint is published by the registered company Springer Nature Switzerland AG The registered company address is: Gewerbestrasse 11, 6330 Cham, Switzerland

To our mentors, for teaching us and guiding us down the path to becoming our best selves To our families, husbands and to our children Francesco, Chiara, Benedetta, and Gregorio Oceano To our patients, for trusting us and for giving us the possibility to learn something new every day To our friends and colleagues To ourselves, because sharing so much made us treasure each other in a way we had not expected

"Who sees further a dwarf or a giant? Surely a giant for his eyes are situated at a higher level than those of the dwarf. But if the dwarf is placed on the shoulders of the giant who sees further? ... So too we are dwarfs astride the shoulders of giants. We master their wisdom and move beyond it. Due to their wisdom we grow wise and are able to say all that we say, but not because we are greater than they."

Isaiah di Trani

Foreword by Ambra Michelotti

This book edited by Tiziana Robba, Carlotta Tanteri, and Giulia Tanteri has the great merit, which is not frequently found, of helping to fill a real gap in the medical-dental literature. The growing knowledge in terms of diagnostic and therapeutic approaches regarding temporomandibular joint pathologies, combined with the incessantly improving technological advances made in the field of diagnostic imaging, have in fact dug a deep groove between the experts of the different disciplines called upon to define the diagnostic and therapeutic path. The project of writing a multidisciplinary book, under the perspective of a dentist, a radiologist, and a maxillofacial surgeon, allows to fill this groove through the union of the clinical and scientific skills of the authors in different fields. The text provides the basic elements needed by the radiologist to understand the different pathologies of the articular tissues and the morphological changes related to them and, at the same time, helps the clinician to interpret the images derived from instrumental investigations. Ultimately, the authors have accomplished an excellent and appreciable work of interdisciplinary linking that will allow all those who will read and study this easily accessible text to face the complex temporomandibular pathologies with greater competence.

> Ambra Michelotti Associate Professor Department of Orthodontics and Gnathology University of Naples Federico II Naples, Italy

Foreword by Daniele Regge

In this volume, Dr. Tiziana Robba, Dr. Carlotta Tanteri, and Dr. Giulia Tanteri provide a very comprehensive overview of temporomandibular joint (TMJ) disorders and of the role of magnetic resonance imaging (MRI) in their diagnosis. All the chapters of this handbook follow the same structure: after a brief introduction on the epidemiology and etiopathogenesis, the authors elaborate the content adopting a multidisciplinary approach. At first a description of the physiology is given followed by a systematic overview of pathological findings both from a radiological and pathological perspective. The understanding of the text is supported by the superb quality of radiological images. Most importantly, clinical and therapeutic implications of radiological finding are thoroughly discussed.

I found this book extremely inspiring and informative, and I recommend it not only to imaging doctors but also to dentists, gnathologists, and maxillofacial surgeons. I am persuaded that this monograph may greatly add to knowledge by providing adequate and precise description of MRI findings and allow accurate treatment planning in patients with TMJ disorders. I hope it will attain the success it rightly deserves.

> Daniele Regge Radiology Unit IRCCS Candiolo Cancer Institute University of Turin Turin Italy

Foreword by Florencio Monje

It is a great honor and privilege to collaborate with this book doing the foreword. The editors form an inspiring team with a clever combination of different specialties involved in the diagnosis and treatment of these pathologies such as radiology, dentistry, and maxillofacial surgery.

The authors indeed make a tour of MRI without ignoring other TMJ imaging modalities. The information of TMJ anatomy applied to MRI, as well as the dynamics of this joint, gives us basic information for the following parts of this book. The key to correct interpretation of TMJ imaging findings lies in a thorough knowledge of the anatomy and an understanding of the function and dysfunction of the TMJs. In other words, the professionals around the pathology of this joint should have enough knowledge about imaging of this area.

The diagnostic process is especially important since an incorrect diagnosis is the most frequent cause of treatment failure. However, radiological findings should never be interpreted in isolation. The decision for any treatment must be based primarily on the combination of clinical and radiological information in conjunction with other factors such as the impact of the disease on the patient and the prognosis in case no treatment is provided. As a maxillofacial surgeon, and someone bewitched by TMJ disease, I truly appreciate this book. It becomes a working tool in TMJ diagnostics, whatever the approach.

> Florencio Monje Department of Oral and Maxillofacial Surgery University Hospital Infanta Cristina Badajoz Spain

Preface

This book is the result of an enduring cooperation between a radiologist, a maxillofacial surgeon, and a dentist, who became specialized in temporomandibular joint pathology throughout the years. It is from our constant exchange of thoughts, competences, and considerations that the necessity to write this book emerged. The urge to share a common language and the constant need of relying on the reciprocal expertise made us realize that we wanted to bridge the gap, first of all for ourselves.

This book aims at providing an easy-to-consult format with the essential knowledge to address TMJ magnetic resonance interpretation. The focus of our work was on presenting TMJ conditions that gnathologists and dental practitioners encounter in their daily practice. Clinicians, in fact, often lack the technical-radiological familiarity with the exams they prescribe and seldom know how to interpret the images they receive. For this reason, we gave plenty of room to the description of MRI TMJ anatomy, to the radiological correlation with the most common disorders, and to technical considerations which lie behind high-quality MR images. At the same time, we wanted to let radiologists understand the clinical presentation which precedes the request to perform radiological examinations for the TMJ, so as to allow them to better appreciate which details should be taken into account while performing the investigation and evaluating the images.

The collaboration between our three disciplines provided insight into each other's difficulties and allowed us to include pieces of information which make this book valuable to all colleagues that deal with temporomandibular disorder patients. The illustrations and the clarifying terminology aim at simplifying concepts which usually create confusion.

Clinicians deal with signs and symptoms, but at times rely too much on their clinical confidence alone. Our approach to TMJ conditions should be consequential, based on shared diagnostic criteria and supported by instrumental analyses. Magnetic resonance shows what is concealed behind signs and symptoms, supporting diagnostics and therapeutic choices.

We sincerely hope that our work will help radiologists to better grasp the clinical reasoning behind TMJ imaging and that it will help clinicians realize how MRI is becoming a fundamental part of everybody's practice.

Tiziana Robba Carlotta Tanteri Giulia Tanteri

Acknowledgments

We would like to thank the contributing authors of this book for their commitment and effort and for the high-quality material they provided.

Thank you Dr. Gino Carnazza and Dr. Eugenio Tanteri, our mentors. Thanks for giving us the possibility of writing this book. You selflessly moved one step back and allowed us to do this by ourselves. Thank you for teaching us, inspiring us, and sharing your knowledge and thank you for your constant support.

Thank you Prof. Gregor Slavicek for giving us the possibility to learn, study and grow up with you.

Thank you to the colleagues who helped up with illustrations and imaging. Thank you Dr. Angelo Bracco and Dr. Nicolò Margolo for the remarkable anatomical drawings.

We would like to thank our mothers and fathers for giving us the necessary peace of mind to go through these hard times and for always having our backs.

Thank you to our husbands and children because we have been blessed by your presence in our lives. Thank you from the bottom of our hearts for your support and your patience.

One final thank you to the doctors, technicians, staff, nurses, and good friends who work with us daily. Thank you for the constant exchange of knowledge, for questioning us and spending your lives with us.

Contents

1	TMJ Magnetic Resonance: Technical Considerations 1 Valeria Clementi and Tiziana Robba 1
2	Other TMJ Imaging Modalities.25Luca Luberto, Sara Garberoglio, and Gino Carnazza
3	TMJ and MRI Anatomy
4	TMJ Dynamics 57Giulia Tanteri, Eugenio Tanteri, Carlotta Tanteri, and Gregor58
5	Developmental Disorders 91 Giovanni Gerbino, Vito Chianca, and Guglielmo Ramieri 91
6	TMJ Trauma 105Claudio Caldarelli, Paolo Busolli, and Giacomo Paolo Vaudano
7	Joint Disorders
8	Joint Diseases. 175 Tiziana Robba, Paolo Tosco, Simone Parisi, Guglielmo Ramieri, Enrico Fusaro, Riccardo Faletti, and Giulia Tanteri
9	Tumors and Tumor-Like Lesions . 219 Paolo Tosco, Vito Chianca, and Guglielmo Ramieri
In	dex

TMJ Magnetic Resonance: Technical Considerations

Valeria Clementi and Tiziana Robba

Key Points

- MR is a multiparametric imaging technique based on absorption of energy by the atomic nuclei of tissues and the subsequent return of the system to its initial state. In order to be performed, the patient has to be inserted into specially generated magnetic fields and non-ionizing electromagnetic radiation is used.
- The main contrast parameters used for image generation are: proton density, T_1 , and T_2 . These last two are intrinsic parameters of any tissue, related to its microscopic structure, which influence the way the system returns to equilibrium after absorption of the radio frequency (RF) energy. Parameters combination can provide for a great variability of the contrast between tissues, and it is selected on the basis of the clinical question.

Electronic supplementary material The online version of this chapter (https://doi.org/10.1007/978-3-030-25421-6_1) contains supplementary material, which is available to authorized users.

V. Clementi (🖂)

Medical Technology Laboratory, IRCCS Istituto Ortopedico Rizzoli, Bologna, Italy

T. Robba

Department of Diagnostic Imaging and Radiotheraphy, Radiology Service, C.T.O. Hospital, A.O.U. Città della Salute e della Scienza, Turin, Italy e-mail: trobba@cittadellasalute.to.it

- Images are generated by sequences of radio frequency pulses and variable magnetic fields. There are two main types of sequences: spin echo and gradient echo. Spin echo sequences are the most commonly used as they provide fine anatomical details, thanks to their SNR. Gradient echo sequences are used to decrease acquisition times, are more sensitive to changes of magnetic susceptibility of tissues, and may provide information regarding deposits such as calcium or hemosiderin.
- MRI is a tomographic imaging technique: it represents anatomical region volumes through 2D images. MR, unlike other imaging techniques, can directly acquire along oblique planes. 3D acquisitions are also possible, allowing isotropic 3D reconstructions of the volume.
- Over the years, clinical MR systems have offered an increasing number of sequences and techniques, many of which are intended to reduce acquisition times through different modalities of data acquisition (fast acquisition techniques) and to suppress fat signal (fat signal suppression techniques).
- MR does not use ionizing radiation and can therefore be considered a low-risk technique. However, MR may still involve risks for operators and patients, and these can be limited by site design and safety procedures applied in the daily practice.

1



1

[©] Springer Nature Switzerland AG 2020

T. Robba et al. (eds.), *MRI of the Temporomandibular Joint*, https://doi.org/10.1007/978-3-030-25421-6_1

1.1 Introduction

Imaging of the temporomandibular joint (TMJ) has been continuously evolving along with the advancement of imaging technologies. Even though many imaging modalities are currently used to evaluate the TMJ (i.e., cone beam computed tomography-CBCT-and multi-detector computed tomography-MDCT), the use of magnetic resonance imaging has increased due to its great contrast resolution, its strength in highlighting soft tissue structures and signs of inflammation, and its capability in acquiring dynamic imaging for demonstration of the functionality of the joint (Bag et al. 2014). Furthermore, MRI does not involve ionizing radiations and this helps in limiting the overall history of patient exposure (Niraj et al. 2016). On the other hand, the relative disadvantages of MRI compared to CT include a more complex scanning technique and a longer acquisition time. The advantages of CT over MRI are enhanced bone details and 3D assessment of congenital, developmental, and traumatic conditions (Bag et al. 2014; Niraj et al. 2016). In this chapter, the reader will be given the essential and basic information to understand the principles of physics that underlie the creation of MR images.

1.2 Principles of Physics of Magnetic Resonance Imaging

1.2.1 Nucleus and Spin

The MR imaging technique is based on absorption of energy by atomic nuclei and the following return of the system to the initial state. In particular, the majority of MR clinical applications are based on hydrogen nuclei, in fact, this is one of the most common elements in nature and very abundant in the human body.

Atoms consist of three main particles: protons, which have a positive charge, neutrons, which have no charge, and electrons, which have a negative charge. The atomic nucleus is positively charged due to the presence of protons and neutrons. Electrons are displaced in orbitals surrounding the nucleus. Each element is defined by a typical number of protons and electrons, while the nucleus can contain a variable number of neutrons, thus characterizing different isotopes of the same element. The hydrogen nucleus contains a single proton and no neutron (Fig. 1.1).

The complete comprehension of the MR phenomenon is based on the quantum mechanics theory, the most complete model to interpret the microscopic world. However, quantum mechanics is usually very far from our intuitive interpretation, which is based on the macroscopic world experience and described by the classical physics models. For this reason, it is common to use quantum mechanics concepts, together with some classical mechanics models, to explain and better understand some aspects of MR.

The atomic nucleus has the intrinsic property of rotating about its axis, like a spinning top. The physical quantity that describes this feature is a vector called *spin angular momentum*, also called *spin* (Fig. 1.2a). From the physics of electromagnetism, it is also known that a moving charge creates a magnetic field. The magnetic field source can be represented in physics by a dipole, that can be imagined as a little magnet, with a north pole and a south pole. Then a nucleus can be represented as a spinning top because of its rotation about its axis, as well as a little magnet because of its little magnetic field (Fig. 1.2b). The little dipoles, generated by the spinning charged atomic nuclei, are at the origin of the MR signal.



Fig. 1.1 Hydrogen atom. Its nucleus is made up of one proton only. Its electron cloud also consists of only one electron



Fig. 1.2 (**a**, **b**) The atomic nucleus of hydrogen rotates about its own axis, like a spinning top. The physical quantity that describes this feature is a vector called *spin angular momentum* (**a**). The positively charged hydrogen nucleus rotates about its own axis like a spinning top and it generates a small magnetic field. It can be represented as a small magnet, with a north and a south pole (**b**)

Based on what has just been described, a volume of patient tissue can be imagined as a pool of little magnets, all hydrogen nuclei (or spins), each of them rotating about their own axis and generating a little microscopic magnet. In normal conditions, out of a strong magnetic field, all spin orientations are possible, the magnetic fields generated from the nuclei cancel each other out and the overall effect is that there is no macroscopic magnetization on the tissue (Fig. 1.3).

1.2.2 Resonance Phenomena and Larmor Precession

When the patient is introduced into a uniform and constant high-intensity magnetic field, specifically created inside the MR scanner, called \mathbf{B}_0 , the hydrogen spins, which were randomly oriented up to that point, move to align along the main magnetic field \mathbf{B}_0 in a parallel or anti-

parallel orientation (Fig. 1.4). The anti-parallel alignment implies a higher energy than the parallel one and the latter is more frequent. In classical physics models, if a spinning top is moved from its axis while it rotates about itself, it also begins to rotate around the direction of gravity. Again, the spin can be considered as a spinning top, and when the spin is exposed to the magnetic field B_0 it behaves like a spinning top on the gravitational field, and it starts to rotate around the axis of B_0 with a typical motion called *precession* (Fig. 1.5).

The Larmor equation describes the relationship between the intensity of the magnetic field \mathbf{B}_0 and the rotation frequency of the spin precession:

$$\omega_0 = \gamma B_0 \tag{1.1}$$

where ω_0 is known as the Larmor precession frequency or resonance frequency and it is expressed in MHz, γ is the gyromagnetic ratio (unique to every atom), expressed in MHz/T, and B_0 is the magnetic field strength in Tesla.

Protons have a gyromagnetic ratio of 42.58 MHz/T, and the corresponding Larmor frequency at 1.5 T is 63.87 MHz. This value can be roughly compared to about 1 KHz, which is the Larmor frequency corresponding to the magnetic field intensity of the Earth.

As a consequence of what has been described, whenever a patient is brought into an external magnetic field, the overall effect is the appearance of a macroscopic magnetization that can be represented as a vector \mathbf{M} , with the same direction and orientation of the external magnetic field \mathbf{B}_0 (Fig. 1.6). There is no magnetization vector when a tissue is not placed in an external magnetic field.

On a quantum mechanics point of view, when the spin component is measured along an axis (the *z*-axis, for instance) it is only possible to obtain a finite number of values (quantized values), related to a number which describe the spin angular momentum: the spin quantum number *I*. This number is different from nucleus to nucleus. Nuclei with a spin quantum number I = 0 cannot be used for MR. The hydrogen nucleus has the spin quantum number $I = \frac{1}{2}$, which makes it suitable for the creation of MR signal. Other nuclei,



for example carbon-13, nitrogen-14, fluorine-19, phosphorus-31, and sodium-23, are characterized by an $I \neq 0$ and could potentially generate MR signal. However, these elements are less abundant in biological tissues compared to hydrogen; therefore, their use in MRI is not standard and is limited to specific research applications.

Out of the B_0 field, when measuring the hydrogen nucleus spin component along the *z*-axis, the only possible results are two values, corresponding to spin states UP and DOWN, both having the same energy. The number of spins UP is equivalent to the number of spins DOWN and there is no macroscopic effect. On the contrary, when a patient is placed into the main magnetic field \mathbf{B}_0 , the two possible hydrogen spins configurations with respect to the \mathbf{B}_0 axis, UP (anti-parallel) or DOWN (parallel)—become energetically different, with UP alignment corresponding to the upper energy state (Fig. 1.7). The distribution of the spins population, between up and down energy states, is no longer equal and it is related to the microscopic thermal motions within the tissue and to the intensity of the external magnetic field \mathbf{B}_0 . At body temperature and with the typical external magnetic field used in clinical practice, there is a minimal excess (approximately 10⁻⁶) of spins on DOWN (lower) energy state. This very weak difference in the distribution of the spins population generates the macroscopic magnetiza-



Fig. 1.5 When the spin is exposed to the magnetic field \mathbf{B}_0 , its axis begins to rotate around the axis of \mathbf{B}_0 , like a spinning top on the gravitational field. This typical motion is called precession

Fig. 1.6 Macroscopic magnetization vector \mathbf{M} is the overall effect of the spin population when the external magnetic field \mathbf{B}_0 is active



tion **M** that is at the origin of the MR signal. This is why one of the main challenges of MR technology is to increase the signal and optimize the signal-to-noise ratio (SNR). In fact the higher the SNR, the more the information, the spatial resolution, and the temporal resolution of dynamic studies or the lower the scan time, on final clinical images. It is important to highlight that as B_0 increases, M intensity increases too. This is the reason why MR scanners are evolving into systems with an always larger B_0 . Moreover, the intensity of the macroscopic magnetization M, which is generated when the patient is inserted into the external field B_0 , is proportional to the number of protons in the tissue volume. Since proton density (PD) in different tissues is different, and it may also be affected by pathology, PD is one of the parameters used to generate contrast on MR imaging technique.

1.2.3 MR Signal Detection

Unlike other imaging techniques (such as CT) in which contrast is essentially based on the attenuation of the X-ray beam, MRI technique uses multiple parameters for contrast generation. Proton density is one of the tissue properties used in MR to create contrast, the macroscopic magnetization being proportional to the number of



protons present in the tissue. More contrastgenerating parameters are described below.

In order to efficiently detect macroscopic magnetization, Faraday's law is adopted. It states that the temporal variation of a magnetic field induces a current in an electric conductor. The macroscopic magnetization M is initially oriented along the \mathbf{B}_0 axis, named z-axis, and therefore the M_z component only will have a value other than zero (Fig. 1.8a). For the purposes of signal acquisition, the orientation of M is changed via a radio frequency (RF) pulse, transmitted by a coil (Fig. 1.8b, c). If the RF pulse frequency is the same as Larmor's, then it matches to the precession frequency of spins around B_0 , and its effect is to rotate M from the z-axis towards the xyplane of an angle, called the flip angle (FA). Flip angle values can depend on the RF pulse duration. Consequently, the M component in the xyplane (M_{xy}) is no longer null. Moreover, when moved out of the B_0 axis, M also behaves like a magnetic dipole, and it starts a precession around the z-axis with the Larmor frequency ω_0 . This motion of M corresponds to a time-varying magnetic field that generates an electrical signal in the coil. In practice, due to technical reasons, it is the M_{xy} component of **M** to be actually detected by the receiving coil, then collected, and analyzed.

The actual measurement of the M_{xy} intensity is however made difficult by some microscopic phenomena. These are responsible for other widely used intrinsic contrast parameters, other than the already mentioned proton density.

1.2.4 Spin Relaxation

It is important to emphasize that the frequency of the RF pulse must match Larmor frequency. Any other frequency would not be effective. In fact, the absorption of the RF pulse by the patient tissue corresponds to energy absorption by the system of spins.

From a classical point of view, the RF pulse corresponds to a further magnetic field, named B_1 , which is rotating at the same frequency of M, and then able to transfer energy to M and rotate it on the *xy*-plane.

From a quantum mechanics point of view, bringing the spin to an excited state requires the transfer of a precise amount of energy that corresponds to the energy gap between the spin energy levels. This is exactly the energy of an electromagnetic wave with the Larmor frequency. The name *nuclear magnetic resonance* refers to energy transfer to a system through appropriate periodic oscillation, and in physics this phenomenon is called *resonance*. The energy-receiving system is made up by the pool of hydrogen nuclear spins within tissues being studied.

When the RF excitation pulse ends, the system naturally tends to its starting condition, through a phenomenon known as *relaxation*.

From a macroscopic point of view, the relaxation after the RF pulse can be represented as the combination of two components of M: the precession movement about B_0 generating a spiral motion and described by M_{xy} (the projection of M on the xy-plane), and the return of M along the \mathbf{B}_0 direction, described by M_z (the **M** component along the z-axis) (Fig. 1.9a, b). These two components describe different microscopic relaxation mechanisms. The longitudinal component M_z depends on the interactions and energy exchanges between the spins and the molecular environment (the "lattice"). M_7 returns to equilibrium according to the increasing curve seen in Fig. 1.9a, characterized by the parameter T_1 , known as the spin-lattice relaxation time. T_1 relaxation time expresses the time needed for the recovery of 63% of the longitudinal magnetization M_{z} value before the RF pulse. T_{1} relaxation time is dependent on the strength of the external magnetic field B_0 and the internal Brownian motion of the molecules.

The relaxation of the transverse component M_{xy} depends on the same phenomena that con-

tribute to T_1 relaxation and on other atomic and molecular interactions including spin-spin interactions. M_{xy} returns to equilibrium according to the decreasing curve seen in Fig. 1.9b, characterized by the T_2 parameter, known as the *spin-spin* relaxation time. T_2 relaxation expresses the time required by the transverse magnetization M_{xy} to decay to 37% of its initial value immediately after the end of the excitation RF pulse. The T_2 relaxation is essentially due to the loss of phase coherence of the spins. Following the 90° RF pulse the spins will begin to precede around B_0 axis all with the same phase, but because of the influence of mutual microscopic magnetic fields, they rotate with a slightly different resonance frequency and tend to be out of phase with each other, thus leading to a progressive destruction of the macroscopic signal in the xy-plane. As a general rule, T_2 is lower in solid tissues and higher in liquid tissues.

In the real measurement process, the loss of phase coherence of the spins, and therefore the relaxation of M_{xy} , can be accelerated by the presence of local magnetic fields, due to variations in



Fig. 1.9 (**a**, **b**) The increasing curve (**a**) is characterized by the parameter T_1 , known as the *spin–lattice relaxation time*. T_1 expresses the time needed for the recovery of 63% of the longitudinal magnetization M_z value before the RF pulse (M_0). The decreasing curve (**b**) is characterized by the T_2

parameter, known as the *spin–spin relaxation time*. T_2 expresses the time required by the transverse magnetization M_{xy} to decay to 37% of the initial value (M_0) immediately after the end of the excitation RF pulse. As a general rule, T_2 is lower in solid tissues and higher in liquid tissues

the local tissue components or an imperfect homogeneity of the external magnetic field \mathbf{B}_0 . This leads to a faster M_{xy} decay, with a similar trend of T_2 decay, but defined by the T_2* parameter, shorter than T_2 . In conclusion T_1 , T_2 (and partially T_2*) are intrinsic parameters of the tissues, they have different values based on different tissues and may be altered by pathology, and therefore they provide solid ground for the creation of image contrast.

The MR technique is accomplished by administering RF pulses to a system and employing magnetic field gradients, in suitable time series, called *sequences*.

A wide variety and a growing number of sequences are currently being used, which allow for the creation of images with different geometric characteristics and contrasts. The principles of creation and optimization of MR pulse sequences are beyond the scope of this chapter, however the basic principles of MR sequences and the main parameters used by the operator during the exam will be covered together with the description of two sequences whose structures are the basis of almost all MR sequences: spin echo and gradient echo (Weishaupt et al. 2008).

1.2.5 Spin Echo Sequences

This is one of the most commonly used family of sequences because of its good SNR.

The spin echo (Fig. 1.10) starts with a 90° RF excitation pulse, which rotates **M** by 90°, from the *z*-axis to the *xy*-plane. As soon as the pulse is

over, the M_{xy} component begins to decay with a T_{2} * relaxation. This signal is called FID (free induction decay) and again, this decay is faster compared to pure T_2 decay. Instead of acquiring the FID, after a TE/2 time interval, a second RF pulse is administered, called 180° RF pulse, with appropriate direction, intensity, and duration, with the aim of rotating the spins by 180° in the xy-plane. Therefore, after another TE/2 time interval, the spins that were out of phase during the T_2 * decay will be again in phase in the xyplane. The signal acquisition starts at this point in time. It is important to stress that the 180° RF pulse is used to retrieve the phase shift of the spins due to the local magnetic variations of the tissue, while the phase shift caused by the spinspin interactions is not recovered. Therefore, the signal acquired at TE will be the intensity of the M_{xy} component decayed according to the spinspin relaxation time T_2 (not T_2 *), and it is then called a T_2 -weighted signal.

As the T_2 parameter is a tissue-related characteristic and it can also change in pathological conditions, by varying TE time interval between the 90° RF pulse and the and 180° RF pulse, a contrast can be generated between tissues with different T_2 decay times (Fig. 1.11).

If the sequence gets repeated several times, as it usually occurs in clinical settings, then it is possible to add the effect of a T_1 relaxation to the signal. TR is the time which runs between two consecutive sequence repetitions.

In fact, after the first 90° RF pulse, while the M_{xy} relaxes and is partially refocused on the *xy*-plane by 180° pulse, the M_z component relaxes



Fig. 1.10 The spin echo sequence starts with a 90° RF excitation pulse that rotates **M** by 90°, from the *z*-axis to the *xy*-plane. After the pulse, the M_{xy} component begins to decay with a T_2 * relaxation



Fig. 1.11 As T_2 is a tissue-specific intrinsic characteristic, contrast can be enhanced by changing TE between the 90° RF pulse and the 180° RF pulse

too and returns to the initial value according to the T_1 relaxation. When the next 90° RF pulse from the following spin echo sequence repetition is given, at a TR time from the previous 90° pulse, the M_z intensity will not necessarily be the maximum M_0 value (starting state), but it will have the value reached at the time TR from the start of the T_1 recovery.

If TR has a value lower than approximately the value of T_1 of the considered tissue multiplied by 3, the M_z component will not have enough time to return to its maximum value; therefore, the signal acquired after the second spin echo repetition will depend on T_1 . This allows for the generation of a contrast based on T_1 relaxation time (Fig. 1.12).

Basically, the T_2 weighting of the signal depends on the TE parameter which is the time that elapses between the rotation of the magnetization in the xy-plane and its acquisition after refocusing. T_1 weighting depends on TR, the time between one repetition of a complete sequence and the next one; therefore, it is the time left for the system to replenish the initial value of the M_z component.

Since patient tissues have different T_1 and T_2 values, and pathological conditions often modify tissue relaxation times, then by suitably varying TE and TR it is possible to obtain anatomical and diagnostic images with different contrast, T_1 weighted or T_2 weighted, based on specific diagnostic questions. Also, TE and TR values can be selected to reduce both the effects of T_2 and T_1



Fig. 1.12 If TR has a value lower than the value of T_1 of the considered tissue multiplied by 3, the M_z component will not have enough time to return to its maximum value, and therefore the signal acquired after the second spin echo repetition will depend on T_1 . This allows the generation of T_1 -weighted images, with contrast between tissues with different T_1

relaxation difference between tissues, thus obtaining proton density images (Figs. 1.13a–e and 1.14a–d).

1.2.6 Gradient Echo Sequences

The second fundamental MR family of sequences is based on gradient echo.

Compared to spin echo, gradient echo sequences show some differences. After the initial RF pulse that moves M out of the z-axis towards the xy-plane, refocusing of the M_{xy} component prior to acquisition is achieved through the use of magnetic field gradients instead of RF pulses. Magnetic field gradients are in fact an essential component of an MR sequence, and they are also involved in image acquisition. These are additional external magnetic fields, appropriately varied in space and time characteristics, generated by specific coils which are positioned around the main magnet. During sequences, these additional magnetic fields are superimposed to the constant external magnetic field B_0 . In gradient echo, the insertion of an appropriate gradient field after the first RF pulse, together with a reverse gradient before signal acquisition, allows refocusing of the magnetization in the xy-plane. However, compared to the spin echo, the signal which is lost because of local variations of the



Fig. 1.13 (**a**–**e**) In spin echo T_1 images fluids are dark, like vitreum (**a**) and cerebrospinal fluid (CSF) (**b**). CSF is bright in SE T_2 images (**c**), gray in SE PD images (**d**), and dark in SE T_1 images (**e**)



Fig. 1.14 (a–d) These coronal SE T_2 -weighted images (a, b), and this sagittal STIR image (c), show joint effusion as bright. The corresponding sagittal PD SE image (d) shows degenerative changes of the posterior band of the disc

magnetic field will not be recovered, and the acquired signal will be T_2 *-weighted. This makes gradient echo images more sensitive to changes in magnetic susceptibility of tissues, and it might, therefore, generate artifacts in some cases. On the other hand, such characteristic may provide information regarding deposits with special magnetic properties and clinical relevance, such as calcium or hemosiderin.

The most important advantage of gradient echo imaging is a faster image generation compared to spin echo. In fact, gradient echo generally uses FA less than 90°, allowing the TR to decrease so that a faster image acquisition can be obtained at the expense of a decreased signal and a lower image quality (Fig. 1.15). Figure 1.16 summarizes the signal features of fluids, adipose tissue, and cortical bone, in the case of spin echo sequences.

1.2.7 Image Reconstruction

As in any tomographic imaging technique, MR imaging represents volumes through 2D images.

A voxel is an elementary volume unit of any investigated anatomical region, while the pixel is the elementary unit of the MR image that repre-



Fig. 1.15 Gradient echo gives a good tissue contrast and fast acquisition time, as in this single image from a kinematic MRI sequence

sents that anatomical region. A voxel has three dimensions, while a pixel is two-dimensional. Anatomical regions can be divided into slices, each containing only one layer of voxels. Each slice will be represented in a single two-dimensional image and therefore a pixel can be considered as the bidimensional representation of a voxel (Fig. 1.17). Voxel depth therefore corresponds to slice thickness, whereas the slice plane is a matrix consisting of rows and columns of single pixels.

Spatial encoding in MR is the process by which the location of a single voxel signal source is identified in space. As already mentioned, according to Larmor equation (Eq. 1.1), the precession frequency ω_0 of the individual spin is directly proportional to the intensity of the static magnetic field B_0 , by the gyromagnetic ratio γ , a physical constant typical of the chemical element to which the spin belongs. If the intensity of the main magnetic field B_0 is no longer constant but varies progressively in space according to a linear gradient *G*, even the precession frequency of the



Fig. 1.16 This image summarizes the signal characteristics of fluids (hyperintensity in T_2 and hypointensity in T_1), adipose tissues (medium hyperintensity in T_1 and T_2) and cortical bone (hypointensity in T_1 and T_2) in SE sequences



Fig. 1.17 The voxel is the elementary volume unit of a given anatomical region, while the pixel is the elementary unit of the MR image that represents that anatomical region. The voxel has three dimensions, while the pixel is two-dimensional. Body regions are divided into several slices, each containing only one layer of voxels. Each slice will be represented in a single two-dimensional image and therefore the pixel can be considered as a bidimensional representation of the voxel

individual spin varies linearly in space, allowing us to identify the position of spins along the direction of the gradient, based on their resonance frequency (Fig. 1.18). The spatial encoding system by means of gradients therefore modulates the intensity of the main magnetic field in space by gradient coils that are additional conductive windings contained in the gantry. Their activation is responsible for the intense noise during the MR examination.

In case of 2D sequences, this is how images are formed. Slice selection is achieved through the application of a linear magnetic field gradient along an axis, for example, the *z*-axis. Then along the *z*-axis, Larmor frequencies will change gradually and for each slice there will be a specific resonance. Once again, protons are excited only by an RF pulse with a frequency equal to their Larmor frequency. An RF pulse that matches the Larmor frequency of the protons located on a desired slice will produce excitation of only protons of this slice, and the remaining slices of the body will be devoid of excitement.

After slice selection, voxel coordinates in the xy-plane are to be identified. Coding in one of the two axes, the y-axis, for instance, takes place through the action of a magnetic field gradient along the axis which is switched on for a given time. This gradient will again induce a variation of the spin resonance frequency along the direction of the gradient, resulting in a different precession rate. When the gradient is switched off, the precession frequency will again be the same for all spins, and hence the precession rate, but in the meantime the spins will have accumulated a phase difference, dependent on their position along the y-axis. This is called phase encoding. Coding of different lines requires repetition with different phase gradient intensities.

Finally, encoding along the *x*-axis is achieved by applying a gradient along *x* during echo sampling. This means simultaneously obtaining all frequencies that correspond to the spins positions along a line parallel to the *x*-axis. Therefore, in the selected slice, the *xy*-coordinates of the single voxel will be identified based on the frequency (*x* coordinate) and phase (*y* coordinate) of the acquired signal.

Ultimately after collection of all data from each slice, the result will be a pool of data, corresponding to the whole volume of interest. The orderly set of these signals gives the *k-space*, also known as *raw data space*.

The *k*-space is not a physical space but a two-dimensional matrix of numeric data, k-space and real space images are both a matrix, but pixels do not correspond directly to each other. The *k*-space contains a lot of information about the real space that it represents, but in a coded form. The highest *k* coefficients (corresponding to the higher signal frequencies), which contribute to anatomical image details, are positioned in the peripheral part of the *k*-space. The lowest *k* coefficients (corresponding to the lower signal frequencies), which contribute to image contrast, are positioned in the peripheral part of the *k*-space.



Fig. 1.18 According to Larmor equation, the precession frequency ω_0 of the individual spin is directly proportional to the intensity of the static magnetic field **B**₀. If **B**₀ intensity (vertical axis of the picture) is no longer constant, but varies progressively on a direction of the space according

to a linear gradient G (along the horizontal axis of the picture), then even the precession frequency of the individual spin varies linearly in the space, based on its position along the direction of G. This allows to identify the position of the spins and it is known as spatial encoding



Fig. 1.19 *K*-space image is a two-dimensional matrix of numeric data and it contains a lot of information about the actual space image. K-space and real space images are both matrices, but *k*-space pixels do not correspond directly to

central area (Fig. 1.19). When all *k*-space points are collected at the end of the scan, data can be reconstructed to produce the image. To switch from the k-space (raw data) to the anatomical image, which consists of pixels with different intensity in relation to the spatial position of the anatomic voxel, a mathematical operation is used: the Fourier transform.

real space image pixels. The highest k coefficients contribute to the image's details and are positioned in the peripheral part of the k-space, while the lowest k coefficients contribute to image contrast and are positioned in the central area

The spatial resolution of the final anatomical image is given by the slice thickness and, on the slice plane, by the size of the field of view (FOV), by the number of phase encodings (*k*-space rows) and frequency encodings (number of points read along a line of the *k*-space).

It is worth pointing out that the SNR of the final image also depends on the amount of tissue

contained in the voxel from which the signal is received, and whose dimensions are defined by the spatial resolution chosen during the sequence prescription. Therefore, when all other parameters are the same, the increase in spatial resolution is in fact limited by the intrinsically weak MR signal of the tissue. Sequence repetition might help increase the signal-to-noise ratio; however, this will be limited by movement artifacts and exam duration.

1.2.8 2D and 3D Acquisition

Two-dimensional acquisition can be single-slice or multislice. In the single-slice mode, the slices are fully acquired in succession. In the multislice technique instead, after acquiring a portion of a slice, the system passes on to another slice, to then go back to completing the first slice only afterwards. This allows to minimize acquisition time given that, during the waiting time between an excitation of one slice and the next (TR), it is possible to excite and acquire *k*-space lines of other slices.

3D acquisition techniques have a high SNR and are increasingly used for scanning volumes with high spatial resolution in all directions. Data can be acquired and reconstructed with any orientation in the space, allowing to view the volume of interest in any plane, with isotropic voxel and remarkable detail. 3D acquisitions are made by using an additional phase gradient that enables phase encoding in the slice direction.

Since spatial directions are encoded by magnetic field gradients made with coils inserted into the scanner gantry, MR acquisitions, unlike other imaging techniques, can be directly made along oblique planes. In fact, a field gradient in any oblique direction can be obtained by the sum of the three components, generated by the gradient coils along the Cartesian axes defined by the scanner geometry. With 3D acquisition, there is also the possibility to reformat data after the acquisition, along planes other than the acquisition plane. Good quality of multiplanar reconstructions, which have been obtained on planes that differ from the acquisition plane, is still due to the use of an isotropic voxel (one with three equal sides).

1.2.9 Fast Acquisition Techniques

Over the years, clinical MR systems have offered an increasing number of sequences and techniques, many of which are intended to reduce acquisition times through different modalities of k-space sampling. Below is a brief description of the most important, with referral to other texts for the in-depth analysis (Weishaupt et al. 2008; Elmaoğlu and Çelik 2012).

Turbo spin echo (TSE), also known as fast spin echo (FSE), is a spin echo sequence in which more 180° pulses will come following the first 180° pulse, each of which generates an echo that is acquired and a sequence repetition fills multiple lines of the k-space. Phase encoding is accomplished by switching on a phase gradient, before each 180° pulse and after the echo acquisition, but with inverted polarity, to cancel the phase shift and allow for a new phase encoding before the next echo. The additional parameters for this sequence are echo train length (ETL, also called turbo factor TF), which is the number of echoes generated during a TR, and echo spacing (ESP), which is the time between an echo and the next one, during the same repetition of the sequence. Overall, the acquisition of the entire k-space will require fewer sequence repetitions and therefore a significant reduction in acquisition time.

Some techniques that are currently being used to reduce acquisition times will reduce the number of rows of the acquired *k*-space, even at the price of a rectangular FOV, a pixel no longer squared but rectangular, or imply a SNR reduction, depending on the technique. The result will, in any case, be an image quality that fulfills clinical needs.

Another fast *k*-space acquisition strategy is the EPI (echo planar imaging). In this case, the frequency gradient quickly alternates positive and negative values. Each value is associated with an echo reading, and the *k*-space lines are alternately filled in different directions. With this technique,

it is also possible to acquire the whole *k*-space after a single excitation pulse.

1.2.10 Fat Signal Suppression

Signals in MR imaging mainly generate from water protons contained in tissues. However in many anatomical regions signals coming from fat protons will make too much an impact, with fatty tissue pixels appearing as hyperintense. Because of this, clinical evaluation becomes more difficult and artifacts can be generated.

With the aim of reducing fat signal, different strategies are used and are referred to as *fat suppression* techniques. Clinical sequences often contain a preliminary contrast preparation step to reduce the fat signal.

A sequence typically used for fat suppression is STIR (short tau inversion recovery), which is a more general version of the IR (inversion recovery) sequence.

In IR, before the 90° pulse which moves magnetization in the *xy*-plane, a 180° RF pulse is given, which inverts the magnetization by 180° with respect to the *z*-axis. At the end of the 180° pulse, the M_z component will tend to equilibrium according to relaxation T_1 , as already described. After a specific time interval, known as TI (inversion time), the 90° pulse will be generated starting the previously described sequence (spin echo or gradient echo). The usefulness of the inversion pulse of 180° is due to the fact that, when returning to equilibrium, signals coming from tissues with different T_1 will cross the zero value at different times. By appropriately selecting the TI value, it is possible to eliminate the signal of a tissue by delivering the 90° excitation pulse when the M_z component of that tissue is null, then the signal on the *xy*plane, M_{xy} , will not contain the component to be deleted.

In the case of STIR sequences, the TI time parameter is chosen in such a way as to eliminate the fat signal (Fig. 1.20). Another technique (fat saturation technique) for reducing fat signal is based on the different resonance frequency between water protons and fat protons. Indeed, as mentioned above, the local magnetic field perceived by spins also depends on the molecular environment surrounding them, this causes the resonance frequency of the protons that belong to the fatty molecules to be slightly different from that of the protons bound to water molecules, which are structurally very different. The frequency difference increases as the external magnetic field B_0 increases. Therefore, fat suppression techniques which are known as frequency-selective techniques, involve the use of an initial 90° excitation pulse only in the fat frequency band, followed by the phase shift in the xy-plane, possibly accelerated by a spoiling gradient. Then the M_{τ} component available for acquisition remains only that of





the water component, which will be the primary signal of the image generated by the following part of the sequence.

Another important type of water separation technique is called *chemical shift imaging*. In this case, the shift between the water signal and the fat signal in the xy-plane is employed. After the 90° pulse, which rotates longitudinal magnetization in the xy-plane, transversal magnetization components for fat and water will begin their precession motion with different speeds, and will reach their maximum phase shift of 180° after a certain time interval which gets chosen as TE (echo time). This will allow for the creation of an "out-of-phase" image. After the same time interval, the two components will be back in phase, and an "in-phase" image of the sum of the water and fat signals can be acquired. Among these sequence types, the Dixon technique is widely used. By combining the two "out-of-phase" and "in-phase" images pixel-by-pixel, images of the water-only and fat-only signal are obtained.

Fat suppression techniques are extremely useful in diagnostics. They can highlight small edema areas, as well as involution of muscle and tissue distress. Replacement of yellow bone marrow with edematous infiltrate can also be detected with fat suppression. This can occur in functional overload processes which result in regressive condylar remodeling, with bone marrow edema as one of the first signs of TMJ arthritis (Fig. 1.21a). With these sequences, supramandibular muscles features can be studied too (Fig. 1.21b).

Finally, the so-called *saturation bands* are commonly used in clinical practice (Fig. 1.22). The operator draws these bands onto a scout image in correspondence of the regions from which the signal is to be suppressed. The purpose



Fig. 1.22 Saturation bands are located in correspondence of those regions whose signal is to be suppressed (arteries, fat tissue) in order to reduce artifacts



Fig. 1.21 (a, b) Sagittal STIR sequence—edema of the superior belly of the lateral pterygoid muscle (\mathbf{a} —*arrow*) and subchondral bone edema (\mathbf{b} —*arrow*)

is generally to eliminate the strong fat or local signal from a region close to the area of interest, thus avoiding artifacts. In this case, 90° RF pulses are used in combination with field gradients, conveniently placed in space so that areas can be selected where the signal is to be saturated. The selected signal will now be in the *xy*-plane and will therefore no longer be available along the *z*-axis, at the time when the imaging sequence will start.

1.2.11 Parameters and Optimization of the Sequences

MRI differs from CT and PET scans in that contrast and resolution depend from a lot of different parameters-some intrinsic, some extrinsicthat affect one another variably, according to anatomy and chosen sequence. During acquisition, every MR sequence infers a selection of a certain number of extrinsic parameters, based on tissue type, the volume of interest, SNR, artifacts reduction, magnetic field, coil to be used, duration of the procedure, desired contrast, and resolution (Manoliu et al. 2016). Besides, within a certain system and region, parameters are intertwined: changing one means changing some others, and the same contrast and resolution can be achieved with different sequences or different settings of the same sequence. Such complexity requires constant optimization to take place at three different levels.

The scanner often comes with a set of protocols prepared by the manufacturer for the most common clinical applications. This is often accompanied by further optimization and selection by users (radiologists, technicians, and medical physicists), depending on the specific preferences and clinical needs. Finally, the operator may modify some parameters on each patient and scan to reduce artifacts or scan time (Elmaoğlu and Çelik 2012).

Criteria and skills used in sequence optimization go far beyond the scope of this chapter, however, for quick consultation purposes, a series of the main parameters and their characteristics is presented at the end of this chapter.

1.3 Kinematic and Positional MRI

Kinematic MRI techniques are regularly used in TMJ exams Video 1.1. These are images of the same anatomical area, acquired sequentially during TMJ movement. Images are then displayed progressively (cine-loop), highlighting the anatomical variations during movement.

Kinematic MRI is given by oblique sagittal spin echo or gradient echo sequence (3–4 sections for each condyle) performed during condylar movement. Usually, due to scan time limitation, the larger the number of acquired images, the poorer the anatomical detail. Cineloops represent the physiological or pathological joint's range of motion, and they allow to understand the interaction of bone, disc, and capsuloligamentous components, in closed mouth as well as in a fully opened position.

Positional MRI has been introduced over the last 15 years and has gained growing attention especially in functional examination of lumbar and cervical spine (Ferreiro Perez et al. 2007; Sonenblum et al. 2013; Zhong et al. 2015). Supine MRI is routinely used in the assessment of low back pain and radiculopathy. However, imaging findings often correlate poorly with clinical findings, and this has to do, to some extent, to positional dependence of spinal stenosis.

Upright MRI in flexed and extended position allows patients to reproduce those situations that trigger their symptoms and may enhance findings that were not visible with routine supine imaging (Ferreiro Perez et al. 2007; Alyas et al. 2008; Gilbert et al. 2010; Tarantino et al. 2013; Lao et al. 2014). In fact, some cervical and lumbar disc herniations are seen only in seated or upright studies (Ferreiro Perez et al. 2007). Similarly, dynamic cervical spine MRI is useful for evaluating craniovertebral junction abnormalities and detecting cord compression in atlantoaxial instabilities that were not visible in the neutral position (Gupta et al. 2007). Moreover, upright MRI may point out the change in postural muscle forces between supine and weight-bearing position (Breul 2007; Stemper et al. 2010; Yang et al. 2014).

Positional TMJ MRI has not yet been reported in the literature, even if clinical examination of TMJ is imperatively performed with patients in a seated position. At present, the standing position is only used in cone-beam CT which, unlike MRI, does not allow disc and ligaments evaluation (Khotari et al. 2016).

Special apparati for supine and upright positional evaluation of the spine can also be equipped with TMJ-dedicated coils although, according to present knowledge, diagnostic accuracy of lowfield MRI in evaluation of TMJ internal derangement has not been reported in the literature yet. Despite the lower image quality of low-field MRI, sporadic cases of change in disc position and change in the condylar-glenoid relationship between supine and standing position at closed and open-mouth are encountered. Nevertheless, it is necessary to remind that the diagnostic accuracy of these available technologies has not been statistically investigated and there are no known trials that compare the impact of these technologies on patient outcome against conventional MRI (Chung et al. 2011).

1.4 MR Contrast Agent

The creation of contrast can be modified by particular substances (endogenous or exogenous) that act as contrast agents. Even though different mechanisms are involved, their action is indirect, and it consists primarily in an alteration of the local magnetic field, and therefore modification of the relaxation properties $(T_1 \text{ and } T_2)$ of the near hydrogen protons. As a result, the MR signal coming from the areas where the contrast agents are concentrated will be altered compared to normal tissues. The exogenous contrast agent most commonly used in clinical practice is gadolinium. Thanks to its electronic structure, gadolinium is a paramagnetic substance, i.e., it has the property to magnetize in the presence of a magnetic field. When bound to a molecule with a specific pharmacokinetic property, it generates a reduction in the T_1 of tissues where it accumulates, whose T_1 -weighted images therefore appear hyperintense.

Among the most common applications of contrast agents is the identification of pathological lesions, angiographic studies, and perfusion sequences.

Contrast media TMJ MR can be performed through intravenous injection or direct intraarticular puncture. Intravenous injection is advised for examination of tissues and their blood supply; it is therefore more often carried out for oncology and rheumatology patients.

Intra-articular delivery of a very diluted gadolinium solution allows performing a joint arthrography (TMJ MR arthrography). This has been described in the literature, but it is practically reserved for preoperative evaluations (Rao et al. 1990; Toyama et al. 2000; White et al. 2002; Yang et al. 2005).

Use of gadolinium must have some clinical justification since it is a drug tout court. Most importantly, one should use it judiciously in kidney disease patients since it may lead to nephrogenic systemic fibrosis. Also, gadolinium deposition in the brain has been reported recently. However, there is no evidence of a correlation between its accumulation in the central nervous system and manifestation of neurological diseases (Idée et al. 2014; Weller et al. 2014; Rogosnitzky and Branch 2016; Stojanov et al. 2016).

1.5 MR Scanner

The MR scanner is a technologically sophisticated system consisting of many components. First of all the magnet, which guarantees the primary static magnetic field \mathbf{B}_0 . The most popular commercial systems currently use superconducting magnets or, less frequently, permanent magnets.

The superconducting magnet has a typical tunnel-like body and consists of a solenoid-shaped conductive circuit. The niobium–titanium alloy allows passage of current without virtually any energy dispersion (superconductivity), but it must be maintained at 4 K with cryogenic liquids. Once the current is induced in the solenoid, it continues to circulate without dispersing energy while maintaining the main field \mathbf{B}_0 constant. Very high and uniform fields can be obtained with this technology, and cryogenic gas management has very much improved, so that cryogenic losses are now uncommon. However, in some rare cases, superconducting magnets may suddenly lose superconductivity (quench). This results in a release of energy with the evaporation of cryogenic gases. Quench is one of the major security issues with these magnets, that is why they are managed through security systems in the magnet room, and emergency procedures are known by the operators.

These systems are named "closed" because of their tunnel shape, which in some patients can create claustrophobia issues. Nevertheless, these are the most widely used systems since they are the only ones that allow for stable and high magnetic field strengths (up to 18 T), which ultimately grant SNR, resolution, and overall image quality. In fact, field strength and homogeneity are among the main indicators of the quality of an MR system.

At present, the most common alternative to superconducting magnets consists of *permanent magnets*, made up of ferromagnetic material, which maintain the field without providing energy from the outside or the need for cryogenic gas. With this technology, only low fields can be achieved. The main benefit from a clinical point of view is the shape of the magnet that can be "open," with a large space between the two poles, allowing to overcome most claustrophobia problems. This is also useful in particular applications, such as orthostatic examination, which can be adopted in the study of the temporomandibular joints.

Finally, there are *resistive magnets*, consisting of electromagnets, in which a circulating current generates the field in a classical conductor with energy dispersion, but which are less and less used due to the high running costs and low performance (low fields and poor homogeneity).

The presence of a strong magnetic field also requires some retention of the field in the proximity of the scanner (shielding). This is usually achieved by inserting metal materials into the walls of the room and through windings around the magnet that reduce the value of the field just away from the magnet.

Around the main magnet are gradient coils that allow achieving the required magnetic field gradient for image generation. The intensity of the gradients and the speed of switch on and switch off are among the parameters that indicate the quality of an MR system. In addition, the turning on and off of the gradients generates a mechanical movement of the gradient coils which is responsible for the strong and distinctive noise of MR scanners.

The use of RF pulses requires an RF generator and a receiver, as well as a set of transmitting and receiving coils. Coils are essential in an MR system and are often specifically designed for dedicated clinical applications.

Coil choice is often a key element to success. Multichannel coils are currently the most widely used. They are convenient in that the number of channels is related to SNR and therefore to resolution and reduction of scanning time too.

The fundamental role of RF pulses and the MR signal weakness make it necessary to isolate the scanner from external potential electromagnetic interference and, reversely, to isolate external devices from the MR RF pulses interference. This is achieved through the Faraday cage, which is a closed conductor structure included in the walls of the magnet room. All room openings (doors, windows, pipes, and cables) must be appropriately designed and periodically controlled to maintain their electromagnetic isolation, which is essential to the good quality of exams.

One or more computers will handle all scanner components and allow the user to plan the exams. Currently, image post-processing takes up an essential part of the exam procedure and often occurs on separate computers, with specific software.

Generally, most of the electronics of an MR system is placed in a different technical room, adjacent to the scanner room, whereas the operator console is in a dedicated room with a window on the magnet room, so that the patient can be seen at all times during the examination.

1.6 MRI Safety

1.6.1 General Principles

Magnetic resonance does not use ionizing radiation and can therefore be considered a low-risk technique compared to techniques that use ionizing radiation, such as X-rays for CT scan. However, MR may be risky for operators and patients who therefore have to be evaluated and protected by appropriate means, whether by MRI site design or day-to-day practice. With this regard, there are international committees for non-ionizing radiations that periodically provide indications and guidance. Furthermore, quality controls and security audits are required continuously and regularly carried out by specialized staff (medical physicist with MR expertise).

Below is a quick glance at the main security issues of MR sites, the reader may refer to specific texts for more information (Schellock and Crues 2014; Schellock 2017).

MR scanners use three types of magnetic field: static magnetic field (B_0) , gradient magnetic fields, and RF pulses. The main static B_0 field, in which the patient is immersed throughout the examination, has strength values ranging from 0.3 to 3 T in currently used systems, and maybe even higher in scientific research settings. These values are much higher than the Earth's magnetic field which reaches approximately 70 µT. When considering MRI, one of the greatest risks is that of an accident caused by the socalled bullet effect, which is the attraction of ferromagnetic objects by the magnet itself. For this reason, all materials that are present or taken into the magnet room must be nonmagnetic, and staff and patients should only access the magnet room after making sure they are not wearing any ferromagnetic objects.

In addition, patients are usually asked to fill in a questionnaire to identify implants or devices (such as pacemakers, clips for intracranial aneurysms, cochlear implants, dental prostheses, etc.), as well as metallic residues which may accidentally be present in the body (bullets, metal chips, etc.) and tattoos. Each of them must be carefully evaluated by operators and doctors, assessing their potential hazard. Depending on the position, object material, and the three types of magnetic field, these materials can cause malfunction, displacement or twist, overheating, or merely generate artifacts in the image, thus invalidating the exam (Schellock and Crues 2014; Schellock 2017). Nowadays, most implanted medical devices are MR compatible. However, there are still potentially critical issues with devices such as pacemakers and aneurysm clips, which need to be carefully evaluated on the basis of the risk-versusbenefit ratio of the exam. There are regularly updated references (for instance, MRI safety at www.mrisafety.com) where operators and radiologist can find the information about the MR compatibility of a given device (Mrisafety.com 2018).

For what concerns possible biological and physiological effects of static magnetic fields, unlike ionizing radiations, it is not possible to quantify any deterioration associated with exposure. However, symptoms like nausea, dizziness, and metallic taste have been reported in the literature. This partly explains why, for precautionary purposes, most countries have defined exposure limits to static magnetic fields for staff and patients, at least in clinical routine.

The second type of magnetic fields used in MR is gradient magnetic fields. These oscillate at kHz frequencies, and can therefore stimulate electrically excitable cells (nerves and muscles), induce discomforts or, in limited cases, ventricular fibrillation. In order to avoid these effects, limitations are imposed on the gradient variation rate. Thresholds can be overruled when necessary, through prior assessment, and still within clear limits to avoid higher risks.

Finally, RF electromagnetic fields lie in the range of radio frequencies (resonance frequency), and their possible risk is related to tissue overheating. For this purpose, a specific absorption rate (SAR), which represents the absorbed power per tissue mass unit, has been defined. SAR values must keep below-defined limits to avoid excessive temperature increase in tissues that are undergoing examination. All MR systems are equipped with software that, based on models simulations, patient weight, and anatomical regions, can calculate SARs during sequence prescription and execution, and that can stop the execution if the SAR exceeds the allowed thresholds. Therefore, especially in the case of sequences that use many RF pulses (fast spin echo) on smaller patients (pediatric patients, for instance), SAR limits are also part of the factors to be considered for sequence optimization.

In the case of superconducting magnets (nowadays the majority of magnets and virtually all those above 1 T), the use of cryogenic gases to maintain the magnet's superconductivity entails the potential risk of damaging patients and operators in case of a magnet quench. As already written before, this is a quite unusual situation in which the magnet suddenly loses its superconducting property, thus releasing a significant amount of energy that causes immediate evaporation of cryogenic gas and a real danger of asphyxia and cold injuries for those close to the magnet. In order to manage this potential threat, MR scanner rooms are designed to monitor the level of oxygen and cryogenic gas leaks continuously, and they are also equipped with a ventilation system that will immediately convey all gas outside the building in case of emergency.

As a final mention, since gradients produce a lot of noise, it is advisable to protect patients with earplugs and headphones.

1.6.2 Safety in Dental and TMJ Patients

MRI safety precautions for TMJ patients deserve a particular mention since these subjects may have dental implants, orthodontic appliances, occlusal splints, and so forth. However, general rules from the previous paragraph apply here too.

All parts made of ferromagnetic metals are subject to deflection forces, whereas only magnetically activated implants are an issue during the procedure. Generally, other dental materials and implants will not overheat or cause damage up to 7 T (Gegauff et al. 1990; Blankenstein et al. 2006; Miyata et al. 2012; Ayyıldız et al. 2013; Wezel et al. 2014; Schellock and Crues 2014; Schellock 2017). Because of the same torsional forces and overheating potential, orthodontic archwires should be removed, whereas single brackets can be kept (Regier et al. 2009; Görgülü et al. 2014).

Glossary of Terms

T_1	Spin–lattice relaxation time,
T_2	Spin–spin relaxation time,
	tissue-specific property
PD	Proton density, tissue-spe-
	cific property
SE	Spin echo
TE	Echo time, sequence param-
	eter related to T_2 weighting
	on spin echo
TR	Repetition time, sequence
	parameter related to T_1
	weighting
FA	Flip angle, sequence
	parameter that defines the
	longitudinal rotation angle
	of magnetization after the
	excitation RF pulse
Bandwidth	RF receiver bandwidth
Acquisition matrix	Number of rows (phase
	encoding) and columns
	(frequency encoding)
	acquired in the k-space.
	The matrix dimension, with
	a fixed FOV, defines the
	spatial resolution of the
	final image and the
	SNR. In addition, for the
	most part of the sequences.
	the number of rows heavily
	affects the acquisition time
FOV	Field of view, with a fixed
	acquisition matrix, it is
	related to the resolution of
	the image and the SNR
NEX	Number of excitation cor-
	responds to the number of
	complete sequence repeti-
	tions. The higher the NEX,
	the higher the SNR but
	also the acquisition time

Echo train length, number of echoes in a single repetition of the multi-echo sequence. When it increases, acquisition times decrease but contrast weighing and image quality are affected too

References

ETL

- Alyas F, Connell D, Saifuddin A. Upright positional MRI of the lumbar spine. Clin Radiol. 2008;63:1035–48. https://doi.org/10.1016/j.crad.2007.11.022.
- Ayyıldız S, Kamburoğlu K, Sipahi C, Murat S, Görgülü S, Pişkin B. Radiofrequency heating and magnetic field interactions of fixed partial dentures during 3-tesla magnetic resonance imaging. Oral Surg Oral Med Oral Pathol Oral Radiol. 2013;116:640–7. https://doi. org/10.1016/j.0000.2013.06.035.
- Bag AK, Gaddikeri S, Singhal A, Hardin S, Tran BD, Medina JA, et al. Imaging of the temporomandibular joint: an update. World J Radiol. 2014;6:567–82. https://doi.org/10.4329/wjr.v6.i8.567.
- Blankenstein FH, Truong B, Thomas A, Schröder RJ, Naumann M. Signal loss in magnetic resonance imaging caused by intraoral anchored dental magnetic materials. Rofo. 2006;178:787–93. https://doi. org/10.1055/s-2006-926817.
- Breul R. Biomechanical analysis of stress distribution in the temporomandibular joint. Ann Anat. 2007;189(4):329–35.
- Chung M, Dahabreh IJ, Hadar N, Ratichek SJ, Gaylor JM, Trikalinos TA et al. Emerging MRI technologies for imaging musculoskeletal disorders under loading stress [Internet]. Rockville, MD: Agency for Healthcare Research and Quality (US). (Comparative effectiveness technical briefs, no. 7.). 2011. https:// www.ncbi.nlm.nih.gov/books/NBK82287/. Accessed 23 Jan 2019.
- Elmaoğlu M, Çelik A. MRI Handbook. MR physics, patient positioning, and protocols. New York: Springer; 2012.
- Ferreiro Perez A, Garcia Isidro M, Ayerbe E, Castedo J, Jinkins JR. Evaluation of intervertebral disc herniation and hypermobile intersegmental instability in symptomatic adult patients undergoing recumbent and upright MRI of the cervical or lumbosacral spines. Eur J Radiol. 2007;62:444–8. https://doi.org/10.1016/j. ejrad.2006.12.007.
- Gegauff AG, Laurell KA, Thavendrarajah A, Rosenstiel SF. A potential MRI hazard: forces on dental magnet keepers. J Oral Rehabil. 1990;17:403–10.
- Gilbert JW, Martin JC, Wheeler GR, Storey BB. Lumbar disk protrusion rates of symptomatic patients using magnetic resonance imaging. J Manip Physiol Ther. 2010;33(8):626–9.

- Görgülü S, Ayyıldız S, Kamburoğlu K, Gökçe S, Ozen T. Effect of orthodontic brackets and different wires on radiofrequency heating and magnetic field interactions during 3-T MRI. Dentomaxillofac Radiol. 2014;43:20130356. https://doi.org/10.1259/ dmfr.20130356.
- Gupta V, Khandelwal N, Mathuria SN, Singh P, Pathak A, Suri S. Dynamic magnetic resonance imaging evaluation of craniovertebral junction abnormalities. J Comput Assist Tomogr. 2007;31(3):354–9. https://doi. org/10.1097/01.rct.0000238009.57307.26.
- Idée JM, Fretellier N, Robic C, Corot C. The role of gadolinium chelates in the mechanism of nephrogenic systemic fibrosis: a critical update. Crit Rev Toxicol. 2014;44:895–913. https://doi.org/10.3109/10408444. 2014.955568.
- Khotari SF, Baad-Hansen L, Hansen LB, Bang N, Sørensen LH, Eskildsen HW, et al. Pain profiling patients with temporomandibular joint arthralgia and osteoarthritis diagnosed with different imaging techniques. J Headache Pain. 2016;17:61. https://doi. org/10.1186/s10194-016-0653-6.
- Lao L, Zhong G, Li Q, Liu Z. Kinetic magnetic resonance imaging analysis of spinal degeneration: a systematic review. Orthop Surg. 2014;6:294–9. https://doi. org/10.1111/os.12137.
- Manoliu A, Spinner G, Wyss M, Filli L, Erni S, Ettlin DA, et al. Comparison of a 32-channel head coil and a 2-channel surface coil for MR imaging of the temporomandibular joint at 3.0T. Dentomaxillofac Radiol. 2016;45:20150420. https://doi.org/10.1259/ dmfr.20150420.
- Miyata K, Hasegawa M, Abe Y, Tabuchi T, Namiki T, Ishigami T. Radiofrequency heating and magnetically induced displacement of dental magnetic attachments during 3.0 T MRI. Dentomaxillofac Radiol. 2012;41:668–74. https://doi.org/10.1259/ dmfr/17778370.
- Mrisafety.com. MRI safety home. 2018. http://www.mrisafety.com/. Accessed 15 Aug 2018.
- Niraj LK, Patthi B, Singla A, Gupta R, Ali I, Dhama K, et al. MRI in dentistry - a future towards radiation free imaging. J Clin Diagn Res. 2016;10:ZE14–9. https:// doi.org/10.7860/JCDR/2016/19435.8658.
- Rao VM, Farole A, Karasick D. Temporomandibular joint dysfunction: correlation of MR imaging, arthrography, and arthroscopy. Radiology. 1990;174:663–7. https:// doi.org/10.1148/radiology.174.3.2305046.
- Regier M, Kemper J, Kaul MG, Feddersen M, Adam G, Kahl-Nieke B, et al. Radiofrequency-induced heating near fixed orthodontic appliances in high field MRI systems at 3.0 Tesla. J Orofac Orthop. 2009;70:485– 94. https://doi.org/10.1007/s00056-009-9923-0.
- Rogosnitzky M, Branch S. Gadolinium-based contrast agent toxicity: a review of known and proposed mechanisms. Biometals. 2016;29:365–76. https://doi. org/10.1007/s10534-016-9931-7.
- Schellock FG. Reference manual for magnetic resonance safety, implants, and devices. Los Angeles: Biomedical Research Publishing Group; 2017.

- Schellock FG, Crues JV III. MRI: bioeffects, safety and patient management. Los Angeles: Biomedical Research Publishing Group; 2014.
- Sonenblum SE, Sprigle SH, Cathcart JM, Winder RJ. 3-dimensional buttocks response to sitting: a case report. J Tissue Viability. 2013;22:12–8. https://doi. org/10.1016/j.jtv.2012.11.001.
- Stemper BD, Baisden JL, Yoganandan N, Pintar FA. Determination of normative neck muscle morphometry using upright MRI with comparison to supine data. Aviat Space Environ Med. 2010;81(9):878–82.
- Stojanov DA, Aracki-Trenkic A, Vojinovic S, Benedeto-Stojanov D, Ljubisavljevic S. Increasing signal intensity within the dentate nucleus and globus pallidus on unenhanced T1W magnetic resonance images in patients with relapsing-remitting multiple sclerosis: correlation with cumulative dose of a macrocyclic gadolinium-based contrast agent, gadobutrol. Eur Radiol. 2016;26:807–15. https://doi.org/10.1007/ s00330-015-3879-9.
- Tarantino U, Fanucci E, Iundusi R, Celi M, Altobelli S, Gasbarra E, et al. Lumbar spine MRI in upright position for diagnosing acute and chronic low back pain: statistical analysis of morphological changes. J Orthop Traumatol. 2013;14(1):15–22. https://doi. org/10.1007/s10195-012-0213-z.
- Toyama M, Kurita K, Koga K, Rivera G. Magnetic resonance arthrography of the temporomandibular joint. J Oral Maxillofac Surg. 2000;58:978–83. https://doi. org/10.1053/joms.2000.8738.

- Weishaupt D, Köchli VD, Marincek B. How does MRI work? An introduction to the physics and function of magnetic resonance imaging. 2nd ed. Berlin: Springer; 2008.
- Weller A, Barber JL, Olsen ØE. Gadolinium and nephrogenic systemic fibrosis: an update. Pediatr Nephrol. 2014;29:1927–37. https://doi.org/10.1007/ s00467-013-2636-z.
- Wezel J, Kooij BJ, Webb AG. Assessing the MR compatibility of dental retainer wires at 7 Tesla. Magn Reson Med. 2014;72:1191–8. https://doi.org/10.1002/ mrm.25019.
- White LM, Schweitzer ME, Weishaupt D, Kramer J, Davis A, Marks PH. Diagnosis of recurrent meniscal tears: prospective evaluation of conventional MR imaging, indirect MR arthrography, and direct MR arthrography. Radiology. 2002;222:421–9. https://doi. org/10.1148/radiol.2222010396.
- Yang C, Zhang SY, Wang XD, Fan XD. Magnetic resonance arthrography applied to the diagnosis of intraarticular adhesions of the temporomandibular joint. Int J Oral Maxillofac Surg. 2005;34:733–8. https://doi. org/10.1016/j.ijom.2005.02.011.
- Yang J, Chu D, Dong L, Court LE. Advantages of simulating thoracic cancer patients in an upright position. Pract Radiat Oncol. 2014;4(1):e53–8.
- Zhong G, Buser Z, Lao L, Yin R, Wang JC. Kinematic relationship between missed ligamentum flavum bulge and degenerative factors in the cervical spine. Spine J. 2015;15:2216–21. https://doi.org/10.1016/j. spinee.2015.06.048.

Studio Futura, Ciriè, Turin, Italy e-mail: gino.carnazza@studiofutura.com

L. Luberto (🖂) · G. Carnazza · S. Garberoglio

Other TMJ Imaging Modalities

Luca Luberto, Sara Garberoglio, and Gino Carnazza

Key Points

- Panoramic (orthopantomogram—DPT: dental panoramic tomography) and plain radiographs may allow a good screening for gross degenerative or traumatic bony changes, but they give limited information because of the anatomy of the region, superimpositions of overlapping structures, and geometric distortion.
- Conventional tomography and computed tomography (CT) overcome these diagnostic difficulties and they should be adopted for specific evaluation of TMJ osseous changes (i.e., fractures), identification of intra-articular calcifications (i.e., pseudogout arthritis), and for all conditions in which a large view of the maxilla is required with multiplanar and 3D reconstructions (i.e., craniofacial malformations).
- Cone beam CT (CBCT) offers some advantages over multidetector computed tomography (MDCT), namely image accuracy, X-ray beam limitation, fast acquisition time, if a head and neck CT study is not required.

 TMJ ultrasound has by now gained widespread acceptance in rheumatology and as a guiding tool in interventional procedures. Ultrasounds are becoming more common in preliminary evaluation of anterior disc dislocations but their capacity and capability of detecting medial, lateral, and posterior dislocations are yet to be confirmed.

Many diagnostic imaging techniques—with and without ionizing radiations—such as panoramic radiography, plain radiographs, tomography, computed tomography (CT), and magnetic resonance imaging (MRI), have been proposed for the assessment of TMJ pathologies.

Over the last 20 years, the study of the temporomandibular joint (TMJ) has mainly moved from radiographic and tomographic imaging to CT and MRI; however, general agreement about TMJ instrumental diagnosis still has to be reached.

While panoramic and plain radiographs allow the detection of gross degenerative or traumatic bony changes, they are often limited due to anatomy of the region, overlapping structures, and geometric distortion. Conventional tomography and the introduction of CT imaging helped overcome these diagnostic difficulties, but they do not have the potential to investigate discal and capsuloligamentous components of TMJ. This is why the advent of MRI marked an important step in the diagnostic evaluation of TMJ disorders.



2

[©] Springer Nature Switzerland AG 2020

T. Robba et al. (eds.), *MRI of the Temporomandibular Joint*, https://doi.org/10.1007/978-3-030-25421-6_2

²⁵
Nevertheless, CT remains the first choice for specific evaluation of TMJ osseous changes (fractures, post-surgical evaluations), for intraarticular calcifications (synovial chondromatosis or metabolic arthritis), and for all conditions in which a large view of the maxillomandibular complex is required with multiplanar and 3D reconstructions (craniofacial malformations, hyperplasia of coronoid and styloid processes, tumors) (Honda et al. 2006; Huntjens et al. 2008; Farronato et al. 2010; Venturin et al. 2010; Meng et al. 2012; Ferreira et al. 2016).

CT can be performed as multislice computerized tomography (MSCT) or as cone beam computerized tomography (CBCT). Cone beam CT (CBCT) was introduced as an alternative to MSCT and is now considered appropriate for a wide range of craniofacial indications. CBCT offers some advantages over MSCT, namely image accuracy, X-ray beam limitation, and fast acquisition time. European guidelines, which have been developed thanks to the SEDENTEXCT project, concluded that CBCT can be considered as an alternative to MSCT, as the radiation dose from CBCT is lower (Holdroyd and Gulson 2009; Radiation Protection 2011). As a consequence, in the last decade, the use of CBCT has increased in various dental specialties for the investigation of maxillofacial structures and this is why CBCT has emerged as a cost- and doseeffective imaging modality for the diagnostic assessment of the TMJ.

2.1 Radiographic Imaging

Several methods are available with conventional extra-oral X-ray equipment. Radiographic examination of the TMJ is possible with orthopanto-mograms, with/without planigraphy (TMJ-specific programs) and plain radiography.

In the past years, radiographic examination of the temporomandibular joint (TMJ) together with linear tomography were the only imaging modalities to investigate TMJ structures (cortical and trabecular architecture of bony structures) and structural changes (extent and progression of osseous changes) (Crow et al. 2005; Ferreira et al. 2016). Moreover, radiographic investigation of TMJ would provide some functional relations between the condyle, the articular tubercle, and the fossa (Ferreira et al. 2016). CBCT has widely replaced radiographic examination of the TMJ as it can overcome the diagnostic difficulties caused by the superimposition of contiguous anatomical structures. Nevertheless one should bear in mind that the effective dose for a TMJ CBCT may be 20–40 times the effective dose of TMJ radiography, depending on the irradiated volume (Holdroyd and Gulson 2009).

2.1.1 Orthopantomograms With or Without Planigraphy

Panoramic radiography can capture an overview of the maxillomandibular complex, it requires a low patient radiation dose and is quite easy to perform. This procedure may be a useful screening tool for differential diagnosis of odontogenic conditions which may mimic TMJ symptoms, and it can highlight gross bony changes in the condyle (as for arthritis in children), in articular morphology (i.e., asymmetries, condylar changes in size and shape) (Fig. 2.1a, b), and intraarticular calcification (Fig. 2.2a, b) (Crow et al. 2005; Ferreira et al. 2016).

Image formation in orthopantomograms follows the basic principles of linear tomography: the X-ray beam is emitted obliquely along the long axis of the condyle in a non-parallel manner, and causes superimpositions and variations in size of structures. It has to be considered that the medial condylar surface maintains a more posterior horizontal rotation with respect to the rear surface, as the condyle is angled between 15° and 33° in a sagittal plane (Fig. 2.3a-c). Moreover, in panoramic radiography, image formation also depends on the position of the TMJ within the image layer, because only a part of the condyle falls within the image layer and is visualized without distortions (Ladeira et al. 2015). Furthermore, projective overlapping (Fig. 2.4) of different anatomical structures can occur, especially in the region of the fossa and of the tubercle and this makes them often difficult to



Fig. 2.1 Orthopantomograms showing mandibular asymmetries, such as an underdeveloped right condyle (**a**—*arrow*) and a small left condylar head due to a major erosion related to rheumatoid arthritis (**b**—*arrow*)



Fig. 2.2 Orthopantomogram showing a large loose intra-articular body of the left TMJ (a). MRI of the same patient is seen in (b)

examine correctly, with the risk of inaccurate diagnosis (Crow et al. 2005; Mawani et al. 2005; Hintze et al. 2009; Ferreira et al. 2016). Therefore, it is generally agreed that the diagnostic pathology TMJ should not be solely based upon panoramic radiography (Hintze et al. 2009).

In addition to panoramic view, any modern device for panoramic radiography will include specific programs to obtain an anatomical overview of the TMJ (*planigraphy* Fig. 2.5a, b). Depending on the different available algorithms, multiple projections for each joint can be obtained on the same film, such as two projections in the sagittal plane (an open mouth and a closed mouth, respectively), documenting the relationship of the condyle in the articular fossa in maximum habitual intercuspation and at the end of excursion in maximum mouth opening. Following this procedure, images can be obtained without major projective overlapping and with a good visualization of the surrounding anatomical structures, such as the styloid process, the mastoid process, and the zygomatic arch. This method is useful to obtain an initial functional evaluation and any basic assessment regarding condyle size, presence of displaced fractures, or ankylosis (Epstein et al. 2001; Hintze et al. 2009; Ferreira et al. 2016).

2.1.2 Plain Radiography

Similarly to panoramic radiography, plain radiographs provide a global anatomical assessment of the condyle, the fossa, and the articular tubercle, but fail to provide an accurate evaluation of the TMJ, because of limitations due to structure



Fig. 2.3 In (a) (Axial SE T1 image) and (b) (Coronal SE T2 image) give an understanding of how the medial and lateral pole project onto different points of



Fig. 2.4 Superimposition of the condyle on the articular eminence and the zygomatic arch (*arrows*)

the panoramic X-ray. In (c) the lateral pole is seen in the antero-inferior region of the condyle (*), whereas the medial pole lies in the postero-superior part (\times)

superimposition and sometimes due to artifacts (for instance, those determined by patient movement) (Barghan et al. 2012).

The literature about this approach is quite outdated; however, in the past years, projections have been set to evaluate TMJ, for instance, using oblique lateral transcranial and Towne projections (Fig. 2.6a–c), but most authors would agree that projections used to evaluate TMJ disorders (i.e., transcranial, transpharyngeal, and transmaxillary) are not reliable from a diagnosis point of view (Lindvall et al. 1976). Furthermore, positioning of the patient can be complex, especially in the case

of trauma. Nevertheless this imaging method is widely available and it can be useful for identifying coarse bone changes and displaced fractures



Fig. 2.5 Planigraphy of the temporomandibular joint, in closed mouth position (a) and open mouth position (b)



Fig. 2.6 Arrows indicate the condyle in closed-mouth oblique lateral transcranial projections (a) and open mouth (b), and Towne projection (c)

Fig. 2.6 (continued)

of the condylar head and neck (Hintze et al. 2009; Ferreira et al. 2016; Caruso et al. 2017). Often this projection will not be carried out correctly, and the patient will need further assessment by means of a CT scan (Fig. 2.7a-c).

2.2 Tomography

Tomography used to be the most accurate imaging technique for TMJ evaluation. In most cases it was performed as lateral tomograms in the sagittal plane, although some authors reported that frontal tomograms could be useful as well (Fig. 2.8a-c) (Hintze et al. 2009).

This approach however proved limited in its reliability, and this imaging modality ended up being replaced by CBCT due to its higher accuracy in depicting condylar bony erosion (Honey et al. 2007).

2.3 Computed Tomography (CT)

2.3.1 **Multislice Computed** Tomography (MSCT)

Multislice computed tomography (MSCT) consists in a rotating gantry with an X-ray source and digital X-ray detectors, which are located directly opposite the X-ray source. During a CT scan, the patient lies on a bed that slowly moves through the gantry while the X-ray tube rotates around the patient (Fig. 2.9a, b). The radiation emitted is a narrow fan-shaped X-ray beam and it is typically emitted at 80-150 mA and 120 kVp (kilovoltage peak) for the maxillofacial region. The emitted radiation passes through the patient and is picked up by the detector as transmitted radiation. The collected data are transmitted to a computer, and a tissue density map in Hounsfield Units (HU) is obtained. Data are represented by numbers in a matrix of pixel (usually 512×512 pixels). The fundamental picture element is the pixel, and every pixel is attributed a tissue density in HU. Every slice is divided in voxel (volumetric picture element), whose base has the pixel dimensions, while the voxel height is the thickness of the slice as set by the radiologist. The thickness of the tissue represented in each image slice can vary depending on the CT machine being used, but it usually ranges from less than 1 mm to a few millimeters. The simultaneous movement of patient and gantry allows to thoroughly cover a volume of interest. Image slices can either be displayed individually or stacked together by the computer to generate a multiplanar and 3D image of the explored volume.

2.3.2 **Cone Beam Computed Tomography (CBCT)**

In its early days, computed tomography of the TMJ was exclusively performed as multislice computed tomography (MSCT). However, in the years that followed the introduction of CBCT, an increased use of this imaging modality for evaluation of the jaws and the TMJ has been observed. This is because of some essential differences between CBCT and MSCT (Palconet et al. 2012; Ferreira et al. 2016; Caruso et al. 2017):

- CBCT uses a three-dimensional cone-shaped X-ray beam instead of the collimated fan beam used in MSCT (Fig. 2.10). The patient's head keeps still during the synchronous movement

С R





Fig. 2.7 Emergency Department Radiology detection of a condylar fracture (suboptimal projection in **a**, **b**), which was subsequently assessed via CT imaging (**c**)

of both the X-ray tube and detector, and the patient may be either in orthostatic position or sitting. The tube detector system performs only one 360° (or 270° , sometimes 180°) rotation around the head of the patient with a constant beam angle. The acquisition lasts from 5 to 70 s depending on the amplitude of the region under examination.

 A relevant difference between CBCT and MSCT is the choice of the *field of view* (FOV). In MSCT the FOV is substantially fixed due to the fan beam angle, achieved by adjustment of collimation. In CBCT the FOV is a cylindrical or spherical volume, because the cone-beam X-ray is coupled with a 2D extended digital array providing a wide area detector. A collimator restricts the beam to match the sensor size in order to focus on the anatomical region of interest. Thus CBCT has its FOV as a flexible volume and most CBCT units will scan small regions of a few centimeters; however, others can scan the entire craniofacial complex as well (Honda et al. 2006; Honey et al. 2007; Barghan et al. 2012). The best imaging quality is



Fig. 2.8 Rheumatoid arthritis—sagittal tomography, right TMJ in closed mouth position (a), open mouth position (b), and coronal view (c)

achieved using small FOVs (allowing to optimize spatial resolution depending on hardware and software with fixed matrix), this also leads to a significant decrease of radiation dose, proportional to the FOV voxel size. According to some authors the best imaging quality is obtained for 6- or 9-in. FOV with a significant decrease of radiation dose, proportional to the FOV voxel size (Librizzi et al. 2011). Depending on patient size, both TMJs can be evaluated with one 9-in. acquisition, while a 6-in. FOV needs 2 scans, hence doubling the exposition. The acquisition of two 6-in. FOV TMJ scans would allow the best image quality and detect small erosions, with a still limited X-ray exposition, compared to the 9- and 12-in. FOV. Small size FOVs allow good quality image with the same matrix. Matrix width is usually fixed (due to computation limits) and small FOVs allow to optimize spatial resolution depending on hardware and software.

 As in CBCT the FOV is volumetric, the voxel is always *isotropic* (meaning that the voxel is a perfect cube, with the slice thickness equal



Fig. 2.9 Multislice computed tomography—the patient lies on a table that slides into the gantry as the tube rotates around the patient (**a**) and emits a narrow fan-shaped

X-ray beam (**b**). The radiation is then caught by a multidetector (a detector with numerous layers)



Fig. 2.10 CBCT—a three-dimensional cone-shaped beam is emitted by the X-ray tube

to the base and the height of the pixel) and its side ranges from 0.075 to 0.400 mm per side. Image acquisition is performed during the X-ray tube movement at certain fixed degree intervals, producing single projection images, known as *basis* images. *Basis* images are similar to lateral teleradiography, each slightly offset from the other. Basis projection images are referred to as the projection data and are reconstructed into a 3D data set. As the voxel is isotropic, CBCT *multiplanar reconstructions* have high accuracy and are performed in orthogonal planes (axial, coronal, sagittal) or curvilinear planes. *Panoramic images* can be obtained and cross-sectional images perpendicular to the curve of the dental arch can be obtained. Additional dedicated software can be used to perform curved reconstructions along some complex anatomical structures, such as the mandibular nerve (Honey et al. 2007; Koyama et al. 2007).

- Exposure parameters are also different in CBCT and in MSCT, being the milliampere (mA) setting very low (1–15 mA), while the peak kilovoltage (kVp) usually ranges from 85 to 120 kVp. Thus the absorbed dose is reduced and a significant reduction of artifacts is reached, especially those determined by X-ray beam hardening (e.g., as in the case of prostheses) (Honey et al. 2007; Barghan et al. 2012).
 - Because of the reduced milliamperage and FOV characteristics, the X-ray dose may be substantially smaller compared to MSCT for TMJ evaluation. Several studies confirm the *reduced dose* in CBCT and some authors focus on CBCT evaluation of the TMJ compared to MSCT examination: MSCT exhibited higher organ doses for all organs except the salivary glands and lymphatic nodes, with a 20% higher effective dose than CBCT

(Hashimoto et al. 2003, 2007; Kadesjö et al. 2015). For bilateral TMJ evaluation, some authors suggested that the estimated effective dose using the optimized exposure parameters would be 92 mSv for a bilateral CBCT examination and 124 mSv for a MSCT; nonetheless, owing to the large range of reported effective doses and large technical differences between the CBCT models, dose comparison between CBCT and MSCT for TMJ diagnostics may be complex (Kadesjö et al. 2015). Existing data suggest that the most important issue to account for is dose optimization (i.e., suitable FOVs and optimized exposure parameters for various diagnostic tasks), rather than the choice of CT modality. The importance of dose exposure should not be overlooked also considering radio-sensitivity of organs and glands in the neck and head district. For instance, recent studies assessed absorbed ionizing radiation by thyroid gland using CBCT, reporting an average of phantom surface radiation dose at the thyroid gland of 0.48 mGy (Setti da Rocha et al. 2017). For dose reduction reasons CBCT operates at much lower mA setting than MDCT resulting in more quantum noise (inconsistent distribution of signal) resulting in grainier images. Increasing radiation dose (mA) reduces noise and may improve spatial resolution. Use of smaller voxel needs an increased amount of radiation to maintain good image quality.

- No significant difference was found between CBCT and MSCT in detecting osseous abnormalities, erosion, and osteophytes (Honda et al. 2006; Zain-Alabdeen and Alsadhan 2012). On the other hand, it should be considered that, from a biological standpoint, CBCT is a quite expensive method when compared to conventional tomography and radiographs (the absorbed dose is 20–30 higher than the dose delivered during conventional radiography) (Holdroyd and Gulson 2009). Thus, some common and easy doubt may be solved by conventional tomography and radiographs.

In summary, CBCT has an acceptable accuracy for diagnosing osseous TMJ abnormalities

with fairly high sensitivity and appears to be cost- and dose-effective. In most studies, high specificity is reported and the diagnostic accuracy of CBCT seems to be comparable with CT for TMJ diagnostics. CBCT was found to be better than conventional radiographic examinations as well as MRI in assessing the TMJ. However, it should be emphasized that the diagnostic information obtained is limited to the morphology of the osseous joint components, cortical bone integrity, and subcortical bone destruction/production. For evaluation of soft-tissue abnormalities, MRI is mandatory (Larheim et al. 2015).

2.4 Ultrasound (US)

TMJ ultrasound has by now gained widespread acceptance because of its acknowledged role in rheumatology and most importantly because of its importance as a guiding tool in interventional procedures (Levorova et al. 2015). US is becoming more common in preliminary evaluation of disc dislocations as it is easily available in most healthcare institutions, is non-invasive, and is readily carried out (Klatkiewicz et al. 2018). Encouraging results currently come from metanalyses reporting sensitivity and specificity between 70% and 85%, with better results in case of anterior disc displacement with reduction in maximum opening (Melis et al. 2007; Li et al. 2012; Dong et al. 2015; Klatkiewicz et al. 2018).

It is yet to be defined US capability of detecting medial, lateral, and posterior dislocations, and this is why further studies are needed and should be carried out with more accurate study procedures so as to assess US effective diagnostic accuracy in all disc dislocations (Li et al. 2012). Metanalyses reach then the conclusion that MRI is fundamental in case of any US finding that could potentially modify the therapeutic approach, or in case of disagreement between a clinical positive finding and a negative US (Li et al. 2012).

One more aspect which is to be standardized is the TMJ method of evaluation, as it is easy to understand that not only a static assessment is needed (closed and open mouth, in coronal, oblique, and axial planes), but also a dynamic orthostatic evaluation during the whole articular excursion (Klatkiewicz et al. 2018).

US oblique–coronal views are taken along the longitudinal axis of the condyle and ramus, whereas axial views are taken along an axis perpendicular to the previous one. These views will show the same anatomical structures that can be seen in a coronal MRI even though they are less comprehensive.

References

- Barghan S, Tetradis S, Mallya S. Application of cone beam computed tomography for assessment of the temporomandibular joints. Aust Dent J. 2012;57:109–18. https://doi.org/10.1111/j.1834-7819.2011.01663.x.
- Caruso S, Storti E, Nota A, Ehsani S, Gatto R. Temporomandibular joint anatomy assessed by CBCT images. Biomed Res Int. 2017;2017:1–10. https://doi.org/10.1155/2017/2916953.
- Crow HC, Parks E, Campbell JH, Stucki DS, Daggy J. The utility of panoramic radiography in temporomandibular joint assessment. Dentomaxillofac Radiol. 2005;34:91–5. https://doi.org/10.1259/ dmfr/24863557.
- Dong Y, He S, Zhu L, Dong TY, Pan SS, Tang LJ, Zhu ZF. The diagnostic value of high resolution ultrasonography for the detection of anterior disc displacement of the temporomandibular joint: a meta-analysis employing the HSROC statistical model. Int J Oral Maxillofac Surg. 2015;44:852–8.
- Epstein JB, Caldwell J, Black G. The utility of panoramic imaging of the temporomandibular joint in patients with temporomandibular disorders. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2001;92:236–9. https://doi.org/10.1067/moe.2001.114158.
- Farronato G, Garagiola U, Carletti V, Cressoni P, Mercatali L, Farronato D. Change in condylar and mandibular morphology in juvenile idiopathic arthritis: cone beam volumetric imaging. Minerva Stomatol. 2010;59:519–34.
- Ferreira LA, Grossmann E, Januzzi E, de Paula MV, Carvalho AC. Diagnosis of temporomandibular joint disorders: indication of imaging exams. Braz J Otorhinolaryngol. 2016;82:341–52. https://doi. org/10.1016/j.bjorl.2015.06.010.
- Hashimoto K, Arai Y, Iwai K, Araki M, Kawashima S, Terakado M. A comparison of a new limited cone beam computed tomography machine for dental use with a multidetector row helical CT machine. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2003;95:371–7. https://doi.org/10.1067/moe.2003.120.
- Hashimoto K, Kawashima S, Kameoka S, Akiyama Y, Honjoya T, Ejima KI, Sawada K. Comparison of image

validity between cone beam computed tomography for dental use and multidetector row helical computed tomography. Dentomaxillofac Radiol. 2007;36:465– 71. https://doi.org/10.1259/dmfr/22818643.

- Hintze H, Wiese M, Wenzel A. Comparison of three radiographic methods for detection of morphological temporomandibular joint changes: panoramic, scanographic and tomographic examination. Dentomaxillofac Radiol. 2009;38:134–40. https://doi. org/10.1259/dmfr/31066378.
- Holdroyd JR, Gulson AD. The radiation protection implication of the use of cone beam computed tomography (CBCT) in dentistry - what you need to know. Oxfordshire: Health Protection Agency; 2009. Available at www.bsdmfr.org.uk/wp-content/ uploads/2014/12/hpaguidance.pdf.
- Honda K, Larheim TA, Maruhashi K, Matsumoto K, Iwai K. Osseous abnormalities of the mandibular condyle: diagnostic reliability of cone beam computed tomography compared with helical computed tomography based on an autopsy material. Dentomaxillofac Radiol. 2006;35:152–7. https://doi.org/10.1259/dmfr/15831361.
- Honey BO, Scarfe WC, Hilgers MJ, Klueber K, Silveira A, Haskell B, Farman A. Accuracy of cone-beam computed tomography imaging of the temporomandibular joint: comparisons with panoramic radiology and linear tomography. Am J Orthod Dentofac Orthop. 2007;132:429–38. https://doi.org/10.1016/j. ajodo.2005.10.032.
- Huntjens E, Kiss G, Wouters C, Carels C. Condylar asymmetry in children with juvenile idiopathic arthritis assessed by cone-beam computed tomography. Eur J Orthod. 2008;30:545–51. https://doi.org/10.1093/ejo/ cjn056.
- Kadesjö N, Benchimol D, Falahat B, Näsström K, Shi XQ. Evaluation of the effective dose of cone beam CT and multislice CT for temporomandibular joint examinations at optimized exposure levels. Dentomaxillofac Radiol. 2015;44:20150041. https://doi.org/10.1259/ dmfr.20150041.
- Klatkiewicz T, Gawriołek K, Pobudek Radzikowska M, Czajka-Jakubowska A. Ultrasonography in the diagnosis of temporomandibular disorders: a metaanalysis. Med Sci Monit. 2018;24:812–7. https://doi. org/10.12659/MSM.908810.
- Koyama J, Nishiyama H, Hayashi T. Follow-up study of condylar bony changes using helical computed tomography in patients with temporomandibular disorder. Dentomaxillofac Radiol. 2007;36:472–7. https://doi. org/10.1259/dmfr/28078357.
- Ladeira DBS, Da Cruz AD, De Almeida SM. Digital panoramic radiography for diagnosis of the temporomandibular joint: CBCT as the gold standard. Original Research Imaginology. Braz Oral Res. 2015;29:1–7. https://doi.org/10.1590/1807-3107BOR-2015.
- Larheim TA, Abrahamsson AK, Kristensen M, Arvidsson LZ. Temporomandibular joint diagnostics using CBCT. Dentomaxillofac Radiol. 2015;44:20140235. https://doi.org/10.1259/dmfr.20140235.

- Levorova J, Machon V, Hirjak D, Foltan R. Ultrasoundguided injection into the lower joint space of the temporomandibular joint. Int J Oral Maxillofac Surg. 2015;44:491–2. https://doi.org/10.1016/j. ijom.2014.12.013.
- Li C, Su N, Yang X, Yang X, Shi Z, Li L. Ultrasonography for detection of disc displacement of temporomandibular joint: a systematic review and meta-analysis. J Oral Maxillofac Surg. 2012;70:1300–9. https://doi. org/10.1016/j.joms.2012.01.003.
- Librizzi ZT, Tadinada AS, Valiyaparambil JV, Lurie AG, Mallya SM. Cone-beam computed tomography to detect erosions of the temporomandibular joint: effect of field of view and voxel size on diagnostic efficacy and effective dose. Am J Orthod Dentofac Orthop. 2011;140:e25–30.
- Lindvall AM, Helkimo E, Hollender L, Carlsson GE. Radiographic examination of the temporomandibular joint. Dentomaxillofac Radiol. 1976;5:24–32.
- Mawani F, Lam EW, Heo G, McKee I, Raboud DW, Major PW. Condylar shape analysis using panoramic radiography units and conventional tomography. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2005;99:341–8. https://doi.org/10.1016/j. tripleo.2004.07.011.
- Melis M, Secci S, Ceneviz C. Use of ultrasonography for the diagnosis of temporomandibular joint disorders: a review. Am J Dent. 2007;20:73–8.
- Meng Q, Chen S, Long X, Cheng Y, Deng M, Cai H. The clinical and radiographic characteristics of condylar osteochondroma. Oral Surg Oral Med Oral Pathol Oral

Radiol. 2012;114:e66–74. https://doi.org/10.1016/j. 0000.2012.01.016.

- Palconet G, Ludlow JB, Tyndall DA, Lim PF. Correlating cone beam computed tomography results with temporomandibular joint pain of osteoarthritic origin. Dentomaxillofac Radiol. 2012;41:126–30. https://doi. org/10.1259/dmfr/60489374.
- Radiation protection: Cone Beam CT for Dental and Maxillofacial Radiology - Evidence Based Guidelines. Energy Protection Radiation n. 172. Directorate General for Energy, Unit D4-Radiation Protection. Euratom, Seventh Framework. Luxembourg: European Commission. 2011; ISSN 1681-6803. Available at http://www.sedentexct.eu/content/guidelines-cbctdental-and-maxillofacial-radiology.htm.
- Setti da Rocha ASP, de Mello Aguiar G, Tulio AP, Ditzel AS, Filipov D. Evaluation of thyroid radiation dose using cone beam computed tomography. Radiat Prot Dosim. 2017;175:368–72. https://doi.org/10.1093/ rpd/ncw360.
- Venturin JS, Shintaku WH, Shigeta Y, Ogawa T, Le B, Clark GT. Temporomandibular joint condylar abnormality: evaluation, treatment planning, and surgical approach. J Oral Maxillofac Surg. 2010;68:1189–96. https://doi.org/10.1016/j.joms.2009.08.002.
- Zain-Alabdeen E, Alsadhan R. A comparative study of accuracy of detection of surface osseous changes in the temporomandibular joint using multidetector CT and cone beam CT. Dentomaxillofac Radiol. 2012;41:185– 91. https://doi.org/10.1259/dmfr/24985971.

37

TMJ and MRI Anatomy

Nicolò Margolo, and Gino Carnazza

Giulia Tanteri, Roberto Prandi, Paolo Lodo,

Key Points

- The temporomandibular joint is characterized by unique features under embryological, anatomical, developmental, and functional perspectives.
- The TMJ is a ginglymoarthrodial synovial joint made up by two separate compartments. The two condyles (left and right) are part of one single mandibular body and these are connected to the cranial base, thanks to capsule and ligaments.
- The components of the temporomandibular joint are the condylar head, the glenoid fossa, and the disc. The latter is made of dense fibrous connective tissue and, in physiological conditions, should be interposed between condyle and fossa.
- The capsule encloses the joint and its inner surface is covered by the synovial membrane which provides nutrition and lubrication to both upper and lower compartment.
- Different ligaments and muscles are strictly correlated to the functions of the TMJ.

R. Prandi · P. Lodo · N. Margolo Private Practice, Turin, Italy

G. Carnazza Studio Futura, Ciriè, Turin, Italy e-mail: gino.carnazza@studiofutura.com

© Springer Nature Switzerland AG 2020

T. Robba et al. (eds.), *MRI of the Temporomandibular Joint*, https://doi.org/10.1007/978-3-030-25421-6_3

For the purpose of this handbook, only relevant embryology and anatomy shall be covered, since the number of notions that TMJ anatomy encompasses cannot all be contained here. Besides, it would be beyond the scope of this chapter, which ultimately is to provide knowledge that corresponds and braids with MRI anatomy.

3.1 Embryology

Not only does the temporomandibular joint possess unique characteristics, but its formation and development, along with the timing, also show peculiar traits that distinguish it from other synovial joints. It is in fact fully developed quite late in life, and its functional surfaces are made of fibrocartilage tissue in place of hyaline cartilage. The skeletal parts of the TMJ are formed from Meckel's cartilage within the embryo's first pharyngeal arch. Meckel's cartilage promotes mandibular intramembranous ossification from mesenchymal tissue. Some slight discrepancies about timing and organizational phases have emerged in previous works; nevertheless, TMJ development can be said to go through three main phases which basically occur between week 7 and 17 of intrauterine life. with the first 4–5 weeks as the most crucial ones (Morimoto et al. 1987; Van der Linden et al. 1987; Ögütcen-Toller and Juniper 1993, 1994; Ögütcen-Toller 1995; Mérida-Velasco et al. 1999). In this section, the following will be outlined:





G. Tanteri (⊠) Studio Tanteri, Turin, Italy e-mail: tanteri@tanteri.it

- the blastematic stage (weeks 7–8 of development);
- the cavitation stage (weeks 9–11 of development);
- the maturation stage (from week 12 of development).

The *blastematic stage* is marked by the beginning of ossification of the mandible and chondrification of the condyle, as well as the ossification of the temporal bone (squamous part). During these weeks mesenchymal cells migrate, grow, and differentiate and in particular, during week 7 a mesenchymal primordiumknown as the condylar anlage-appears externally and adjacent to the lateral pterygoid muscle and Meckel's cartilage. The masseteric and auriculotemporal nerve are already present. Superolaterally lies the zygomatic process of the temporal bone which begins its intramembranous ossification the following week and so does the mandibular ramus. The articular disc blastema and the discomalleolar ligament also appear at this stage, and the primordium capsule reaches the disc and condyle more inferiorly, where the lateral pterygoid muscle is now connected on their medial aspect. During week 9 chondrification of the condyle starts to take place, while ossification continues nearby and the lower compartment is formed. In fact, up to this point there are no joint spaces as such. But it is during week 9 that the cavitation stage begins with the lower compartment forming first, followed by the upper at week 11.

As the condylar portion acquires a larger, more elongated conical shape with its tip facing the mandibular nerve, blood vessels form and ossification of the mandible continues and organization of deciduous teeth buds is present. The squamous part of the temporal bone grows and the discomalleolar ligament maintains its attachments to Meckel's cartilage and the tympanic bone.

During the *maturation stage*, the temporal part of the joint acquires a concave shape. The capsule displays posterior insertions on the temporal and condylar surfaces. In this area the auriculotemporal nerve and new vessels (venous plexus mainly) are visible. Joint compartments are by now established and masticatory muscles follow the development of the ramus and condyle, where endochondral ossification occurs anteriorly. Fibrous projections originate from the disc and the upper head of the lateral pterygoid shows its attachments to the disc and the condyle, while a smaller anterolateral part of the disc is linked to the temporal and the masseter muscle (Mérida-Velasco et al. 1999).

At this stage vascularization of the condylar cartilage is visible in the form of vascular canals whose role is to provide support in mandibular growth. As weeks go by, mandible formation is complete and Meckel's cartilage undergoes a process of resorption. Around the sixteenth week, it is in fact entirely taken in, leaving behind the incus, malleus, and sphenomandibular ligament as its remnants (Symons 1952; Mérida-Velasco et al. 1999).

From these stages until birth hematopoiesis and growth take place. The S-shaped eminence and fossa are fully formed and continue their growth until late childhood years, under the influence of function (suckling and mastication), but before then there are no true anterior boundaries as such.

3.2 Anatomy and MRI Anatomy

The temporomandibular joint is a ginglymoarthrodial synovial joint made up by two separate compartments, which both take part in complex mandibular movements. This joint comprises the articular fossa and eminence, the articular disc, and the mandibular condyle (Fig. 3.1). Diarthroses have hyaline cartilage covering the functional surfaces, however the TMJ has fibrous tissue instead. It is a double joint as the two condyles (left and right) are part of one single mandibular body. These are connected to the cranial base, thanks to the joint capsule and ligaments, and (indirectly) to the hyoid bone, because of longer mandibular ligaments.



Fig. 3.1 Simplified sagittal relationship between condyle, disc, and eminence

3.2.1 Bone Components and Joint Surfaces

The condylar head is the most cranial part of the condylar process of the mandible. It is ovoidal in shape. Its latero-lateral diameter is approximately 20 mm and its width is 8–10 mm. Variability in shape and dimension has been described, however a lateral and a medial pole are always recognizable and when these are connected on both sides, two separate condylar axes are obtained (Fig. 3.2a–c) (Yale 1969; Solberg et al. 1985; Christiansen et al. 1987). Condylar axes are not parallel on an axial view, instead they are oblique and directed posteromedially, so that if they are extended they will meet at the foramen magnum with an angle of 150–170° (Fig. 3.3a, b).

The functional surface of the condylar head is covered by fibrocartilage and it faces upward and forward. The lateral and medial ends of the condyle display small tubercles where the collateral ligaments are attached. More medially a small depression is present for the insertion of the lateral pterygoid muscle.

The glenoid fossa is part of the squamous temporal bone and it is also oriented posteromedially (Fig. 3.3a). Together with the articular eminence, it has a first concave then convex shape: it is concave at the fossa and becomes convex moving down toward the slope of the eminence and tubercle, where there is a layer of fibrocartilage. The articular eminence is the most anterior boundary of the fossa. The petrotympanic fissure divides the fossa between the extra-articular portion (in front of the ear canal) and the intraarticular portion, where the hinge movements take place.

The bony structures here described are the ones used to set planes for MRI sequence acquisition. The sagittal plane has to be oriented along the vertical ramus of the mandible and the short (or anteroposterior) condylar axis (Fig. 3.4a). The coronal plane has to be oriented along the vertical ramus and the long (or latero-lateral) axis of the condyle (Fig. 3.4b). The axial plane is oriented along the coronal intercondylar plane and it corresponds to the anatomical axial plane in the sagittal plane (Fig. 3.4c).

3.2.2 Articular Disc, Capsule, and Ligaments

Articular Disc If one considers the functional aspect of this joint, then it is true to say that the two bony parts of the joint are both convex. The articular disc that lies in between is oval in shape and concave on both faces, thus making all profiles fit together in rest position and during movements. It is made of resistant dense fibrous connective tissue (avascular fibrocartilage) and it resembles a biconcave lens in a sagittal cross section, with a thinner center (pars intermedia, 1.5 mm), a thick anterior portion (anterior band, 2 mm), and an even thicker posterior portion (posterior band, 3 mm) (Gaa and Hüls 1989; Bumann and Lotzmann 2000). The thinner central part lies where the condyle and the eminence face each other (see Chap. 7).

Disc properties are not only addressed to the geometrical suitability of the joint components, but also to the biomechanics of the TMJ. The disc acts as a viscoelastic shock and stress absorber as it has a role in load distribution (Detamore and Athanasiou 2003; Tanaka et al. 2003).

Animal studies and reports have suggested that the cell population of the TMJ disc is slightly different from the expected chondrocyte cell.



Fig. 3.2 Condylar morphology can vary greatly among individuals. It can have a convex profile (a), it can be slightly rounded (b) or flat (c), and still be considered within the normal range

There seems to be a variable amount of cells, with a denser distribution where the disc is thicker, surrounded by extracellular matrix and lacunae, and these exhibit features such as vimentin microfilaments that can associate disc cells to myofibroblasts with some contractile role. Collagen fibers within the disc, mostly type I and III, have been described extensively. They are believed to be responsible for the tensile strength of the disc. They possess a wavy appearance and their direction is mostly anteroposterior in the central region of the disc. A ring-like pattern is found just outside the thinner part, where collagen fibers tend to arrange themselves in the direction of the disc boundaries. The result is that the anterior and posterior region have a mediolateral pattern of fibers and the remaining areas have a more anteroposterior organization. Elastin is also present, dispersed among collagen fibers (Detamore and Athanasiou 2003). Overall, up to 65-80% of the extracellular matrix is made up of water, whereas the disc is made up of collagen fibers (65–80%) and proteoglycans (10%). The latter together with the various glycosaminoglycans are responsible for the load-bearing characteristic of the disc and for



Fig. 3.3 Condylar axes intersect at the foramen magnum with an angle of $150-170^{\circ}$. The same orientation is true for the glenoid fossae (*left*) and for the condyles (*right*) (**a**). Axial MR image showing the same condylar axes (**b**)

resistance to compression. Mechanical loading differentially influences proteoglycan mRNA expression and proteoglycans are believed to be responsible for two-thirds of the dynamic elastic properties of the disc (Tanaka et al. 2003).

More in detail, when considering the structure of the disc, it is important to emphasize that the extracellular matrix of the disc is mainly composed of collagen, glycosaminoglycans (such as hyaluronic acid), proteoglycans (mostly highmolecular-weight chondroitin sulfate), elastic fibers, and water (Fig. 3.5) (Kiga 2012).

The predominant type of collagen is type I and it creates a reticulum (*lattice*) which forms the



Fig. 3.4 Condylar axes (short and long) together with the ramus axis are the reference points for setting the sagittal plane (**a**), the coronal plane (**b**), and the axial plane (**c**) in MRI



Fig. 3.5 Extracellular matrix of the disc is mostly composed of collagen, glycosaminoglycans (such as hyaluronic acid), proteoglycans (mostly high-molecular-weight chondroitin sulfate), elastic fibers, and water

substrate of discal tissue. Fibers of type I collagen in the intermediate zone of the disc are oriented parallel to the disc surface. Most of these fibers extend into the anterior band and posterior band where they either join with transversally or vertically oriented fiber groups or pass through the bands into the posterior attachment. Vertical bundles are present in all disc regions, but were found to be more frequent in the posterior band, the latter being the area of the disc in which the largest amount of collagen fibers is found (Katzberg et al. 2016). Along the inferior surface of the disc, type III collagen is present, thus originating more delicate fibers (Kiga 2012). Type II collagen is present pericellularly and in the interstices of the type I lattice and is co-distributed with the chondroitin sulfate proteoglycan.

Proteoglycans are macromolecules which are made up by a protein core to which a high number of polysaccharide chains, named glycosaminoglycans (GAG), are bound. GAGs are polysaccharides in which one of the repeated dimeric units (disaccharide) is always an amino sugar. GAGs are rarely found free within the matrix, with the exception of hyaluronic acid which does not take part into the formation of proteoglycans. Chondroitin sulfate proteoglycan and type II collagen make up a matrix which shows great affinity for water (Tanaka et al. 2003; Lu et al. 2009; Katzberg et al. 2016). Elastic fibers are present in all regions, but appear to be more plentiful along the borders of the disc rather than the central part. The posterior attachment (see below) is the structure that contains the highest density of elastic fibers, and these are more abundant in the superior lamina than in the inferior one. The anterior band contains significantly fewer fibers than the posterior attachment, but more than the posterior band. Pars intermedia displays no (or few) elastic fibers (Clément et al. 2006; Scapino et al. 2006). Elastic fibers of the disc appear smaller than those of the attachments.

Knowledge of tissue characteristics and molecular structure is fundamental for the understanding of MRI disc appearance. In physiological conditions, the posterior band of the disc shows a hyperintensity in its central area in all sequences. This is due to the presence of transversely oriented collagen fibers. The typical hypointensity seen both in the anterior band and in the intermediate zone is instead caused by tight, linearly organized collagen. Vertically oriented, compact collagen fibers account for the prominent, vertical, low signal-intensity line which is often seen at the junction of the posterior band and in the bilaminar zone (Fig. 3.6a) (Drace et al. 1990). MRI sequences, however, may not clearly show the junction between the posterior band and the posterior attachment, as the posterior band also contains loosely associated collagen fibers which reach into the ones of the posterior attachment (Fig. 3.6b) (Scapino et al. 2006; Katzberg et al. 2016). This might affect the diagnosis of discal displacements, especially when partial. The thin, horizontal, low signal-intensity lines representing the inferior and superior surfaces of the posterior band of the disc could be easily confused with more anterior prolongations of the two laminae of the posterior attachment (Drace et al. 1990).

The posterior attachment is made up by the superior retrodiscal lamina (sometimes called temporal posterior attachment or disco-temporal ligament) and by the thinner inferior retrodiscal lamina (sometimes called condylar posterior attachment or disco-condylar ligament) (Fig. 3.7). In maximum intercuspation, retrodiscal laminae may not be fully visible, whereas upper lamina fibers will be stretched out (if the disc is not displaced) and clearer during mouth opening (Fig. 3.8a). The inferior lamina can at times be harder to assess, regardless of the disc position (Fig. 3.8b) (Katzberg et al. 2016). Again, in



Fig. 3.6 The boundary between posterior band and retrodiscal laminae can be clearly observed in this sagittal SE PD image (**a**, black arrow). This is due to the collagen fibers of the posterior band which are vertically oriented.

In other cases, the boundary between posterior band and retrodiscal laminae is not so clear (**b**) and this is probably due to collagen fibers which reach the laminae from the posterior band (*white arrows*)



Fig. 3.7 In closed mouth, static position, the condyledisc complex is kept in place and maintains a correct relationship with the eminence, thanks to the action of the upper head of the lateral pterygoid muscle. In this position, the disco-condylar ligament (inferior lamina) is tense, while the disco-temporal ligament (superior lamina) is relaxed. As protrusion or mouth opening takes place, the condyle–disc complex moves along the eminence. The ligament activity is now opposite, so the inferior lamina is relaxed and the upper is stretched. This creates the posterior space which is needed for the hydraulic cushion to replenish



Fig. 3.8 Open mouth sagittal STIR. In (a) the disc shows reduced mobility and the posterior band lies slightly behind the condyle. As a consequence, the retrodiscal laminae are well visible and everted. The temporal and condylar attachments of the upper and lower lami-

nae are indicated by the *arrows*. Open mouth sagittal STIR in (**b**) shows that the inferior lamina can hardly be seen in normal conditions, while the superior one runs parallel to the glenoid fossa (*arrow*) and its attachment is well visible

between these two layers is the adaptable, innervated vascular area known as the *bilaminar zone* (or retrodiscal area, confined within the retrodiscal laminae) made of loose elastic fibers, adipose tissue, and blood vessels, which plays a role in joint stabilization and homeostasis (Fig. 3.9a, b). The disc is then attached to the joint capsule posteriorly and anteriorly, and the anteromedial aspects of the disc and capsule also receive the insertion of the upper head of the lateral pterygoid muscle (Fig. 3.10). Laterally and medially there are only discal connections to the condyle so that the condyle–disc unit can move on its own (rotation).





Fig. 3.9 Open mouth—bilaminar zone (**a**). Open mouth sagittal SE PD fat saturated (**b**, *arrow*). In normal conditions, the retrodiscal laminae are intact and the retrodiscal



Fig. 3.10 Sagittal SE PD in maximum intercuspation showing the anteromedial part of the joint, the superior head of the lateral pterygoid muscle and its insertion on the disc (*arrow*)

Capsule The *capsule* is made of dense connective tissue and it encloses the joint. On its inner surface the synovial membrane covers the walls and the discal surfaces, and it provides for lubrication and nutrition in both joint compartments,

adipose tissue is hyperintense due to the abundant quantity of nerve fibers and blood vessels

through the hyaluronate-rich synovial fluid. On the outer aspect, its attachments are toward the temporal region of the joint and to the neck of the mandibular condyle.

Ligaments The capsule is reinforced by the socalled temporomandibular ligament (also known as lateral ligament), a thick fan-shaped ligament that has a deeper horizontal component and a more superficial oblique component (Fig. 3.11). This ligament extends from the temporal bone posteriorly to the lateral pole of the condyle and to the condylar neck. The significance of the temporomandibular ligament lies in the protection from anteroposteriorly directed traumas and movements (especially the horizontal sheet), whereas the oblique portion can be considered like a suspensory ligament of the mandible. The horizontal arrangement limits posterior and lateral movements, whereas the oblique arrangement affects mouth opening.

Although not strictly part of the temporomandibular joint, the other suspensory ligaments which should be mentioned are the sphenomandibular ligament, the stylomandibular ligament, and the stylohyoid ligament. These are important for mandibular suspension and are involved in the control of mandibular posture (Tanteri et al. 2009).

Collateral ligaments have been mentioned together with the articular disc (Fig. 3.12).

3.2.3 Muscles

Muscles that will be described here are those actively involved in mandibular movements and that are spatially related to the TMJ (Fig. 3.13).



Fig. 3.11 Oblique (**a**) and horizontal (**b**) portions of the temporomandibular ligament



Fig. 3.13 Masticatory muscles

Temporal Muscle The temporal muscle has its wide origin in the temporal fossa, then it narrows down to run through the zygomatic arch to finally insert onto the coronoid process and part of the anterior margin of the ramus (Fig. 3.14). It is a powerful closing muscle (elevator of the mandible). It can be divided into three parts: anterior, middle, and posterior. The anterior part can be considered as the joint-stabilizing belly for the final closing phase. It is covered by the deep sur-









Fig. 3.14 Temporal muscle

face of the temporal fascia, it is vascularized by the deep temporal branches of the internal maxillary artery, and it is innervated by cranial nerve V (mandibular nerve, deep temporal nerves).

Masseter Muscle The masseter muscle runs from the zygomatic arch to the border and angle of the mandible (Fig. 3.15). It is an important chewing muscle which is also part of the elevators. It can be described in its two parts: the deep masseter and the superficial masseter. The superficial masseter is the largest, almost rectangular muscle which inserts into the lateral face of the ramus and the angle. The deep masseter is smaller, it originates from the deeper surface of the zygomatic arch and inserts onto the cranial part of the ramus (Fig. 3.16). These shorter fibers run slightly divergent as opposed to the superficial layer and can also be considered as joint stabilizers. The masseter muscle is vascularized by the masseteric artery from the internal maxillary artery and it is innervated by the masseteric nerve (mandibular nerve, cranial nerve V).

Medial Pterygoid Muscle The medial pterygoid muscle (or internal pterygoid) is a rectangular muscle which originates from the medial face of

Fig. 3.15 Masseter muscle

the lateral pterygoid plate (sphenoid bone), from the tuber and from the pyramidal process of the palatine bone (Fig. 3.17). It runs posterolaterally to reach the angle and medial face of the ramus. The simultaneous bilateral activation of this muscle protrudes and elevates the mandible. Monolateral activation is responsible for lateral movements of the mandible. The medial pterygoid muscle is vascularized by the pterygoid branches of the internal maxillary artery, although further arteries may supply this muscle, and it is innervated by the medial pterygoid nerve (mandibular nerve, cranial nerve V) (Kwak et al. 2008).

Lateral Pterygoid Muscle The lateral pterygoid muscle (or external pterygoid) is a thick strong muscle with an upper and a lower head (Fig. 3.18). The upper head originates from the greater wing of the sphenoid bone, while the lower head originates from the lateral face of the pterygoid plate. These two bellies insert posterolaterally, reaching the anterior part of the condylar neck, the capsule, and the disc (upper head). Electromyographic studies have shown that these two work as antagonist muscles: the upper head being actively engaged in closing and the lower



Fig. 3.16 Deep (left side) and superficial masseter muscle (right side)



Fig. 3.17 Medial pterygoid muscle-ventral view

head being actively engaged during opening and forward movement (Fig. 3.19a–h) (Molin 1973; Mahan et al. 1983; Widmalm et al. 1987). It is why the upper head is considered a joint stabilizer. The simultaneous bilateral activation of the lower head protrudes the mandible and is active



Fig. 3.18 Lateral pterygoid muscle—upper and lower bellies

during mouth opening. Monolateral activation is responsible for lateral movements of the mandi-



Fig. 3.19 Opening movement (a-e). The lower head of the lateral pterygoid is active during this movement. Closing movement (e-h). The upper head is active, stabilizing the disc during the opposite movement

ble. The lateral pterygoid muscle is vascularized by the pterygoid branches of the internal maxillary artery and it is innervated by the lateral pterygoid nerve (mandibular nerve, cranial nerve V).

Masticatory muscles can be visualized in TMJ MRI, as shown in Figs. 3.20.

3.2.4 Blood Supply and Innervation to the TMJ

Blood supply to the TMJ intuitively comes from branches of the external carotid artery. The superficial temporal artery (terminal branch of



Fig. 3.20 Atlas figures

the external carotid artery), the transverse facial artery (from the superficial temporal artery), the anterior tympanic, middle meningeal, and deep temporal arteries (all from the internal maxillary artery), the ascending pharyngeal artery, and the auricular posterior artery (branches of the external carotid) all participate in the vascularization of this joint (Macias et al. 2014). Venous blood reaches the internal jugular vein



Fig. 3.20 (continued)

through the venous plexus of the internal maxillary vein and the superficial temporal vein.

Sensory innervation comes from branches of the mandibular nerve (auriculotemporal and masseteric nerves, cranial nerve V). These innervate the lateral and medial aspect of the joint, respectively. Motor innervation has been outlined with each masticatory muscle.

Knowledge of facial nerve anatomy is mandatory in case operative techniques are to be performed in this region (arthrocentesis, arthroscopy, open surgery).



Fig. 3.20 (continued)



Fig. 3.20 (continued)



Fig. 3.20 (continued)

References

- Bumann A, Lotzmann U. Diagnostica Funzionale e Terapia. Milan: Masson; 2000. p. 23.
- Christiansen EL, Chan TT, Thompson JR, Hasso AN, Hinshaw DB Jr, Kopp S. Computed tomography of the normal temporomandibular joint. Scand J Dent Res. 1987;95(6):499–509.
- Clément C, Bravetti P, Plénat F, Foliguet B, Haddioui AE, Gaudy JF, et al. Quantitative analysis of the elastic fibres in the human temporomandibular articular disc and its attachments. Int J Oral Maxillofac Surg. 2006;35(12):1120–6. https://doi.org/10.1016/j. ijom.2006.06.017.
- Detamore MS, Athanasiou KA. Structure and function of the temporomandibular joint disc: implications for tissue engineering. J Oral Maxillofac Surg. 2003;61(4):494–506. https://doi.org/10.1053/ joms.2003.50096.
- Drace JE, Young SW, Enzmann DR. TMJ meniscus and bilaminar zone: MR imaging of the substructurediagnostic landmarks and pitfalls of interpretation. Radiology. 1990;177:73–6. https://doi.org/10.1148/ radiology.177.1.2399341.
- Gaa U, Hüls A. Biophysical properties of the articular disc tissue and their functional evaluation. Dtsch Zahnarztl Z. 1989;44:S75–8.
- Katzberg RW, Hatcher D, Ethier J. Specialty imaging: temporomandibular joint. In: Tamimi D, Hatcher D, editors. Specialty imaging: temporomandibular joint. 1st ed. Philadelphia: Elsevier; 2016. p. 490–6.

- Kiga N. Histochemistry for studying structure and function of the articular disc of the human temporomandibular joint. Eur J Histochem. 2012;56:e11. https:// doi.org/10.4081/ejh.2012.e11.
- Kwak HH, Hu KS, Hur MS, Won SY, Kim GC, Park BS, et al. Clinical implications of the topography of the arteries supplying the medial pterygoid muscle. J Craniofac Surg. 2008;19(3):795–9. https://doi. org/10.1097/SCS.0b013e31816aab4b.
- Lu XL, Mow VC, Guo XE. Proteoglycans and mechanical behavior of condylar cartilage. J Dent Res. 2009;88:244–8. https://doi. org/10.1177/0022034508330432.
- Macìas D, Ganan Y, Macìas Y. Anatomy and embryological development of the temporomandibular joint. In: Monje Gil F, editor. Surgical management of temporomandibular joint: vol. 1. Arthroscopy. ebook. Philadelphia: W.B. Saunders; 2014. p. 10–29.
- Mahan PE, Wilkinson TM, Gibbs CH, Mauderli A, Brannon LS. Superior and inferior bellies of the lateral pterygoid muscle EMG activity at basic jaw positions. J Prosthet Dent. 1983;50(5):710–8.
- Mérida-Velasco JR, Rodríguez-Vázquez JF, Mérida-Velasco JA, Sánchez-Montesinos I, Espín-Ferra J, Jiménez-Collado J. Development of the human temporomandibular joint. Anat Rec. 1999;255:20–33. https://doi.org/10.1002/ (SICI)1097-0185(19990501)255:1<20::AID-AR4>3.0.CO:2-N.
- Molin C. An electromyographic study of the function of the lateral pterygoid muscle. Sven Tandlak Tidskr. 1973;66:203–8.

- Morimoto K, Hashimoto N, Suetsugu T. Prenatal development process of human temporomandibular joint. J Prosthet Dent. 1987;57:723–30.
- Ögütcen-Toller M. The morphogenesis of the human discomalleolar and sphenomandibular ligaments. J Craniomaxillofac Surg. 1995;23:42–6.
- Ögütcen-Toller M, Juniper RP. The embryologic development of the human lateral pterygoid muscle and its relationships with the temporomandibular joint disc and Meckel's cartilage. J Oral Maxillofac Surg. 1993;51:772–8.
- Ögütcen-Toller M, Juniper RP. The development of the human lateral pterygoid muscle and the temporomandibular joint and related structures: a three-dimensional approach. Early Hum Dev. 1994;39:57–6.
- Scapino RP, Obrezb A, Greisingc D. Organization and function of the collagen fiber system in the human temporomandibular joint disk and its attachments. Cells Tissues Organs. 2006;182:201–25. https://doi. org/10.1159/000093969.
- Solberg WK, Hansson TL, Nordström B. The temporomandibular joint in young adults at autopsy: a mor-

phologic classification and evaluation. J Oral Rehabil. 1985;12:303–21.

- Symons NB. The development of the human mandibular joint. J Anat. 1952;86(3):326–32.
- Tanaka E, Aoyama J, Tanaka M, van Eijden T, Sugiyama M, Hanaoka K, et al. The proteoglycan contents of the temporomandibular joint disc influence its dynamic viscoelastic properties. J Biomed Mater Res A. 2003;65(3):386–92. https://doi.org/10.1002/ jbm.a.10496.
- Tanteri E, Bracco A, Prandi R. Elementi di Gnatologia. Dalla diagnosi alla riabilitazione. Milan: RC Libri; 2009. p. 133–87.
- Van der Linden EJ, Burdi AR, Jongh HJ. Critical periods in the prenatal morphogenesis of the human lateral pterygoid muscle, the mandibular condyle, the articular disk, and medial articular capsule. Am J Orthod Dentofac Orthop. 1987;91:22–8.
- Widmalm SE, Lillie JH, Ash MM. Anatomical and electromyographic studies of the lateral pterygoid muscle. J Oral Rehabil. 1987;14:429–46.
- Yale SH. Radiographic evaluation of the temporomandibular joint. J Am Dent Assoc. 1969;79:102–7.

TMJ Dynamics



4

Giulia Tanteri, Eugenio Tanteri, Carlotta Tanteri, and Gregor Slavicek

Key Points

- The TMJ is a synovial joint. The disc, which lies between fossa and condyle, creates two compartments which may be functionally considered as two different joints.
- Mandibular position continually changes to efficiently perform mastication, swallowing, breathing, and speech; therefore, various spatial relationships between upper and lower dental arches will continuously exist.
- Opening, closing, and lateral movements require finely coordinated mechanisms.
- Rotation and translation, together with protrusion, retraction, and retrusion, are the premise for TMJ and mandibular movement.
- Condylography is a jaw-tracking technique which allows to assess all components of mandibular movement and its spatial coordinates. It is a helpful diagnostic and program-

Electronic supplementary material The online version of this chapter (https://doi.org/10.1007/978-3-030-25421-6_4) contains supplementary material, which is available to authorized users.

G. Tanteri (⊠) · E. Tanteri · C. Tanteri Studio Tanteri, Turin, Italy e-mail: tanteri@tanteri.it

G. Slavicek Steinbeis Transfer Institut - Biomedical Interdisciplinary Dentistry, Steinbeis University, Stuttgart, Germany e-mail: gregor.slavicek@stw.de

© Springer Nature Switzerland AG 2020 T. Robba et al. (eds.), *MRI of the Temporomandibular Joint*, https://doi.org/10.1007/978-3-030-25421-6_4 ming tool, to be used in conjunction with clinical functional analysis and imaging.

The temporomandibular joint (TMJ) is the joint between the mandible and the temporal bone where all movements of the lower jaw are developed. Such movements occur according to specifically required functions and allow the mandible to be positioned appropriately. Mandibular position continually changes to efficiently perform mastication, swallowing, breathing, and speech; therefore, various spatial relationships—between teeth of the upper and lower dental arches—will continuously and necessarily exist.

Mandibular dynamics and precise changes of position are particularly crucial, for example, during chewing, which is a highly differentiated and specialized task. Depending on the appropriate function being performed (cutting, tearing, squashing, or fragmentation), the relationship between jaws and teeth must adapt. This is possible, thanks to both the morphological differentiation of dental elements and to mandibular movement itself.

Adequate TMJ function assists postural stabilization mechanisms of the whole body and, in addition to this, the role of the stomatognathic system in stress-management is more and more discussed and recognized (Ahlberg et al. 2013; Lobbezoo et al. 2018). An orthopedically stable system is needed in order to clench and brux with limited damage to the participating structures. If such parafunctions occur in functionally and structurally unstable situations, and for prolonged periods, significant damage to joints, muscles, and teeth-periodontium may be triggered.

One of the unique traits of the TMJ—which makes it different from any other joint—is that there is a single bone (the mandible) articulating with the cranium via two symmetrical joints. The two TMJs can move with a certain degree of freedom; however, they will always influence one another. Movements will therefore depend on the mobility of both joints, and knowledge of their dynamics is fundamental to understand physiology, pathology, and to perform diagnostics.

When considering mandibular movements, one should also bear in mind the difference between border movements and functional movements. Any mandibular movement is naturally limited by articular surfaces, ligaments, and teeth (both in terms of dental anatomy and dental position/alignment). Border movements are those which can be performed up to the maximum range of motion and that are also more reproducible. Functional movements are those freely performed during function. They are highly variable, for example, chewing on different food textures will imply a different width of movement and differently orientated chewing strokes, and they occur within the envelope of border movements. This means that the opening movement can occur either at its maximum possible extension (maximum opening-border movement) or up to a certain extent (during chewing or speech). Understanding the different possible extent of motion is also important when performing MRI assessment for disc disorders.

Neurological, muscular, and occlusal features related to chewing and to the other functions are fascinating, complex, and finely coordinated, but it is not within the aim of this handbook to describe them. In this chapter, TMJ dynamics will therefore be discussed.

4.1 TMJ and Mandibular Dynamics

4.1.1 Rotation and Translation

The TMJ, the only movable joint of the skull, is a synovial joint. The disc, which lies between fossa

and condyle, creates two compartments which may be anatomically and functionally considered as two different joints. The disco-temporal joint represents the upper (cranial) compartment, while the disco-condylar joint constitutes the lower (caudal) compartment. The disc may erroneously be referred to as a meniscus, a terminology which is out-of-date and apparently difficult to eradicate. A meniscus is a functionally passive, semi-solid, and wedge/crescent-shaped cartilage structure, which has ligamentous attachments but does not bear two distinct compartments since it does not divide the joint space. The disc instead separates the articular space into two systems (upper and lower compartments), thus preventing communication between the different joint heads. Structural union between the articular surfaces is therefore lacking; however, functional contact is needed to ensure joint stability. This is why a balanced activity of muscles (muscle tone) is also essential (Okeson 2014).

Two types of movement can take place in the temporomandibular joint: *rotation* and *translation*. Hence why the TMJ can be defined as a ginglymoarthrodial joint (it is both hinged and sliding) (Meyer 1990).

Rotation The condyle–disc joint (lower compartment) is where *rotation* occurs. The medial and lateral collateral ligaments connect the disc to the condyle and rotation of the condyle onto the disc is the only possible movement of the lower compartment in physiological conditions. If one thinks of all movements happening at a condylar level, then they can be visualized as taking place along three planes: horizontal (transverse), frontal (coronal), and sagittal plane, as well as around a rotational axis (Fig. 4.1a–c).

• *Transverse (horizontal) rotation axis*: this axis passes through the two condyles. When the condyles rotate around such axis, opening and closing movements are performed. This is a functional axis and not an anatomical one. It is commonly referred to as *intercondylar axis* and, in gnathological terminology, it is the so-called *hinge axis*. The assumption behind hinge axis location is that it is where a "pure rotation" takes place, with no combined translation. When the hinge axis rotation occurs in



Fig. 4.1 Transverse (a), vertical (b), and sagittal (c) rotation axes of the condyle

centric relation (condyle and disc are centered onto one another and are found in the uppermost part of the glenoid fossa), the result is a *terminal hinge axis*. According to Dawson:

the axis of rotation passing through the medial poles of both joints causes the possibility of a pure rotation for the first 20 mm of interincisal opening,

which is then stopped by the temporomandibular ligament (Fig. 4.2a, b) (Posselt 1956;

Dawson 2000, 2006). A pure rotation can be demonstrated and recorded, but it rarely takes place as such during function (Okeson 2014);

• Vertical (frontal/coronal) rotation axis: this axis passes through the condyle from its superior to its inferior aspect. The condyle rotates around this axis during ipsilateral lateral movements (i.e., when the mandible moves toward the right side, the right condyle will rotate along its vertical axis);



• Sagittal rotation axis: this axis crosses the condyle with a postero-anterior direction. The condyle rotates around such axis when the condyle and mandible on the opposite side lower down (for example, when food is placed between dental arches on one side only).

The above-described movements have been outlined in a very simplified manner for better understanding but in actual facts, they seldom take place separately.

Translation The upper compartment (also known as disco-temporal joint) is where *transla-tion* occurs. This movement takes place between the superior aspect of the disc and the inferior aspect of the glenoid fossa, and results in the condyle shifting along the articular eminence.

For full range of movements to occur, a simultaneous combination of motion in both compartments is needed (Lindauer et al. 1995). Again, partial opening of the mouth may be achieved by rotation alone, but maximum opening can only be obtained if translation in the upper compartment takes place too (Fig. 4.3a, b) (Dawson 2006).

Mandibular activity therefore takes place predominantly in one of the compartments or both, according to the function which has to be performed. In addition to this, when eccentric movements are considered, both condyles move asymmetrically and asynchronously, and one compartment may be mostly engaged in one joint, whereas the other compartment may be more active in the contralateral.

4.1.2 Opening–Closing

On a theoretical level and up to a certain extent, the opening movement may involve the lower compartment alone (rotation). Such opening would only allow a limited separation between lower and upper incisors of approximately 20 mm (Posselt 1956). In the presence of such a small movement, the disc sits and rests in its original, almost unchanged position, and only its ligaments will have some variation of tension. Needless to say, this amount of mouth opening is not sufficient in order to bite and to take food into the oral cavity. An optimal opening can only be achieved when the upper compartment comes into play, thus adding translational motion to rotation. The disc-condyle complex can then slide onto and against the articular eminence (Fig. 4.4a, b). The latter has a downward and forward inclination, which will cause the disc-condyle complex to move along a downward and forward vector, thus originating a wider opening and an interincisal separation which can reach 45-50 mm (even more in some cases). Rotation between condyle and disc can occur anywhere during the forward condylar movement. Closing takes place with the involvement of both compartments and a direction which is opposite to that of opening.

Under a functional perspective, muscles which are engaged during opening are the so-called depressors (suprahyoids and lateral pterygoid muscles), while during closing the elevators are involved (temporalis, masseter, and medial pterygoid muscles). Muscle vectors' action on the condyle is relatively straightforward. The disc, under physiological conditions, maintains its



Fig. 4.4 Pure rotation only allows for a small mouth opening (**a**). Translation and rotation together are responsible for maximum opening (**b**). Courtesy of Dr. A. Bracco

position, thanks to the effect of ligaments and muscles (upper head of the lateral pterygoid, see below and Chap. 3) which prevent displacements. The translational component requires a much more sophisticated coordination between muscles and ligaments (Fig. 4.5a–h).

4.1.3 Protrusion, Retraction, and Retrusion

Protrusion This is a forward-directed movement, away from the central position of the joint (where the condyles are in their physiological position in the articular fossa). It is described as the anterior movement capacity in the sagittal plane (Fig. 4.6). **Retrusion** This movement is defined in two different ways:

- Retrusion from maximum protrusion (*retrac-tion*) is a movement away from the maximum protruded position, defined as the position where the condyles are in an eccentric position along the articular eminence, and where the relationship between condyle and disc is still maintained under physiological conditions. It is therefore the inversion of protrusion.
- Retrusion from a joint-central position (*retrusion*) is a movement further backward from the point where the condyles are in their physiological position in the glenoid fossa. It is described as the posterior movement capacity



Fig. 4.5 Opening (**a**–**e**) and closing movement (**e**–**h**)—rotation and translation can be seen as well as the upper head of the lateral pterygoid muscle which is at rest during

opening and becomes active during closing, thus stabilizing the disc and the joint



Fig. 4.6 Protrusion—condyle and dental points move forward


Fig. 4.7 Retrusion—the condyle moves further backward

of the mandible in the sagittal plane, still in close relation to and along the articular eminence (Fig. 4.7).

Protrusion and retraction cannot be considered primary movements. In fact, the protrusive and retrusive components are actually intrinsic within the opening-closing movement, as there can be no maximum opening without a certain amount of mandibular protrusion. Similarly, the mandible cannot move back to its starting point if the condyles are not retracted. Protrusion is performed, for example, when whistling, when pronouncing dental consonants, or when divers bite onto their mouthpiece. The muscles which are mainly responsible for mandibular protrusion (translation) are the medial pterygoid muscle and the lower head of the lateral pterygoid, whereas retraction takes place under the combined action of the posterior part of the temporalis and the digastric muscle. Protrusion and retraction are, by definition and under physiological circumstances, symmetrical movements. If, for instance, protrusion takes place on one side only, the result will be an overall lateral movement.

The upper head of the lateral pterygoid has a stabilizing effect onto the disc–condyle complex and plays a major role during retraction. At rest, the tone of the upper head prevails over the retrusive pull exerted by the disco-condylar ligament. When protrusion begins, the disc-condyle complex starts to slide against and along the eminence. At this point, the upper head can be considered inactive. It is, in fact, the lower head to be actively engaged and to be responsible for the movement itself. The collateral ligaments are placed under tension accordingly to the rotation of the condylar head and, at the same time, maintain the disc-condyle complex stable. At maximum protrusion the following will occur:

- the disco-temporal ligament will be tense to its maximum and the disco-condylar ligament will be relaxed,
- the lower head of the lateral pterygoid will show maximum activity and the upper head will be inactive,
- the bilaminar zone, found between the discotemporal and disco-condylar laminae, will be stretched out and replenished (hydraulic cushion effect) (Fig. 4.8).

Retrusion can be observed during physiological activities such as swallowing. It can be voluntary (by asking the patient to slide further back from maximum intercuspation), or it can take place during parafunctions or because of malocclusions (teeth surfaces sliding against



one another and forcing the subject to further retrude so as to reach a proper maximum intercuspation).

4.1.4 Lateral Movements

Mandibular laterotrusion is an eccentric hemimandibular movement. In lateral movements, the mandible moves either to the left or to the right as a unit, but the movements which simultaneously take place in the two temporomandibular joints are different. In order to better explain such movement, the midsagittal plane can be taken as a reference plane and the mandible is virtually divided into a right side and a left side. For instance, a mandibular movement is assumed to occur toward the right side. The right side of the mandible will move away from the median sagittal line, thus carrying out a laterotrusive movement. The left mandibular side will simultaneously move toward the midline, thus carrying out a *mediotrusive* movement. Laterotrusion is an outward movement and mediotrusion is an inward movement from the central joint position (Fig. 4.9a, b).

- Bodily mandibular movement to the right = right laterotrusion = left mediotrusion.
- Bodily mandibular movement to the left = left laterotrusion = right mediotrusion.

This necessarily implies that two simultaneous different movements take place in the left and right TMJ. During lateral movements, different muscles are involved. The one which is the most active and responsible for this is the lower head of the lateral pterygoid on the mediotrusive side (mandibular movement to the right, lower head of the lateral pterygoid on the left is mostly active). The protrusive muscles will be active unilaterally on the mediotrusive side so that the mandible can shift toward the opposite side (mandibular movement to the right, protrusive muscles on the left are active). On the laterotrusive side (in this example the right side), elevators will be active, as they maintain the disc–condyle complex in the fossa.

The condyle on the laterotrusive side (in this example the right side) will mainly perform a rotation close to horizontal, and, because of its ovoid shape, an overall movement directed backward and outward. This condyle is also called the laterotrusive condyle, the rotating or the working condyle, as this is the side where tooth contacts are taking place (or chewing, for instance). The condyle on the mediotrusive side carries out a bodily movement directed downward, forward, and inward, following the direction of the fibers of the lower head of the lateral pterygoid. This condyle is also named non-working or orbiting condyle, as no tooth contacts should be present on this side during mediotrusion. In prosthodontics, this is also referred to as balancing condyle as complete dentures can be balanced on this side.

All of the above show how complex mandibular and condylar movements are, and how finely controlled they must be. Once again, the analysis of occlusion, its functions, and how occlusal



Fig. 4.9 Lateral movement of the mandible. Lateral movement to the right (a) can be described as right laterotrusion and left mediotrusion. Lateral movement to the left (b) can be described as right mediotrusion and left laterotrusion

architecture is fundamental for a correct distribution of loads is beyond the scope of this handbook. A few considerations must be briefly exposed nonetheless. The thickness of the cartilage differs along the condyle and tubercle. The disc itself shows different thicknesses along its structure. This is because different parts must bear a different load (functional or nonfunctional) and for a certain period of time. Teeth are also all different in terms of morphology, dimension, and position. During mandibular movements, they must interact correctly to avoid interferences and allow for optimal muscular activity. When the condyle–disc complex slides against the eminence, upper and lower teeth slide against each other too. It is quite easy to understand how the steepness of occluding surfaces must be in harmony with the steepness of the eminence in order to allow for the correct alternation of compression and decompression within



Fig. 4.10 The palatal aspect of the upper incisor (*left*) is the guiding structure of teeth-guided protrusion, and its shape is inversely related to that of the articular eminence (*right*) (from A.I.G. 1994)

the joint. Moreover, an inverse correspondence between the shape of upper front teeth lingual anatomy and the anatomy of the joint surfaces has been studied extensively (Slavicek 1984). During mandibular movements, when structural contact is taking place on a very steep part within the joint, the dental contact is taking place on a flatter one and vice versa (Fig. 4.10).

4.2 Focus on Condylography: Understanding Mandibular Movement

As seen in the previous chapters, the temporomandibular joint is a complex entity, in both structure and function. A profound understanding of its complexity is of central importance during clinical and instrumental examination and will influence treatment planning. This characteristic is clearly highlighted in a quote from Mariano Rocabado:

In order to understand how a joint that maintains such a close environment can become unstable, lose its normal mechanics, and start functioning against the rules of synovial joints (movable, friction-free, and pain-free joints), we must understand the normal physiology of joints and of periarticular connective tissue, mainly, ligaments and capsules (Rocabado 1983).

Condylography is a tool that can address these demands. It completes and supports the field of

clinical functional analysis and, as such, it is attributed to the so-called instrument-assisted clinical functional analysis. It provides the operator with graphical representation of the movements carried out by the joints, and it gives the possibility to take a closer look at the movement pattern of virtually any mandibular structure, as the recording system and the software enclose the subject into precise three-dimensional geometrical coordinates.

In order to do so, a clutch is temporarily secured to mandibular teeth onto their vestibular surface (paraocclusal clutch) or on their occlusal surface (occlusal clutch) (Fig. 4.11). An upper bow bears the digital recording flags, whereas a lower bow is attached to the mandible through the clutch and bears the recording stili on both sides (Fig. 4.12a–d). These are double stili as one is dedicated to the hinge axis and one is for rotational evaluations. The recording apparatus is connected to a transducer and thus to a computer where the software interface allows for visualization of the live recording and for storing it.

It should be emphasized at this point, that using condylography to record joint path movements by no means replaces a thorough clinical functional analysis or manual examination and palpation of the temporomandibular joint. Instead, it should be seen as a necessary complement to these procedures, in terms of providing a



Fig. 4.11 Paraocclusal clutch used for condylographic recording

depth of diagnosis that has the clear potential to detect pathological changes in their early stages. Condylography can enable the well-trained and experienced user to detect signs of disorder before they become clinically apparent and even before the patients themselves become aware of any discomfort. More clearly, this analysis allows to identify findings as belonging to bony structure, disc/ligaments, and muscles. Condylography should not be understood as an alternative to radiological imaging, but as a possibility to come up with a valid confluent diagnosis. Not to mention that it could also be a powerful tool in the perspective of saving useful resources in the hospital setting, or at least use them wisely, so that in selected patients and conditions, they can be employed for other purposes rather than interim and final analysis. Reliability studies still have to validate this instrument, which is traditionally part of gnathology, together with MRI findings, as it gives more refined information than clinical findings alone.

This overview demonstrates where condylography fits into the analytic process that has to be considered in any TMJ patient that is referred to a dentist or maxillofacial surgeon:

- · Clinical functional analysis
- Instrument-assisted clinical functional analysis (condylography, cephalometry)
- Instrumental functional analysis (articulator)
- Additional diagnostic methods (MRI, CT scan, X-rays, US)

Condylography aims at detecting early changes in the biodynamics of the temporomandibular joint and its associated structures. Diagnosis is based on the principle of recording the dynamics of the hinge axis. The careful interpretation of the condylographic diagnostic findings can highlight alterations related to the articular disc, the ligaments of the craniomandibular system, the joint capsule, the condyle, the fossa, the neuromuscular system, and occlusion. This section is not aimed at providing the reader with an "atlas" of condylographic interpretation, but rather at walking beginners and advanced users through the biomechanics principles that can be evaluated and understood instrumentally.

4.2.1 Principles of Condylography

Condylography is based on the idea that the entire range of movements of the lower jaw can be repeatedly and reproducibly recorded and presented in graphical form. Condylography as a method for evaluating mandibular movements has its predecessors dating as back as the last decades of the 1800s. The first recordings were reported by Ulrich and Walker, and invaluable contributions to the study of hinge axis, Bennett movement, jaw tracking, articulator programming and for diagnostic purposes have been made by Bennett, Eltner, Gysi, McCollum, Posselt, Messerman, Bewersdorff, Jankelson, Lundeen, Alsawaf, Missert, and Slavicek (Piehslinger et al. 1991). Continuous improvements in the technique have been observed until today, together with technological advances, so that electronic condylography performed nowadays links the geometrical knowledge of traditional condylography together with the possibilities of the so-called advanced condylography (insights into translational and rotational movements, quantity of rotation, velocity assessments, graphical representation of the end feel, and so on).

The movements of the mandible are highly diverse, complex, and three-dimensional, limited in range and motion by few structures: teeth,



Fig. 4.12 Condylographic hardware—upper bow with digital flags and lower bow with double stili (**a**). The double stili allow for recording of the hinge axis movement and rotational information. Right mediotrusion movement

while being performed and recorded $(\mathbf{b}-\mathbf{d})$. The still can be seen to intrude more and more inside their slot as the movement takes place

ligaments, muscles, and soft tissue (Rocabado 1983; Koolstra and Van Eijden 2007). Teeth represent the vertical limiting structures for the closing movement of the mandible. This is of course also true for dentures or the alveolar ridge in the edentulous mouth. Dental surfaces are modifying determinants of protrusion, laterotrusion, and retrusion. Ligaments limit the range of posterior motion, while muscles and soft tissues limit the opening and sideways movements.

In healthy humans, there are no limitations to lower jaw movements due to bone. The fossa and the articular eminence give the mandible a great deal of freedom. And when considering the dynamics of the temporomandibular joint, it would be wrong to assume that the glenoid fossa of the temporomandibular joint holds a dominant control function in terms of locating and determining mandibular position. On the other hand, the articular eminence controls the forward movement during protrusion, opening, and mediotrusion and allows the condyle–disc complex to slide along it. But it can be clearly seen in joint path recording that this movement usually stays within the tubercle and does not necessarily go over and beyond this bony structure. This can be observed in physiological situations, particularly in the functional movements of the mandible, especially during chewing.

Mandibular movements are often described as *border movements*, based on their spatial complexity. Once again border movements are those

that display maximum range in all directions of space, and these cannot be exceeded under physiological conditions. This applies to forward movement, lateral movement, opening and posterior movement. These extreme movements can now be reproduced in two-dimensional representations, i.e., on the sagittal, transverse, and cranial planes (Fig. 4.13), and as points of a mandibular movement pattern (Fig. 4.14a, b) (Posselt 1952, 1962).

At this point one essential factor should be stressed about the description of mandibular









movement: it is of major importance to specify the physical (anatomical) point at which the border movements are observed. In fact, one occlusal point will show a very different movement pattern as opposed to another occlusal point and opposed to jaw joints (Koolstra et al. 2001; Tanaka and Van Eijden 2003). The reason for this lies in the fact that during movement, the temporomandibular joint does not stay in one position within the articular fossa. The joint permits rotational movements and, in fact, rather wide translational motions, and the original position in relation to the fossa is abandoned all the time. The result is a compound motion that is a combination of translation and rotation. In addition, the movement of the mandible is determined not just by one but by both temporomandibular joints, moving in either a symmetric (opening and protrusion) or asymmetric (laterotrusion and mediotrusion, respectively) way. Within the range of the above-mentioned border movements, the actual movement sequences now take place with the execution of movements such as the ones for chewing, swallowing, speaking, breathing, pressing, grinding, postural support, and body balance.

Having said this, recording such mandibular movements represents a very complex and demanding task for the recording instruments which are used, whether mechanical or electronic. In the analysis of mandibular movement, it is fundamental to always distinguish between analyzing the movement of the TMJs themselves and the movement of other components expressed as TMJ movement. This should generally be considered in the underlying method and instruments used (i.e., intra- or extra-oral recording) (Lundeen and Wirth 1973; Mauderli and Lundeen 1991; Miller et al. 1995; Currie 2010).

4.2.2 Mandibular Movements

Mandibular movements are versatile and complex. When defining and describing such movements, one necessarily has to specify which part of the recorded system is being displayed: this could be the hinge axis, any point of occlusion, and any mandibular part such as the chin. Typically, mandibular movement is first analyzed through standardized movements (Fig. 4.15a–d):

- Protrusion
- Retraction
- Retrusion
- Laterotrusion
- Lateroretrusion (from maximum laterotrusion back to the central starting position)
- Mediotrusion
- Medioretrusion (from maximum mediotrusion back to the central starting position)
- Opening movement (abduction)
- Closing movement (adduction)





Fig. 4.15 (continued)

All these mandibular movements can now be performed during the study in several ways:

- *Free movement*: the test subject or patient moves spontaneously without any contact of teeth. These are the standard movements that are primarily used for the evaluation. The influence of occlusion on movement patterns and dynamics of the mandible is reduced, but not eliminated completely, as the programming (or memory) of the neuromuscular system will still show its flaws.
- *Guided movement*: the test subject or patient is asked to move the mandible under the guide of tooth contacts. The influence of occlusion on the paths of movement can be seen here. These pathways provide a basis for further analysis of functional movements. Such movements cannot be recorded in case an occlusal clutch is used.
- Manipulated movement: the examiner influences the movement through active manipulation of the mandible in one direction. These movements enable the differential diagnosis of various pathologies of the TMJ (such as

internal derangement) and the stomatognathic system.

- *Functional movements* (functions): the test subject or patient is asked to perform functions, as standardized as possible. Typically, chewing (with or without swallowing), talking, clenching, and grinding will be recorded.
- *Extra* (specific movements): depending on anamnestic data, clinical functional analysis results, or findings from instrumental analysis, it may be advisable to record specific movement patterns that are to be incorporated into the overall analysis. An example of this is the possibility of showing movement patterns on an occlusal splint to evaluate its therapeutic effects.

Besides movement analysis, other parameters can be recorded and analyzed during condylography. This is the case of *end feel*, for instance. This is adopted from traditional orthopedics where it is defined as the quality of resistance at the end of a movement. An end feel is pathological if it has a different quality than expected, and if it occurs at a point in time other than under physiological conditions. Assessing the end feel involves a passive movement test (Freesmeyer 2011). Four typical physiological end feels are found in the assessment of joints:

- soft-elastic: caused by interposition or stretching of muscles.
- firm-elastic: caused by the limiting effect of the capsular ligaments.
- hard-elastic: caused by the physiological function of the cartilage.
- hard-inelastic: limitation of movement by bony structures.

The TMJ is usually soft-elastic (in protrusion, laterotrusion, and opening) and firm-elastic (in retrusion and in caudal and cranial manipulation).

4.2.3 Forward Movement (Protrusion)

During protrusion, the lower jaw moves forward, out of a position where both condyles are in the fossa. The movement path is largely determined by the articular eminence and dental structures. Rotation only occurs to a limited extent and this depends on the dynamic connection between the lingual morphology of the front teeth and the morphology of the fossa and eminence (Slavicek and Lugner 1976; Slavicek 1984; Slavicek 2011). Forward movement is limited due to increased tension in the muscles and soft tissues, giving a soft end feel. A hard end feel does not occur with this motion under physiological conditions.

The determinants of protrusion are:

- Articular eminence (on both sides)
- · Condyle-disc complex
- · Lingual morphology of the upper front teeth

The modulators (major influential factors) of protrusion are:

- Occlusion in the posterior region
- Skeletal and/or dental asymmetry

- Shape and orientation of the condyles
- Skeletal class
- Neuromuscular system

The Posselt diagram shows a characteristic image of movements of mandibular dental structures—for example, the lower incisor (Posselt 1952, 1962). This can be represented as a three-dimensional envelope curve. Viewed from the side, the movement of the jaw is seen to be very simple in principle, due primarily to the defined trajectory of the articular eminence. Viewed from the cranial direction, and equally from the front, the physiological protrusive movement path is seen to be straight with no deviation to the side. This is a symmetric movement of the mandible (Figs. 4.15a and 4.16).

In the forward movement of the lower jaw, the temporomandibular joints move along a trajectory, which is largely determined by the curvature and slope of the articular eminence under physiological conditions. For the lower front teeth, under physiological conditions, protrusion is dominated by the upper front teeth and can be represented by the characteristic movement seen in the Posselt diagram (Fig. 4.17a, b).

4.2.4 Backward Movement (Retraction)

In terms of the TMJ, the backward movement from maximum protrusion should be, under physiological conditions, identical to that of protrusion. Again, the path of the front teeth may be traced over the previous; however, this does not normally happen, instead the movement is performed either with the same or with a greater opening rotation than in protrusion, without anterior control, and the movement ends with a closing rotation. There are no bony or dental structures that limit the backward movement of the mandible from the maximum excursion. Ligaments do play a certain role, but the action of centering the mandible that concludes the backward movement is essentially controlled by the muscles of the craniomandibular system.





Fig. 4.16 Protrusion tracing for the right and left joint. Each side has its movement displayed on all three planes. Protrusion is symmetrical and no lateral displacement

should be seen on the frontal and cranial plane (where a straight line is observed). Sagittal view shows a physiological concave characteristic of the curve



As the mandible moves backward from the maximum protrusive position, the temporomandibular joints perform a movement that, under physiological conditions, is largely determined by the curvature and slope of the articular eminence. Here, it is possible to see influence of the articular disc and position of the ligaments and to note that they come into play earlier than in protrusion. The reason for this lies in the direction of muscle vectors that are active in protrusion and retraction/ retrusion, respectively (Fig. 4.18a, b). The front teeth are said to have an indirect influence on this movement, in the sense of avoiding contact, so that the upper front teeth have no contact in retraction





and are bypassed. The retraction and protrusion paths of the lower front teeth will differ according to physiological conditions and will be characterized by a larger opening rotation of the mandible in moving backward, and a late-occurring closing rotation on occlusal contact. To put it another way, while protrusion is executed as a border movement, this is not expected to be the case for retraction. In retraction, the movement paths of the teeth in the posterior regions differ from both the TMJ motion path and that of the front teeth of the mandible. However, it should be noted-again assuming physiological conditions-that the left and right sides of the occlusion move almost identically. At the end, when the condyles reach their position in the joint fossa, the movement is controlled and regulated by muscles. As there are no bony structures to protect the back of the temporomandibular joint in humans, the end feel is elastic. A hard end feel at the end of this movement is not present under physiological conditions and, if present, it would indicate pathology.

4.2.5 Backward Motion (Retrusion)

The mandible moves backward from the position where both condyles are positioned in the fossa (Fig. 4.15b). The movement pattern is fundamentally determined not only by the articular fossa and

the articular disc, but also by dental structures. Rotation only occurs to a limited extent and this depends on the dynamic connection between the steepness and position of tooth contacts during this movement. The front teeth are usually not involved, except in the event of an anterior crossbite. Contact in the molar region, in the areas behind the transverse crest (crista transversa) of the first upper molar, is particularly critical to these movements. Backward movement is limited due to increased tension in the ligaments (in this case primarily the temporomandibular ligament). In humans, there is no bony restriction to such movements, due to the absence of a distinct post-glenoidal process and the orientation of the articular fossa and the TMJ (Fig. 4.19a, b). In any case, a hard end feel is not observed with this movement, although, with wellaligned ligaments, the difference in the end feel between active movement (engaged by the patient) and passive movement (manipulated by the practitioner) is negligible.

The determinants of this movement are:

- Articular fossa (on both sides)
- Condyle–disc complex
- Active retrusive occlusal contact (ideally in the upper premolar area)

The modulators (major influential factors) of this movement are:



Fig. 4.19 (a) Post-glenoid process limits posterior movement of the condyle; however, this structure is different in the human TMJ (b), where there are no bone limitations to movement other than the fossa

- Individual articular position
- Occlusion in the posterior region (often one-sided)
- Skeletal and/or dental asymmetries
- Shape and orientation of the condyles
- · Skeletal class
- Neuromuscular system

4.2.6 Lateral Movements (Laterotrusion, Mediotrusion)

In lateral movements, the mandible moves from the position at which both condyles are in the glenoid fossa, toward one side, left or right (Fig. 4.9a, b). For the most part, the movement pattern is determined by the articular eminence and the articular fossa on the mediotrusive side, the shape and position (in terms of orientation) of the condyles on both the medio- and laterotrusive sides, and the dental structures on the laterotrusive side. Rotation only occurs to a limited extent, depending on the dynamic connection between the lingual morphology of the canines and the morphology of both the eminence and the fossa on the left and right sides (Slavicek and Lugner 1976, 1979; Fialka et al. 1990; Slavicek et al. 1990). Sideways movement is limited with increased tension of the muscles and soft tissue, giving a soft end feel. A hard end feel does not occur with this motion under physiological conditions.

The determinants of this movement are:

- Articular eminence (especially on the mediotrusive side)
- Condyle–disc complex (on both sides)
- Lingual morphology of the canines and upper lateral incisors
- Any guiding (controlling) premolar or first maxillary molar structures on the laterotrusive side
- Articular fossa (on the laterotrusive side)

The modulators (major influential factors) of this movement are:

- Occlusion in the posterior region
- Skeletal class
- Individual articular position of the condyle in the articular fossa (habitual position)
- Skeletal and/or dental asymmetries
- Shape and orientation of the condyles
- Neuromuscular system

In lateral movement, the teeth of the mandible on the mediotrusive side move with a forward, inward, and downward motion. In the same movement, the laterotrusive side of the occlusion moves with a sideways, downward, and outward motion. Due to the geometry of the mandible, the paths of movement at various points of the mandible are significantly different. Albeit a simplification, this can be illustrated in the form of a triangle with two points at the left and right temporomandibular joints and a third at the lower anterior point.

In a lateral mandibular movement, the nonworking condyle makes a wide and sweeping motion, forward, downward, and inward. In the same movement, the range of motion of the working side is, by contrast, small and often only perceived in the sense of rotation. Depending on the geometrical position of the rotation axis and on the shape of the condylar head itself, some part of the condyle may undergo a backward rotation or a forward rotation. A pure rotation may also be the case. Anyway, the scope of condylar movement is comparatively very different on the latero- and mediotrusive sides. The lower incisal edge moves forward (due to the extensive movement of the non-working condyle), toward the laterotrusive side (again, due to the wide motion of the non-working condyle), and downward (because of the connection between the nonworking and working condyles). For simplicity, it is assumed here that the incision inferior is

positioned in the medial sagittal plane and that the path of movement is not influenced by the upper front teeth. The downward component of the movement is influenced by the action of the working condyle: a more extensive backward and upward movement of the working condyle flattens the trajectory of the lower incisor, while an excessive backward and downward movement of the working condyle gives a significantly steeper trajectory of the lower incisor (Fig. 4.20).

Occlusal structures of the mandible, such as the cusps of canines, premolars, and molars, move differently: the laterotrusive side moves noticeably out and down, while the mediotrusive side moves forward, inward, and downward. The influence of the working condyle should be noted on the laterotrusive side. The movement paths of the cusps on the laterotrusive side can then vary in direction and achieve a forward or backward movement component. In the Posselt diagram, this movement can be seen when viewed from the front or from a cranial view (Posselt 1952, 1962).



Fig. 4.20 Mediotrusion of the right joint and laterotrusion of the left joint. This compound movement corresponds to mandibular lateral movement to the left. The

mediotrusive side moves inward, downward, and forward and the graphical representation of the laterotrusive side results in an upward, outward, and backward movement

Observing the Working Condyle (Laterotrusive Condyle) The working condyle is the one situated on the side toward which the mandible moves. In this sideways movement, the working condyle only has a limited range of motion that is often described as rotational. In gnathology, and particularly in condylography, the direction of movement of this side is specified with the terms pro-, sur-, re-, and de- (Fig. 4.21). The movement components of the working condyle are called pro-trusion, sur-trusion, re-trusion, and de-trusion:

- PRO-trusion: a forward direction of the movement
- SUR-trusion: an upward direction of the movement
- RE-trusion: a backward direction of the movement
- DE-trusion: a downward direction of the movement

Naturally, combinations of these movements are possible:

- PRO-DE-trusion: forward and downward direction of motion
- PRO-SUR-trusion: forward and upward direction of motion



Fig. 4.21 Terminology of condylographic movements according to their direction in the sagittal plane

- RE-SUR-trusion: backward and upward direction of motion
- RE-DE-trusion: backward and downward direction of motion

It should be noted that these movements and descriptions of movements are based on paths recorded using hinge axis recording techniques and they are always subject to some distortion. Due to the dimension of the condyle, especially the transversal one, it would be an improper simplification to describe only the working condyle as "rotating." If a vertical rotation axis exists exactly at the geometrical center of the condyle, around which the working condyle should rotate in a mediotrusive mandibular movement, then the lateral pole of the working condyle may also be seen to move backward and the medial pole of the condyle forward. It should also be noted that this vertical rotation axis is not stable or fixed, and that it is subject to a significant shift with the sideways movement of the mandible when it will be progressively tilted downward and outward. Consequently, there is then a backward and upward movement of the lateral pole of the working condyle, while the medial pole moves forward and downward. However, it is not assumed that the working condyle vertical rotation axis is always located at the center of the condyle: the more eccentric-median this axis is, the more distinct the PRO-, SUR-, RE-, and DE- movements will look for the lateral parts of the condyle (Fig. 4.22).

Non-Working Observing the Condyle (Mediotrusive Condyle) This condyle performs a forward, downward, and inward movement. The shape and slope of the articular fossa and articular eminence are determining factors. However, the inward movement component must be considered in even greater detail (Fig. 4.23). The inward movement path has a characteristic shape if the border movement is executed. Here, border movement is used to understand that the condyle-disc complex maintains a contact with the medial wall of the articular fossa. Since a very long time, this movement has been of great importance in gnathology and for prosthodontic reconstruction of



Fig. 4.22 Sagittal view—movements of the hinge axis start from the zero (where the axes meet) and are described depending on their direction of movement

occlusal surfaces. The movement was defined by Sir Norman Godfrey Bennett (1870–1947), from whom the term *Bennett movement* originated (Bennett 1908). The equivalent components of an articulator are also often referred to as Bennett elements or inserts (Fig. 4.24a–d).

The Bennett movement, which is the pronounced inward movement of the non-working condyle, influences not only the movement of the mandible, but also individual points of occlusion, particularly on the mediotrusive and the laterotrusive side. The more developed the Bennett movement, the more backward oriented are the movement paths of the cusps in the premolar and molar, which must of course be considered in reconstructive occlusal procedures.

Bennett likewise established the term *immediate side shift* (ISS) of the non-working condyle. This is described as a movement whereby the non-working condyle performs a direct inward



Fig. 4.23 Mediotrusion is an overall downward and inward movement (*straight line*); however, the condylographic representation of this condylar motion can be visualized as a *curve* (*curved dotted line*) which traces the border Bennett movement

motion of between 0.5 mm and 2 mm, without the essential sagittal translational (forward and down) motion (Fig. 4.25). This phenomenon has been discussed extensively in various publications, with interpretations varying considerably, from physiological to pathological joint conditions. It is becoming increasingly accepted that there is a pathological cause. Today, ISS is known as *immediate mandibular lateral translation* and is defined as follows:

the translatory portion of lateral movement in which the non-working side condyle moves essentially straight and medially as it leaves the centric relation position (Academy of Prosthodontics 2017).



Fig. 4.24 Interchangeable mediotrusion inserts (Bennett inserts) that are commonly used for articulator programming (**a**–**d**). Not only does the shape of the path change, but its horizontal inclination can also be adjusted to fit

individual characteristics that are drawn from Instrumental Functional Analysis. Not shown here is the possibility to also change the steepness and shape of the fossa/eminence part of the articulator accordingly



Fig. 4.25 Immediate side shift—this non-physiological feature may be noticed on the mediotrusive (non-working) condyle. Its relevance is still debated

4.2.7 Opening Movement

This is a mandibular movement from a position at which both condyles are in the fossa, then move forward along the articular eminence, with substantial mandibular rotation (Fig. 4.26). The movement path is largely determined by the articular eminence, with dental structures only having a modulating influence in the initial phase. In contrast to protrusion, a significant degree of rotation of the hinge axis occurs. Forward and downward movement is limited with increased tension in the muscles and soft tissues, giving a soft end feel under physiological conditions. A hard end feel does not occur with this motion under physiological conditions.

The determinants of this movement are:

- Articular eminence (on both sides)
- Condyle–disc complex (on both sides)

The modulators (major influential factors) of this movement are:

- Front teeth (overjet, overbite)
- Skeletal and dental asymmetry
- Shape and orientation of the condyles
- Skeletal class
- Neuromuscular system

The Posselt diagram shows a characteristic image of this motion that is largely determined by the intact structures of the temporomandibular



Fig. 4.26 Rotation and translation are involved in mouth opening. The effect of ligaments can be observed when looking at the changing shape of the opening movement as seen in the most posterior contour of the Posselt scheme

joint (Posselt 1952, 1962). The characteristic path can only be observed where there are good ligament structures and an effective execution of the border movement during the recording: this means a movement starting with pure rotation, leading to a tightening of ligaments, with transition to combined translation and rotation (Fig. 4.27a, b).

In condylography, when viewed from the side, TMJ movement is seen to be very simple in principle and largely consistent with the protrusion, which is due primarily to the defined trajectory of the articular eminence. Viewed from the cranial direction, and equally from the front, the physiological opening movement path is straight, showing no deviation to the side. This is a symmetrical mandibular movement.

4.2.8 Closing Movement

The closing movement of the lower jaw is typically a more or less precise reverse of the opening movement. However, it should be noted that the combination of translation and rotation can differ **Fig. 4.27** The more posterior part of the Posselt scheme corresponds to the movement of the lower incisor during opening, with its two typical curved parts (**a**). At a joint level, the condylographic aspect of opening is a concave curve (**b**)



considerably in opening and closing, respectively. Nowadays, no explanatory models and accepted general paradigm are available to serve as a basis for detecting pathological conditions. The relevance of occlusion can be studied during the closing movement as dental structures get closer and closer. The clearer the occlusal inhibiting factors, in terms of elongated and misaligned teeth or groups of teeth, or steeply angled front teeth (i.e., deep bite or overbite), the sooner a typical movement pattern with already fully complete translational component and only pure rotation instead of closing can emerge, to achieve occlusion. As a consequence, research of a functional disorder is encouraged (Pullinger et al. 1993; Widmalm et al. 1994; LeResche 1997; Pullinger and Seligman 2000; Thilander et al. 2002; LeResche et al. 2003; Mohlin et al. 2007; Wang et al. 2009).

The closing movement is stopped at the point of occlusion, the end feel is usually hard and will be different only in case of severe periodontal disease or in some complete denture subject.

4.2.9 The Posselt Diagram

The Posselt diagram is used to describe the movements of the mandible. It was defined and its relevance shown in the 1950s and early 1960s (Posselt 1952, 1962). Usually the Posselt diagram is used to represent the movement at the lower incisor level. In most publications, it illustrates the border movements of the mandible in the lower anterior region. The diagram presents not only the lateral, frontal, and cranial, but also the three-dimensional perspective.

Naturally, similar diagrams can be presented for each point of occlusion. Condylography software makes it possible to generate individual Posselt diagrams for the individual patient.

4.2.10 The Hinge Axis

The hinge axis and the actual rotation axis of the mandible must always be considered separately. Any synonymous use of these terms is inaccurate and can lead to considerable misinterpretation. It is incorrect to equate mandibular hinge axis with the rotation axis. Due to the complex functional anatomy and biomechanics of the TMJ, mandibular motion sequences are determined by a range of interrelated axes that are also subject to spatial displacement. The mandibular hinge axis is the axis around which the mandible rotates if a pure rotation takes place in the temporomandibular joints of both sides.

It is important to consider the relation of the condyle-disc complex to the fossa, on the sagittal, vertical, and transverse planes. Determining joint centric relation has always been a central theme of function-oriented dentistry and gnathology, and still the end to scientific debate on this particular subject is not in sight. As a basic principle, however, the so-called retral, posterior, terminal, or most dorsal position of the hinge axis determines the centric relation, in case a pure rotation is being performed. At the same time, although more rarely and less consistently, the transverse centering of the condyle in the fossa is to be defined, but this is difficult to determine because of the geometric and spatial relationships involved. Nonetheless, the temporomandibular joints and the "centric" must always be seen in three-dimensional, dynamic terms. A static, two-dimensional view is at best a didactic method that must ultimately be put back into the context of a complex spatial approach. With the help of certain recording instruments (i.e., kinematic face bow), the hinge axis is determined using geometric relationships. Pure rotation of the mandible takes place around what is known as the terminal hinge rotation (THR). Here, the incisal edge of the lower front tooth is moved by about 15-20 mm downward and backward, corresponding to an approximate hinge axis rotation of about 12° (Fig. 4.2b). It is assumed here that the joints are correctly positioned in the transverse and vertical respects and that there is a functional and physiological relation between the condyle and disc. A translational hinge axis movement must take place for any further opening due to the tension in the ligaments (especially of the temporomandibular ligament) and capsular ligaments.

However, it would be fundamentally wrong to assume that it is possible to determine the hinge axis in every patient. In case it is not possible to determine the hinge axis, this represents a medical finding that must be documented in patient records and taken into consideration with respect to any treatment plan. Two possible strategies are briefly outlined as follows: in the first instance, attempts can be made during the initial treatment to achieve better determinability, or reproducibility, of this position. Secondly, it may be necessary to accept that the hinge axis is unstable and to establish a treatment plan based on other parameters (Sato and Kawamura 2006; Koolstra and Van Eijden 2007; Slavicek 2009).

4.2.11 DC/TMD and Condylography

In accordance to and as an advancement of the Research Diagnostic Criteria (RDC/TMD) for the use in research and science, the Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) have been developed for clinical purposes (Dworkin et al. 1992, 2002a, b; John et al. 2006; Dworkin 2010; Schiffman et al. 2014). The rationale behind this is that a consistent analytic scheme for temporomandibular disorders, using medical history, anamnestic data, and clinical examination leads to the correct diagnosis. The consequent application of these decision trees leads to the following groups of diagnosis:

- 1. pain-related TMD, eventually in combination with headache;
- 2. intra-articular jaw joint dysfunctions;
- 3. degenerative jaw joint dysfunctions.

Within the first group, myalgia, local myalgia, myofascial pain, myofascial pain with referral and arthralgia are combined. Correlations to headaches, especially tension-type headache, are possible. The second and third group are described in the appropriate chapters within this handbook.

One task of condylography is articulator programming and transfer of relevant information to the interdisciplinary team during the design and fabrication of a new individual occlusal restoration. Moreover, condylography considerably contributes to detailed diagnostics of temporomandibular disorders. The aim of condylographic diagnostics is the differential identification of structures and functions involved in the craniomandibular dysfunction: muscles, disc and ligaments, morphological changes of the condyle. In addition, the possible influence of occlusion on the dysfunction should be illustrated by the condylographic records.

At times, assigning the pathology of a patient to only one specific category proves to be a hard task (1. pain-related TMD, eventually in combination with headache; 2. intra-articular jaw joint dysfunctions; 3. degenerative jaw joint dysfunctions). In such cases, condylography enables to better clarify the findings. Many patients suffer from a combination of muscle problems (myalgia with or without referral, myofascial pain), internal derangements (disc displacement with or without reduction), alterations of mandibular mobility (deviation, deflection, limitation), headneck-throat pain, and degenerative changes of the condyle and/or the eminence. A problemoriented diagnosis enables the interdisciplinary team to establish and coordinate the therapeutic procedures and to apply them in a consequent chronological sequence. The therapeutic process can be controlled accordingly to the treatment strategy, and an adequate rigorous documentation equips the team with an objective orientation and will reveal whether the treatment objectives are truly achieved.

The aim of condylographic descriptive analysis is to systematically gather and analyze all possible phenomena from a condylographic standpoint. Each individual TMJ path will be collected and then an overall picture of the condylography will be created by merging the single paths. The aim of the interpretation is to correlate the observed phenomenon in condylography with aspects from the physiological and pathophysiological function of the temporomandibular joint and its separate components.

Before starting to plan any treatment, it is imperative that all components and structures of the stomatognathic system which are responsible for the reproducible phenomenon recorded in the condylography are identified. Needless to say, these findings have to be combined with the subjective symptoms of the patient and the results of clinical and instrumental function analyses. It is therefore possible to identify causal relations early on, as functional and structural changes start to occur in the temporomandibular joint area. On this basis, therapeutic consequences can be inferred. Here are structures and functional units that are generally concerned:

- Structures
 - Neuromuscular system
 - Capsule–ligament–disc
 - Capsule and ligaments
 - Disc
 - Occlusion
 - Bony structures
- Functions
 - Chewing and swallowing
 - Phonation
 - Stress-management
 - Posture and balance

Proper assignment based on condylographic findings to one or more of the above categories is only possible with sound knowledge of the anatomy, physiology, and biomechanics of the temporomandibular joint and of all of the involved components.

Interpretation of Condylography in Relation to the Neuromuscular System When neuromuscular problems are present, a condylography is typically described as follows:

- quantity is reduced but can be increased by repeating the movement several times (warming-up phenomenon);
- quality is poor-average with a tendency to poor/very poor quality;
- descriptions of the quality of partial movements are often very different;
- descriptions of quality in the excursive and incursive movements are often very different;
- a very poor quality can lead to difficulties in evaluating characteristics;
- incursion and excursion paths are not superimposable;
- start and end points are not coincident;
- start and end points do not coincide with the reference point (Fig. 4.28).



Fig. 4.28 Patient with myalgia. In this example, quality of tracing is poor; however, quantity is preserved. Start and end points do not coincide

Interpretation of Condylography in Relation

to the Disc Under physiological conditions, the condyle and the disc have a very close and stable relationship that is maintained in the whole range of movement. There are only minimal position changes of the disc in relation to the condyle during movement, and these generally do not influence the condylographic recordings, or only influence them minimally, and are not included in the description of condylography.

Because of its morphology, the disc is also able to influence the position of the condyle and therefore the hinge axis. However, it can also be the case that the extremely stable relation between the disc and condyle, guaranteed by the lateral ligaments and the joint capsule, is loosened. The condyle will then be able to leave the thinner central part of the discus and be positioned permanently, or even just temporarily, on the thicker posterior edge. In condylography, this can lead to clear, typical phenomena:

- changing characteristic of the tracing;
- initial convexity, if the hinge axis is determined with a dorsal "drive" during hinge axis location;
- hypermobility, the quantity is increased;

- separation of excursion and incursion tracings;
- early or complete separation between the superimposed tracings of protrusion and mediotrusion;
- clear influenceability with manipulation;
- immediate side shift—spontaneous or triggered;
- signals in the recording indicating disc displacement with reduction;
- typical condylography characteristics indicating disc displacement without reduction.

Typical Findings in Condylography with an Unstable Relation Between Disc and **Condyle** The correct technique to locate the hinge axis and perform a centric registration is still discussed with extreme controversy. The concept of the centric relation is however a central element in daily clinical dentistry. There is a daily need to determine lower jaw positions which can be reproduced and are not determined by dental structures (Orthlieb et al. 2011). Various ideas and schools of thought have established themselves regarding the right technology to use when determining centric relation. This however has led to confusion, doubt, and ultimately resignation because of the variety of overlapping and unclear definitions (Slavicek et al. 1983). Nevertheless, a disc displacement with reduction (DDwR), clinically called a "reciprocal click," will show a typical tracing in condylography (*crossing signal*). In such condition, the disc is not in the physiological position in centric relation but is mostly shifted forward and inward—in front of the condyle, during the opening movement. During protrusion and mediotrusion, a repositioning of the disc takes place, the functional condyle–disc unit is restored, and the disc will now remain in place during the rest of excursion and during part of incursion. Only just before reaching centric relation, a displacement will take place again-the disc and physiological condyle lose their relation (Figs. 4.29a-f and 4.30a, b). On the other hand, a disc displacement without reduction (DDWoR), clinically noiseless, is presented by a typical tracing in condylography as well. At rest, the disc is not in the physiological position in centric relation but dislocated forward and inward-in front of the condyle. In the case of the protrusion movement and mediotrusion, there is no restoration of the disc-condyle unit during the overall excursive and incursive movement. The movement is reduced in quantity, and the characteristic of the tracing is straight but it may show some convex-



Fig. 4.29 (**a**–**f**) Condylographic sign of DDwR (*reciprocal click* or *crossing signal*). Reduction is seen in (**b**). Disc displacement occurs again in (**e**)



Fig. 4.30 (a, b) Bilateral DDwR as seen on a real condylographic tracing. Protrusion–retrusion is here displayed (a). Close-up of the first part of the same tracing (right side) is seen in (b). The cursor (+) is placed where the second click (closing click) takes place. Xyz coordi-

ity, especially in the case of a chronic displacement (Figs. 4.31a–d and 4.32). Unlike the acute joint block, in chronic locked joints the patient mostly indicates no pain, both in a relaxed state and when the lower jaw is moving.

nates are given by the software and can be used in the articulator for diagnostic/therapeutic purposes as these are the spatial points where disc displacement is about to occur again during closing

The system used in Fig. 4.12, 4.28, 4.30 and 4.32 is CADIAX (GAMMA Medizinischwissenschaftliche Fortbildungs-GmbH, Austria) The articulator displayed in Fig. 4.24 is SAM 2P (SAM Präzisionstechnik, Germany)



Fig. 4.31 (a–d) Condylographic sign of DDWoR (straight characteristic) with reduced quantity, a trait which is especially visible in patients with acute closed lock



Fig. 4.32 Right side DDWoR (acute) as seen on a real condylographic tracing—protrusion–retraction is here displayed. The right side has a straight characteristic with a reduced quantity

References

- A.I.G. Associazione Italiana di Gnatologia. Ricostruzione delle Superfici Masticatorie dei Denti Anteriori. Published for Members; 1994. p. 41.
- Academy of Prosthodontics. The glossary of prosthodontic terms: ninth edition. J Prosthet Dent. 2017;117(5S):e1–e105. https://doi.org/10.1016/j. prosdent.2016.12.001.
- Ahlberg J, Lobbezoo F, Ahlberg K, Manfredini D, Hublin C, Sinisalo J, et al. Self-reported bruxism mirrors anxiety and stress in adults. Med Oral Patol Oral Cir Bucal. 2013;18:7–11.
- Bennett NG. A contribution to the study of the movements of the mandible. Proc R Soc Med. 1908;1:79–89.
- Currie P. Age and gender as factor in temporomandibular joint movement in adolescents, as determined by computerized 3D electronic condylography. Int J Stomatol Occlusion Med. 2010;3:76–82. https://doi. org/10.1007/s12548-010-0048-2.
- Dawson PE. Commentary from Dawson Center for Advanced Dental Study. 2000. http://www.tcbsc. net/pdfs/Peter%20Dawson%20Commentary.pdf. Accessed 9th May 2018.
- Dawson PE. Functional occlusion: e-book. From TMJ to smile design. St Louis: Elsevier, Mosby; 2006. p. 142.
- Dworkin SF. Diagnostic criteria for temporomandibular disorders: current status and future relevance. J Oral Rehabil. 2010;37:734–43. https://doi. org/10.1111/j.1365-2842.2010.02090.x.
- Dworkin SF, Von Korff MR, LeResche L. Epidemiologic studies of chronic pain: a dynamic-ecologic perspective. Ann Behav Med. 1992;14:3–11. https://doi. org/10.1093/abm/14.1.3.
- Dworkin SF, Huggins KH, Wilson L, Mancl L, Turner J, Massoth D, et al. A randomized clinical trial using research diagnostic criteria for temporomandibular disorder-axis II to target clinic axes for a tailored self-care TMD treatment program. J Orofac Pain. 2002a;16:48–63.
- Dworkin SF, Turner JA, Mancl L, Wilson L, Massoth D, Huggins KH, et al. A randomized clinical trial of a tailored comprehensive self-care treatment program for temporomandibular disorders. J Orofac Pain. 2002b;16:259–76.
- Fialka V, Weber KH, Slavicek G, Vinzenz K. Die elektronische Axiographie zur Steuerung der physikalischen. Therapie Biomed Techn. 1990;35:169–70.
- Freesmeyer W. Therapieorientierte Gliederung der Krankheitsbilder. In: Ahlers MO, Jakstat HA, editors. Klinische Funktionsanalyse: Manuelle Strukturanalyse-Interdisziplinäre Diagnostik. Hamburg: Denta-Concept; 2011.
- John MT, Hirsch C, Reiber T, Dworkin SF. Translating the research diagnostic criteria for temporomandibular disorders into German: evaluation of content and process. J Orofac Pain. 2006;1:43–52.
- Koolstra JH, Van Eijden TM. Consequences of viscoelastic behavior in the human temporomandibular

joint disc. J Dent Res. 2007;86:1198–202. https://doi. org/10.1177/154405910708601211.

- Koolstra JH, Naeije M, van Eijden TM. The threedimensional active envelope of jaw border movement and its determinants. J Dent Res. 2001;80:1908–12. https://doi.org/10.1177/00220345010800100901.
- LeResche L. Epidemiology of temporomandibular disorders: implications for the investigation of etiologic factors. Crit Rev Oral Biol Med. 1997;8:291–305.
- LeResche L, Mancl L, Sherman JJ, Gandara B, Dworkin SF. Changes in temporomandibular pain and other symptoms across the menstrual cycle. Pain. 2003;106:253–61.
- Lindauer SJ, Sabol G, Isaacson RJ, Davidovitch M. Condylar movement and mandibular rotation during jaw opening. Am J Orthod Dentofacial Orthop. 1995;107:573–7. https://doi.org/10.1016/ S0889-5406(95)70099-4.
- Lobbezoo F, Ahlberg J, Raphael KG, et al. International consensus on the assessment of bruxism: report of a work in progress. J Oral Rehabil. 2018;00:1–8. https:// doi.org/10.1111/joor.12663.
- Lundeen HW, Wirth C. Condylar movement patterns engraved in plastic blocks. J Prosthet Dent. 1973;30:866–75.
- Mauderli AP, Lundeen HC. Simplified condylar movement recorders for analyzing temporomandibular derangement. Cranio. 1991;4:208–12.
- Meyer RA. Chapter 163: The temporomandibular joint examination. In: Walker HK, Hall WD, Hurst JW, editors. Clinical methods: the history, physical, and laboratory examinations. 3rd ed. Boston: Butterworths; 1990.
- Miller D, et al. Mechanische und elektronische Kondylographie, ein Methodenvergleich. Inf Orthod Kiefer Orthop. 1995;27:15–23.
- Mohlin B, Axelsson S, Paulin G, Pietilä T, Bondemark L, Brattström V, et al. TMD in relation to malocclusion and orthodontic treatment. Angle Orthod. 2007;77:542–8. https://doi. org/10.2319/0003-3219(2007)077[0542:TIRTMA]2. 0.CO;2.
- Okeson JP. Il trattamento delle disfunzioni e dei disordini temporomandibolari. 7a edizione. Edizioni Martina; 2014. Chapter 1. Translated from the original Management of temporomandibular disorders and occlusion. 7th ed. St Louis: Elsevier, Mosby; 2013.
- Orthlieb JD, Hernandez G, Darmouni L, Rè JP, Girardeau A, Slavicek G. Myostabilized centric relation. Int J Stomatol Occlusion Med. 2011;4:87–94. https://doi. org/10.1007/s12548-011-0014-7.
- Piehslinger E, Celar AG, Celar RM, Slavicek R. Computerized axiography: principles and methods. Cranio. 1991;9(4):344–55.
- Posselt U. Studies in the mobility of the human mandible. Acta Odontol Scand. 1952;10:19–160.
- Posselt U. Hinge opening axis of the mandible. Acta Odontol Scand. 1956;14:c1.

- Posselt U. Physiology of occlusion and rehabilitation. Oxford: Blackwell Scientific Publications; 1962.
- Pullinger AG, Seligman DA. Quantification and validation of predictive values of occlusal variables in temporo-mandibular disorders using a multifactorial analysis. J Prosthet Dent. 2000;83:66–75. https://doi. org/10.1016/S0022-3913(00)70090-4.
- Pullinger AG, Seligman DA, Gornbein JA. A multiple logistic regression analysis of the risk and relative odds of temporo-mandibular disorders as a function of common occlusal features. J Dent Res. 1993;72:968– 79. https://doi.org/10.1177/00220345930720061301.
- Rocabado M. Arthrokinematics of the temporomandibular joint (symposium on temporomandibular joint dysfunction and treatment). Dent Clin N Am. 1983;27:573–94.
- Sato S, Kawamura H. Changes in condylar mobility and radiographic alterations after treatment in patients with non-reducing disc displacement of the temporomandibular joint. Dentomaxillofac Radiol. 2006;35:1–8. https://doi.org/10.1259/dmfr/92464710.
- Schiffman E, Ohrbach R, Truelove E. 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;28:6–27. https://doi. org/10.11607/jop.1151.
- Slavicek R. Die funktionellen Determinanten des Kauorgans. München: Zahnärztlich-Medizinisches Schrifttum; 1984.
- Slavicek G. Okklusion im Schatten Evidenz basierter. Medizin Stomatol. 2009;106:17–22.

- Slavicek G. Kephalometrie/Cephalometry: Transfer— Dokumentation—Report 1. Stuttgart: Steinbeis Edition; 2011.
- Slavicek R, Lugner P. Der schädelbezüglich teiladjustierbare Artikulator—eine mathematischemechanische Analyse und Bewertung. Stomatologie. 1976;73:84–102.
- Slavicek R, Lugner P. Über die Möglichkeit der Bestimmung des Bennettwinkels bei sagittaler Aufzeichnung. Stomatologie. 1979;75:270–84.
- Slavicek R, Avril C, Collesano V. Centric relation. Riv Ital Stomatol. 1983;52(2):101–2.
- Slavicek G, Weber KH, Fialka V, Vinzenz K. Use of electronic axiography in diagnosis and therapy. Biomed Tech. 1990;2:219–20.
- Tanaka E, Van Eijden T. Biomechanical behavior of the temporomandibular joint disc. Crit Rev Oral Biol Med. 2003;14:138–50.
- Thilander B, Rubio G, Pena L, de Mayorga C. Prevalence of temporomandibular dysfunction and its association with malocclusion in children and adolescents: an epidemiologic study related to specified stages of dental development. Angle Orthod. 2002;72:146–54. https:// doi.org/10.1043/0003-3219(2002)072<0146:POTDA I>2.0.CO;2.
- Wang MQ, Xue F, He JJ, Chen JH, Chen CS, Raustia A. Missing posterior teeth and risk of temporomandibular disorders. J Dent Res. 2009;88:942–5. https:// doi.org/10.1177/0022034509344387.
- Widmalm SE, Westesson PL, Kim IK, Pereira FJ Jr, Lundh H, Tasaki MM. Temporomandibular joint pathosis related to sex, age, and dentition in autopsy material. Oral Surg Oral Med Oral Pathol. 1994;78:416–25.

G. Gerbino (🖂) · G. Ramieri

guglielmo.ramieri@unito.it

e-mail: giovanni.gerbino@unito.it;

Italy

V. Chianca

Developmental Disorders

Giovanni Gerbino, Vito Chianca, and Guglielmo Ramieri

Key Points

- · This chapter focuses on mandibular abnormalities that can be encountered when assessing temporomandibular joint dysfunctions and their imaging.
- Growth disturbances of the condyle may be unilateral or bilateral, and they may affect both the morphology of the face and the functions of the stomatognathic system.
- Condylar aplasia and hypoplasia determine ٠ ramus and mandibular underdevelopment as a consequence of growth disturbances of the condylar cartilage.
- · Bilateral forms include Treacher Collins syndrome, Nager syndrome, and Stickler syndrome. These are frequently associated with Pierre Robin sequence.
- Unilateral forms are accompanied by facial asymmetry and include hemifacial microsomia and Goldenhar syndrome.
- · Condylar hyperplasia can cause mandibular asymmetry due to excessive growth on one side.

Department of Surgical Sciences, Division of

Maxillofacial Surgery, University of Turin, Turin,

IRCCS Istituto Ortopedico Galeazzi, Milan, Italy

- Multidetector computed tomography has to ٠ enclose the whole facial skeleton so as to obtain multiplanar reconstructions (MPR) and panoramic three-dimensional (3D) volumerendering (VR) reconstructions for diagnosis and treatment planning.
- Contemporary management of these disorders is based on a team approach and contemplates anatomical, functional, and psychosocial issues.

Growth disturbances of the mandibular condyleand its related structures-can be unilateral or bilateral, and they may affect both morphology of the face and functions of the stomatognathic system. These conditions arise from a variety of anomalies that present with different onsets and various morpho-functional characterization.

For the purpose of this handbook, and in the light of the existing classifications, developmental disorders shall be distinguished according to morphology, etiology, and time of appearance (Obwegeser 2001; Kaneyama et al. 2008; Pirttiniemi et al. 2009). The following forms can be identified:

- condylar aplasia/hypoplasia, including its two subgroups:
 - embryonic-affecting the first and second branchial arches, due to genetic conditions or to systemic forms (exposition to toxic agents, local factors);



[©] Springer Nature Switzerland AG 2020

T. Robba et al. (eds.), MRI of the Temporomandibular Joint,

https://doi.org/10.1007/978-3-030-25421-6_5

- acquired postnatal—consequence of an adverse event during active growth such as trauma, infection of the joint or the surrounding structures (i.e., the middle ear), tumors and their therapies (i.e., radiation therapy);
- condylar hyperplasia, depending on condylar hyperactivity during growth, usually unilateral.

5.1 Condylar Aplasia/Hypoplasia

Condylar aplasia or hypoplasia is a consequence of growth disturbances of the condylar cartilage determining ramus and mandibular underdevelopment (Walker 1968; Sarnat 1969; Obwegeser 2001; Ferri et al. 2006).

Condylar aplasia or hypoplasia is frequently associated with other craniofacial conditions like the *Pierre Robin sequence* (PRS), where mandibular growth disturbances hinder rotation and advancement of the tongue. The consequence of this is glossoptosis, with the tongue remaining pressed against the posterior pharynx, leading to airway obstruction, lack of palatal fusion, and U-shaped palatal cleft (Buchanan et al. 2014).

The most important forms of developmental condylar aplasia and hypoplasia are described below.

5.1.1 Bilateral Mandibulofacial Dysostosis

These include purely bilateral forms that are frequently associated with Pierre Robin sequence.

Treacher Collins Syndrome Treacher Collins syndrome (TCS), also known in Europe as Franceschetti–Klein syndrome, is a mandibulofacial dysostosis characterized by symmetrical bilateral abnormalities of structures that develop from the first and second branchial arches.

Affecting 1/50,000 live births, TCS is an autosomal dominant disorder in up to 40% of

cases. albeit with variable expressivity (Magalhães et al. 2007). Facial features include bilateral zygomatic arch and malar hypoplasia, as well as mandibular hypoplasia with micro and retrogenia (Fig. 5.1a-d). Eyes have an antimongoloid slant, lower lid and external ear anomalies are present too. Hearing is frequently affected as a result of hypoplasia of external auditory canal and ossicles in the middle ear (Posnick and Ruiz 2000; Gorlin et al. 2001). The maxillary and mandibular bones are hypoplastic with a typical skeletal class II profile. Anterior open bite is often present and a steep occlusal plane usually develops because of an overall clockwise rotation which determines a bird- or fish-like face appearance. These features have variables effect on the temporomandibular joints (TMJ) and the masticatory muscles, with a higher incidence of TMJ dysfunction and ankylosis (Chang and Steinbacher 2012).

Nager Syndrome This rare syndrome, known as acrofacial dysostosis, was first described by Nager and De Reynier and it has craniofacial features that are very similar to TCS. Associated anomalies include those at the radial aspect of the hand (preaxial deficiency, hypoplasia of the radius and the thumb) (Nager and De Reynier 1948).

Stickler Syndrome Stickler syndrome, or hereditary arthro-ophthalmopathy, is a connective tissue disorder affecting the eyes, the craniofacial skeleton, and the joints. The estimated incidence is around 1/10,000 births (Printzlau and Andersen 2004).

From a craniofacial point of view, it is associated with PRS sequence, with severe micrognathia and midface hypoplasia. Ophthalmological manifestations (early-onset cataract, retinal detachment, vitreous anomalies with myopia) are present in newborns and are usually nonprogressive. As collagen synthesis is impaired or deficient, joint conditions include early-onset spondylolisthesis and hypermobility which may require complex orthopedic treatment.



Fig. 5.1 Treacher Collins syndrome—adolescent patient (**a**, **b**). Facial features include zygomatic hypoplasia, micro and retrogenia, antimongoloid slant of the eyes, lower lid and external ear anomalies (a hearing aid is visible on one side). Treacher Collins syndrome—child

5.1.2 Unilateral Condylar Hypoplasia

This form includes typically unilateral conditions which are mostly defined by remarkable facial asymmetry.

patient (\mathbf{c} , \mathbf{d}). Multiplanar volume rendering reconstructions (MPVR) (\mathbf{c}) and 3D in volume rendering (\mathbf{d}) highlight the mandibular and zygomatic bone hypoplasia which severely modifies the appearance of the TMJ, the zygomatic arch, and the zygomatic part of the orbit

Hemifacial Microsomia Hemifacial microsomia (HFM), also known as otomandibular dysostosis or craniofacial microsomia, is a relatively common congenital craniofacial anomaly (1/3200–5600 births) characterized by asymmetric underdevelopment of structures which originate

from the first and second branchial arches (Hartsfield 2007).

HFM affects in various ways skeletal, soft tissue, and neuromuscular components, resulting in underdevelopment of the lower half of the face (Galea et al. 2018). Deformities usually involve ear, mandible, maxilla, zygomatic arch and bone, temporal bone, and facial muscles. Multiple cranial nerves abnormalities can be observed.

Although clinical patterns are variable, mandibular deformity typically correlates with soft tissue and muscular deformity (Moulin-Romsée et al. 2004; Meazzini et al. 2011a, b).

Etiology is heterogeneous, with chromosomal disorders, exposition to teratogen agents and vascular anomalies reported as causal factors (Cousley and Calvert 1997). Poswillo hypothesized that a hematoma in the stapedial artery area may play a role in interfering with the branchial arches in utero-blood supply (Poswillo 1973; Hartsfield 2007).

Growth of the affected side does not match normal growth of the opposite side and, depending on the degree of condylar and mandibular ramus involvement, this leads to deformities which typically show a progressive worsening.

Mildest forms, characterized by a small condyle with thinner than normal condylar cartilage but fairly normal endochondral ossification, exhibit a less severe asymmetry. In such cases, treatment decision-making may lead to correction by means of functional orthodontics and conventional orthognathic surgery (Fig. 5.2a–e) (Caccamese et al. 2006; Pirttiniemi et al. 2009).

Severe forms of HFM with aplasia or severe hypoplasia of the condyle, with complete absence of condylar cartilage and endochondral ossification, cause severe facial asymmetry along with maxillary canting. This anticipated occurrence justifies early intervention by restoring the ramus–condyle unit with autogenous grafts (rib grafts) or osteodistraction (Buchanan et al. 2014) (Fig. 5.3a–c).

Because of these aspects, HFM classification is traditionally based on the degree of TMJ mandibular deficiency, ranging from type I (small glenoid fossa, condyle, and ramus) to type III (absent condyle and/or glenoid fossa) (Mulliken and Kaban 1987).

A more-detailed and operatively oriented classification was provided by Kaban (Kaban et al. 1988).

Lack or underdevelopment of structures was therefore classified as follows:

- Type I: mild glenoid, ramus, and condylar hypoplasia;
- Type IIA: moderate glenoid, ramus, and condylar hypoplasia, with satisfactory TMJ function;
- Type IIB: mild-to-moderate glenoid, ramus, and condylar hypoplasia, with incongruous function and positioning of the joint components;
- Type III: absence of ramus, condyle, and TMJ.

Not only does this approach take into account anatomical relationships, but also the function of the TMJ, and it allows to manage indications for a tailored surgical treatment plan.

In growing children, long-term results of asymmetry surgical treatment, unfortunately, show a consistent recurrence towards asymmetry during subsequent growth, and the neuromuscular deficit seems a critical factor which drives the pattern of craniofacial growth (Meazzini et al. 2008). This particular aspect lies behind the relevant post hoc remarks made by Meazzini, who distinguished true HFM from another asymmetry condition (condylar coronoid collapse-CCC), based on the latter's successful long-term post-surgical behavior (Meazzini et al. 2011a, b). Both conditions show a similar mandibular hypoplastic pattern; however, a different morphological appearance can be identified for soft tissues (normal masticatory muscles and ears) and most importantly for coronoid, sigmoid notch, and condyle (deep sigmoid notch, condylar and coronoid process collapsed against one another).

Goldenhar Syndrome Goldenhar syndrome, also known as oculoauriculovertebral syndrome (OAVS) is considered to be a variation of HFM (Goldenhar 1952; Gorlin et al. 1963; Caccamese



Fig. 5.2 Mild mandibular hypoplasia—orthognathic surgery candidate. Panoramic radiography (**a**) and lateral cephalogram (**b**) show slight asymmetry of condylar development as well as retrogenia. Sagittal SE PD

Jr et al. 2006). OAVS includes the distinguishing unilateral (rarely bilateral) hypoplastic mandible associated with epibulbar dermoids and images of the left TMJ displays a flattened tubercle (*black arrow*, **c**). The condyles are reduced in size as seen on the axial plane (**d**). In a coronal view the left disc is displaced anterolaterally (e)

vertebral anomalies, which are sometimes accompanied by genitourinary, renal, and cardiac malformations.



Fig. 5.3 Hemifacial microsomia—type IIb. Right side soft tissue deficiency and microtia can be observed along with a severe mandibular asymmetry (**a**, **b**). The chin deviates towards the affected side and the position of the

Etiologic heterogeneity of this syndrome is similar to that of HFM. The classification used here is also used for HFM and it takes into account the major anomalies including orbital, mandible, ear, nerve, soft tissue, and any extracranial manifestation of the disease. The acronym used is OMENS+ (O for orbital distortion, M for mandibular hypoplasia, E for ear anomaly, N for facial nerve involvement, S for soft tissue

corners of the mouth is affected. Volume rendering CT reconstructions clearly show right mandibular hypoplasia and occlusal plane canting (c)

deficiency, + for extra-craniofacial features) (Vento et al. 1991; Horgan et al. 1995).

5.1.3 Acquired Postnatal Condylar Hypoplasia

An adverse event in the condylar region during late postfetal or early postnatal growth can result

in abnormal growth and malformation of the mandible with unilateral mandibular hypoplasia and facial deviation, combined with transversal upper occlusal plane canting, chin deviation, curved facial midline, gonion, and lips asymmetry.

Within this group of patients, mandibular shape is constantly similar as presentation basically depends on the degree of damage and on the developmental stage of the mandible, regardless of the type of adverse event (Obwegeser 2001; Meazzini et al. 2011a, b; Gerbino et al. 2014). The most common causes are trauma, joint infections, middle ear infections, juvenile idiopathic arthritis (JIA), tumors, and outcomes of tumorrelated therapies (Fig. 5.4a–d).

Facial anomalies can first become evident long time after the damage has occurred. It is not difficult to expect ankylosis as part of the development and worsening of a hypoplastic mandible,



Fig. 5.4 Right unilateral mandibular hypoplasia—adolescent patient (**a**, **b**). Facial asymmetry with deviation towards the affected side is visible along with mandibular retrognathism. Mandibular angles and lips asymmetry are

related to the underdevelopment of right side. Coronal reformatted CT scan and 3D assessment show condylar and ramus asymmetry, as well as occlusal plane canting (\mathbf{c}, \mathbf{d})

also given the relevance that muscle function carries out in physiological mandibular growth (Meazzini et al. 2008).

5.2 Condylar Hyperplasia

Mandibular asymmetries due to condylar overgrowth are traditionally outlined from a morphological and clinical perspective as follows:

- Type I—hemimandibular elongation: excessive horizontal growth of the mandible with a "normal" condyle and elongated neck and ramus with a significant deviation of the chin and lower interincisal line towards the unaffected site;
- Type II—hemimandibular hyperplasia: mandibular enlargement with increased height and width of the mandibular body on one side, usually without significant chin deviation. Condylar head and neck are often enlarged;
- Type III: combination of type I and II (Obwegeser and Makek 1986).

Condylar hyperactivity manifests itself during childhood and can be straightforwardly detected when it is unilateral (Obwegeser 2001).

This condition is characterized by distortion of the midface and asymmetry of the lower third (Fig. 5.5a–c). Etiology is controversial and largely unknown. Theories include neoplasia, trauma, infection, abnormal loading, hormonal influences, and vascularity (Kaneyama et al. 2008). Regardless of the underlying cause, one condylar growth center becomes largely more active than the other or its activity lasts for a longer time, causing a gradually worsening mandibular asymmetry for which patients usually seek treatment.

5.3 Imaging

Assessment of shape, size, volume, and symmetry of cranial bones is crucial for the evaluation of developmental anomalies. Today, computed tomography (CT) in the form of multidetector computed tomography (MDCT) has to enclose the whole facial skeleton so as to obtain multiplanar reconstructions (MPR) and panoramic three-dimensional (3D) volume-rendering (VR) reconstructions for diagnosis and treatment planning.

Especially in the case of unilateral forms, one possible scenario is that typical clinical objective findings are not remarkable. In such mild cases, detection of a developmental disorder is at times occasional. Orthopantomogram can easily highlight mandibular asymmetries and it is still a widespread first assessment (Fig. 5.6a, b). For similar reasons, cone beam computed tomography (CBCT)-reformatted panoramic imaging can be used for a general mandibular overview (Tsiklakis et al. 2004). Computed tomography can undoubtedly give a sharper anatomical information as opposed to conventional radiology; however, CBCT is a sufficient imaging technique most of the times and even more so in milder asymmetries-for it has a lower absorbed dose than MDCT (Fig. 5.7a-d) (Larheim et al. 2015; Mupparapu and Nadeau 2016).

For more severe cases, digital imaging and communications in medicine (DICOM) files from CT can be used to generate stereolithographic models and STL files that can be imported into planning software. The advantages are the possibility to perform 3D surgical planning and fabrication of patient-specific computer-generated surgical tools such as templates, cutting guides, patient-specific implants, and osteosynthesis plates. Evaluation of muscle volumes and insertions is also viable and satisfactory with this imaging modality. Magnetic resonance imaging (MRI) has consequently limited relevance because of the poor definition of hard tissue and the required time for scanning, which is an important issue to consider since patients are often children and perhaps uncompliant. Within this group of patients, MRI has a role in case of detectable TMJ alterations that may be related to the dysmorphic skeletal condition and which require in-depth TMJ functional assessment (Fig. 5.8a-c) (Larheim et al. 2015). Condyle-disc relationships are known to vary considerably among the conditions that have been here described and, especially in the case of HFM, the underdevelopment of the skeletal component of the joint does not necessarily associate with the characteristics of the disc
5 Developmental Disorders



Fig. 5.5 Right condylar hyperplasia—adult patient (**a**). Deviation of the chin towards the left side with asymmetry of the corners of the mouth and of the mandibular angles. Posteroanterior cephalogram (**b**) shows remark-

able ramus elongation and an increased size of the right condyle (*arrow*), which is associated with high scintigraphic activity (c)



Fig. 5.6 Orthopantomogram (a) can be useful in detecting asymmetries of the two mandibular sides. In this case, the left side is smaller than the right one, with a smaller

(absence, presence, and displacement). This therefore implies that MRI has a significance both because knowledge about TMJ soft tissue status cannot be inferred from the patient's skeletal features detected on conventional radiography and CT, and because it allows planning of distraction osteogenesis procedures with more insight into prognosis (Kitai et al. 2004).

5.4 Management

Surgical treatment protocols and orthognathic considerations are beyond the scope of this handbook and are extensively described elsewhere. Contemporary management of these disorders is based on a team approach and contemplates ana-

ramus and a smaller condyle (*arrow*). Tomographic images (**b**) show a flattened and remodeled condyle (*arrows*)

tomical, functional, and psychosocial issues. Careful multidisciplinary evaluation is needed to provide for an individualized coordinated treatment plan, with a programmed timing for every step of therapy (Buchanan et al. 2014; Galea et al. 2018).

At birth, treatment priority should focus on airway stabilization, feeding, and eyesight. This stage is particularly crucial for bilateral hypoplasia patients. Depending on the severity of airway obstruction and hypoplasia, different procedures may be required, including special infant positioning, glossopexy, tracheostomy, and early mandibular osteodistraction, which can be considered a life-saving procedure (Andrews et al. 2013; Flores et al. 2014; Khansa et al. 2017). Once the child is stable from a respiratory and



Fig. 5.7 Multidetector computed tomography shows better details of the same patients shown in Fig. 5.6. Oblique sagittal plane reconstructions (**a**) display a reduced condylar size (*asterisk*) and a wider glenoid cavity (*arrow*), as

nutritional standpoint, a staged approach is normally adopted, depending upon the severity of the deformity, facial growth pattern, and psychosocial factors.

From a surgical point of view, different protocols have been proposed but as a general rule zygomatic reconstruction is planned at about 8–10 years of age and microtia is addressed from age 7. TMJ and ramus reconstruction are staged according to the severity of the deformity: Kaban types I and IIA are normally reconstructed at early skeletal maturity (13–15 years of age) via conventional orthognathic surgery or staged protocols which combine a stepped sequence of distraction and orthognathic surgery (Gerbino et al. 2014; Galea et al. 2018).

also visible in coronal (**b**) and 3D reconstructions (**d**). Axial reconstructions (**c**) show a smaller left condyle (*arrow*) which is also placed more anteriorly with respect to the contralateral

Kaban type IIB requires reconstruction of the ramus–condyle unit via multiple stepped procedures in which orthognathic surgery surely has a role together with grafts, distraction, and total TMJ alloplastic reconstruction. Secondary procedures for correction of soft tissue deficiencies should also be planned in these patients (Obwegeser 2001; Mommaerts and Nagy 2002; Wolford et al. 2012).

In Kaban type III, reconstruction of the missing ramus–condyle and fossa is accomplished with the use of costochondral grafts or with total TMJ alloplastic reconstruction, which is gaining wider acceptability even in growing patients due to the predictability of clinical outcomes (Wolford et al. 2012; Galea et al. 2018).



Fig. 5.8 Oblique sagittal PD weighted images show condylar remodeling (**a**, *arrow*) and anterior disc displacement (**b**, *arrow*) without reduction in maximum opening (**c**, *arrow*)

Lipofilling and fat grafting are more and more common for soft tissue correction, and nasal and touch-up procedures are normally completed after skeletal maturity.

Ideal management of complex skeletal abnormalities demands a coordinated effort from a team, with extensive use of up-to-date 3D morphologic analysis and 3D computer-assisted planning and surgery, and close cooperation with patients' families. Reconstruction needs to be staged in order to coordinate surgical procedures which have a time-specific significance that is matched to both the development phase and the need to preserve growth potential.

Timing of therapy for condylar hyperplasia is dictated by an assessment on condylar growth, and this is normally performed with a combination of periodical clinical observation and bone scanning with technetium-99 planar bone scintigraphy or PET-scintigraphy (Fig. 5.5a–c). During active growth, high or proportional condylectomy is indicated, while orthognathic surgery should be chosen when condylar growth has burned itself out.

References

- Andrews BT, Fan KL, Roostaeian J, Federico C, Bradley JP. Incidence of concomitant airway anomalies when using the university of California, Los Angeles, protocol for neonatal mandibular distraction. Plast Reconstr Surg. 2013;131(5):1116–23. https://doi.org/10.1097/ PRS.0b013e3182865da0.
- Buchanan EP, Xue AS, Hollier LH Jr. Craniofacial syndromes. Plast Reconstr Surg. 2014;134(1):128e–53e. https://doi.org/10.1097/PRS.00000000000308.
- Caccamese JF Jr, Costello BJ, Mooney MP. Novel deformity of the mandible in oculo-auriculo-vertebral spectrum: case report and literature review. J Oral Maxillofac Surg. 2006;64(8):1278–82. https://doi. org/10.1016/j.joms.2006.03.034.
- Chang CC, Steinbacher DM. Treacher Collins syndrome. Semin Plast Surg. 2012;26(2):83–90. https://doi.org/1 0.1055/s-0032-1320066.
- Cousley RR, Calvert ML. Current concepts in the understanding and management of hemifacial microsomia. Br J Plast Surg. 1997;50(7):536–51.
- Ferri J, Carneiro JM, Lemiere E, Vereecke F, Baralle MM. Severe congenital hypoplasia of the mandibular condyle-diagnosis and treatment: a report of 2 cases. J Oral Maxillofac Surg. 2006;64(6):972–80. https://doi. org/10.1016/j.joms.2006.02.019.
- Flores RL, Tholpady SS, Sati S, Fairbanks G, Socas J, Choi M, et al. The surgical correction of Pierre Robin sequence: mandibular distraction osteogenesis versus tongue-lip adhesion. Plast Reconstr Surg. 2014;133(6):1433–9. https://doi.org/10.1097/ PRS.00000000000225.
- Galea CJ, Dashow JE, Woerner JE. Congenital abnormalities of the temporomandibular joint. Oral Maxillofac Surg Clin North Am. 2018;30(1):71–82. https://doi. org/10.1016/j.coms.2017.09.003.
- Gerbino G, Bianchi FA, Verzé L, Ramieri G. Unilateral mandibular hypoplasia in adult patients: distraction osteogenesis and conventional osteotomies in a standardized sequence. J Craniofac Surg. 2014;25(6):1959–66. https://doi.org/10.1097/ SCS.0000000000000975.
- Goldenhar M. Associations malformatives de l'oeil et de l'oreille, in particular le syndrome dermoide epibulbaireappendices auriculares-fistula auris congenita et ses relations avec la dysostose mandibulofaciale. J Genet Hum. 1952;1:243–82.
- Gorlin RJ, Cohen MM Jr, Hennekam RCM. Syndromes of the head and neck. Oxford: Oxford University Press; 2001.
- Gorlin RJ, Jue KL, Jacobsen U, Goldschmidt E. Oculoauriculovertebral dysplasia. J Pediatr. 1963;63:991–9. https://doi.org/10.1016/ S0022-3476(63)80233-4.
- Hartsfield JK. Review of the etiologic heterogeneity of the oculo-auriculo-vertebral spectrum (hemifacial microsomia). Orthod Craniofac Res. 2007;10(3):121–8. https://doi.org/10.1111/j.1601-6343.2007.00391.x.

- Horgan JE, Padwa BL, LaBrie RA, Mulliken JB. OMENS-Plus: analysis of craniofacial and extracraniofacial anomalies in hemifacial microsomia. Cleft Palate Craniofac J. 1995;32(5):405–12.
- Kaban LB, Moses MH, Mulliken JB. Surgical correction of hemifacial microsomia in the growing child. Plast Reconstr Surg. 1988;82(1):9–19.
- Kaneyama K, Segami N, Hatta T. Congenital deformities and developmental abnormalities of the mandibular condyle in the temporomandibular joint. Congenit Anom (Kyoto). 2008;48(3):118–25. https://doi. org/10.1111/j.1741-4520.2008.00191.x.
- Khansa I, Hall C, Madhoun LL, Splaingard M, Baylis A, Kirschner RE, Pearson GD. Airway and feeding outcomes of mandibular distraction, tongue-lip adhesion, and conservative management in Pierre Robin sequence: a prospective study. Plast Reconstr Surg. 2017;139(4):975e–83e. https://doi.org/10.1097/ PRS.0000000000003167.
- Kitai N, Murakami S, Takashima M, Furukawa S, Kreiborg S, Takada K. Evaluation of temporomandibular joint in patients with hemifacial microsomia. Cleft Palate Craniofac J. 2004;41(2):157–62. https:// doi.org/10.1597/02-108.
- Larheim TA, Abrahamsson AK, Kristensen M, Arvidsson LZ. Temporomandibular joint diagnostics using CBCT. Dentomaxillofac Radiol. 2015;44(1): 20140235. https://doi.org/10.1259/dmfr.20140235.
- Magalhães MH, da Silveira CB, Moreira CR, Cavalcanti MG. Clinical and imaging correlations of Treacher Collins syndrome: report of two cases. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2007;103(6):836– 42. https://doi.org/10.1016/j.tripleo.2006.04.011.
- Meazzini MC, Donati V, Garattini G, Brusati R. Maxillary growth impairment in cleft lip and palate patients: a simplified approach in the search for a cause. J Craniofac Surg. 2008;19(5):1302–7. https://doi. org/10.1097/SCS.0b013e31814fb711.
- Meazzini MC, Brusati R, Diner P, Giannì E, Lalatta F, Magri AS, Picard A, Sesenna E. The importance of a differential diagnosis between true hemifacial microsomia and pseudo-hemifacial microsomia in the postsurgical long-term prognosis. J Craniomaxillofac Surg. 2011a;39(1):10–6. https://doi.org/10.1016/j. jcms.2010.03.003.
- Meazzini MC, Brusati R, Caprioglio A, Diner P, Garattini G, Giannì E, Lalatta F, Poggio C, Sesenna E, Silvestri A, Tomat C. True hemifacial microsomia and hemimandibular hypoplasia with condylar-coronoid collapse: diagnostic and prognostic differences. Am J Orthod Dentofac Orthop. 2011b;139(5):e435–47. https://doi.org/10.1016/j.ajodo.2010.01.034.
- Mommaerts MY, Nagy K. Is early osteodistraction a solution for the ascending ramus compartment in hemifacial microsomia? A literature study. J Craniomaxillofac Surg. 2002;30(4):201–7. https://doi. org/10.1054/jcms.2002.0314.
- Moulin-Romsée C, Verdonck A, Schoenaers J, Carels C. Treatment of hemifacial microsomia in a

growing child: the importance of co-operation between the orthodontist and the maxillofacial surgeon. J Orthod. 2004;31(3):190–200. https://doi. org/10.1179/146531204225022407.

- Mulliken JB, Kaban LB. Analysis and treatment of hemifacial microsomia in childhood. Clin Plast Surg. 1987;14(1):91–100.
- Mupparapu M, Nadeau C. Oral and maxillofacial imaging. Dent Clin N Am. 2016;60(1):1–37. https://doi. org/10.1016/j.cden.2015.08.001.
- Nager FR, De Reynier JP. Das Gehörorgan Bei Den Angeborenen Kopfmissbildungen. Pract Otorhinolaryngol (Basel). 1948;10:3–52. https://doi. org/10.1159/000273559.
- Obwegeser HL. Mandibular growth anomalies: terminology - aetiology diagnosis - treatment. Berlin: Springer; 2001.
- Obwegeser HL, Makek MS. Hemimandibular hyperplasia—hemimandibular elongation. J Maxillofac Surg. 1986;14(4):183–208.
- Pirttiniemi P, Peltomäki T, Müller L, Luder HU. Abnormal mandibular growth and the condylar cartilage. Eur J Orthod. 2009;31(1):1–11. https://doi.org/10.1093/ejo/ cjn117.
- Posnick JC, Ruiz RL. Treacher Collins syndrome: current evaluation, treatment, and future directions. Cleft Palate Craniofac J. 2000;37(5):434. https://doi. org/10.1597/1545-1569(2000)037<0434:TCSCET> 2.0.CO;2.
- Poswillo D. The pathogenesis of the first and second branchial arch syndrome. Oral Surg Oral Med Oral Pathol. 1973;35(3):302–28.

- Printzlau A, Andersen M. Pierre Robin sequence in Denmark: a retrospective population-based epidemiological study. Cleft Palate Craniofac J. 2004;41:47–52. https://doi.org/10.1597/02-055.
- Sarnat BG. Developmental facial abnormalities. In: Facial pain and mandibular dysfunction. Philadelphia: Saunders; 1969. p. 83–101.
- Tsiklakis K, Syriopoulos K, Stamatakis HC. Radiographic examination of the temporomandibular joint using cone beam computed tomography. Dentomaxillofac Radiol. 2004;33(3):196–201. https://doi.org/10.1259/ dmfr/27403192.
- Vento AR, LaBrie RA, Mulliken JB. The O.M.E.N.S. classification of hemifacial microsomia. Cleft Palate Craniofac J. 1991;28(1):68–76; discussion 77. https://doi.org/10.1597/1545-1569_1991_028_0068_ tomens_2.3.co_2.
- Walker RV. Developmental deformities of the jaws. In: Guralnick WC, editor. Textbook of oral surgery. Boston: Little, Brown and Company; 1968. p. 317–84.
- Wolford LM, Bourland TC, Rodrigues D, Perez DE, Limoeiro E. Successful reconstruction of nongrowing hemifacial microsomia patients with unilateral temporomandibular joint total joint prosthesis and orthognathic surgery. J Oral Maxillofac Surg. 2012;70(12):2835–53. https://doi.org/10.1016/j. joms.2012.02.010.

Check fr update

TMJ Trauma

6

Claudio Caldarelli, Paolo Busolli, and Giacomo Paolo Vaudano

Key Points

- Trauma can cause intracapsular damage, including hyperemia of the capsule, hemarthrosis, capsular tear, synovial ecchymosis, fragmentation of the disc and of the articular surfaces, and disc displacement.
- Condylar fractures are the most frequent mandibular fractures and the most frequently missed.
- Orthopantomogram plus CT scan is currently used for condylar fractures assessment.
- MRI is gaining importance in identifying traumatic soft tissue damages that may be responsible for ankylosis and late TMJ dysfunctions.

Fracture of the mandibular condyle is the most renowned TMJ traumatic event; however, TMJ soft tissue damage exists as well, even when the mandible is not involved directly, as in the case of whiplash injuries. Both fractures and soft tissue injuries may harm the joint and play a role in the

C. Caldarelli (🖂)

Department of Otolaryngology and Maxillofacial Surgery, San Giovanni Bosco Hospital, Turin, Italy e-mail: claudio.caldarelli@fastwebnet.it development of chronic dysfunctions that can range from disc disorders to osteoarthrosis, fibrous ankylosis, and bone ankylosis. Recent clinical and experimental studies have pointed out the relevance of trauma-induced disc displacement and damage to the condylar cartilage in the development of these complications, with or without the presence of associated condylar fractures (Miyamoto et al. 1999; Li et al. 2006; Zhang and He 2006; Duan and Zhang 2011; Arakeri et al. 2012; Yan et al. 2013; Xiang et al. 2014; Dai et al. 2016; Han et al. 2017). Especially in the case of condylar head fractures (CHFs or diacapitular fractures), which are known to be often treated conservatively by most maxillofacial surgeons all over the world, these findings may advocate indication for open reduction and rigid internal fixation (ORIF), and most importantly for soft tissue repair, in order to prevent joint ankylosis and decrease the risk of future functional issues, as well as to address signs and symptoms related to the fracture itself (joint noises, mandibular deflection on opening).

TMJ injury treatment seems no longer just a matter of restoring skeletal anatomy and, with this in mind, MRI is at times, together with CT, important in identifying the correct indications for surgical treatment, for surgical approach choice, and ultimately for understanding pathogenesis of late trauma-induced dysfunctions.

© Springer Nature Switzerland AG 2020

G. P. Vaudano · P. Busolli Neuroradiology, San Giovanni Bosco Hospital, Turin, Italy

T. Robba et al. (eds.), *MRI of the Temporomandibular Joint*, https://doi.org/10.1007/978-3-030-25421-6_6

6.1 Nonfracture and Soft Tissue Injuries of the TMJ

If condylar fractures are absent, soft tissue damage may lead to injury features that differ from those commonly related to fractures; however, early diagnosis is often missed at the time of trauma because of lack of radiographic signs. Arthroscopy and MRI findings have shown that trauma can cause intracapsular damage, including hyperemia of the capsule, stretching and effusion of retrodiscal tissue, hemarthrosis, capsular tear, synovial ecchymosis, fragmentation of the disc and of the articular surfaces, and disc displacement (Goss et al. 1990; Sullivan et al. 1995; He et al. 2013; Hirjak et al. 2017; Krishnan 2017). Relevance of these findings lies in potentially causing irreversible conditions that are doomed to undergo a number of conservative and invasive treatments which may further jeopardize TMJ status.

Epidemiology Incidence of TMJ soft tissue injury after mandibular trauma is unknown (He et al. 2013). Diagnosis is often missed because symptoms are not specific and X-ray findings are negative.

Etiology and Pathogenesis While nonfracture and soft tissue TMJ injuries share the same etiology and pathogenesis as mandibular condylar fractures (discussed below), little is known about the reason behind the fact that not all nonfracture patients develop TMJ disorders (He et al. 2013).

Presentation Lack of findings on clinical examination is typical for most TMJ soft tissue injuries in the acute setting. It is not unusual that patients are in pain and do not include the condylar region in their main complaints at presentation, so it is mandatory for the clinician to investigate for further details such as the mechanism of injury and the injured areas. Diffuse pain in the preauricular region and occlusion discomfort (minimal posterior open bite and inability to get to habitual maximum intercuspation) may be reported, as well as a mandibular midline shift if one side is mainly affected. In the case of *hemarthrosis*, because of capsule distension induced by bleeding into the joint and effusion of the soft tissues, pain may increase with mouth opening and there can be a limitation of movement. Swelling of the preauricular region on the affected side can be observed, but ecchymosis is rare.

In case of *disc displacement* patients may experience some difficulty in opening their mouth.

Chronic discomfort and function impairment which are known to develop from these conditions only become evident over time (Fig. 6.1a, b).

Imaging In case of traumatic lesions which are not associated with condylar fractures nor with TMJ luxation, conventional radiology and CT scans are performed in order to exclude the presence of fractures. Conventional radiology (orthopantomogram and plain film radiography) plays a minor role, whereas CT proves to be fundamental for traumatic TMJ diagnostics, also because it is easily available in the emergency room. Furthermore, CT allows to detect late bone



Fig. 6.1 (**a**, **b**) Bite opening has developed gradually in this patient with a history of mandibular trauma without condylar fracture (**a**). Tooth wear pattern confirms that the open bite is recent (courtesy of Dr. Giulia Tanteri)

changes (erosions, subchondral sclerosis) which may develop in case of post-traumatic TMJ arthrosynovitis (He et al. 2013).

MRI is the only imaging technology which allows to detect soft tissue injury-related changes if performed soon after trauma has occurred (Dwivedi et al. 2012). The extent of capsule– disc–ligaments lesions is proportional to the entity of trauma. This will affect residual function and pain symptoms in the medium and long term (Tripathi et al. 2015). Performing TMJ MR immediately after a trauma can therefore correctly address prognosis and the therapeutic approach of traumatic TMJ lesions.

Studies based on TMJ arthroscopy revealed that, in case of articular trauma without condylar fractures, damage of capsule and ligaments prevails. These are associated with hemarthrosis, capsular tear, and lesions of the bilaminar zone. Conversely, whenever a condylar fracture is present, disc lesions prevail. The disc is mainly dislocated anteromedially and less frequently ruptured. Hemarthrosis and disc displacement seem to be the elements that mainly influence functional outcome (Dwivedi et al. 2012).

MR signs of TMJ traumatic lesions are not particularly specific (Fig. 6.2a–d). A careful analysis of the medical history and a thorough clinical examination still prove to be fundamental for diagnosis and they provide data which lead to a correct interpretation of MR sequences. MR signs which need to be investigated are:

- hemarthrosis, which is bleeding within the joint space. This will appear wider in size. Signal intensity of hemarthrosis is similar to that of fluids, even though in the acute stage hemoglobin can be detected due to its hyperintensity in T2 and fat suppression sequences;
- disc displacement, which is most commonly anteromedial in traumas (Fig. 6.3a–e). This is due to the pull exerted by the lateral pterygoid muscle (as discussed in Chap. 7) (Goss et al. 1990; Takaku et al. 1996). Discriminating a recent post-traumatic displacement from a previous one may be challenging at times.

If the displaced disc does not show degenerative changes, and if signs suggesting a recent trauma are present (e.g., bone edema without articular remodeling, ligament edema), then it is possible to confirm that the dislocation is recent and post-traumatic (Fig. 6.4a-c) (Dwivedi et al. 2012). The opposite situation is harder to interpret. In fact, if a displaced disc is affected by pre-existing degenerative changes, it is difficult to understand whether the displacement is recent or longwithstanding in MR sequences. In such cases the retrodiscal laminae will perhaps show prominent signal alterations, which may suggest a recent dislocation. Furthermore, degenerative changes and discal involution (size reduction) may be secondary to disc dislocation;

- disc rupture (Fig. 6.5a, b) may occur but with no degenerative disc changes. In such case, a recent, post-traumatic disc rupture is to be suspected;
- bilaminar zone lesions will show a further increase of signal hyperintensity in fat suppression sequences. This is due to edema and active hyperemia, which are particularly noticeable in this vessel-rich area. Difficulty in identifying the superior retrodiscal lamina (which marks the temporal insertion of the disc) indicates a bilaminar zone lesion, which is functionally comparable to a disc rupture (Takaku et al. 1996);
- capsular tear (Fig. 6.3d, e) is generally asso-٠ ciated with a ruptured bilaminar zone. This is due to the fact that the disc, the retrodiscal laminae, and the capsule-ligament structures constitute a functional unit (Gerhard et al. 2007). This condition is often featured by a variation in the layout of capsule-ligament structures. Coronal sequences show such as at the medial and lateral condylar insertions, where collateral ligaments may be hardly visible or where the retrodiscal laminae and the capsule, anterior to the tubercle, insert (sagittal sequences) (Takaku et al. 1996). In acute stages it is possible to observe the spread of serous infiltration into the soft tissues



Fig. 6.2 Patient with a previous trauma of the left TMJ (which occurred 12 months before this examination was carried out). In the sagittal SE T1 image (**a**) the disc (*arrow*) is displaced anteriorly and has a rounded morphology due to degenerative changes. Effusion is present. Sagittal image GE T1 (**b**) in maximum opening shows that the disc is not recaptured (*arrow*). In the coronal SE PD

image (c) the presence of effusion is well visible in the medial and lateral articular spaces without signs of capsular tear (*arrows*) and the condyle (*star*) is characterized by hypointensity due to subchondral sclerosis and irregular articular profile. Also in the coronal reformatted CT image (d) the articular profile is finely irregular and eroded (*arrow*), with sclerosis of the subchondral bone

surrounding the condyle. This is particularly evident in T2 and fat suppression sequences (Dwivedi et al. 2012);

• *bone edema of the condyle* is not a constant finding in TMJ traumas and it is less fre-

quent in capsule–ligament lesions (Takaku et al. 1996). Nevertheless, it must be carefully investigated because deterioration of fibrocartilage and periosteal reaction, associated with hemarthrosis, may favor the formation of heterotopic bone and consequently promote ankylosis (Krishnan 2017). Bone edema appears as a hyperintense signal in fat suppression sequences (Fig. 6.6a, b).

Management TMJ soft tissue injuries are often neglected because of lack of early radiological

and clinical signs. Trauma-related disc displacement, cartilage erosions, and soft-tissue shredding have been studied retrospectively and they are associated with chronic TMJ dysfunction, although there is little evidence concerning early management and its adequacy (He et al. 2013). Some authors suppose that



Fig. 6.3 84-year-old patient. Mandibular trauma occurred 8 days before this examination was performed. In the sagittal PD image (**a**) it is possible to observe disc displacement (*arrow*), articular effusion, and bone edema of the condyle (*star*). Sagittal STIR image in open mouth position show that the disc is not recaptured and that the retrodiscal laminae are uneven and not clearly visible

(**b**—*arrow*), whereas the anterior temporal insertion of the capsule (**c**—*arrow*) is irregular. Also in coronal SE T2 images, capsular tear (*arrow*) is seen both in the temporal (**d**) and in the medial condylar area (**e**—*arrow*), where it is associated with edema of the medial pterygoid (*asterisk*)



Fig. 6.3 (continued)

early detection of articular surface and disc damage or displacement on MRI may be an indication for arthroscopic or open surgery (He et al. 2013; Hirjak et al. 2017). In the case of disc displacement with tearing, posterior disc repositioning with suturing or repositioning with fixation to the condyle has been described (Yang et al. 2012; Zhang and He 2006). This straightforward assumption on management can prevent degenerative joint disease and ankylosis and reduce the need for more aggressive surgeries, and at times inadequate in the long run, such as gap arthroplasty and use of costochondral graft or even prosthetic replacement.

Traditionally, management of soft tissue injuries remains mostly conservative, its aim being restoration of occlusion, function, and pain relief. Soft diet and anti-inflammatory medication along with close daily observation are shown to be frequently successful. Limitation of function is to be avoided since it has an important role in the development of joint ankylosis (Ferretti et al. 2005).

In case of hemarthrosis, arthrocentesis and/or arthroscopy may be indicated. In the last decade, clinical and experimental international research focus on traumatic TMJ ankylosis (TTMJA), put in evidence some crucial aspects on its etiopathogenetic mechanisms, explaining the rationale for a more interventionist approach to condylar trauma, as well as identifying the goals of ankylosis treatment. TTMJA develops in case of retrodiscal and lateral capsular tear rather than disc displacement alone. Ossification is more likely when there is no disc interposition between damaged articular surfaces. All of the damage features above are typically present in condylar fractures, but are also observed in nonfracture TMJ injuries (Miyamoto et al. 1999; Li et al. 2006; Zhang and He 2006; Duan and Zhang 2011; Arakeri et al. 2012; He et al. 2013, 2014; Yan et al. 2013; Xiang et al. 2014; Dai et al. 2016; Han et al. 2017; Abdala-Júnior et al. 2018).



Fig. 6.4 Patient referring a low-energy trauma 2 months before these images were obtained. In sagittal STIR closed-mouth sequence (**a**) the disc, which is partially displaced anteriorly, does not show degenerative changes and effusion is visible in the upper joint space (*arrow*). In the open-mouth sagittal STIR sequence (**b**), the disc is not

recaptured and the upper retrodiscal lamina, even if intact, appears to be elongated and deflected as a consequence of traumatic distraction. Also in the coronal T2 sequence (c) the condylar insertion of the lateral collateral ligament is regular (*arrow*)



Fig. 6.5 Contusion of the left condyle following mandibular trauma. Sagittal GE T1 weighted sequence (a) shows a fragmented disc, with small stumps (*arrows*) anteriorly and posteriorly placed with respect to the con-

dyle. Coronal GE T2 weighted fat sat sequence (**b**) shows an irregular disc with a wavy profile (*arrow*). The condylar profile is irregular as well (*star*)



Fig. 6.6 12-year-old patient—trauma directed against the chin which occurred 10 days earlier. Sagittal SE PD in closed mouth position (a) and sagittal STIR with open

mouth (**b**) do not show fracture lines nor effusion, but a faded, post-contusive edematous area of the condylar bone is present (*arrows*)

6.2 Condylar Fractures

Any mandibular fracture located above the lingula and running from the angle of the mandible to the sigmoid notch or towards the condylar head can be defined as a condylar fracture. For the purpose of this section focus will be on soft tissue joint damage associated with condylar head fractures along with mention to fractures of the condylar base and condylar neck.

Condylar fractures (CF) are considered the most frequent mandibular fractures (Neff 2014a,

b). In spite of this, CF management has been controversial over the years. Techniques have evolved and approaches have been implemented in order to deal with this demanding area characterized by the presence of the main branches of the facial nerve and vessels, whose iatrogenic damage is clearly undesirable. Recently, there has been an increasing consensus on CF surgical treatment, and ORIF is now considered the gold standard for both condylar base and neck fractures with displacement and dislocation (Neff 2014a, b; Al-Moraissi and Ellis 2015). Again, benefits of ORIF have also been demonstrated for condylar head fractures (CHFs), but there is still no definitive agreement about surgical management and age-appropriate approach (treatment of fractures in children will not be reviewed here). Not to mention that matters regarding training for this kind of surgery have different aspects to be taken into consideration, one of them being the learning curve. Joint damage in case of fractures is due to the trauma itself; however, long-term functional aspects are potentially related to the choice of management too, as seen in joints which underwent closed treatment only (Tripathi et al. 2015; Krishnan 2017).

Since there will be more and more understanding regarding potential benefits of joint soft tissue repair, further controversies are to be expected, as even just for the "skeletal" issue surgical treatment of CHF is still not widespread. MRI will surely have a key role in understanding the injured TMJ and in identifying joint damage that may need repair in order to prevent post-traumatic TMJ disorders and ankylosis. In this perspective, MRI could advise in favor of combined bone and soft tissue surgical repair of CHF.

CF Classification A wide range of CF classification has been developed in the last decades, some with more radiological/anatomical remarks, some with consideration of condyle–fossa relationship, and some others blending all elements for surgical implications, with the result that outlining prevalence of fracture types and comparing treatment outcomes can be quite troublesome (Spiessl and Schroll 1972; Lindahl and Hollender 1977; Loukota et al. 2005; Powers 2017).

The AO (Arbeitsgemeinschaft für Osteosynthesefragen-Association for the Study of Internal Fixation) classification system for condylar fractures is the most widely used today, and it provides decision-making guidance for maxillofacial surgeons worldwide. This classification outlines fractures of the condylar head, neck, and base according to specific anatomic landmarks and reference lines, with the purpose of promoting clinical research and guidance among colleagues about surgical and nonsurgical management, choice of surgical approach, reduction method, and type of plating (Neff et al. 2014b). The landmarks that describe head, neck, and base of condyle are summarized in Fig. 6.7.

In 2018, Kozakiewicz observed that condylar fractures run mostly oblique, often involving more than one of the three currently used regions (Kozakiewicz 2019). With this knowledge, subdivision lines that lie parallel to fracture lines were postulated based on CT scan-identified anatomical landmarks, and this author suggested that they



Fig. 6.7 A: the posterior ramus line (tangent to the most prominent points of the masseteric tuberosity and of the lateral pole of the condyle); B: the condylar head reference line (perpendicular to A below the lateral pole of the condylar head); C: the sigmoid notch line (Loukota lineperpendicular to A and running through the deepest point of the sigmoid notch); E: the masseteric tuberosity notch line (perpendicular to A and running through the upper posterior edge of the masseteric tuberosity, that is located at the lower one third of the distance between the most prominent point of the posterior border of the masseteric tuberosity (D) and the sigmoid notch line (C). Given the above, a CHF will involve the area above the B line; a fracture that remains below B and lies above C for more than one-third is a condylar neck fracture; a fracture that is below C for more than two-thirds is a condylar base fracture (one-third to two-thirds rule)



Fig. 6.8 Recent classification of mandibular condylar fractures based on oblique reference lines and landmarks that are: a line oblique to the sigmoid notch line or Loukota line (A) running from the most prominent point of the masseteric tuberosity to the deepest point of the sigmoid notch (B); the head anterior border point (C), which lies immediately below the condylar head, marking the boundary between the condylar head and neck; a line parallel to B, passing through C, and a third line in between and parallel to the previous two (E)

could be more practical and useful in distinguishing the different fracture levels (especially neck fractures). Preciseness in distinguishing between low and high-neck fractures is promising from an epidemiological point of view, but also in therapeutic terms in that these fractures may require different surgical approach, fixation methods, and equipment (Fig. 6.8).

Despite new proposals, the AO coding still encompasses all parameters that make this the most complete classification nowadays, so that condylar fractures are systemized according to:

- location (head, neck, base);
- presence of fragmentation (none, minor, major);
- degree of vertical apposition (complete, partial, none);
- sideward displacement and direction (none, partial, full; anterior, posterior, medial, lateral);
- angulation (degrees and direction);
- displacement of the cranial fragment/fossa;
- displacement of the caudal fragment/fossa;
- distortion of the condylar head;
- loss of ramus height (none, loss, increase) on orthopantomogram.



Fig. 6.9 Measurement of ramus height. *A*: a horizontal line passing through the most prominent point of the masseteric tuberosity; *B*: a horizontal line passing through the upper border of condylar heads; C: the posterior ramus line. The *red arrow* indicates loss of vertical height

According to Eckelt, overall loss of ramus height is calculated comparing the affected side to the contralateral non-affected side on an orthopantomogram (Fig. 6.9) (Eckelt et al. 2006).

CHF Classification CHF nomenclature has been revised over the last years. The name being used now is *diacapitular fracture*, also because of evidence showing that these fractures frequently extend beyond the borders of the joint capsule anyway (Neff et al. 2014b). As surgery for CHFs is becoming more common, it is quite logical that their classification system should follow more practical implications rather than research/anatomical ones (as it was the case during the conservative treatment days). This is why in the AO system fractures are divided into fractures:

- medial to the lateral pole/involving the lateral pole (Fig. 6.10);
- with/without fragmentation (minor or major);
- with vertical apposition (total or partial)/loss of vertical apposition.

In 2002 Neff proposed a division of CHF into three types, subsequently modified by Loukota. In order to better clarify surgical indications, CHF type C, B, and A were condensed in type 1 and type 2, with or without ramus shortening, respectively (Neff et al. 2004; Loukota et al. 2010). The Strasbourg Osteosynthesis Research Group (SORG) accepted this nomenclature for their research (Fig. 6.11).



Fig. 6.10 Medial (*blue*) and lateral (*orange*) pole areas of the condylar head

In 2009 He identified three regions in the condylar head on the basis of axial and coronal CT scans (lateral, central, and medial third) and classified CHF into four types according to the location of the midpoint of the fracture line, with type A corresponding to Neff's type B/C and to Loukota's type 1 (He et al. 2009).

A retrospective study more recently introduced a further CHF grading based on the relation between the stump of the ramus and the glenoid fossa. Assuming that the higher the grade, the higher the risk of ankylosis, they put an accent on the role of soft tissue damages and the need for their surgical repair (Fig. 6.12) (He et al. 2013). Both Neff and He classification identified comminuted CHFs (type M).

Epidemiology CFs represent about 11–16% of all facial fractures and approximately 30–60% of all mandibular fractures (Chrcanovic 2015). About half of all CFs are associated with another mandibular fracture at the body or the angle, and approximately 84% are unilateral. While in literature it is mostly reported that condylar base fractures are the most common type (62%), followed by condylar neck fractures (24%), it has been



Fig. 6.11 CHF classification by Neff. Type A: diacapitular head fracture without loss of vertical height; type B: diacapitular head fracture involving the intracapsular lateral pole zone with loss of vertical height; type C: diacapitular head fracture involving the extracapsular lateral pole zone with loss of vertical height. Loukota in 2010 simplified nomenclature identifying a type 1 (previously type B and C) as a CHF with loss of vertical height and a type 2 (previously type A) as a CHF without loss of vertical height

recently stated that CHFs can represent up to the 65% of all CFs if a coronal CT scan is performed, as they can be easily missed on conventional X-ray imaging (Loukota and McCann 2003; He et al. 2009). Gender ratio M:F ranges from 3:1 to 2:1. Incidence is higher between the twenties and the forties for base and neck fractures, while CHFs seem to occur more often in older population (Loukota and McCann 2003).

Etiology and Pathogenesis Interpersonal violence, falls, sports injuries, and road traffic accidents are the main causes of CF with a predominance of accidental falls in case of CHF. Most are caused by an indirect trauma to



Fig. 6.12 CHF grading based on the relationship between mandibular ramus stump and articular fossa. Grade 0: mandibular stump is in the fossa and retrodiscal tissue is lengthened; grade 1: there is contact between the stump and the fossa and retrodiscal tissue is torn or perforated; grade 2: mandibular stump is laterally displaced out of the

the condylar region due to a traumatic load applied elsewhere to the mandible and transmitted by the mandibular arch itself. A symmetric load seems related to bilateral fractures, while unilateral injury produces contralateral fractures (Fonseca et al. 2009).

Presentation Among mandibular fractures, condylar fractures are the most commonly missed. An associated chin skin laceration is an accepted sign that should alert the clinician about a CF. Patients may refer pain during mouth opening, limited range of motion, and occlusion discomfort. The lateral pole of the condyle may not be palpable at the preauricular fossa in case of medial dislocation. On the contrary, bulging of the preauricular fossa may be palpable when dislocation is lateral. At examination, deflection may be present towards the fractured side, where premature contact could be experienced, and a contralateral open bite can

fossa and retrodiscal tissue and capsule are torn. Images of three patients can here be seen (CT scans, *left* and MRI, *right*). The relationship between mandibular stump and fossa can be assessed on CT, while soft tissue lesions are evaluated with MRI

be observed on the opposite side. In the case of bilateral fractures, an anterior open bite may be observed along with a straight mouth opening. Bleeding or hematoma of the external auditory canal may be detected too. Dental fractures are common in close mouth trauma and should be investigated for forensic issues.

Imaging Orthopantomogram and plain film radiography may prove useful in case of lowenergy traumas so as to exclude mandibular fractures (Schuknecht and Graetz 2005). Furthermore, closed mouth and open mouth transcranial oblique lateral projection and Towne projection can be used with the patient's cooperation (Fig. 6.13a, b) (see Chap. 2). Nevertheless, in case of mandibular trauma CT scans are often necessary. In fact, whenever X-rays are negative and fracture-suggesting symptoms persist, a CT exam is needed. In addition to this, if a mandibular fracture has



Fig. 6.13 Right condylar fracture—in the postero-anterior radiographic projection (**a**) and closed-mouth oblique lateral transcranial (**b**) of the right TMJ, the displaced fracture of the mandibular condyle (*arrows*) is well visible. Technical quality of the projections is suboptimal due to the

presence of the cervical collar. Coronal reconstruction (c) of the subsequently performed CT scan confirms the fracture on the right side (*arrow*). Axial scan highlights a parasymphyseal fracture (*arrow* in **d**). Both fractures (condyle *star*, parasymphyseal *arrow*) are seen in 3D CT (e)

been detected on either an orthopantomogram or a plain film X-ray, a CT scan is likely needed for preoperative assessment or to verify the frequently occurring presence of bifocal mandibular fractures and dentoalveolar lesions (Fig. 6.13c–e) (Schuknecht and Graetz 2005; Alimohammadi 2018). Finally, if a high-energy trauma has occurred, CT is indicated first and foremost, as mandibular fractures are in these cases often associated with cervical and facial fractures. Multidetector computed tomography (MDCT) will therefore be performed, so as to include the upper and lower jaw as well as the zygomaticomaxillary complex (Patel et al. 2012). CT scan provides a higher diagnostic accuracy and details which allow for a correct conservative or surgical planning. In particular, CT multiplanar reconstructions prove particularly useful for describing and classifying fractures (Patel et al. 2012; Neff 2014a).

As previously stated, functional outcome depends greatly on the extent to which damage has involved the capsule–disc–ligament structures. In the acute stage, for instance, a decreased articular excursion may depend on disc ruptures and condylar head dislocations (Fig. 6.14a–c). After a condylar fracture, the unopposed pull of the lateral pterygoid muscle



Fig. 6.14 Triple mandibular fracture—coronal CT reconstruction (**a**) shows fractures affecting both condylar necks (the parasymphyseal fracture does not appear in this

image). Subsequent MR investigation shows in GE oblique sagittal of right (**b**) and left side (**c**) that both discs (*arrows*) are anteriorly displaced

on the condylar fragment results in its anteromedial and inferior displacement. This will cause a mechanical obstacle as well as a compression of disc and retrodiscal tissue against the articular eminence. If the disc is placed between the fractured segment and the glenoid cavity, joint function will be affected but most probably preserved (Fig. 6.15a-e). Vice versa, if the disc is either dislocated or ruptured, mandibular segments will get into direct contact with the tubercle, thus secondary arthrosynovitis and degenerative phenomena will follow (Fig. 6.16a-e). Arthroscopic findings recently shed some light on soft tissue damage MRI interpretation and its differences from arthroscopic direct view. Hemarthrosis, disc rupture, and posterior tearing may be overestimated in MRI and be absent in the actual setting (Hirjak et al. 2017).

Management As stated in the Second Consensus conference held in Marseille in 2012, ORIF is the gold standard for both condylar base and neck fractures with displacement and dislocation. Management of CHF in adults is still debated, but ORIF is highly recommended, though requiring highly skilled surgeons. Occlusal stability is crucial, even only for temporary intraoperative intermaxillary fixation; therefore, dental status or dental prostheses availability is to be evaluated. Surgical approach should be selected according to fracture location and considering the osteosynthesis method (Neff 2014a; Bischoff et al. 2017). The most preferred surgical approaches are:

- the antero or transparotideal preauricular approach;
- the retroauricular transmeatal approach;
- the antero or transparotideal retromandibular approach;
- the high submandibular approach.

Endoscopically assisted transoral procedure has shown a lower risk of facial nerve injury, but specific training is needed. Neck and base fractures undergo internal fixation by means of miniplates. Shape, thickness, and number of needed screws are elements affecting the osteosynthesis type which best fits the fracture line(s). Intermaxillary postoperative fixation time varies from 0 to 7–10 days, followed by intensive rehabilitation with the aim to restore mandibular movements.

Focusing on CHF, the objectives of surgical treatment should encompass restoration of the functional anatomy of disc and condyle, restoration of ramus height, decrease in long-term discomforts, and prevention of TMJ ankylosis and dysfunction. Screws, resorbable pins and sutures are currently used. Intraoperative CT scan use is offered only at some institutions but it is useful especially in case of comminuted CHF (Cuddy et al. 2018). For these fractures virtual planning can also prove useful in restoring condyle unity.

Once again, reduction and fixation may be not sufficient to prevent TMJ dysfunctions and ankylosis, and immediate soft tissue repair with disc repositioning and fixation may be increasingly performed in the future. Insight into this matter has by now explained that traumatic ankylosis has, in retrospect, a well-defined origin. In CHFs, joint space is occupied by the formation of a hematoma in close contact with the fractured bone surface. Capsule rupture and discoligamental dislocation allow for flooding close by, and they are not able to confine tissue healing and organization so that when differentiation begins in this load-less environment (in case of immobilization, for instance), bone production and excessive growth will be promoted (Ferretti et al. 2005).

Although forecasts on the development of ankylosis are unlikely in the acute setting, MRI can visualize CT-invisible risk factors such as hematoma, capsule rupture, and disc dislocation.



Fig. 6.15 Patient presenting a consolidated and longstanding fracture of the left condylar head, with prominent residual post-traumatic dysmorphism (*arrow*—**a**, **b**) when compared to the contralateral. In the closed-mouth

oblique sagittal PD sequences (c, d) the disc is found between tubercle and condyle (*arrows*), ensuring an adequate articular excursion during opening (e). An anterior capsular tear (*arrow*) can also be detected



Fig. 6.16 (a–e) Diacapitular fracture of the left mandibular condyle. The medial stump (*arrow*) is dislocated antero-inferiorly (axial SE T1 image (a) and coronal b), as it is pulled by the lateral pterygoid muscle. In sagittal STIR closed-mouth (c) and coronal (b) images, the disc (*arrow*) looks displaced anteriorly, away from the lateral

stump (*arrows*), whereas open-mouth sequences show that it is found between tubercle and medial stump (d). In the open-mouth sagittal STIR sequence (e), the disc lies underneath the articular tubercle, and the lateral stump impacts against the tubercle and shows some degree of bone edema (*arrow*)

References

- Abdala-Júnior R, Cortes A, Aoki E, Ferreira S, Luz J, Arita E, et al. Impact of temporomandibular joint discectomy on condyle morphology: an animal study. J Oral Maxillofac Surg. 2018;76(5):955.e1–5. https:// doi.org/10.1016/j.joms.2017.12.019.
- Alimohammadi R. Imaging of dentoalveolar and jaw trauma. Radiol Clin N Am. 2018;56(1):105–24. https://doi.org/10.1016/j.rcl.2017.08.008.
- Al-Moraissi EA, Ellis E 3rd. Surgical treatment of adult mandibular condylar fractures provides better outcomes than closed treatment: a systematic review and meta-analysis. J Oral Maxillofac Surg. 2015;73(3):482–93. https://doi.org/10.1016/j. joms.2014.09.027.
- Arakeri G, Kusanale A, Zaki GA, Brennan PA. Pathogenesis of post-traumatic ankylosis of the temporomandibular joint: a critical review. Br J Oral Maxillofac Surg. 2012;50:8–12. https://doi. org/10.1016/j.bjoms.2010.09.012.
- Bischoff EL, Carmichael R, Reddy LV. Plating options for fixation of condylar neck and base fractures. Atlas Oral Maxillofac Surg Clin North Am. 2017;25(1):69– 73. https://doi.org/10.1016/j.cxom.2016.11.003.
- Chrcanovic BR. Surgical versus non-surgical treatment of mandibular condylar fractures: a meta-analysis. Int J Oral Maxillofac Surg. 2015;44(2):158–79. https://doi. org/10.1016/j.ijom.2014.09.024.
- Cuddy K, Khatib B, Bell RB, Cheng A, Patel A, Amundson M, et al. Use of intraoperative computed tomography in craniomaxillofacial trauma surgery. J Oral Maxillofac Surg. 2018;76(5):1016–25. https:// doi.org/10.1016/j.joms.2017.12.004.
- Dai J, Ouyang N, Zhu X, Huang L, Shen G. Injured condylar cartilage leads to traumatic temporomandibular joint ankylosis. J Craniomaxillofac Surg. 2016;44(3):294–300. https://doi.org/10.1016/j. jcms.2015.12.006.
- Duan DH, Zhang Y. A clinical investigation on disc displacement in sagittal fracture of the mandibular condyle and its association with TMJ ankylosis development. Int J Oral Maxillofac Surg. 2011;40(2):134– 8. https://doi.org/10.1016/j.ijom.2010.11.011.
- Dwivedi AND, Tripathi R, Gupta PK, Tripathi S, Garg S. Magnetic resonance imaging evaluation of temporomandibular joint and associated soft tissue changes following acute condylar injury. J Oral Maxillofac Surg. 2012;70(12):2829–34. https://doi.org/10.1016/j. joms.2012.08.026.
- Eckelt U, Schneider M, Erasmus F, Gerlach KL, Kuhlisch E, Loukota R, et al. Open versus closed treatment of fractures of the mandibular condylar process– a prospective randomized multi-centre study. J Craniomaxillofac Surg. 2006;34(5):306–14. https:// doi.org/10.1016/j.jcms.2006.03.003.
- Ferretti C, Bryant R, Becker P, Lawrence C. Temporomandibular joint morphology following post-traumatic ankylosis in 26 patients. Int J Oral

Maxillofac Surg. 2005;34(4):376–81. https://doi. org/10.1016/j.ijom.2004.09.003.

- Fonseca RJ, Marciani RD, Turvey T. Oral and maxillofacial surgery. 2nd ed. St. Louis: Saunders/Elsevier, 2009.
- Gerhard S, Ennemoser T, Rudisch A, Emshoff R. Condylar injury: magnetic resonance imaging findings of temporomandibular joint soft-tissue changes. Int J Oral Maxillofac Surg. 2007;36(3):214–8. https://doi. org/10.1016/j.ijom.2006.09.013.
- Goss AN, Fracds OMS, Bosanquet AG. The arthroscopic appearance of acute temporomandibular joint trauma. J Oral Maxillofac Surg. 1990;48(8):780–3.
- Han L, Long T, Tang W, Liu L, Jing W, Tian WD, et al. Correlation between condylar fracture pattern after parasymphyseal impact and condyle morphological features: a retrospective analysis of 107 Chinese patients. Chin Med J. 2017;130:420–7. https://doi. org/10.4103/0366-6999.199836.
- He D, Yang C, Chen M, Jiang B, Wang B. Intracapsular condylar fracture of the mandible: our classification and open treatment experience. J Oral Maxillofac Surg. 2009;67(8):1672–9. https://doi.org/10.1016/j. joms.2009.02.012.
- He D, Yang C, Chen M, Yang X, Li L. Effects of soft tissue injury to the temporomandibular joint: report of 8 cases. Br J Oral Maxillofac Surg. 2013;51(1):58–62. https://doi.org/10.1016/j.bjoms.2012.02.005.
- He D, Cai Y, Yang C. Analysis of temporomandibular joint ankylosis caused by condylar fracture in adults. J Oral Maxillofac Surg. 2014;72(4):763.e1–9. https:// doi.org/10.1016/j.joms.2013.12.015.
- Hirjak D, Machon V, Beno M, Galis B, Kupcova I. Surgical treatment of condylar head fractures, the way to minimize the postraumatic TMJ ankylosis. Bratisl Lek Listy. 2017;118(1):17–22. https://doi. org/10.4149/BLL_2017_004.
- Kozakiewicz M. Classification proposal for fractures of the processus condylaris mandibulae. Clin Oral Investig. 2019;23(1):485–91. https://doi.org/10.1007/ s00784-018-2459-1.
- Krishnan DG. Soft tissue trauma in the temporomandibular joint region associated with condylar fractures. Atlas Oral Maxillofac Surg Clin North Am. 2017;25(1):63–7. https://doi.org/10.1016/j. cxom.2016.11.002.
- Li ZB, Li Z, Shang ZJ, Zhao JH, Dong YJ. Potential role of disc repositioning in preventing postsurgical recurrence of traumatogenic temporomandibular joint ankylosis: a retrospective review of 17 consecutive cases. Int J Oral Maxillofac Surg. 2006;35(3):219–23. https://doi.org/10.1016/j.ijom.2005.06.021.
- Lindahl L, Hollender L. Condylar fractures of the mandible. II. A radiographic study of remodeling processes in the temporomandibular joint. Int J Oral Surg. 1977;6(3):153–65.
- Loukota RA, McCann PJ. In: Ward Booth P, Eppley BL, Schmelzeisen R, editors. Maxillofacial trauma and esthetic facial reconstruction. St. Louis: Elsevier Saunders; 2003.

- Loukota RA, Eckelt U, Bont LD, Rasse M. Subclassification of fractures of the condylar process of the mandible. Br J Oral Maxillofac Surg. 2005;43(1):72–3. https://doi.org/10.1016/j. bjoms.2004.08.018.
- Loukota RA, Neff A, Rasse M. Nomenclature/classification of fractures of the mandibular condylar head. Br J Oral Maxillofac Surg. 2010;48(6):477–8. https://doi. org/10.1016/j.bjoms.2009.08.036.
- Miyamoto H, Kurita K, Ogi N, Ishimaru JI, Goss AN. The role of the disk in sheep temporomandibular joint ankylosis. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1999;88:151–8. https://doi.org/10.1016/ S1079-2104(99)70109-5.
- Neff A, Mühlberger G, Karoglan M, Kolk A, Mittelmeier W, Scheruhn D, et al. Stabilität der Osteosynthese bei Gelenkwalzenfrakturen in Klinik und biomechanischer Simulation (Stability of osteosyntheses for condylar head fractures in the clinic and biomechanical simulation). Mund Kiefer Gesichtschir. 2004;8(2):63–74.
- Neff A, Chossegros C, Blanc JL, Champsaur P, Cheynet F, Devauchelle B, International Bone Research Association, et al. Position paper from the IBRA Symposium on Surgery of the Head – the 2nd International Symposium for Condylar Fracture Osteosynthesis, Marseille, France 2012. J Craniomaxillofac Surg. 2014a;42(7):1234–49. https:// doi.org/10.1016/j.jcms.2014.03.005.
- Neff A, Cornelius CP, Rasse M, Torre D, Audigé L. The comprehensive AOCMF classification system: condylar process fractures - level 3 tutorial. Craniomaxillofac Trauma Reconstr. 2014b;7(Suppl 1):S044–58. https:// doi.org/10.1055/s-0034-1389559.
- Patel R, Reid RR, Poon CS. Multidetector computed tomography of maxillofacial fractures: the key to high-impact radiological reporting. Semin Ultrasound CT MR. 2012;33:410–7. https://doi.org/10.1053/j. sult.2012.06.005.
- Powers DB. Classification of mandibular condylar fractures. Atlas Oral Maxillofac Surg Clin North Am. 2017;25(1):1–10. https://doi.org/10.1016/j. cxom.2016.11.001.
- Schuknecht B, Graetz K. Radiologic assessment of maxillofacial, mandibular, and skull base trauma.

Eur Radiol. 2005;15:560–8. https://doi.org/10.1007/ s00330-004-2631-7.

- Spiessl B, Schroll K. Gelenkfortsatz-und Kieferköpfchenfrakturen. Spezielle Frakturenund Luxationslehre. Band 1/1: Gesichtsschädel ed. Stuttgart: Georg Thieme Verlag; 1972. p. 136–52.
- Sullivan SM, Banghart PR, Anderson Q. Magnetic resonance imaging assessment of acute soft tissue injuries to the temporomandibular joint. J Oral Maxillofac Surg. 1995;53(7):763–6. https://doi. org/10.1016/0278-2391(95)90326-7.
- Takaku S, Yoshida M, Sano T, Toyoda T. Magnetic resonance images in patients with acute traumatic injury of the temporomandibular joint: a preliminary report. J Craniomaxillofac Surg. 1996;24(3):173–7.
- Tripathi R, Sharma N, Dwivedi AND, Kumar S. Severity of soft tissue injury within the temporomandibular joint following condylar fracture as seen on magnetic resonance imaging and its impact on outcome of functional management. J Oral Maxillofac Surg. 2015;73(12):2379.e1–7. https://doi.org/10.1016/j. joms.2015.09.003.
- Xiang G, Long X, Deng M, Han Q, Meng Q, Li BA. Retrospective study of temporomandibular joint ankylosis secondary to surgical treatment of mandibular condylar fractures. Br J Oral Maxillofac Surg. 2014;52(3):270–4. https://doi.org/10.1016/j. bjoms.2014.01.002.
- Yan YB, Zhang Y, Gan YH, An JG, Li JM, Xiao E. Surgical induction of TMJ bony ankylosis in growing sheep and the role of injury severity of the glenoid fossa on the development of bony ankylosis. J Craniomaxillofac Surg. 2013;41(6):476–86. https://doi.org/10.1016/j.jcms.2012.03.011.
- Yang C, Cai XY, Chen MJ. New arthroscopic disc repositioning and suturing technique for treating an anteriorly displaced disc of the temporomandibular joint: part I—technique introduction. Int J Oral Maxillofac Surg. 2012;41:1058. https://doi.org/10.1016/j. ijom.2012.05.025.
- Zhang Y, He DM. Clinical investigation of early posttraumatic temporomandibular joint ankylosis and the role of repositioning discs in treatment. Int J Oral Maxillofac Surg. 2006;35(12):1096–101. https://doi. org/10.1016/j.ijom.2006.09.003.

Check for updates

Joint Disorders

7

Carlotta Tanteri, Tiziana Robba, Roberta Cimino, and Giulia Tanteri

Key Points

- This chapter describes the epidemiology, etiopathogenesis, clinical presentation, radiological features, and management of joint disorders, namely disc disorders, hypomobility and hypermobility disorders.
- Disc disorders may have different subjective and objective manifestations. The disc may be displaced in various directions and may or may not be reduced on opening. In addition to this, the disc may preserve its morphology or may be affected by degenerative changes. Due to heterogeneity of displacements, a careful

C. Tanteri (⊠) · G. Tanteri Studio Tanteri, Turin, Italy e-mail: tanteri@tanteri.it

T. Robba

Department of Diagnostic Imaging and Radiotheraphy, Radiology Service, C.T.O. Hospital, A.O.U. Città della Salute e della Scienza, Turin, Italy e-mail: trobba@cittadellasalute.to.it

R. Cimino

Section of Orthodontics and Gnathology, University of Naples Federico II, Naples, Italy e-mail: rocimino@unina.it analysis needs to be carried out in order to appropriately differentiate, diagnose, and manage them.

- Hypomobility disorders are not very common, nonetheless radiologists and dental practitioners should be able to recognize signs and symptoms so as to formulate a correct diagnosis. They do, in fact, limit mouth opening and this can lead to severe consequences. The patient therefore needs to be referred to the specialist for prompt management.
- Hypermobility disorders are more common and often overlooked both by the patient and by the practitioner. Nevertheless they may, over time, create severe damage to articular structures.
- MRI allows to perform the evaluation of disc position and morphology and to assess the presence and position of intra-articular adhesions. Kinematic MRI allows to evaluate the quality of articular excursion and timing of disc recapture.
- Skeletal details of TMJ ankylosis and coronoid hyperplasia are best analyzed by means of computed tomography.
- Luxation and subluxation can be diagnosed with both MRI and CT scans, but magnetic resonance allows to observe associated discal changes if present.

Electronic supplementary material The online version of this chapter (https://doi.org/10.1007/978-3-030-25421-6_7) contains supplementary material, which is available to authorized users.

[©] Springer Nature Switzerland AG 2020 T. Robba et al. (eds.), *MRI of the Temporomandibular Joint*, https://doi.org/10.1007/978-3-030-25421-6_7

7.1 Disc Disorders

7.1.1 Physiological Relationship Between Condyle and Disc (Normal Position of the Disc)

During maximum intercuspation of teeth, the disc should be centered onto the condyle, and the disc-condyle complex should be centered into the fossa. According to different authors, in such a position the condyle shows an average concentric position in the fossa with symmetric joint space widths (Pullinger et al. 1985, 1986; Ren et al. 1995; Rammelsberg et al. 2000). The posterior band of the disc should therefore sit on top of the condyle whereas the pars intermedia rests where condyle and tubercle are at their closest. If one were to imagine the condylar head profile as a clock, then the area between the posterior border and the bilaminar zone should lie at 12 o'clock (Fig. 7.1a, b). This physiological position has been named superior disc position (Tasaki et al. 1996). In gnathological terms, the term centric relation is often used when describing the position of the condyle-disc-fossa complex. It is a term which historically aroused controversy and its definition changed throughout the years. However, when the condyle-disc unit is functionally intact, any spatial location during functional movements in which condyle and disc are related as above can be defined as a *centric relation*. The centric relation position in the uppermost and most anterior position of the fossa is named *terminal centric relation*, and it is often used for prosthetic rehabilitation purposes (Tanteri et al. 2009).

There is no general consensus in the literature on this spatial relation because the above description would imply an excess of diagnosed discal displacements among the general population. Due to this reason, various authors have suggested a wider range of positions of the posterior band for diagnosis of "normality." The posterior band could, in fact, be placed at around 10 o'clock or even up to 20° in front of 12 o'clock (Drace and Enzmann 1990; Rammelsberg et al. 1997; Tomas et al. 2006) (Fig. 7.2a, b). A functionally relevant definition for a physiological position of the disc would be with the intermediate part of the disc lying between condyle and tubercle, independently of the posterior band reference (Orsini et al. 1998).



Fig. 7.1 (a, b) Oblique sagittal SE T1 (a) and oblique sagittal SE PD fat saturated (b) images show the physiological position of the disc in maximum intercuspation. The pars intermedia is between the eminence and the

condylar head (*arrow*), and the border between the retrodiscal lamina and the posterior band lies at 12 o'clock (*dotted line*). The condyle-disc complex is centered with respect to the fossa



Fig. 7.2 (**a**, **b**) Same patient, right side (**a**) and left side (**b**). The disc can still be considered *non-dislocated* on the right side (**a**) since the border between the retrodiscal lamina and the posterior band lies at $+15^{\circ}$ (*dotted line*), even if the pars intermedia tends to slide anteriorly with

During mandibular movements, the disc should smoothly and harmoniously follow the condyle and move along in a position between the condyle and the articular tubercle. In physiological conditions, the disc-condyle complex allows for an adequate amount of *translation* (upper compartment) and *rotation* (lower compartment) so as to perform all movements (protrusion, mediotrusion, laterotrusion, opening, closing, and in some cases retrusion), as explained in Chap. 4. In normal conditions the disc will therefore show mobility with respect to the condyle and the tubercle, thus resting between the two at all times, as shown in kinematic MRI images (Fig. 7.3a–d).

There is a balance between the anteromedially oriented pull of the upper head of the lateral pterygoid muscle on the disc, and the elastic resistance of the retrodiscal laminae which prevents the disc from being anteriorly displaced (Tomas et al. 2006). When disc and ligaments are intact, the intermediate part will keep its position between condyle and eminence even in condylar hypermobility situations (Fig. 7.4a, b). Conditions which cause the loss of such balance (such as trauma and overload) may lead to disc displacement.

respect to the condyle. The left disc instead (**b**) is dislocated. In fact, the border between the retrodiscal lamina and the posterior band lies at approximately $+40^{\circ}$ (*dotted line*), and the pars intermedia is not found between condyle and eminence any longer

7.1.2 Disc Displacement

Overview As described in previous chapters, the lower joint compartment is the one in which rotation occurs, whilst translation takes place in the upper compartment (disc-fossa compartment). Displacement of the disc (sometimes referred to as DD-disc dislocation/displacement or ADD—articular disc displacement) is an intracapsular mechanical disorder in which the physiological relationship between disc, condyle, and fossa is lost. This may be associated with pain and muscle dysfunction, and it can manifest with or without disc reduction and with or without limited mouth opening (Pertes and Gross 1995; Tanteri et al. 2009; Schiffman et al. 2014). These conditions present different clinical manifestations which will be discussed in the corresponding sections. Furthermore, the disc can be dislocated in different directions. Nevertheless, according to the Diagnostic Criteria for Temporomandibular Disorders (DC-TMD), the direction of the discal displacement might not be so relevant. The most significant features include instead the possibility of recapturing the disc, the presence of intermittent locking episodes and



Fig. 7.3 (a–d) Oblique sagittal Gradient Echo fat saturated sequences. These four images have been selected from a kinematic-MR and show sequences from intercus-

clinical information regarding mouth opening limitation (Schiffman et al. 2014). In particular, TMD diagnostic criteria contemplate for disc disorders:

- · disc displacement with reduction,
- disc displacement with reduction with intermittent locking,
- disc displacement without reduction with limited opening,
- disc displacement without reduction without limited opening (Fig. 7.5).

pation (closed mouth) to maximum opening. During excursion, the pars intermedia of the disc (*arrow*) is constantly found between condyle and tubercle

Epidemiology Females show a higher prevalence (F:M = 5:1), and a reason for this genderrelated difference could be the ligamentous laxity which men somewhat lack. According to the literature, a percentage which varies from 18% to 34% of the general asymptomatic population may be affected by disc displacement. DDs are found in 38–73% of TMD patients, and up to 65% of displacements come along with clicking. Data regarding the direction of displacement reveal that anterior dislocations are found in approximately 30% of the asymptomatic popula-



Fig. 7.4 Oblique sagittal SE PD (**a**) and STIR (**b**) sequences. Same patient as in Fig. 7.3. Hypermobile condyle with a correctly placed disc. In maximum intercuspation, the condyle and the disc retain a normal position in

the fossa (**a**) and fibers of the superior head of the lateral pterygoid muscle (*arrow*) are inserted in the disc. Upon maximum opening the bilaminar zone is undamaged (*arrows*) (**b**)



INTRA-ARTICULAR JOINT DISORDERS

Fig. 7.5 Diagnostic flowchart based on history and clinical examination for disc displacements according to the TMDdiagnostic criteria

tion. Few studies report specific prevalence of disc displacement with reduction (DDwR) versus disc displacement without reduction (DDWoR). According to these, the prevalence of DDwR in the general population is about 40%, while that of DDWoR is 4%. In patients affected by temporomandibular disorders, the prevalence of DDwR ranges from 24% to 47%, and from 11% to 26% in case of DDWoR (Tasaki et al. 1996; Ribeiro et al. 1997; Larheim et al. 2001; Santos et al. 2013; Amaral Rde et al. 2013; Su et al. 2018). Studies performed on MRI sequences of pre-orthodontic adolescents suggest that disc displacements occur frequently, with a confirmed higher prevalence in girls with respect to boys (Agerberg and Helkimo 1987; Helkimo and Westling 1987; Nebbe 2000).

Etiology and Pathogenesis Despite the growing diagnostic capacity, controversy still surrounds the etiology of disc displacements (Rammelsberg et al. 2000). All factors which may potentially lead to mechanical shifts and sliding between disc and condyle can contribute to disc displacement. Most authors have identified traumas (either micro- or macrotrauma) as the main cause for internal derangements. Macrotraumas include joint contusions, mandibular traumas (sport, whiplashes), and iatrogenic traumas (such as those due to intubation, extraction surgery, endoscopic maneuvers, and tonsillectomies). Such events are usually reported by the patient whilst completing the medical history. Even though car accidents involving whiplashes have often been taken into consideration as possible causative factors, they may most probably play a role in acute disc displacement. TMJ whiplash-related damages should not be seen as an independent clinical entity as they can only manifest on pre-existing, possibly asymptomatic, TMJ conditions (McKay and Christensen 1998). Microtraumas are trickier to detect and typically include stress-related parafunctions (such as bruxing, nail/pen biting), orthopedic instability (facilitated by ligamentous laxity and occlusal features), and hypoxia/reperfusion damage (Okeson 2014a). Posterior displacement of the condyle caused by trauma or changes of centric occlusion have been considered as a preceding condition of disc displacement (Rammelsberg et al. 1996). Some static and dynamic occlusal features (such as Class II Division II occlusion or slide from centric relation to occlusal relation >2 mm) and interferences in occlusion have long been considered as risk factors for joint instability and overload, but studies are too heterogeneous in methodology and design to be adequately compared. Data in the literature, therefore, do not provide unequivocal evidence (Solberg et al. 1986; Seligman and Pullinger 1989; Jiménez-Silva et al. 2017). Overall, one might consider disc displacement to be the consequence of an incorrectly distributed overload combined with orthopedic instability and a decreased adaptive capacity under predisposing circumstances (Okeson 2014a; Ernberg 2017).

According to Nitzan, an important etiological role might be played by impairment of the lubrication system. Such system relies on phospholipids plus hyaluronic acid and proves to be essential for enabling translation of the disc. If uncontrolled free radicals are present, the lubrication system may break down, thus leading to a high friction between surfaces. It is believed that such friction may be the trigger to loosen disc attachments to the condyle (Nitzan 2001). Finally, some authors report frequent disc displacement associated with condylar fractures. In such cases the disc is often dislocated anteromedially and remains connected to the proximal fragment of the condyle, thus being pulled by the lateral pterygoid muscle (Gerhard et al. 2007; Krishnan 2017).

Presentation The clinical presentation of disc dislocations with and without reduction will be discussed in the corresponding section. Nevertheless, a brief clarification should be made about the terms deflection and deviation. These are often used and misused when describing mandibular movements, especially when disc displacements are analyzed. A deflection is a movement away from the midline during the excursive movement, with no return towards the midline at the end of the movement. A deviation is a movement away from the midline during



Fig. 7.6 (a, b) Deflection and deviation are here described as the movement of a point located on the midline at the interincisal level between the lower central incisors during opening. A *deflection* is a movement away from the midline, with no return towards it at the comple-

tion of opening (**a**). A *deviation* is a movement away from the midline, with return towards it at the completion of the excursions (**b**). Both deflection and deviation can be observed in opening and closing of the mouth

excursion, with return to the midline upon the end of the movement. If the movement of one of the temporomandibular joints is for some reason hindered, the mandible typically deflects towards the affected side (which is moving less than the non-affected side) (Fig. 7.6a, b).

Management Management of disc dislocations is needed whenever pain or dysfunction is present, as these affect the patients in terms of discomfort and range of motion, thus influencing oral functions. Management of DDwR and DDWoR is discussed in the corresponding sections.

7.1.3 Direction of Displacement

Authors have widely debated methods to radiologically define and classify the direction of disc displacements (Drace et al. 1990; Tasaki et al. 1996; Foucart et al. 1998; Whyte et al. 2006). Different experts believe that knowledge of the direction of disc displacement may prove useful for decision-making in therapy. Some find it sufficient to describe such displacement according to six main vectors: anterior, posterior, lateral,

medial, anterolateral, and anteromedial. Others believe it is important to add horizontal rotational information when describing the direction of disc displacement (Tasaki et al. 1996; Foucart et al. 1998; Larheim et al. 2001; Aiken et al. 2012). In his work, Tasaki drew a line between partial anterior disc displacement in the lateral aspect of the joint, and rotational anterolateral disc displacement. The same can be done when differentiating between partial anterior disc displacement in the medial aspect of the joint, and rotational anteromedial disc displacement. This is why, besides from superior disc position, eight more disc positions have been described: anterior disc displacement, partial anterior disc displacement in the lateral part of the joint, partial anterior disc displacement in the medial part of the joint, rotational anterolateral disc displacement, rotational anteromedial disc displacement, medial displacement, lateral displacement, and posterior displacement. Nevertheless, it is widely known that anterior and anterolateral disc displacements account for nearly 80% of all displacements, and the remaining possibilities are less frequent (among all, posterior displacement is the least frequently described in the literature) (Paesani et al. 1992).

Independently from clinical relevance and clinical considerations, MRI allows to identify the direction of disc displacement. For didactic purposes disc displacements shall be classified as follows (Tasaki et al. 1996) (Fig. 7.7a–h):

- anterior displacement, partial or complete,
- anteromedial and anterolateral partial displacement,
- rotational anteromedial and anterolateral partial displacement,
- medial and lateral displacement, partial or complete,
- posterior displacement.

It is fundamental to remark that MRI studies performed solely in the sagittal plane can be misleading in that they can only detect sagittal displacements, thus underestimating lateral or medial displacements. Detection of an anterior displacement in a sagittal projection may prove to be an easy task but as a matter of fact, only a three-dimensional evaluation (especially on the coronal plane) will allow to define the disc position and will help to highlight the rotational component of displacement. In addition to this, detection of posterior displacements also requires open mouth MRI evaluation (all disc displacements need closed mouth and maximum opening MRI for accurate diagnosis).



Fig. 7.7 (**a**–**h**) Directions of disc displacement as seen on the right condyle. Anterior disc displacement (**a**), partial anterior disc displacement in the medial part of the joint (**b**), partial anterior disc displacement in the lateral part of

the joint (c), rotational anteromedial disc displacement (d), rotational anterolateral disc displacement (e), lateral disc displacement (f), medial disc displacement (g), posterior disc displacement (h)

Anterior Displacements In anterior displacements, the border between the posterior band/part of the disc and the bilaminar zone is found anterior to the condyle (beyond 12 or 10 o'clock according to the different classification systems). Coronal views will not show any lateral or medial slide of the disc with respect to the condyle as it is in front of it. Anterior displacements can be either partial or complete.

 a partial anterior displacement somehow maintains a contact between the posterior part of the dislocated disc and the anterior surface of the condyle, and the two maintain the same distance with respect to each other throughout the sagittal MRI sequences. This means that all acquired images along the sagittal plane, from the medial to the lateral pole, will show the same constant extent of anterior sliding of the posterior band with respect to the condyle (Fig. 7.8a–c). In coronal sequences, the disc may still be interposed between condyle and fossa.

 a complete anterior displacement shows a complete loss of the relationship between condyle and disc. The latter manifests important



Fig. 7.8 (**a**–**c**) Partial anterior disc dislocation. Sagittal STIR from the medial pole (**a**), along the median line of the condyle (**b**), to the lateral pole (**c**). The disc (*arrow*)

shows the same degree of dislocation and maintains contact with the condyle



Fig. 7.9 In complete anterior dislocation, the disc (*arrow*) loses the relationship with the condyle and the posterior band is usually affected by degenerative changes

morphological changes (such as disc fragmentation, globular aspect, crumpled disc) whose entity varies according to the chronicity of the situation (see Sect. 7.1.6). These morphological changes are often seen in long-standing displacements as the disc is not involved in function and articular dynamics any longer. This causes the disc to undergo involutive processes (Fig. 7.9).

Anteromedial and Anterolateral Partial Displacements In these, the anterior band of the disc does not lay in a fully anterior position but it slides either towards the *medial joint space* (partial anterior disc displacement in the medial aspect of the joint or anteromedial partial disc displacement) or towards the *lateral joint space* (partial anterior disc displacement in the lateral aspect of the joint according to Tasaki, also called anterolateral partial displacement) (Tasaki et al. 1996; Katzberg et al. 2016).

• In case of an *anteromedial partial displacement*, sagittal MRI views show that the disc leaves the medial pole of the condyle uncovered. In anterolateral partial displacement, sagittal MRI views show that the disc leaves the lateral pole of the condyle uncovered (Fig. 7.10a–d).

Rotational Anteromedial and Anterolateral Partial Displacement In such displacements, the disc rotation leaves both the medial and lateral poles of the condyles uncovered.

- In *rotational anteromedial partial displacements*, the disc slides anteriorly and rotates medially, thus showing that also the lateral pole of the condyle is uncovered in sagittal images.
- In rotational anterolateral partial displacements, the disc slides anteriorly and rotates laterally, and also the medial pole appears uncovered on sagittal images (Fig. 7.11a–d).

Medial and Lateral Displacement In medial and lateral displacements, the disc migrates into the medial articular recess and into the lateral articular recess, respectively, but never sits anterior to the condyle. This explains why lateral and medial displacements are easily diagnosable in coronal views, as the disc is positioned on one of the two sides of the condyle. It proves harder to perform a correct diagnosis of a medial or lateral dislocation when the disc has completely slidden off the head of the condyle. MR sequences apparently show a missing disc, which is in fact visible as a globular entity located on the side of either the medial or the lateral pole (Fig. 7.12a–d).

Posterior Displacement A posterior displacement will show the disc with its pars intermedia on top of the condyle as a "hood," whilst the more voluminous posterior band will fall behind the condyle (Bellot et al. 2000; Chossegros et al. 2001). The posterior band of the disc appears in contact with the bilaminar zone, and its anterior band is found in the 2 o'clock or 3 o'clock position (Gil et al. 2012). According to the authors' personal experience, during articular movements, the disc quite often retains this posterior deranged position. Nevertheless, at the end of the



Fig. 7.10 (**a**–**c**) Sagittal SE PD images: anterolateral partial dislocation. The medial pole of the condyle is left uncovered by the disc (**a**) which progressively dislocates

excursions, the condyle is often seen to regain a centered position with respect to the pars intermedia of the disc, thus showing an apparent reduction. Posterior displacement is quite a rare finding: studies do, in fact, report a prevalence in TMD patients of approximately 1% (Afroz et al. 2018). Some authors suggest that posterior displacements may be underdiagnosed and often misdiagnosed (Fig. 7.13a, b), also due to the fact that thinned out posterior bands can be seen with much trouble in maximum intercuspation

in an anterolateral direction (\mathbf{b}, \mathbf{c}) , until it reaches the lateral joint space (*arrow*) visible in coronal SE T2 image (**d**)

(Fig. 7.14a, b). The literature reports diverse descriptions of this entity, in terms of both the position of the disc and to what extent it diverges from the norm, and in terms of clinical manifestations. Bilateral coronal MR views are needed for proper investigation, and posterior dislocation can be often associated to a medial or lateral sliding component; images in maximum opening and kinematic MRI can show a cranially positioned disc (Westesson et al. 1998; Okochi et al. 2008; Afroz et al. 2018).


Fig. 7.11 (**a**–**d**) Anterolateral rotational partial dislocation. The disc slides anteriorly and rotates laterally, leaving both the medial (**a**) and lateral pole (**b**) uncovered. The

disc *(arrow)* is found anterolaterally placed with respect to the condyle (**c**) as visible in coronal view (**d**)

7.1.4 Disc Displacement with Reduction (DDwR)

In anteriorly displaced discs, maximum intercuspation of teeth will provide the position at which displacement best manifests itself. At some point during the opening movement, the disc is repositioned (or *recaptured*) on the condyle, and the disc-condyle complex maintains a correct relationship until the end of the excursive movement. At some point upon incursion (i.e., closing of the mouth) the disc is displaced again, thus recreating the same situation that was present before the opening movement began, with the disc displaced in front of the condyle. Whenever the disc is recaptured or dislocated, a clicking sound is perceived (Figs. 7.15 and 7.16).

Patients report a history of joint noises during mandibular function which have been present for at least the past 30 days or which may be verifiable during the clinical examination. Clinically, the operator must perform repetitions of joint



Fig. 7.12 (**a**–**d**) In maximum intercuspation, the disc cannot be seen on the lateral pole (**a**) whilst its position is correctly preserved along the condylar median axis (**b**).

Simultaneous study on the coronal view (\mathbf{c}) and on the sagittal plane (\mathbf{d}) allows to detect the disc (*arrow*) medial to the medial pole of the condyle (medial displacement)

palpations in order to check for the presence of clicking, popping or snapping noises during opening, closing, lateral movements, and protrusion (for full details about diagnostic criteria please refer to Schiffman et al. 2014). Mandibular movements may be affected by deviations which usually correspond in time with the joint noises. The presence and stage during opening and closing at which the clicking sound appears, give useful information regarding displacement (and

its potential for reduction/prognosis). The timing of the clicking sound on opening, that is to say the *recapture*, allows to classify such phenomenon as early, intermediate, and late recapture of the disc (Isberg 2001). Under a prognostic perspective, the point at which the disc is dislocated and the point at which it is recaptured may prove to be very important (Tanteri et al. 2009). If the opening click (reduction in opening) occurs at the very first instants of the opening movement



Fig. 7.13 (a, b) The posterior band (*arrow*) can be seen posterior to the condyle (a) due to a posterior disc displacement. In maximum opening (b) the disc (*arrow*) is not repositioned and the displacement is seen even more clearly



Fig. 7.14 (**a**, **b**) Posterior dislocations are not clearly visible in closed mouth sequences (**a**). Sometimes, only open mouth sequences (**b**) allow to diagnose a posterior dislo-

cation of the disc (*arrow*), such as in this case in which an elongated condyle is present

(early recapture), it means that the correct condyle-disc relationship is re-established during the translation of the condyle from the fossa to the inflection point of the posterior slope of the eminence (Isberg 2001). An early recapture implies a fairly recent displacement and it should have a better prognosis (Fig. 7.17a–c). This is usually accompanied by MRI images which show a disc which has maintained its morphology. Conversely, intermediate and late recaptures take place, respectively, between inflection point and vertex of eminence and beyond the eminence



Fig. 7.15 (**a**–**c**) Disc displacement with reduction. Right TMJ, opening movement. In maximum dental intercuspation the disc is displaced (**a**). During excursion the condyle at first moves against the disc and may push it

forward (**b**). At some point the condyle recaptures the disc (**c**) and a click is perceived. Condyle and disc maintain the correct relationship until the end of opening



Fig. 7.16 (**a**–**c**) Disc displacement with reduction. Right TMJ, closing movement. In maximum opening, the disc and the condyle are correctly placed with respect to each other (**a**). In the first stages of incursion the two may

maintain such a relationship (**b**). At some point upon incursion the disc is displaced again and the closing click is perceived. In maximum intercuspation the disc remains anteriorly dislocated (c)

and can be accompanied by degenerative changes of the disc and articular surfaces (Fig. 7.18a–d) (Isberg 2001).

Recapture upon opening and loss of the disc upon closing can take place in different spatial points during the movement (reciprocal click). As a rule of thumb, if the opening and closing click occur in the same spatial point, it is very unlikely that the phenomenon is of discal origin. When in doubt, the operator may ask the patient to protrude and then perform maximum opening and closing movements from this protruded position. Whenever this maneuver leads to the disappearance of the click, the operator has a useful indicator of a DDwR (Tanteri et al. 2009). On a condylographic tracing a disc displacement with reduction is often accompanied by a socalled "crossing signal" in two points of the overall tracing (see Chap. 4). The closing click will often be more cranial than the first opening click and it will look like a quick change in direction and velocity of the closing path, which may or may not cross the opening movement tracing. The closing click usually is the closest to the reference position (RP) and its spatial coordinates can be used for diagnostic and therapeutical purposes as condylography has the power of displaying space-time characteristics of discal phenomena.

According to some authors, disc dislocations with reduction are not only characterized by an anterior displacement of the disc, but most often a concomitant posterior positioning of the condyle occurs as well. This causes an increased width of the anterior joint space and a reduced posterior joint space. Such a condition is seen in the majority of bilateral DDwR (Pullinger et al. 1986; Ren et al. 1995; Rammelsberg et al. 2000).



Fig. 7.17 (**a**–**c**) Anterior dislocation (**a**) with reduction in maximum opening (**b**). Kinematic-MR images (**c**) show that the recapture of the disc *(arrow)* takes place at the

beginning of excursion. Remodeling of the condyle is visible, and degenerative changes of the posterior band of the disc are moderate

In some cases, there may be an insufficient reduction of the disc due to a lack of mobility of the disc itself (see Sect. 7.2). When the teeth are closed together the disc will appear in front of the condyle as predicted, during opening the condyle will slide against a disc which is fixed against the eminence and temporarily reduce the displacement by positioning itself at the pars intermedia.

In summary, if the displacement is recent and the disc shows regular mobility and morphology, the reduction of the displacement (recapture) is usually complete and MRI images in maximum opening rarely show any anomaly at all. The pars intermedia of the disc lays between condyle and tubercle, and the bilaminar zone is intact without inflammatory signal alterations.

In the personal experience of the authors and the editors, a partial recapture of the disc can be observed in patients in which the posterior margin of the disc has lost its thickness and is thinned out (Fig. 7.19). Clinically, the patient may have a delayed reduction (a clear click appears towards



Fig. 7.18 (**a**–**d**) Anterior dislocation (**a**) with reduction in maximum opening (**b**). Articular heads show remodeling and the glenoid cavity is deep, with severe degenerative changes of the disc. Kinematic-MR images (**c**, **d**)

show that recapture of the disc (*arrow*) takes place only when the condyle is below the articular eminence, i.e., a late recapture

the end of the opening movement), but the closing click might not be noticeable as the posterior band of the disc, or at least part of it, has lost its shape.

Kinematic-MRI allows to evaluate the condyle-disc complex during excursion and to visualize the point at which the disc is recaptured. The optimal temporal resolution (each image is obtained in few seconds) leads to the unfortunate consequence of a poorer quality of the acquired images, both in terms of contrast and spatial resolution. This is why kinematic-MR should be solely adopted with the aim of evaluating the quality of articular excursion of the condyle-disc complex and not to study anatomical alterations of the joint. Furthermore, this type of examination allows to distinguish early recaptures (which indicate a more recent onset of dislocation) and late recaptures (which are seen in longstanding dislocations instead).



Fig. 7.19 Sagittal SE PD image showing a thinned disc. The *arrow* indicates the posterior band

The presence of a click is not an indicator of need for treatment per se. If painless, disc dislocations with reduction need no treatment, although the patient should be informed about the situation. However, since dislocation could be the sign of joint changes which have not been detected yet, monitoring is advised and the sounds should be carefully evaluated before commencing any sort of dental rehabilitation. In addition to this, studies which have evaluated the natural course of DDwR over a period of time report that 43% of patients would develop other signs and/or symptoms in time (intermittent locking, clicking on opening appearing at a later stage, disappearance of the reciprocal click). Only few of these would develop a closed lock (Kalaykova et al. 2010; Naeije et al. 2013). In case treatment is needed, authors report among the possible interventions: counselling/habits modification, systemic or topical pain management (typically Nonsteroidal Anti-Inflammatory Drugs—NSAIDs) in case of painful conditions, physical therapy, manual therapy, splints (Huang et al. 2011; Young 2015).

Disc Displacement with Reduction (DDwR) with Intermittent Locking Disc displacement with reduction with intermittent locking is a form of DDwR, the difference being that in the closed mouth position the disc is found anterior to the condyle and only sometimes—hence the term *intermittent*—the disc is reduced on opening. Whenever the disc is not recaptured, the patient experiences a limited mouth opening. In such cases, a maneuver might be necessary to perform an unlocking of the temporomandibular joint (Peck et al. 2014). Given the intermittent nature of this condition, if the locking is not present when performing the MRI, the imaging criteria are the same as for DDwR. A clinical confirmation of the intermittent locking episodes will therefore be needed. If locking episodes are sporadic, the therapeutic considerations are the same as those adopted for DDwR.

7.1.5 Disc Displacement Without Reduction (DDWoR)

Disc displacement without reduction (DDWoR) may be the natural progression of a disc dislocation with reduction. The disc is displaced in maximum intercuspation and it is not recaptured on opening, thus maintaining an anterior position with respect to the condyle (Figs. 7.20 and 7.21). As for DDwR, the disc may be displaced in various directions. Disc displacement without reduction may be associated with limited opening, in which case it may be referred to as *closed lock* (for some authors acute lock with limited mouth opening). In such case the maximum assisted opening (including incisal overlap) is less than 40 mm, no maneuver (neither by the clinician nor by the patient) can be performed to reduce the limitation and a late opening clicking sound may still be present (Peck et al. 2014; Schiffman et al. 2014). Limited opening may instead be absent, in which case the condition is named disc displacement without reduction without limited opening (some authors refer to this condition as chronic lock) (Okeson 2007; Sembronio et al. 2008; Al-Baghdadi et al. 2014a). In case of disc displacement without reduction, when the patient is asked to open the mouth, the mandible deflects towards the affected side. If the disc is not morphologically compromised, and the bilaminar zone somehow preserves its function, the disc maintains mobility with respect to the articular tubercle



Fig. 7.20 (**a**–**c**) Disc displacement without reduction. Right TMJ, opening movement. In maximum dental intercuspation the disc is displaced (**a**). During excursion the condyle at first moves against the disc and may push it forward (b), but the disc is never recaptured and maximum opening may be reduced (c)



Fig. 7.21 (**a**–**c**) Disc displacement without reduction. Right TMJ, closing movement. In maximum opening the disc is displaced (**a**) and during the closing movement, it is not recaptured (b) thus remaining dislocated at the end of the closing movement when teeth are in maximum intercuspation (c)



Fig. 7.22 (a, b) Anterior dislocation (a) without reduction in maximum opening (b). The disc shows good mobility whilst moving from the glenoid cavity and beyond the tubercle during excursion

(Fig. 7.22a, b). Vice versa, whenever the disc is affected by degenerative changes, with reduced dimensions and a globular form with a damaged bilaminar zone, it will be found anterior to the

tubercle with immobility during joint excursions (Fig. 7.23a, b).

Treatment of disc dislocation without reduction should be conservative whenever possible.



Fig. 7.23 (a, b) Anterior dislocation (a) without reduction in maximum opening (b). In closed mouth sequences the disc is placed almost in front of the condyle and shows

scarce mobility during excursion. Condylar head is affected by arthritic changes, bone edema, and erosion

Data in the literature are too heterogeneous to provide clear guidelines, but unlocking of the joint should not be attempted unless the displacement is acute and the operator is trained to do so. NSAIDs (either systemic or topical) can be prescribed if the patient is in pain. Manual therapy, exercises, and behavioral modifications can be suggested. Custom-designed splints may alleviate the muscle pain and TMJ overload. In case pain and/or limitation persist, more invasive procedures such as arthrocentesis, arthroscopy, or even arthroplasty have been described in the literature. Some authors describe surgical disc repositioning, which may be combined with disc reshaping, either in arthroscopy or open joint surgery. This can be performed if the disc is structurally intact and free from degenerative changes and its mobility is sufficiently preserved so as to allow repositioning without tension. Conversely, if the disc is damaged, unrecoverable, and its dislocation limits function or causes pain, discectomy may be taken into account. Current evidence is not sufficient to support the use of one technique against the others (Ebrahim 2012; Al-Baghdadi et al. 2014b; Young 2015; Kraus and Prodoehl 2017; Renapurkar 2018).

7.1.6 Morphological Discal Changes

7.1.6.1 Degenerative Discal Changes

In physiological conditions, a balance is found between production and degradation of the extracellular matrix, with a constant cellular turnover within the disc. Whenever such an equilibrium is altered, degenerative discal phenomena may occur, and complex molecular mechanisms are involved (Ernberg 2017).

Changes or anomalies in temporomandibular joint load are thought to be involved in the pathogenesis of disc displacements and degenerative joint diseases. This holds true for all joints, and the TMJ is no exception. In vivo studies demonstrated that disc dislocations can rapidly induce profound degenerative alterations (Ali and Sharawy 1994; Sharawy et al. 2000). One week after a surgically induced disc dislocation, the cartilage of the disc becomes hypercellular and hypervascularized at first, with the loss of the ordered distribution of collagen fibers which used to be parallel to the articular surface. After only 2 weeks, fibrous areas interbedded with vacuoles, adipose degeneration, and hyperplastic nodules appear. After 6 weeks, debundling of collagen fibers is observed, whilst chondrocytes become agglomerated and fewer in number. Alterations of the retrodiscal laminae appear, with fibrosis, hyalinization, and loss of the adipose tissue within the bilaminar zone (Ali and Sharawy 1994; Sharawy et al. 2000). Such modifications rely on refined molecular mechanisms which involve the inflammatory cascade and the release of cytokines and enzymes which ultimately lead to changes in cellular and extracellular matrix composition, with impairment of collagen fibers (Ernberg 2017).

Cytokines are cell-signalling proteins which may be released in response to inflammation. Interleukins (IL) and tumor necrosis factor (TNF) belong to the family of cytokines, and their levels increase in case of inflammation. Higher quantities of ILs and TNFs have been found in the synovial fluid of patients affected by internal derangement with degenerative changes, thus indicating that chronic inflammation may impair the adaptive capacity of the TMJ. The levels of prostaglandins and other inflammatory mediators also seem to increase in internal derangement (Kawashima et al. 2013; Ernberg 2017).

Specific interleukins (IL-1beta) stimulate the release of matrix metalloproteinases (MMPs). These proteases are involved in tissue repair and neoformation, angiogenesis, differentiation, and apoptosis. Their activation may be responsible for degenerative alterations of the extracellular matrix in internal derangements. Patients with internal derangement show increased levels of some MMPs-collagenases, stromelysin, and gelatinases-in the synovial fluid, condylar cartilage and disc. These metalloproteinases are activated as a consequence of inflammation and are responsible for the degradation of the extracellular matrix. In addition to this, metalloproteinases regulate the viscosity of the synovial fluid, which increases already in early stages of degenerative processes. This reduces the lubrication capacity and leads to increased friction, thus worsening the disc dislocation and making disc reduction more difficult (Srinivas et al. 2001; Yoshida et al. 2006).

Some studies demonstrated that the expression of lumican and fibromodulin (proteoglycans) changes in discs affected by degenerative changes. In these, in fact, lumican (which is also enhanced by IL-1beta) is expressed in areas of the disc that contain many fibroblast-like cells and is associated with the regulation of the neoformation of collagen fibers as well as with cell migration. This is why lumicans may promote regeneration of the disc following discal degeneration and deformation (Kiga 2012).

Cellular alterations taking place within the disc are also related to apoptotic phenomena. An excess of programmed death of disc cells is a potential cause of degenerative disc disease, and if apoptotic cells are not removed they may lead to inflammatory responses that can promote chronic inflammatory conditions, thus feeding the previously cited inflammatory cascade (Ernberg 2017). Some works showed an involvement of caspases (cysteine-containing aspartate-specific proteases), which are enzymes that cleave proteins, thus resulting in cell destruction. Such studies suggested that a greater proportion of caspases is found in dislocated discs, especially in the posterior band which is mostly loaded in case of altered compressive forces. In case of disc dislocations with reduction, caspases are also found within the anterior band (Loreto et al. 2011). These data suggest that an abnormal mechanical load is responsible for causing a greater expression of this apoptosis-bound enzyme. Mechanical overload of the disc can have an effect on synovial fluid viscosity as well. Such a phenomenon may cause discal hypoperfusion (and consequently hypoxia) and reperfusion cycles (when the loading is reduced). This hypoperfusion-reperfusion releases free oxygen radicals that inhibit the polymerization of high molecular weight hyaluronic acid and induce instead the synthesis of low molecular weight hyaluronic acid. In addition to this, the overload and the consequent hypoxia trigger the upregulation of specific growth factors (such as vascular endothelial growth factors), which in turn regulate the expression of MMPs and recruit chondrocytes, endothelial cells, and osteoclasts (Matsumoto et al. 2010; Ernberg 2017).

MR Imaging of Degenerative Disc Changes As previously shown, degenerative discal alterations and the consequent disc deformities are more frequently secondary to disc dislocation. Under the histological perspective, the posterior band initially shows a reduction of elastic fibers and an unorganized pattern of the collagenous fibers, with loss of the expected architecture and an increase of transversely oriented collagen fibers (Scarpino et al. 2006). This is why initial degenerative changes are characterized by an increased thickness of the posterior band of the disc, particularly the inferior surface, whilst the upper surface remains relatively flat (Fig. 7.24a, b). Successively, degenerative alterations involve the anterior band as well.

In this acquired altered pattern of collagen fibers, chondroid metaplasia, myxomatous degeneration and, in more advanced cases, neovascularization can be present (Isacsson et al. 1986). In fact, blood vessels may increase due to the action of pro-angiogenic factors released by discal chondrocytes in a paracrine way (Kiga 2012). MRI sequences show an increased signal which is typical of initial degenerative alterations (Fig. 7.25a, b) (Larheim 2005; Katzberg et al. 2016). As the degenerative processes evolve, the disc goes through regressive phenomena: its dimensions decrease and it becomes constantly hypointense. Both disc bands, especially the posterior one, become globular in shape. The disc appears stiff, bulgy, and biconvex (Fig. 7.26). Less frequently, as the architecture of the collagen fibers of the posterior band is lost, an extensive collagen deposition which may reach the anterior portion of the bilaminar zone is seen (Drace et al. 1990). The posterior band is usually dimensionally reduced and thinned out, appearing lamellar in shape (Fig. 7.27).

7.1.6.2 Disc Perforation

Traumatic lesions involving the disc, capsule, and ligaments are more frequently associated with condylar fractures and will be more extensively discussed in Chap. 6. This section is dedicated to MRI signs in traumatic and degenerative ruptures of the articular disc in the absence of condylar fractures.

Traumatic Disc Perforation and Rupture The incidence of traumatic disc ruptures in absence of condylar fractures has been the subject of a few studies in literature. The finding of a traumatic disc



Fig. 7.24 (a, b) Initial phases of degenerative changes. The thickness of the posterior band (*arrow*) of the disc is increased, and the inferior surface appears more convex.

Anterior DD in sagittal SE PD closed mouth (**a**); anterior DDWoR in sagittal STIR open mouth (**b**)



Fig. 7.25 (**a**, **b**) Anterior disc displacement with severe degenerative changes of the disc. Sagittal PD (**a**); sagittal STIR (**b**). Chondroid metaplasia, myxomatous degenera-

tion, and neovascularization processes determine an increased signal of both the posterior and the anterior band of the disc



Fig. 7.26 Sagittal SE PD: in more severe degenerative forms, both disc bands (*arrow*) show globular morphology and decreased size with a hypointense signal

perforation represents an unlikely event in the case of a condylar fracture, whereas disc displacement has been reported with variable frequency (Goss and Bosanquet 1990; Gerhard et al. 2007; Dwivedi et al. 2012; Yang et al. 2015; Krishnan 2017).

Whenever a disc, either displacement or ruptured, preserves its signal strength and band



Fig. 7.27 Sagittal SE PD: in other degenerative forms, the disc becomes smaller and thinned out

morphology within normal limits in an MR investigation performed soon after the traumatic event, then it can be assumed that the dislocation or disc rupture is of recent onset, and therefore related to trauma (Dwivedi et al. 2012). The traumatic event is also indicated by the presence of other elements such as capsular rupture and

hemarthrosis, whose severity and frequency are correlated with the type of trauma (Gerhard et al. 2007; Tripathi et al. 2015).

The situation is different when there is no history of trauma caused by an external agent, but a functional overload of excessive duration (prolonged openings) or excessive forces (chewing against a resistance) have been applied. In these cases the pre-existence of degenerative discal changes promotes the onset of the disc rupture, but it is difficult to quantify the causal role of the trauma and the favoring role of degenerative changes (Fig. 7.28a–c). **Degenerative Disc Perforation** Even in the absence of trauma, in a percentage of patients affected by degenerative joint disease (DJD) with disc displacement which ranges from 5% to 15%, a disc rupture can be found (Kuribayashi et al. 2008; Shen et al. 2014). The most common cause of disc perforation appears to be an abnormal relationship of the disc to the condyle and to the temporal bone. Direction of disc displacement may have a direct influence over the location of disc perforation. As the most frequent direction, the most frequently found disc perforations are those



Fig. 7.28 (a–c) Patient affected by TMJ DJD, with anterior marginal osteophytosis of the condyle and sclerotic flattening of the tubercle. The patient complained of left TMJ pain after intubation. Sagittal SE PD (a) and STIR

images (b) show lesions of the retrodiscal laminae and posterior band (*arrow*) which appears thinned out and edematous. The disc is displaced anteromedially (*arrow*) in coronal SE T2 (c)

of the pars intermedia, of the posterior band and the bilaminar zone. However, patients affected by an anterolateral displacement without reduction are at greater risk of developing a disc perforation, with percentages reaching up to 55%, with a more frequent involvement of the medial part of the disc (Liu et al. 2010).

The acute onset of disc rupture, regardless of its traumatic or degenerative origin, is accompanied by arthrosynovial alterations, with articular effusion and edematous infiltration of the capsule-ligamentous structures and retrodiscal laminae, which explain the pain symptoms. Joint heads may be affected by bone marrow edema and this can either have a post-traumatic meaning or be an expression of chronic functional overload. It may involve only the subchondral condylar bone or sometimes extend up to the whole ramus of the mandible (Fig. 7.29a–d). MRI signs of acute suffering of the joint, in particular joint effusion and bone edema, may however decrease over time, when the joint recovers



Fig. 7.29 (a–d) Patient affected by TMJ DJD, complaining about acute pain which had its onset after chewing hard foods. Broad flattening of tubercle and condylar surfaces is visible. The condyle is also affected by anterior marginal osteophytosis. The pars intermedia of the disc is

perforated, and the disc appears ring-shaped and surrounds the condyle (*arrows*). (**a**) sagittal SE PD closed mouth; (**b**) sagittal STIR open mouth; (**c**) coronal SE T2 weighted; and (**d**) axial SE T1 weighted. Abundant effusion and intense bone edema of articular surfaces can be observed



Fig. 7.30 (**a**, **b**) Patient affected by Juvenile Idiopathic Arthritis in remission. The condyle is typically deformed, and no signs of arthrosynovitis are present (effusion or bone edema). SE PD sagittal images in closed mouth (**a**)

and STIR open mouth (\mathbf{b}) show a wide lesion of the pars intermedia of the disc, but this does not impede articular excursion

its functional balance. In fact, disc rupture per se does not necessarily imply loss of joint function (Fig. 7.30a, b).

Fluid-sensitive sequences, in particular T2-weighted and fat-suppression ones above all, are the optimal sequences for the diagnosis of discal, degenerative, or traumatic rupture, because joint effusion allows to better identify the margins of the disc abutments. For the same purpose the intra-articular contrast medium (MR arthrography) can be adopted, the use of which is reported in the literature only sporadically (Venetis et al. 2011; Yura et al. 2012).

7.2 Hypomobility Disorders

Mandibular hypomobility implies a persistent restriction of mandibular movement. It is frequently painless but may show progressive worsening. Pain is generally triggered only when applying forces while attempting to increase mouth opening (Okeson 2014b). This section aims at discussing hypomobility disorders other than the most common disc disorders (such as disc displacement without reduction), degenerative joint disease, and other restrictions of mandibular movement which are independent from the TMJ and the masticatory muscles (such as scar tissue involving lips/other structures, ulceration of oral mucosa and trismus, including radiation therapy-induced and post-surgical conditions) which may indeed limit movements.

Hypomobility is firm and unyielding and may be due to:

- intra-articular adhesions (with or without stuck disc),
- · temporomandibular joint ankylosis,
- coronoid hyperplasia.

7.2.1 Intra-Articular Adhesions

Overview Physiological temporomandibular joint movements primarily depend on free sliding of articular surfaces. Mobility is guaranteed by the sophisticated movement taking place within the condyle-disc-eminence complex and by the lubrication system (Nitzan et al. 2004). Whenever the latter is somehow altered, friction and adhesion inhibit the physiological joint function and may give rise to intra-articular adhesions (IAs), with different degrees of articular and discal involvement. Synovial adhesions and fringes may only be partially and occasionally visible in MRI sequences, even when intra-articular contrast is used in arthrography. Different classifications of IAs have been proposed according to shape, size, location, correlation with symptoms, and residual TMJ function. In maximum intercuspation the disc may be either dislocated or in a physiological position and its mobility may be preserved or reduced, in which case a stuck disc is diagnosed (Nitzan and Marmary 1997; Nitzan 2001; Kaneyama et al. 2007; Zhang et al. 2009; Venetis et al. 2011; Millon-Cruz et al. 2015).

The term *anchored disc phenomenon* (ADP) is often misused. It is a phenomenon which manifests with a severe and sudden limited mouth opening, pain on forced opening, adherences, and is accompanied by the MRI sign of a disc stuck to the glenoid fossa. The possible pathogenetic explanation for ADP is intermittent joint overloading which causes an alteration of the lubrication system (due to oxidation and consequent degradation of hyaluronic acid) which in turn creates friction and abrupt adherences which can be accompanied by secondary disc dislocation and osteoarthrosis (Nitzan and Dolwick 1991; Nitzan and Marmary 1997; Rao et al. 1993; Sanroman 2004).

Epidemiology The incidence of IAs was reported to be 44% in a study on 134 TMJs of selected patients who underwent bilateral arthroscopy. IAs were found in 28.9% of the joints with disc displacement with reduction, and in 58.3% of those affected by disc displacement without reduction. In joints with well-positioned discs, adhesions were also found in 15% of the cases (Murakami et al. 1992; Millon-Cruz et al. 2015). The same authors claim that intra-articular adhesions are a common finding during arthroscopy in patients with internal derangement with a percentage ranging from 28.7% to 100% of treated joints. Such a wide range is probably due to the inclusion of patients based on different classification systems and severity of pathology. Other

authors agree with these data and suggest intraarticular adhesions to be present in 91% of all TMJ internal derangements (Murakami et al. 1992; Millon-Cruz et al. 2015).

Etiology and Pathogenesis In order for the disc to slide down the eminence, an efficient lubrication system is needed (Nitzan 2003). The most recent theories suggest that joint overload exceeding its adaptive mechanisms results in tissue damage (such as synovitis, adhesions, and osteoarthritis) as a consequence of the release of free radicals and inflammatory mediators (Israel et al. 2006). Adherences, which are generally caused by a prolonged static load of the joint, represent a temporary sticking of the articular surfaces. They normally disappear whenever a sufficient force is applied during mandibular movement. If the adherence is instead maintained, the more permanent condition of *adhesion* will develop due to the formation of fibrotic connective tissue between articular surfaces (Akheel and Hussain 2014; Okeson 2014c). Other hypotheses have been formulated regarding the etiology and pathogenesis of intra-articular adhesions, and these include fibrin deposition due to synovitis which decreases joint lubrication, altered hypoxia-reperfusion cycles which in turn alter lubrification and finally hemarthrosis or inflammation caused by macrotrauma, surgery, or systemic conditions (such as a polyarthritic disease) which attract fibroblasts and fibrocytes and cause scar tissues formation (Kaminishi and Davis 1989; Murakami et al. 1992; Nitzan et al. 2001; Okeson 2014d).

Presentation Adherences and adhesions may occur either in the superior articular space, between disc and fossa, or in the inferior compartment between disc and condyle. When adherences develop into adhesions, the patient generally complains of limited function (limited opening) with or without pain. Patient history is usually negative for clicking or previous episodes of loss of jaw mobility (Peck et al. 2014). Adhesions in the superior compartment are more frequent. They cause a limited condylar translation, thus the residual movement is given by rotation alone. This is very similar to what happens in DDWoR, the only difference being the absence of triggered intracapsular pain during a bilateral manipulated load (Nitzan and Marmary 1997; Yura et al. 2003; Okeson 2014c). Clinically, unilateral adhesions show a deflection of the mandible towards the affected side and marked limitation of lateral movement towards the contralateral side whilst bilateral adhesions will show no deflection (Peck et al. 2014). Inferior compartment adhesions (between condyle and disc) are trickier to diagnose. Both superior and inferior compartment adhesions may cause a fixed disc, in which case the disc adheres either to the tubercle or to the condyle (Okeson 2013; Akheel and Hussain 2014; Kim et al. 2016).

Imaging In the majority of adherences, a moderately limited excursion of the condyle with preserved or slightly reduced disc mobility is present. In case of disc adhesions, if moderate quantities of synovial fluid are present in the upper compartment, some authors describe small areas of synovial tissue with fibrotic aspects in T2-weighted and fat-suppression sequences (Zhang et al. 2009; Yura et al. 2012). This fibrotic MR finding is not constant and not very sensitive for the diagnosis of discal adhesions, although highly specific (Shen et al. 2014). Magnetic resonance arthrography possesses a higher specificity and sensitivity for the diagnosis of intra-articular adhesions, but its use it is limited by the fact that it is invasive, expensive and requires trained operators (Yang et al. 2005; Venetis et al. 2011). Intra-articular adhesions with fixed disc are easier to diagnose. In these cases, the disc loses its mobility and becomes fixed, that is to say it adheres just like a suction cup to the articular tubercle or to the condyle (the latter condition being less frequent).

When the disc is stuck to the glenoid fossa, hypomobility is generated by the reduced articular translation. In such condition, the disc might

maintain a correct condyle-disc relationship in maximum intercuspation, with adequate interposition of the pars intermedia between condyle and tubercle. During the limited articular excursion, the condyle moves against the anterior band and the disc strictly adheres against the glenoid cavity (Fig. 7.31a-c). It is however possible for the disc to appear anteriorly dislocated in maximum intercuspation and, being "adherent" to the tubercle, it can show an apparent reduction during excursion. At the end of the movement, the condyle will obtain a correct position with respect to the pars intermedia (Fig. 7.32a-c). Vice versa, other cases show in maximum intercuspation an anteriorly dislocated disc which appears to impede the excursion of the condyle and the disc is not recaptured. In such situations the disc shows altered morphology such as degenerative changes causing thickening of the posterior band (Fig. 7.33a-c) or longstanding adhesions causing a thinned disc (Fig. 7.34a–c).

The disc may adhere to the condyle as well. In such situation it is deranged and stuck on top of the condyle and appears smaller, thinner, and less mobile, thus more likely to maintain this pathological position. These characteristics make it prone to posterior dislocation (Fig. 7.35a, b).

Management Lysis of adhesions and lavage of the upper compartment of the TMJ using arthrocentesis or during arthroscopy seem to provide positive effects, even though the superiority of one technique over another cannot be demonstrated and neither of these two methods can effectively change the morphology or the position of the disc (Nitzan 2002; Sanroman 2004; Zhang et al. 2011). Some data however suggest that high-pressure arthrocentesis provides better results in breaking or releasing severe anchorage of the upper compartment, whilst others confirm the success of low-pressure arthrocentesis in treating acute lock with anchored disc phenomenon (Yura et al. 2003; Sanroman 2004; Monje-Gil et al. 2012).



Fig. 7.31 (a, c) 48 years old female. The patient complained about pain and reduced mouth opening. Maximum intercuspation in sagittal STIR (a) and sagittal SE PD (b). The disc appears moderately hyperintense due to degenerative changes, it is thin but maintains a correct condyle-disc relationship. Moderate effusion is visible in

both joint spaces and articular surfaces are flattened out due to remodeling, with subchondral edema of the condyle. Upon maximum opening (c), the articular excursion is reduced and the disc loses its mobility with respect to the condyle

7.2.2 Articular Ankylosis

Overview Temporomandibular joint ankylosis (TMJA) is a severe disabling structural condition that can cause impairment of speech, mastication, nutrition, difficulty in achieving adequate oral hygiene, and disorders of facial and man-

dibular growth with functional and aesthetic consequences. The patient might be totally unable to open or to perform lateral movements in the most severe cases (Adekeye 1983; Posnick and Goldstein 1993; Akama et al. 2009; Katsnelson et al. 2012). Diverse classifications have been proposed and combined based on involved tissue



Fig. 7.32 (a–c) 57 years old female. Patient complaining of limited mouth opening. Articular surfaces are severely remodeled, the glenoid fossa is deepened and the condyle has reduced dimensions, with no bone edema. SE PD sagittal sequences in maximum intercuspation (a) and sagittal STIR (b) show a posterior band (*arrows*) which has

become thicker due to degenerative changes and the disc is anteriorly dislocated. The excursion is reduced, as seen in maximum opening (c), and the disc adheres to the tubercle. During articular excursion, the condyle carries out a translation on the disc, reaching the pars intermedia

(osseous sometimes referred to as *true*, fibrous sometimes referred to as *false* or fibro-osseous), site (intra-articular versus extra-articular), and degree of fusion (complete or incomplete). Bony ankylosis is a fusion of condylar head and glenoid fossa. Fibrous ankylosis is formed by scar tissue or fibrous adhesions instead and it can progress into bony ankylosis (Tripathy et al.

2009; Perciaccante and Krishnan 2014; Al-Moraissi et al. 2015). This condition, along with other restricted mouth opening situations, must lead to careful consideration of upper airway management. Oral intubation in case of emergency or planned surgery is often not possible and tracheostomy or fiberoptic nasotracheal intubation has to be chosen instead.



Fig. 7.33 (\mathbf{a} - \mathbf{c}) Articular surfaces appear remodeled, with a deepened glenoid fossa and a condyle which shows reduced dimensions and no bone edema. Sagittal SE PD (\mathbf{a}) and sagittal STIR (\mathbf{b}) images in maximum inter-

cuspation display degenerative changes of the posterior band of the disc, which is anteriorly dislocated thus hindering condylar excursion. The disc is not recaptured upon maximum opening (c)

Epidemiology Non-traumatic TMJ ankylosis most often develops before the age of 10, with a diagnostic peak in the age group 20–30 (Bello et al. 2012; Babu et al. 2013; Al-Moraissi et al. 2015). A study which included 21,720 children aged 3–15 from the general population revealed 10 cases of TMJ ankylosis, 6 of which were bilateral and 4 monolateral. The reported male to female ratio was 1:9 (Gupta et al. 2012). In contrast with this, other studies report only a slight and nonsignificant difference between males and females with unilateral TMJA being slightly more common than the bilateral form (Al-Moraissi et al. 2015; Braimah et al. 2018). The overall incidence is apparently decreasing, but is still significant in some developing countries (Chidzonga 1999).



Fig. 7.34 (\mathbf{a} - \mathbf{c}) Degenerative changes of the disc are associated with a reduced thickness in this case. The tubercle is remodeled and flatter. Sagittal SE PD (\mathbf{a}) and sagittal STIR images in maximum intercuspation (\mathbf{b})

show a thinner disc (*arrow*) which is anteriorly dislocated. Upon maximum opening (c) the disc (*arrow*) maintains this fixed appearance and adheres to the tubercle like a suction cup. The condyle moves against the disc

Etiology and Pathogenesis Ankyloses most commonly develop following macrotraumas which have caused bleeding (hemarthrosis) and consequent scarring within the joint (Gupta et al. 2012; Al-Moraissi et al. 2015; Liu et al. 2015). Typical traumas include condylar fractures, in some cases accompanied by concomitant mandibular fractures (He et al. 2008). Further reported etiological factors are local infections (middle ear and mastoid infections), systemic infections, systemic disease (such as inflammatory rheumatic diseases), and inadequate surgical management of the condylar area (Sarma and Dave 1991; Kobayashi et al. 2001; Arakeri et al. 2012; Cunha et al. 2012; Peck et al. 2014; Madhumati et al. 2015).

When considering pediatric forms of TMJ ankylosis, the literature reports both acquired



Fig. 7.35 (**a**, **b**) These images show a disc which is reduced in thickness and a flattened tubercle. Sagittal SE PD sequence in maximum intercuspation (**a**) shows a thinner pars intermedia which is located at 10–11 o'clock

thus indicating a posterior dislocation. Upon maximum opening in sagittal STIR sequence (b) the disc appears immobile with respect to the condyle

forms (i.e., perinatal traumas or infections) and congenital temporomandibular joint ankylosis. The latter is possibly caused by genetic factors or by blood disruption in embryo (Domarus 1990; Cheong et al. 2016). Under the pathogenical perspective, fibrous (false) ankyloses may develop from fibrous articular adhesions of the joint and from fibrotic changes of the capsular ligaments (Okeson 2014b).

Presentation Patients typically report a longterm, progressive, painless and severe limited opening capacity which involves all movements. Joint sounds are usually not present. Interincisal opening is an indicator of the severity of ankylosis. Clinically, as condylar rotation is still possible, 20-25 mm of opening could be achievable (Posselt 1956; Okeson 2014f). Complete bilateral bony ankylosis may present with an opening which is often less than 5 mm (Fig. 7.36). When monolateral, a deflection towards the affected side is usually visible on opening and further forced opening is allowed by the mandibular elasticity and the minimal mobility of the cranial sutures (Guyot et al. 1995; Güven 2008; Arakeri et al. 2012; Peck et al. 2014; Okeson 2014b).

Imaging Computed tomography is fundamental for the diagnosis and pre-surgical evaluation of TMJ ankyloses. Such a technique allows, in fact, to accurately evaluate the extent of bone fusion, whilst MRI retains a marginal role as it would not show TMJ skeletal details as precisely (Li et al. 2014). CT scans also allow to correctly plan surgery and placement of prostheses if necessary (Figs. 7.37, 7.38a, b, 7.39a, b, 7.40) (Zhao et al. 2017; Chen et al. 2018).

Management Management of TMJ ankylosis is challenging due to the residual distorted anatomy and to the presence of nerve and vessels which risk to be damaged. The aim is not that of reducing pain, as this condition is painless. Surgical intervention is instead necessary to solve the limitation of mouth opening and to restore joint function. Fibrous ankylosis can be managed by removing fibrous scars and performing either coronoidectomy or coronoidotomy according to the specific case. Excision of the ankylotic part, gap arthroplasty and reconstruction of the ramuscondyle unit with a costochondral graft have all been described in the literature to treat bony



Fig. 7.36 (**a**, **b**) Clinical presentation of bilateral bony ankylosis. In (**a**) the patient is in closed mouth position. The attempt to perform a maximum mouth opening (**b**)

shows an extremely limited movement (Courtesy of Prof. Guglielmo Ramieri and Prof. Giovanni Gerbino)



Fig. 7.37 Orthopantomogram showing bilateral TMJ bony ankylosis. Signs of previous surgeries and wire osteosyntheses are visible, and so is the skeletal dysmorphism

ankylosis (Perciaccante and Krishnan 2014; Madhumati et al. 2015; Braimah et al. 2018). Subjects affected by temporomandibular joint ankylosis who underwent gap arthroplasty showed greater improvement in terms of interincisal opening when compared to those undergoing resection of the ankylotic mass and ramus-condyle unit reconstruction by means of a costochondral graft (Katsnelson et al. 2012). Nowadays it is possible to restore anatomy, allow function and long-term success with TMJ prostheses. Total (fossa and condyle) replacement with stock prostheses is still an option, however computer-aided design and manufacturing (CAD/ CAM) techniques have the enormous advantage of designing and providing a patient-specific prosthesis as well as planning for correction of skeletal deformities altogether (Hu et al. 2017). Post-operative management and monitoring, with early mobilization and intense physiotherapy, must aim at re-ankylosis prevention.



Fig. 7.38 (a, b) Bony ankylosis. Coronal reformatted CT (a) shows that on the left side a bony bridge between articular surfaces is visible. Such features can be seen also in

3D images (b) (Courtesy of Prof. Guglielmo Ramieri and Prof. Giovanni Gerbino)



Fig. 7.39 (a, b) CT-guided intra-operative navigation can be used for resection within the typically distorted anatomy of severely compromised ankylotic TMJs (a). Virtual volume

rendering planning incorporates resection and reconstruction by means of a patient-specific TMJ prosthesis (**b**) (Courtesy of Prof. Guglielmo Ramieri and Prof. Giovanni Gerbino)



Fig. 7.40 Immediate post-operative control of bilateral custom-made prostheses on an orthopantomogram

7.2.3 Coronoid Hyperplasia

Overview Coronoid hyperplasia is an enlargement of the coronoid process with histologically normal bone. This causes a progressive and slow decrease of mouth opening (McLoughlin et al. 1995). It must not be mistaken for other causes of coronoid enlargement such as osteomas and osteochondromas, which are histologically and radiographically different.

Epidemiology The literature lacks comparable epidemiological data regarding coronoid hyperplasia. This is probably due to the fact that it develops progressively, it is generally painless and it initially mimics other TMJ disorders, causing it to be often overlooked (Zhong et al. 2009). Data suggest that coronoid hyperplasia accounts for 5% of all limited mouth openings. The first signs appear at the onset of puberty, with males being mostly affected with a 5:1 male-to-female ratio. The bilateral-to-unilateral ratio is 4.7:1, and the mean age at the time of diagnosis is approximately 25 years (Isberg et al. 1987; McLoughlin et al. 1995; Satoh et al. 2006; Tavassol et al. 2012).

Etiology and Pathogenesis Causative factors include traumas (especially involving the zygomatic bone), infections, increased activity of the temporalis muscle (whose fibers attach to the coronoid), endocrine abnormalities, and hereditary factors (Zhong et al. 2009). Finally, TMJ

internal derangement on the ipsilateral side of coronoid hyperplasia, such as disc displacement, causes condylar translation to be inhibited but rotation is not impeded. This may again lead to increased activity in the temporalis muscle and could be an etiological factor (Isberg and Eliasson 1990). Bilateral hyperplasia of the coronoid processes may also evolve from unilateral hyperplasia (Zhong et al. 2009). The exact pathogenesis is still unclear. Regardless of the onset mechanism the effect is that, as the coronoid enlarges, it impacts against the posterior surface of the zygomatic bone and the medial surface of the zygomatic arch, thus creating a mechanical interference during jaw function (Satoh et al. 2006).

Presentation Patients present a painless, restricted mouth opening, and they may be able to report a progressive onset. The limitation involves all movements, but is more pronounced during protrusion (Okeson 2014b). On examination end feel is hard. When coronoid hyperplasia is unilateral, facial asymmetry may be present and a deflection towards the affected side is visible during opening.

Imaging The diagnosis of coronoid process hyperplasia, when suspected from panoramic radiography, can be confirmed by CT scan (Fig. 7.41a–e). This condition involves bones alone and MRI is therefore not necessary. Conversely, a CT scan allows to investigate the entire maxillofacial complex. Three-dimensional reconstructions provide detailed imaging of the coronoid processes and their relationship with the zygomatic bone and they are useful in case surgery is indicated and needs to be planned (Satoh et al. 2006).

Management Coronoid process hyperplasia is generally treated surgically by intraoral coronoidectomy, or coronoidotomy alone, so as to relieve impingement on the zygoma. In order to improve prognosis it is important to associate physiotherapy post-operatively (Gerbino et al. 1997; Zhong et al. 2009).



Fig. 7.41 (a–e) CT scan has been obtained by means of multidetector computed tomography (MDCT), which allows 3D volume rendering reconstructions (\mathbf{a} , \mathbf{b}) and by means of multiplanar reconstructions MPRs (\mathbf{c} –e). All images show a prominent coronoid process whose dimen-

sions are considerable when compared to those of the condyle. The coronoid process gets caught in the masticatory muscle space between the zygomatic arch and the pterygoid process of the sphenoid bone (Courtesy Dr. Roberto Magnato)

7.3 Hypermobility Disorders

Temporomandibular joint *hypermobility* is a term used to describe the hypertranslation of the mandibular condyle anterior to the articular eminence during wide mouth opening. Depending on how it is reduced, it can be classified as either *subluxation* or *luxation* (Okeson 2014c; De Almeida et al. 2016; Nagori et al. 2018). One should bear in mind that the condyle may frequently be found anterior to the eminence at full mouth opening and this by itself is not pathognomonic for hypermobility disorders (Kalaykova et al. 2006).

7.3.1 Subluxation

Overview Subluxation of the temporomandibular joint (also described in the literature as *hypermobility*) is an abnormal movement of the condyle-disc complex anterior to the eminence which occurs on wide mouth opening. When closing, the patient is unable to return to a normal closed mouth position unless he performs a specific maneuver which allows the condylar head to return into the glenoid fossa. This is why a subluxation is said to be spontaneously reduced by the patient alone.

Epidemiology A study reported TMJ hypermobility to be found in 27.3% of randomly selected girls aged 15–16 versus a prevalence of 43% of generalized joint laxity (Winocur et al. 2000). Other researchers found subluxation in 8.28% of all joints in a group of 19-year-old males and females (Kondrat et al. 2018).

Etiology and Pathogenesis Subluxation is not a pathology itself, instead it should rather be considered the consequence of anatomical variations of the fossa. Such a phenomenon is usually associated to tubercles which are steeper in the posterior part and become flatter anteriorly. This leads to an incoordination of the physiological maximal rotation-translation combination which should occur synchronously. When subluxation occurs, maximal rotation takes place before maximal translation because of the initial steepness. This leaves the final amount of translation to take place at the end of the movement and the disccondyle complex "jumps" farther anteriorly. In case of recurrent subluxation episodes, ligaments may become looser and other TMJ disorders may develop (Okeson 2013).

Presentation Signs of subluxation are visible once the patient has reached what appears to be the maximum extent of mouth opening. At this point a small pause of the movement is seen, followed by an abrupt forward dash which causes the mouth to open even further. This "jump" may be accompanied by a dull sound (not a click), and the lateral pole of the condyle becomes more visible underneath the skin (a pre-auricular depression might be seen as well) (Okeson 2014e).

Imaging Literature widely reports that morphostructural alterations of the osteoarticular components of the TMJ predispose to joint instability. In particular, all radiological examinations (cephalogram, dental panoramic tomography, cone beam computed tomography, and magnetic resonance imaging) allow to demonstrate the presence of a joint tubercle with a steeper posterior surface and a flatter anterior one. This is known to be a predisposing factor to the development of instability (Nam and Lee 2013; Tuijt et al. 2018). On the other hand, there are no morphological nor signal alterations of the capsule-ligament components that reflect those genetically determined connective anomalies which are the basis of ligamentous laxities (either involving the TMJ or systemic). According to many authors, in 95% of patients affected by joint instability linked to ligamentous laxity, the condition does not lead to the development of any morphological or functional alteration of the condylar-discal complex (Chang et al. 2015; Ögren et al. 2012) (Fig. 7.42a,



Fig. 7.42 (a-f) Joint instability associated with an excessive condylar excursion (the condyle moves well beyond the tubercle upon maximum opening) can be observed in different situations. These images show such condition in patients with no TMJ alterations (a, b) patients with a disc dislocation (c, d) or DJD (e, f). Sagittal SE T1 sequences in closed mouth (a) and open mouth (b) show a tubercle with an almost vertical posterior surface and a deepened glenoid fossa (which does not show remodeling). Condyle-disc relationship is maintained. Open mouth sequences display a condyle which moves beyond the tubercle and which is located anterosuperiorly with respect to the tubercle itself. Retrodiscal laminae appear intact and their thickness is within the normal range, with no edema. Sagittal SE T1 images with closed mouth (c) and sagittal STIR open mouth (d) display a prominent tubercle which is affected by slight degenerative subchondral sclerosis. Closed mouth sequences (c) show a condyle which is posteriorly located with respect to fossa and disc. The latter is anteriorly dislocated and its posterior band underwent degenerative changes and appears thicker. Open mouth sequences (d) display recapture of the disc, with a good condylar excursion and well represented retrodiscal laminae. Figures (e) and (f) show severe DJD with smaller condyle affected by bone marrow edema. The tubercle is almost flat and displays subchondral sclerosis. The disc is reduced in thickness and in maximum intercuspation is anteriorly dislocated (e), without reduction upon maximum opening (f). Articular excursion is pronounced, the condyle is well located in front of the tubercle



Fig. 7.42 (continued)

b). In these cases the only anomaly in MR findings is the marked amplitude of the condylar joint excursion, which at maximum opening is placed well in front of the tubercle without any alteration of the retrodiscal laminae or articular effusion. In these cases there is no remodeling of the articular heads, which maintain an adequate bone marrow signal without bone marrow edema. Only a small part of the patients develop joint changes, but it has been shown that patients with instability have a risk which is about seven times greater than that of the general population to develop a functional and morphological alteration. In fact, it is frequent to observe patients with instability and consequent marked joint excursion that have developed disc dislocation (Fig. 7.42c, d) or DJD (Fig. 7.42e, f) (Ögren et al. 2012). Furthermore, in advanced forms of DJD, joint instability is exacerbated by the flattening due to remodeling of the articular tubercle.

Management Subluxation is not a pathology, thus no specific intervention is required apart from the movement performed by the patient in order to restore the correct condyle-disc complex position. However, recurrent subluxation episodes may, in time, damage and loosen the ligaments and may therefore lead to the development of other disorders. No univocal correlation between subluxation and temporomandibular disorders has yet been established (Kavuncu et al. 2006).

7.3.2 Luxation

Overview Luxation (sometimes referred to as spontaneous condylar dislocation, recurrent dislocation or open lock) is the excessive anterior translation of the condyle outside the glenoid fossa which becomes fixed in front of and superior to the eminence, where it cannot be selfreduced by the patient alone. The patient is unable to close his mouth, and the clinician must perform a manipulation in order to reduce the dislocation. In the long term, recurrent luxation may lead to damage of disc, ligaments, and capsule (Kuttenberger and Hardt 2003). Some authors have suggested a further differentiation between condylar dislocation and open lock, based on the differences shown in different radiological exams (Nitzan and Etsion 2002).

Epidemiology Luxation is most commonly seen in young adults aged 25–45, but it may also occur in elderly subjects predisposed to TMJ dislocation (Ziegler et al. 2003; Sang et al. 2010;

Marqués-Mateo et al. 2016). Data regarding its incidence are diverse and range from a reported annual incidence of TMJ dislocation of 5.3 per 100,000 patients presenting to emergency departments each year to 1.8% in the general population (Lowery et al. 2004; Triantafillidou et al. 2012; Prechel et al. 2018).

Etiology and Pathogenesis The phenomenon usually occurs after the patient has performed a wide mouth opening such as yawning, eating large bites of food, or laughing. Iatrogenic triggers such as long dental appointments, endoscopy, and intubation are less common (Katti et al. 2016; Marqués-Mateo et al. 2016). The anteriorly dislocated position of the condyle triggers reflex contraction of chewing muscles which prevent the condylar head from returning to its resting position (De Almeida et al. 2016). The literature reports that condylar dislocation occurs more frequently in patients who are affected by internal derangement, subjects who present a loss of vertical dimension (often edentulous patients who are not wearing dentures), joint laxity, spasms of lateral pterygoid muscles, morphological alteration or variation of condyle and/or fossa-eminence (flattening), neurological disorders, muscular dystonias, and syndromes such as Ehlers-Danlos and Marfan (Undt et al. 1997; Nitzan and Etsion 2002; Medra and Mahrous 2008; Vasconcelos et al. 2009; Sharma et al. 2015). Medial, lateral, cranial, and posterior condylar dislocations are not frequent and are mostly associated with trauma (Harstall et al. 2005).

Presentation Patients typically present with a wide mouth opening and later refer performing an excessive excursion followed by inability to close the mouth and/or occlusal impairment. Clinically, the patient is unable to fully close the mouth so upper and lower incisors are separated, and palpation should reveal an empty joint socket on one or both sides. Medical history reveals that previous episodes, if any, required a maneuver to be performed by a clinician (Katti et al. 2016; Marqués-Mateo et al. 2016). Pain may occur at the time of dislocation with residual pain

following the episode (Peck et al. 2014). In most cases the dislocations are bilateral, in few cases are unilateral (Akinbami 2011).

Some authors suggest a differentiation between open lock and luxation (condylar dislocation) according to the radiological features. In both conditions the patient is unable to fully close, but in an *open lock*, the condyle has reduced mobility and is found beneath the eminence with a disc which may show reduced mobility and appears at times behind the condyle. In *luxation* instead, the maximum opening is increased and the condyle is beyond the eminence, thus resulting anterior and superior to it. A detailed differentiation is however not the purpose of this handbook (Kai et al. 1992; Nitzan and Etsion 2002).

Imaging If correctly diagnosed and treated, acute non-traumatic mandibular luxations can be promptly reduced. This implies that patients do not undergo radiological examinations in these cases. Longstanding chronic or relapsing luxations may instead be further investigated by means of CT or MRI. This is done in order to confirm the diagnosis and to detect predisposing anatomical conditions, but also for accurate planning of any surgical intervention (Martins et al. 2014). Chronic mandibular luxations, which are more frequent in the elderly and in patients affected by neurological diseases, show a condyle which is anterosuperiorly located with respect to the tubercle, with no edema or effusion. Sometimes disc changes are absent as well (Fig. 7.43a–d). Nitzan further differentiates open lock and condylar dislocation according to their radiological features (Fig. 7.43a-d) (Nitzan and Etsion 2002).

Management Treatment for TMJ luxation is mainly conservative. Manual repositioning of the condyle into the glenoid fossa is performed, followed by placement of a head bandage (also known as Barton bandage) for hours to guarantee some stabilization and analgesics may be prescribed if required (Sharma et al. 2015). Reduction of luxation can be done on awake



Fig. 7.43 (**a**–**d**) Long withstanding non-traumatic luxation of the left TMJ. Orthopantomogram (**a**) shows that the left condyle is anteriorly displaced with respect to the tubercle (*arrow*). Axial SE T1 images (**b**) show anterior dislocation of the left condyle (*arrow*), especially when compared

to the right side. Sagittal SE PD in maximum intercuspation (c) shows an anterior luxation of the condyle. Articular excursion is limited (d) but allows the condyle to move further anteriorly. Both the disc (*arrow*) and the retrodiscal laminae are undamaged, without joint effusion

patients most of the time and, the earlier it is performed, the greater are the chances of successful, although maybe only temporary, reduction. Traditionally, the technique is compound (intra and extra-oral) and bimanual, with the thumbs positioned intra-orally bilaterally, even in case of monolateral luxation. Various techniques have been described nonetheless, and specific training for emergency professionals is advisable since many patients resort to the ER because of this. Following repositioning, conservative treatments include physiotherapy and muscular exercises (Cheng 2010; De Almeida et al. 2016) (Fig. 7.44).

Management of condylar dislocation most often becomes surgical whenever luxations are persistent (present for 3–4 weeks) or recurrent, which happens in approximately 5% of patients (Nitzan and Etsion 2002; Triantafillidou et al.



Fig. 7.44 (**a**–**d**) Bilateral open lock, patient is unable to close his mouth (*arrows* indicate the posterior band). Sagittal SE T1 images during the attempt to perform a maximum intercuspation show the right (**a**) and left TMJ (**c**). The condyle is anteriorly positioned in the glenoid fossa and already in relationship with the tubercle. On the right side the disc, which is degenerated and thicker, adheres to the tubercle. Condylar excursion is reduced and

the disc appears immobile with respect to both condyle and tubercle (b). On the left side the disc appears fragmented and the condyle shows marginal osteophytosis. Joint excursion is reduced on this side as well (d). Movement artifacts can be seen and this is not an uncommon finding in patients who are in pain and who are unable to open their mouth in a stable and adequate manner

2012; Prechel et al. 2018). Various conservative and surgical treatments are available (eminectomy, miniplates positioning, and so on), but to this date no evidence-based principle supports one choice over another (De Almeida et al. 2016). Choice for surgical treatment is to be made on an individual basis, taking into consideration the benefits and risks of a surgical intervention under general anesthesia which in a certain category of patients might not be advisable nor indicated (for example edentulous neurologically impaired subjects in elderly facilities).

References

- Adekeye EO. Ankylosis of the mandible: analysis of 76 cases. J Oral Maxillofac Surg. 1983;41:442–9.
- Afroz S, Naritani M, Hosoki H, Takechi K, Okayama Y, Matsuka Y. Prevalence of posterior disc displacement of the temporomandibular joint in patients with temporomandibular disorders: systematic review and meta-analyses. J Oral Facial Pain Headache. 2018;32:277–86. https://doi.org/10.11607/ofph.1924.
- Agerberg G, Helkimo M. Symptomatology of patients referred for mandibular dysfunction: evaluation with the aid of a questionnaire. Cranio. 1987;5:157–63.
- Aiken A, Bouloux G, Hudgins P. MR imaging of the temporomandibular joint. Magn Reson Imaging Clin N Am. 2012;20:397–412. https://doi.org/10.1016/j. mric.2012.05.002.
- Akama MK, Guthua S, Chindia ML, Kahuho SK. Management of bilateral temporomandibular joint ankylosis in children: case report. East Afr Med J. 2009;86:45–8. https://doi.org/10.4314/eamj. v86i1.46930.
- Akheel M, Hussain SMD. Temporomandibular joint disorders. Morrisville: Lulu Press; 2014. p. 253–5.
- Akinbami BO. Evaluation of the mechanism and principles of management of temporomandibular joint dislocation. Systematic review of literature and a proposed new classification of temporomandibular joint dislocation. Head Face Med. 2011;7:10. https://doi. org/10.1186/1746-160X-7-10.
- Al-Baghdadi M, Durham J, Steele J. Timing interventions in relation to temporomandibular joint closed lock duration: a systematic review of 'locking duration'. J Oral Rehabil. 2014a;41:24–58. https://doi. org/10.1111/joor.12126.
- Al-Baghdadi M, Durham J, Araujo-Soares V, Robalino S, Errington L, Steele J. TMJ disc displacement without reduction management: a systematic review. J Dent Res. 2014b;93:37S–51S. https://doi. org/10.1177/0022034514528333.
- Ali AM, Sharawy MM. Histopathological changes in rabbit craniomandibular joint associated with experimentally induced anterior disk displacement (ADD). J Oral Pathol Med. 1994;23:364–74.
- Al-Moraissi EA, El-Sharkawy TM, Mounair RM, El-Ghareeb TI. A systematic review and meta-analysis of the clinical outcomes for various surgical modalities in the management of temporomandibular joint ankylosis. Int J Oral Maxillofac Surg. 2015;44: 470–82. https://doi.org/10.1016/j.ijom.2014.10.017.
- Amaral Rde O, Damasceno NN, de Souza LA, Devito KL. Magnetic resonance images of patients with temporomandibular disorders: prevalence and correlation between disk morphology and displacement. Eur J Radiol. 2013;82:990–4. https://doi.org/10.1016/j. ejrad.2013.01.002.
- Arakeri G, Kusanale A, Zaki GA, Brennan PA. Pathogenesis of post-traumatic ankylosis of the temporomandibular joint: a critical review. Br J

Oral Maxillofac Surg. 2012;50:8–12. https://doi. org/10.1016/j.bjoms.2010.09.012.

- Babu L, Jain MK, Ramesh C, Vinayaka N. Is aggressive gap arthroplasty essential in the management of temporomandibular joint ankylosis?—a prospective clinical study of 15 cases. Br J Oral Maxillofac Surg. 2013;51:473–8. https://doi.org/10.1016/j. bjoms.2012.11.004.
- Bello SA, Aluko Olokun B, Olaitan AA, Ajike SO. Aetiology and presentation of ankylosis of the temporomandibular joint: report of 23 cases from Abuja, Nigeria. Br J Oral Maxillofac Surg. 2012;50:80–4. https://doi.org/10.1016/j.bjoms.2010.12.006.
- Bellot V, Chossegros C, Cheynet F, Guyot L, Sarrat P, Paris J, Blanc JL. Posterior disk displacement of the temporomandibular joint. Apropos 2 cases. Rev Stomatol Chir Maxillofac. 2000;101:23–9.
- Braimah R, Taiwo A, Ibikunle A, et al. Clinical experience in managing temporomandibular joint ankylosis: fiveyear appraisal in a Nigerian subpopulation. J Korean Assoc Oral Maxillofac Surg. 2018;44:112–9. https:// doi.org/10.5125/jkaoms.2018.44.3.112.
- Chang TH, Yuh DY, Wu YT, Cheng WC, Lin FG, Shieh YS, Fu E, Huang RY. The association between temporomandibular disorders and joint hypermobility syndrome: a nationwide population-based study. Clin Oral Investig. 2015;19:2123–32. https://doi. org/10.1007/s00784-015-1422-7.
- Chen K, Xiao D, Abotaleb B, Chen H, Li Y, Zhu S. Accuracy of virtual surgical planning in treatment of temporomandibular joint ankylosis using distraction osteogenesis: comparison of planned and actual results. J Oral Maxillofac Surg. 2018;76:2422.e1. https://doi.org/10.1016/j.joms.2018.07.003.
- Cheng D. Unified hands technique for mandibular dislocation. J Emerg Med. 2010;38:366–7. https://doi. org/10.1016/j.jemermed.2008.12.022.
- Cheong RC, Kassam K, Eccles S, Hensher R. Congenital temporomandibular joint ankylosis: case report and literature review. Case Rep Otolaryngol. 2016;2016:5802359. https://doi. org/10.1155/2016/5802359.
- Chidzonga MM. Temporomandibular joint ankylosis: review of thirty-two cases. Br J Oral Maxillofac Surg. 1999;37:123–6.
- Chossegros C, Cheynet F, Guyot L, Bellot-Samson V, Blanc JL. Posterior disk displacement of the TMJ: MRI evidence in two cases. Cranio. 2001;19:289–93. https://doi.org/10.1080/08869634.2001.11746180.
- Cunha CO, Pinto LM, de Mendonça LM, Saldanha AD, Conti AC, Conti PC. Bilateral asymptomatic fibrousankylosis of the temporomandibular joint associated with rheumatoid arthritis: a case report. Braz Dent J. 2012;23:779–82. https://doi.org/10.1590/ S0103-64402012000600025.
- De Almeida VL, Vitorino Nde S, Nascimento AL, da Silva Júnior DC, de Freitas PH. Stability of treatments for recurrent temporomandibular joint luxation: a systematic review. Int J Oral Maxillofac Surg. 2016;45: 304–7. https://doi.org/10.1016/j.ijom.2015.10.022.

- Drace JE, Enzmann DR. Defining the normal temporomandibular joint: closed-, partially open-, and open-mouth MR imaging of asymptomatic subjects. Radiology. 1990;177:67–71. https://doi.org/10.1148/ radiology.177.1.2399340.
- Drace JE, Young SW, Enzmann DR. TMJ meniscus and bilaminar zone: MR imaging of the substructurediagnostic landmarks and pitfalls of interpretation. Radiology. 1990;177:73–6. https://doi.org/10.1148/ radiology.177.1.2399341.
- Dwivedi AN, Tripathi R, Gupta PK, Tripathi S, Garg S. Magnetic resonance imaging evaluation of temporomandibular joint and associated soft tissue changes following acute condylar injury. J Oral Maxillofac Surg. 2012;70:2829–34. https://doi.org/10.1016/j. joms.2012.08.026.
- Ebrahim S, Montoya L, Busse JW, Carrasco-Labra A, Guyatt GH, Medically Unexplained Syndromes Research Group. The effectiveness of splint therapy in patients with temporomandibular disorders: a systematic review and meta-analysis. J Am Dent Assoc. 2012;143:847–57.
- Ernberg M. The role of molecular pain biomarkers in temporomandibular joint internal derangement. J Oral Rehabil. 2017;44:481–91. https://doi.org/10.1111/joor.12480.
- Foucart JM, Carpentier P, Pajoni D, Marguelles-Bonnet R, Pharaboz C. MR of 732 TMJs: anterior, rotational, partial and sideways disc displacements. Eur J Radiol. 1998;28:86–94. https://doi.org/10.1016/ S0720-048X(97)00102-2.
- Gerbino G, Bianchi SD, Bernardi M, Berrone S. Hyperplasia of the mandibular coronoid process: long-term follow-up after coronoidotomy. J Craniomaxillofac Surg. 1997;25:169–73. https://doi. org/10.1016/S1010-5182(97)80010-8.
- Gerhard S, Ennemoser T, Rudisch A, Emshoff R. Condylar injury: magnetic resonance imaging findings of temporomandibular joint soft-tissue changes. Int J Oral Maxillofac Surg. 2007;36:214–8. https://doi. org/10.1016/j.ijom.2006.09.013.
- Gil C, Santos KC, Dutra ME, Kodaira SK, Oliveira JX. MRI analysis of the relationship between bone changes in the temporomandibular joint and articular disc position in symptomatic patients. Dentomaxillofac Radiol. 2012;41:367–72. https://doi. org/10.1259/dmfr/79317853.
- Goss AN, Bosanquet AG. The arthroscopic appearance of acute temporomandibular joint trauma. J Oral Maxillofac Surg. 1990;48:780–3.
- Gupta VK, Mehrotra D, Malhotra S, Kumar S, Agarwal GG, Pal US. An epidemiological study of temporomandibular joint ankylosis. Natl J Maxillofac Surg. 2012;3:25–30. https://doi. org/10.4103/0975-5950.102146.
- Güven O. A clinical study on temporomandibular joint ankylosis in children. J Craniofac Surg. 2008;19:1263– 9. https://doi.org/10.1097/SCS.0b013e3181577b1b.
- Guyot L, Chossegros C, Cheynet F, Gola R, Lachard J, Blanc JL. Interposition of a full-thickness skin graft in the surgery of temporomandibular joint

ankylosis. A study of 31 cases of which 20 had long-term follow-up. Rev Stomatol Chir Maxillofac. 1995;96:372–8.

- Harstall R, Gratz KW, Zwahlen RA. Mandibular condyle dislocation into the middle cranial fossa: a case report and review of literature. J Trauma. 2005;59:1495–503. https://doi.org/10.1097/01.ta.0000199241.49446.80.
- He D, Ellis E 3rd, Zhang Y. Etiology of temporomandibular joint ankylosis secondary to condylar fractures: the role of concomitant mandibular fractures. J Oral Maxillofac Surg. 2008;66:77–84. https://doi. org/10.1016/j.joms.2007.08.013.
- Helkimo E, Westling L. History, clinical findings, and outcome of treatment of patients with anterior disk displacement. Cranio. 1987;5:269–76. https://doi.org /10.1080/08869634.1987.11678200.
- Hu Y, Zhang L, He D, Yang C, Chen M, Zhang S, Li H, Ellis E III. Simultaneous treatment of temporomandibular joint ankylosis with severe mandibular deficiency by standard TMJ prosthesis. Sci Rep. 2017;7:45271. https://doi.org/10.1038/srep45271.
- Huang IY, Wu JH, Kao YH, Chen CM, Chen CM, Yang YH. Splint therapy for disc displacement with reduction of the temporomandibular joint. Part I: modified mandibular splint therapy. Kaohsiung J Med Sci. 2011;27:323–9. https://doi.org/10.1016/j. kjms.2011.03.006.
- Isacsson G, Isberg A, Johansson AS, Larson O. Internal derangement of the temporomandibular joint: radiographic and histologic changes associated with severe pain. J Oral Maxillofac Surg. 1986;44:771–8.
- Isberg A. Temporomandibular joint dysfunction: a practitioner's guide London. Isis Medical Media. London: Martin Dunitz; 2001. p. 80.
- Isberg A, Eliasson S. A cephalometric analysis of patients with coronoid process enlargement and locking. Am J Orthod Dentofac Orthop. 1990;97:35–40.
- Isberg A, Isacsson G, Nah KS. Mandibular coronoid process locking: a prospective study of frequency and association with internal derangement of the temporomandibular joint. Oral Surg Oral Med Oral Pathol. 1987;63:275–9.
- Israel HA, Langevin CJ, Singer MD, Behrman DA. The relationship between temporomandibular joint synovitis and adhesions: pathogenic mechanisms and clinical implications for surgical management. J Oral Maxillofac Surg. 2006;64:1066–74. https://doi. org/10.1016/j.joms.2006.03.012.
- Jiménez-Silva A, Tobar-Reyes J, Vivanco-Coke S, Pastén-Castro E, Palomino-Montenegro H. Centric relationintercuspal position discrepancy and its relationship with temporomandibular disorders. A systematic review. Acta Odontol Scand. 2017;75:463–74. https:// doi.org/10.1080/00016357.2017.1340667.
- Kai S, Kai H, Nakayama E, Tabata O, et al. Clinical symptoms of open lock position of the condyle. Oral Surg Oral Med Oral Pathol. 1992;74:143–8. https://doi. org/10.1016/0030-4220(92)90372-w.
- Kalaykova S, Naeije M, Huddleston Slater JJ, Lobbezoo F. Is condylar position a predictor for functional signs of

TMJ hypermobility? J Oral Rehabil. 2006;33:349–55. https://doi.org/10.1111/j.1365-2842.2005.01572.x.

- Kalaykova S, Lobbezoo F, Naeije M. Two-year natural course of anterior disc displacement with reduction. J Orofac Pain. 2010;24:373–8.
- Kaminishi RM, Davis CL. Temporomandibular joint arthroscopic observations of superior space adhesions. Oral Maxillofac Surg Clin North Am. 1989;1:103–9.
- Kaneyama K, Segami N, Shin-Ichi T, Fujimura K, Sato J, Nagao T. Anchored disc phenomenon with a normally positioned disc in the temporomandibular joint: characteristics and behaviour. Br J Oral Maxillofac Surg. 2007;45:279–83. https://doi.org/10.1016/j. bjoms.2006.08.021.
- Katsnelson A, Markiewicz MR, Keith DA, Dodson TB. Operative management of temporomandibular joint ankylosis: a systematic review and meta-analysis. J Oral Maxillofac Surg. 2012;70:531–6. https://doi. org/10.1016/j.joms.2011.10.003.
- Katti G, Shahbaz S, Chaubey SS, Khan M. Management of temporomandibular joint dislocation: review of literature. Int J Sci Res. 2016;5:574–7. https://doi. org/10.15373/22778179.
- Katzberg RW, Hatcher D, Ethier J. Specialty imaging: temporomandibular joint. In: Tamimi D, Hatcher D, editors. Specialty imaging: temporomandibular joint. 1st ed. Philadelphia: Elsevier; 2016. p. 490–6.
- Kavuncu V, Sahin S, Kamanli A, Karan A, Aksoy C. The role of systemic hypermobility and condylar hypermobility in temporomandibular joint dysfunction syndrome. Rheumatol Int. 2006;26:257–60. https://doi. org/10.1007/s00296-005-0620-z.
- Kawashima M, Ogura N, Akutsu M, Ito K, Kondoh T. The anti-inflammatory effect of cyclooxygenase inhibitors in fibroblast-like synoviocytes from the human temporomandibular joint results from the suppression of PGE2 production. J Oral Pathol Med. 2013;42:499– 506. https://doi.org/10.1111/jop.12045.
- Kiga N. Histochemistry for studying structure and function of the articular disc of the human temporomandibular joint. Eur J Histochem. 2012;56:e11. https:// doi.org/10.4081/ejh.2012.e11.
- Kim J, Kim MJ, Kho HS. Posterior disk displacement in the temporomandibular joint: a report of two cases. J Oral Med Pain. 2016;41:137–43. https://doi. org/10.14476/jomp.2016.41.3.137.
- Kobayashi R, Utsunomiya T, Yamamoto H, Nagura H. Ankylosis of the temporomandibular joint caused by rheumatoid arthritis: a pathological study and review. J Oral Sci. 2001;43:97–101. https://doi. org/10.2334/josnusd.43.97.
- Kondrat W, Sierpińska T, Radke J. Assessment of the temporomandibular joint function in young adults without complaints from the masticatory system. Int J Med Sci. 2018;15:161–9. https://doi.org/10.7150/ ijms.21665.
- Kraus S, Prodoehl J. Outcomes and patient satisfaction following individualized physical therapy treatment for patients diagnosed with temporomandibular disc displacement without reduction with limited opening:

a cross-sectional study. Cranio. 2017;4:1–8. https:// doi.org/10.1080/08869634.2017.1379260.

- Krishnan DG. Soft tissue trauma in the temporomandibular joint region associated with condylar fractures. Atlas Oral Maxillofac Surg Clin North Am. 2017;25:63–7. https://doi.org/10.1016/j.cxom.2016.11.002.
- Kuribayashi A, Okochi K, Kobayashi K, Kurabayashi T. MRI findings of temporomandibular joints with disk perforation. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2008;106:419–25. https://doi. org/10.1016/j.tripleo.2007.11.020.
- Kuttenberger JJ, Hardt N. Long-term results following miniplate eminoplasty for the treatment of recurrent dislocation and habitual luxation of the temporomandibular joint. Int J Oral Maxillofac Surg. 2003;32:474–9.
- Larheim TA. Role of magnetic resonance imaging in the clinical diagnosis of the temporomandibular joint. Cells Tissues Organs. 2005;180(1):6–21. https://doi. org/10.1159/000086194.
- Larheim TA, Westesson P, Sano T. Temporomandibular joint disk displacement: comparison in asymptomatic volunteers and patients. Radiology. 2001;218:428–32. https://doi.org/10.1148/radiology.218.2.r01fe11428.
- Li JM, An JG, Wang X, Yan YB, et al. Imaging and histologic features of traumatic temporomandibular joint ankylosis. Oral Surg Oral Med Oral Pathol Oral Radiol. 2014;118:330–7. https://doi.org/10.1016/j. 0000.2014.05.007.
- Liu XM, Zhang SY, Yang C, et al. Correlation between disc displacements and locations of disc perforation in the temporomandibular joint. Dentomaxillofac Radiol. 2010;39:149–56. https://doi.org/10.1259/ dmfr/72395946.
- Liu X, Shen P, Zhang S, Yang C, Wang Y. Effectiveness of different surgical modalities in the management of temporomandibular joint ankylosis: a meta-analysis. Int J Clin Exp Med. 2015;8:19831–9.
- Loreto C, Almeida LE, Trevilatto P, Leonardi R. Apoptosis in displaced temporomandibular joint disc with and without reduction: an immunohistochemical study. J Oral Pathol Med. 2011;40:103–10. https://doi. org/10.1111/j.1600-0714.2010.00920.x.
- Lowery LE, Beeson MS, Lum KK. The wrist pivot method, a novel technique for temporomandibular joint reduction. J Emerg Med. 2004;27:167–70. https://doi.org/10.1016/j.jemermed.2004.03.007.
- Madhumati S, Shruthi R, Mitul S, Karan A, Aziz A. TMJ ankylosis: management with reconstruction and interpositional arthroplasty. Niger J Med. 2015;24:374–9.
- Marqués-Mateo M, Puche-Torres M, Iglesias-Gimilio ME. Temporomandibular chronic dislocation: the long-standing condition. Med Oral Patol Oral Cir Bucal. 2016;21:e776–83. https://doi.org/10.4317/ medoral.21221.
- Martins WD, Ribas Mde O, Bisinelli J, França BH, Martins G. Recurrent dislocation of the temporomandibular joint: a literature review and two case reports treated with eminectomy. Cranio. 2014;32:110–7. https://doi.org/10.1179/0886963413Z.00000000017.

- Matsumoto T, Inayama M, Tojyo I, Kiga N, Fujita S. Expression of hyaluronan synthase 3 in deformed human temporomandibular joint discs: in vivo and in vitro studies. Eur J Histochem. 2010;54:e50. https:// doi.org/10.4081/ejh.2010.e50.
- McKay DC, Christensen LV. Whiplash injuries of the temporomandibular joint in motor vehicle accidents: speculations and facts. J Oral Rehabil. 1998;25:731–46.
- McLoughlin PM, Hopper C, Bowley NB. Hyperplasia of the mandibular coronoid process: an analysis of 31 cases and a review of the literature. J Oral Maxillofac Surg. 1995;53:250–5.
- Medra AM, Mahrous AM. Glenotemporal osteotomy and bone grafting in the management of chronic recurrent dislocation and hypermobility of the temporomandibular joint. Br J Oral Maxillofac Surg. 2008;46:119–22.
- Millon-Cruz A, Martin-Granizo R, Encinas A, Berguer A. Relationship between intra-articular adhesions and disc position in temporomandibular joints: magnetic resonance and arthroscopic findings and clinical results. J Oral Maxillofac Surg. 2015;43:497–502. https://doi.org/10.1016/j.jcms.2015.02.010.
- Monje-Gil F, Nitzan D, González-Garcia R. Temporomandibular joint arthrocentesis. Review of the literature. Med Oral Patol Oral Cir Bucal. 2012;17:e575–81. https://doi.org/10.4317/ medoral.17670.
- Murakami K, Segami N, Moriya Y, Iizuka T. Correlation between pain and dysfunction and intra-articular adhesions in patients with internal derangement of the temporomandibular joint. J Oral Maxillofac Surg. 1992;50:705–8. https://doi. org/10.1016/0278-2391(92)90102-6.
- Naeije M, Te Veldhuis AH, Te Veldhuis EC, Visscher CM, Lobbezoo F. Disc displacement within the human temporomandibular joint: a systematic review of a 'noisy annoyance'. J Oral Rehabil. 2013;40:139–58. https:// doi.org/10.1111/joor.12016.
- Nagori SA, Jose A, Gopalakrishnan V, Roy ID, Chattopadhyay PK, Roychoudhury A. The efficacy of dextrose prolotherapy over placebo for temporomandibular joint hypermobility: a systematic review and meta-analysis. J Oral Rehabil. 2018;45:998–1006. https://doi.org/10.1111/joor.12698.
- Nam JN, Lee JY. Skeletal factors related to open lock of the temporomandibular joint. J Oral Med Pain. 2013;38(3):267–74. https://doi.org/10.14476/ JOMP.2013.38.3.267.
- Nebbe B, Major PW. Prevalence of TMJ disc displacement in a pre-orthodontic adolescent sample. Angle Orthod. 2000;70:454–63. https://doi. org/10.1043/0003-3219(2000)070<0454:POTDDI>2 .0.CO;2.
- Nitzan DW. The process of lubrication impairment and its involvement in temporomandibular joint disc displacement: a theoretical concept. J Oral Maxillofac Surg. 2001;59:36–45. https://doi.org/10.1053/ joms.2001.19278.
- Nitzan DW. Temporomandibular joint "open lock" versus condylar dislocation: signs and symptoms, imag-

ing, treatment, and pathogenesis. J Oral Maxillofac Surg. 2002;60:506–11. https://doi.org/10.1053/joms.2002.31846.

- Nitzan DW. 'Friction and adhesive forces'-possible underlying causes for temporomandibular joint internal derangement. Cells Tissues Organs. 2003;174: 6–16. https://doi.org/10.1159/000070570.
- Nitzan DW, Dolwick MF. An alternative explanation for the genesis of closed-lock symptoms in the internal derangement process. J Oral Maxillofac Surg. 1991;49:810–5.
- Nitzan DW, Etsion I. Adhesive force: the underlying cause of the disc anchorage to the fossa and/or eminence in the temporomandibular joint—a new concept. Int J Oral Maxillofac Surg. 2002;31:94–9. https://doi. org/10.1054/ijom.2001.0153.
- Nitzan DW, Marmary Y. The "anchored disc phenomenon": a proposed etiology for sudden-onset, severe, and persistent closed lock of the temporomandibular joint. J Oral Maxillofac Surg. 1997;55:797–802.
- Nitzan DW, Nitzan U, Dan P, Yedgar S. The role of hyaluronic acid in protecting surface-active phospholipids from lysis by exogenous phospholipase A. Rheumatology (Oxford). 2001;40:336–40. https:// doi.org/10.1093/rheumatology/40.3.336.
- Nitzan DW, Kreiner B, Zeltser R. TMJ lubrification system: its effect on the joint function, dysfunction, and treatment approach. Compend Contin Educ Dent. 2004;25:437–40.
- Ogren M, Fältmars C, Lund B, Holmlund A. Hypermobility and trauma as etiologic factors in patients with disc derangements of the temporomandibular joint. Int J Oral Maxillofac Surg. 2012;41:1046–50. https://doi. org/10.1016/j.ijom.2012.02.024.
- Okeson JP. Joint intracapsular disorders: diagnostic and nonsurgical management considerations. Dent Clin N Am. 2007;51:85–103. https://doi.org/10.1016/j. cden.2006.09.009.
- Okeson JP. Management of temporomandibular disorders and occlusion. 7th ed. St. Louis: Elsevier; 2013. p. 244–7, 330–2.
- Okeson JP. Trattamento delle patologie dell'articolazione temporomandibolare. In: Okeson JP. Il Trattamento delle disfunzioni e dei disordini temporomandibolari. 7a Edizione. Edizione Italiana a cura di B. Giuliano Maino. Bologna: Edizioni Martina; 2014a. p. 317.
- Okeson JP. Diagnosi dei disordini temporomandibolari. In: Okeson JP. Il Trattamento delle disfunzioni e dei disordini temporomandibolari. 7a Edizione. Edizione Italiana a cura di B. Giuliano Maino. Bologna: Edizioni Martina; 2014b. p. 252–3.
- Okeson JP. Diagnosi dei disordini temporomandibolari. In: Okeson JP. Il Trattamento delle disfunzioni e dei disordini temporomandibolari. 7a Edizione. Edizione Italiana a cura di B. Giuliano Maino. Bologna: Edizioni Martina; 2014c. p. 245–6.
- Okeson JP. Diagnosi dei disordini temporomandibolari. In: Okeson JP. Il Trattamento delle disfunzioni e dei disordini temporomandibolari. 7a Edizione. Edizione
Italiana a cura di B. Giuliano Maino. Bologna: Edizioni Martina; 2014d. p. 255.

- Okeson JP. Eziologia e diagnosi delle alterazioni funzionali del sistema masticatorio. In: Okeson JP. Il Trattamento delle disfunzioni e dei disordini temporomandibolari. 7a Edizione. Edizione Italiana a cura di B. Giuliano Maino. Bologna: Edizioni Martina; 2014e. p. 150.
- Okeson JP. Trattamento dell'ipomobilità mandibolare cronica e delle patologie dell'accrescimento. In: Okeson JP. Il Trattamento delle disfunzioni e dei disordini temporomandibolari. 7a Edizione. Edizione Italiana a cura di B. Giuliano Maino. Bologna: Edizioni Martina; 2014f. p. 362–72.
- Okochi K, Ida M, Honda E, Kobayashi K, Kurabayashi T. MRI and clinical findings of posterior disk displacement in the temporomandibular joint. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2008;105(5):644– 8. https://doi.org/10.1016/j.tripleo.2007.07.034.
- Orsini MG, Kuboki T, Terada S, Matsuka Y, Yamashita A, Clark GT. Diagnostic value of 4 criteria to interpret temporomandibular joint normal disk position on magnetic resonance images. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1998;86:489–97. https:// doi.org/10.1016/S1079-2104(98)90380-8.
- Paesani D, Westesson PL, Hatala M, Tallents RH, Kurita K. Prevalence of temporomandibular joint internal derangement in patients with craniomandibular disorders. Am J Orthod Dentofac Orthop. 1992;101:41–7. https://doi.org/10.1016/0889-5406(92)70080-T.
- Peck CC, Goulet JP, Lobbezoo F, Schiffman EL, et al. Expanding the taxonomy of the diagnostic criteria for temporomandibular disorders. J Oral Rehabil. 2014;41:2–23. https://doi.org/10.1111/joor.12132.
- Perciaccante VJ, Krishnan DG. Ankylosis of temporomandibular joint. In: Bagheri SC, Jo C, editors. Clinical review of oral and maxillofacial surgery, a case based approach. 2nd ed. St. Louis: Elsevier; 2014. p. 333–51.
- Pertes RA, Gross SG. Clinical management of temporomandibular disorders and orofacial pain. Chicago: Quintessence; 1995. p. 71.
- Posnick JC, Goldstein JA. Surgical management of temporomandibular joint ankylosis in the pediatric population. Plast Reconstr Surg. 1993;91:791–8.
- Posselt U. Hinge opening axis of the mandible. Acta Odontol Scand. 1956;14:c1.
- Prechel U, Ottl P, Ahlers OM, Neff A. The treatment of temporomandibular joint dislocation. Dtsch Arztebl Int. 2018;115:59–64.
- Pullinger AG, Hollender L, Solberg WK, Peterson A. A tomographic study of mandibular condyle position in an asymptomatic population. J Prosthet Dent. 1985;53:706–13.
- Pullinger AG, Solberg WK, Hollender L, Guichet D. Tomographic analysis of mandibular condyle position in diagnostic subgroups of temporomandibular disorders. J Prosthet Dent. 1986;55:723–9.
- Rammelsberg P, Pospiech P, Gernet W, Heumann C, Toutenburg H. Etiologic factors of disk dis-

placements. A statistical analysis of risk factors using MRI as diagnostic standard. Ätiologische Faktoren für Diskusverlagerungen im Kiefergelenk. Eine statistische Risikoanalyse mit der Magnetresonanztomographie als Diagnosestandard. Dtsch Zahnärztl Z. 1996;51:211–7.

- Rammelsberg P, Pospiech PR, Jäger L, Pho Duc JM, Böhm AO, Gernet W. Variability of disk position in asymptomatic volunteers and patients with internal derangements of the TMJ. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1997;83(3):393–9. https:// doi.org/10.1016/s1079-2104(97)90248-1.
- Rammelsberg P, Jäger L, Duc JM. Magnetic resonance imaging-based joint space measurements in temporomandibular joints with disk displacements and in controls. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2000;90:240–8. https://doi.org/10.1067/ moe.2000.107361.
- Rao VM, Liem MD, Farole A, Razek AAKA. Elusive "stuck" disk in the temporomandibular joint: diagnosis with MRI imaging. Radiology. 1993;189:823–7. https://doi.org/10.1148/radiology.189.3.8234710.
- Ren Y-F, 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;80:101–7.
- Renapurkar SK. Discectomy versus disc preservation for internal derangement of the temporomandibular joint. Oral Maxillofac Surg Clin North Am. 2018;30:329–3. https://doi.org/10.1016/j.coms.2018.05.002.
- Ribeiro RF, Tallents RH, Katzberg RW, et al. The prevalence of disc displacement in symptomatic and asymptomatic volunteers aged 6 to 25 years. J Orofac Pain. 1997;11:37–47.
- Sang LK, Mulupi E, Akama MK, Muriithi JM, Macigo FG, Chindia ML. Temporomandibular joint dislocation in Nairobi. East Afr Med J. 2010;87:32–7. https:// doi.org/10.4314/eamj.v87i1.59949.
- Sanroman JF. Closed lock (MRI fixed disc): a comparison of arthrocentesis and arthroscopy. Int J Oral Maxillofac Surg. 2004;33:344–8. https://doi. org/10.1016/j.ijom.2003.10.005.
- Santos KC, Dutra ME, Warmling LV. Correlation among the changes observed in temporomandibular joint internal derangements assessed by magnetic resonance in symptomatic patients. J Oral Maxillofac Surg. 2013;71:1504–12. https://doi.org/10.1016/j. joms.2013.04.033.
- Sarma UC, Dave PK. Temporomandibular joint ankylosis: an Indian experience. Oral Surg Oral Med Oral Pathol. 1991;72:660–4.
- Satoh K, Ohno S, Aizawa T, Imamura M, Mizutani H. Bilateral coronoid hyperplasia in an adolescent: report of a case and review of the literature. J Oral Maxillofac Surg. 2006;64:334–8. https://doi. org/10.1016/j.joms.2005.10.032.
- Scarpino RP, Obrez A, Greising D. Organization and function of the collagen fiber system in the human temporomandibular joint disk and its attachments.

Cells Tissues Organs. 2006;182:201–25. https://doi. org/10.1159/000093969.

- 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;28:6–27. https://doi. org/10.11607/jop.1151.
- Seligman DA, Pullinger AG. Association of occlusal variables among refined TM patient diagnostic groups. J Craniomandib Disord. 1989;3:227–36.
- Sembronio S, Albiero AM, Toro C, Robiony M, Politi M. Is there a role for arthrocentesis in recapturing the displaced disc in patients with closed lock of the temporomandibular joint? Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2008;105:274–80. https:// doi.org/10.1016/j.tripleo.2007.07.003.
- Sharawy M, Ali AM, Choi WS, Larke V. Ultrastructural characterization of the rabbit mandibular condyle following experimental induction of anterior disk displacement. Cells Tissues Organs. 2000;167:38–48. https://doi.org/10.1159/000016765.
- Sharma NK, Singh AK, Pandey A, Verma V, Singh S. Temporomandibular joint dislocation. Natl J Maxillofac Surg. 2015;6:16–20. https://doi. org/10.4103/0975-5950.168212.
- Shen P, Huo L, Zhang SY, Yang C, Cai XY, Liu XM. Magnetic resonance imaging applied to the diagnosis of perforation of the temporomandibular joint. J Craniomaxillofac Surg. 2014;42:874–8. https://doi.org/10.1016/j.jcms.2014.01.001.
- Solberg WK, Bibb CA, Nordström BB, Hansson TL. Malocclusion associated with temporomandibular joint changes in young adults at autopsy. Am J Orthod. 1986;89:326–30.
- Srinivas R, Sorsa T, Tjäderhane L, et al. Matrix metalloproteinases in mild and severe temporomandibular joint internal derangement synovial fluid. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2001;91:517–25. https://doi.org/10.1067/moe.2001.115136.
- Su N, van Wijk AJ, Visscher CM, Lobbezoo F, van der Heijden GJMG. Diagnostic value of ultrasonography for the detection of disc displacements in the temporomandibular joint: a systematic review and meta-analysis. Clin Oral Investig. 2018;22:2599–614. https://doi.org/10.1007/s00784-018-2359-4.
- Tanteri E, Bracco A, Prandi R. Terminologia Gnatologica. In: Tanteri E, Bracco A, Prandi R, editors. Elementi di Gnatologia. Dalla diagnosi alla riabilitazione. Volume I. Milan: RC Libri; 2009. p. 133–87.
- Tasaki MM, Westesson PL, Isberg AM, Ren YF, Tallents RH. Classification and prevalence of temporomandibular joint disk displacement in patients and symptomfree volunteers. Am J Orthod Dentofac Orthop. 1996;109:249–62.
- Tavassol F, Spalthoff S, Essig H, Bredt M, Gellrich NC, Kokemüller H. Elongated coronoid process: CT-based quantitative analysis of the coronoid process and review of literature. Int J Oral Maxillofac

Surg. 2012;41:331–8. https://doi.org/10.1016/j. ijom.2011.10.033.

- Tomas X, Pomes J, Berenguer J, et al. Imaging of temporomandibular joint dysfunction: a pictorial review. Radiographics. 2006;26:765–81. https://doi. org/10.1148/rg.263055091.
- Triantafillidou K, Venetis G, Markos A. Short-term results of autologous blood injection for treatment of habitual TMJ luxation. J Craniofac Surg. 2012;23:689–92. https://doi.org/10.1097/SCS.0b013e31824dba9e.
- Tripathi R, Sharma N, Dwivedi AN, Kumar S. Severity of soft tissue injury within the temporomandibular joint following condylar fracture as seen on magnetic resonance imaging and its impact on outcome of functional management. J Oral Maxillofac Surg. 2015;73:2379.e1–7. https://doi.org/10.1016/j. joms.2015.09.003.
- Tripathy S, Yaseen M, Singh NN, Bariar LM. Interposition arthroplasty in post-traumatic temporomandibular joint ankylosis: a retrospective study. Indian J Plast Surg. 2009;42:182–7. https://doi. org/10.4103/0970-0358.59277.
- Tuijt M, Parsa A, Koutris M, Berkhout E, Koolstra JH, Lobbezoo F. Human jaw joint hypermobility: diagnosis and biomechanical modelling. J Oral Rehabil. 2018;45:783–9. https://doi.org/10.1111/joor.12689.
- Undt G, Kremer E, Rasse M. Treatment of recurrent mandibular dislocation, part I: eminectomy. Int J Oral Maxillofac Surg. 1997;26:92–7. https://doi. org/10.1016/S0901-5027(05)80634-4.
- Vasconcelos BC, Porto GG, Neto JP, Vasconcelos CF. Treatment of chronic mandibular dislocations by eminectomy: follow-up of 10 cases and literature review. Med Oral Patol Oral Cir Bucal. 2009;14:e593– 6. https://doi.org/10.4317/medoral.14.e593.
- Venetis G, Pilavaki M, Triantafyllidou K, Papachristodoulou A, Lazaridis N, Palladas P. The value of magnetic resonance arthrography of the temporomandibular joint in imaging disc adhesions and perforations. Dentomaxillofac Radiol. 2011;40: 84–90. https://doi.org/10.1259/dmfr/13255885.
- von Domarus H, Scheunemann H. Congenital prearticular temporo-mandibular ankylosis in two siblings. J Craniomaxillofac Surg. 1990;18(7):299–303.
- Westesson PL, Larheim TA, Tanaka H. Posterior disc displacement in the temporomandibular joint. J Oral Maxillofac Surg. 1998;56:1266–73.
- Whyte AM, McNamara D, Rosenberg I, Whyte AW. Magnetic resonance imaging in the evaluation of temporomandibular joint disc displacement—a review of 144 cases. Int J Oral Maxillofac Surg. 2006;35: 696–703. https://doi.org/10.1016/j.ijom.2005.12.005.
- Winocur E, Gavish A, Halachmi M, Bloom A, Gazit E. Generalized joint laxity and its relation with oral habits and temporomandibular disorders in adolescent girls. J Oral Rehabil. 2000;27:614–22. https://doi. org/10.1046/j.1365-2842.2000.00546.x.
- Yang C, Zhang SY, Wang XD, Fan XD. Magnetic resonance arthrography applied to the diagnosis of intraarticular adhesions of the temporomandibular joint.

Int J Oral Maxillofac Surg. 2005;34:733–8. https://doi. org/10.1016/j.ijom.2005.02.011.

- Yang X, Yao Z, He D, Cai Y, Dong M, Yang C. Does soft tissue injury affect intracapsular condylar fracture healing? J Oral Maxillofac Surg. 2015;73:2169–80. https://doi.org/10.1016/j.joms.2015.05.030.
- Yoshida K, Takatsuka S, Hatada E, et al. Expression of matrix metalloproteinases and aggrecanase in the synovial fluids of patients with symptomatic temporomandibular disorders. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2006;102:22–7. https://doi. org/10.1016/j.tripleo.2005.07.013.
- Young AL. Internal derangements of the temporomandibular joint: a review of the anatomy, diagnosis, and management. J Indian Prosthodont Soc. 2015;15:2–7. https://doi.org/10.4103/0972-4052.156998.
- Yura S, Totsuka Y, Yoshikawa T, Inoue N. Can arthrocentesis release intracapsular adhesions? Arthroscopic findings before and after irrigation under sufficient hydraulic pressure. J Oral Maxillofac Surg. 2003;61(11):1253–6. https://doi.org/10.1016/ s0278-2391(03)00724-9.
- Yura S, Nobata K, Shima T. Diagnostic accuracy of fatsaturated T2-weighted magnetic resonance imaging in the diagnosis of perforation of the articular disc of the temporomandibular joint. Br J Oral Maxillofac Surg. 2012;50:365–8. https://doi.org/10.1016/j. bjoms.2011.05.017.

- Zhang S, Liu X, Yang C, Cai X, Chen M, Haddad MS, Yun B, Chen Z. Intra-articular adhesions of the temporomandibular joint: relation between arthroscopic findings and clinical symptoms. BMC Musculoskelet Disord. 2009;10:70. https://doi. org/10.1186/1471-2474-10-70.
- Zhang S, Huang D, Liu X, Yang C, Undt G, Haddad SM, Chen Z. Arthroscopic treatment for intraarticular adhesions of the temporomandibular joint. J Oral Maxillofac Surg. 2011;69:2120–7. https://doi. org/10.1016/j.joms.2010.12.039.
- Zhao J, He D, Yang C, et al. 3-D computed tomography measurement of mandibular growth after costochondral grafting in growing children with temporomandibular joint ankylosis and jaw deformity. Oral Surg Oral Med Oral Pathol Oral Radiol. 2017;124:333–8. https://doi.org/10.1016/j.oooo.2017.06.002.
- Zhong SC, Xu ZJ, Zhang ZG, Zheng YH, Li TX, Su K. Bilateral coronoid hyperplasia (Jacob disease on right and elongation on left): report of a case and literature review. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2009;107:e64–7. https://doi. org/10.1016/j.tripleo.2008.10.017.
- Ziegler CM, Haag C, Mühling J. Treatment of recurrent temporomandibular joint dislocation with intramuscular botulinum toxin injection. Clin Oral Investig. 2003;7:52–5. https://doi.org/10.1007/ s00784-002-0187-y.

Joint Diseases

0

Tiziana Robba, Paolo Tosco, Simone Parisi, Guglielmo Ramieri, Enrico Fusaro, Riccardo Faletti, and Giulia Tanteri

Key Points

- Degenerative joint disease is a progressive, irreversible disease characterized by remodeling, deterioration of cartilage, sclerosis of subchondral bone and osteophyte formation.
- Etiology is multifactorial: joint instability, previous traumas, mechanical overload, disc displacement without reduction (DDWoR) are believed to be predisposing factors.
- CT has high spatial resolution and high diagnostic accuracy for degenerative TMJ altera-

P. Tosco Clinica Fornaca di Sessant, Turin, Italy

S. Parisi · E. Fusaro

Rheumatology Unit, Department of General and Specialized Medicine, A.O.U. Città della Salute e della Scienza, Turin, Italy

G. Ramieri

e-mail: guglielmo.ramieri@unito.it

R. Faletti

Department of Surgical Sciences, Radiology Unit, University of Turin, Turin, Italy e-mail: riccardo.faletti@unito.it

G. Tanteri Studio Tanteri, Turin, Italy e-mail: tanteri@tanteri.it tions (erosion, subchondral and generalized sclerosis, osteophytes, bone edema, subcortical cysts).

- MRI can simultaneously detect disc displacements, joint effusion and bone marrow edema and has a crucial role in early and differential diagnosis of all DJDs.
- Inflammatory TMJ Rheumatic such as Rheumatoid Arthritis (RA), Juvenile Idiopathic Arthritis (JIA) and Spondyloarthritis (SpA) can involve the TMJ. Common arthritis signs are bone marrow edema, joint effusion, synovial thickening, joint erosion.
- Idiopathic Condylar Resorption (ICR) is a progressive resorption of condyles with consequent facial deformity and no known cause. ICR is a diagnosis of exclusion. ICR treatment depends on the extent of joint damage and on the associated facial features and may include orthodontic treatment only, orthognathic surgery and a combination of orthognathic surgery and TMJ prosthetic replacement.
- Depending on diagnosis and severity, DJD treatment may range from conservative non-invasive therapies, to total joint replacement.

8.1 Degenerative Joint Disease

The terms Degenerative Joint Disease (DJD), Osteoarthritis, and Osteoarthrosis (OA) have often been used as synonyms in the past. Peck and

© Springer Nature Switzerland AG 2020 T. Robba et al. (eds.), *MRI of the Temporomandibular Joint*, https://doi.org/10.1007/978-3-030-25421-6_8

T. Robba (🖂)

Department of Diagnostic Imaging and Radiotheraphy, Radiology Service, C.T.O. Hospital, A.O.U. Città della Salute e della Scienza, Turin, Italy e-mail: trobba@cittadellasalute.to.it

Department of Surgical Sciences, Division of Maxillofacial Surgery, University of Turin, Turin, Italy

colleagues, in the '*expanded Temporomandibular Disorders (TMD) taxonomy*', enveloped osteoarthritis and osteoarthrosis as separate entities within the DJD group. The two show the same anatomical features, however osteoarthritis refers to a high inflammatory status along with a painful joint condition (arthralgia), a clinical symptom which is instead absent in DJD subjects that present with signs of osteoarthrosis (Ahmad et al. 2009; Schiffman et al. 2014; Peck et al. 2014). For convenience, the two terms shall here fall under the same label *osteoarthrosis/osteoarthritis* (OA).

Degenerative joint disease is characterized by loss of cartilage, remodeling of subchondral bone, osteophyte formation, and it can be combined with the previously-described disc disorders.

8.1.1 Epidemiology

Recent studies on Temporomandibular Disorders (TMD), suggest an increasing prevalence of TMDs in children and adolescents, with peaks at age 15 and 19, but most importantly a female to male ratio which ranges from 3.5:1 up to 5:1. (Thilander et al. 2002; Köhler et al. 2009; Winocur et al. 2009; Zhao et al. 2011; Horton et al. 2016; Lei et al. 2017). These data however, encompass all of the TMD spectrum, whereas signs and symptoms of OA are usually more predominant in adults between 20 and 40 years of age (Yap et al. 2003). Prevalence of TMJ OA has been described as similar to that of generalized OA, and several authors outlined prevalence and findings according to the different age groups: prevalence based on clinical aspects and MRI was found to be 25% in a 20-49-year age group, and 70% in subjects over 70 years of age (Bernhardt et al. 2007; Schmitter et al. 2010; Das 2013; Bansal 2016).

8.1.2 Etiology

The etiology of TMJ OA is complex and multifactorial. Long-sustained mechanical joint overloading (with oral parafunctional habits), joint instability (i.e., hypermobility of joint components due to ligamentous laxity as well as trauma), previous injuries and macrotrauma are credited for being predisposing factors (Kalladka et al. 2014; Chisnoiu et al. 2015; Manfredini et al. 2016). These conditions can lead to or be associated with disc disorders of the TMJ as well as its subsequent stages, in which damaged intra-articular tissues lead to disturbances in the harmonic biomechanical functioning of the temporomandibular joint. Besides, according to some Authors, disc disorders can be considered as the forerunner of a degenerative process (Dimitroulis 2005). Although the literature offers conflicting standpoints over a potential correlation between disc displacement (DD) and DJD, disc displacement without reduction (DDWoR) has been correlated with osteoarthrotic changes of the TMJ (Toller 1973; Takatsuka et al. 2005; Campos et al. 2008; Cortés et al. 2011; Dias et al. 2012; Gil et al. 2012; Roh et al. 2012; de Melo 2015). Displaced discs might interfere with condylar mobility and lead to an increased load on condylar surfaces (Takatsuka et al. 2005). The articular disc, in fact, protects the underlying tissues, and its displacement could expose articular surfaces to excessive additional pressure and ultimately degenerative changes (Maydana et al. 2010). Other authors highlighted that osteoarthrotic bone degenerative changes not only tend to increase in case of DDWoR, but that they are also associated with oral parafunction (Honda et al. 2008). In this respect, occlusal modifications have also been blamed, especially when immediate, like those that follow orthognathic surgery. These correlations have been widely debated in the literature and are part of clinicians' familiar observations (Hoppenreijs et al. 1999; Gill et al. 2008; Catherine et al. 2016).

Cartilage and subarticular bone destruction occur gradually, and degenerative alterations will most likely show up a variable amount of time after initial damage, disc displacement included (Manfredini et al. 2016).

8.1.3 Pathogenesis

TMJ OA is a chronic irreversible disease, characterized by deterioration and abrasion of the articular cartilage and by subarticular bone loss. All of the conditions that trigger and take part in the process of degeneration seem to bring to a cascade of events that flow into production and release of free radicals, cytokines, catabolites, and enzymes that will ultimately degrade joint components.

In healthy joints, and this is especially true for the TMJ which has special post-natal development and transformation, these substances are responsible for tissue remodeling as an adaptive response to functional forces (Stegenga et al. 1989; Tanaka et al. 2008). However, if the balance between adaptive capacity and joint load is unfavorable, cartilage breakdown will eventually begin (Ernberg 2017). The physiological joint environment is therefore altered so that the habitual catabolic-anabolic equilibrium fails to maintain its homeostatic status.

In the early stages of OA, synovial fluid qualities change so that lubrication is less efficient and friction increases. Cartilage matrix is affected by this, and a pro-inflammatory status originates due to cytokines release and proteinases action. Degradation with focal loss of cartilage occurs and pain may be triggered (Chen et al. 2013; Kalladka et al. 2014; Ernberg 2017).

Hypoperfusion-reperfusion mechanisms may begin where friction and loading have an on-off presence and this furtherly exhausts synovial fluid viscosity and worsens attrition between disc and cartilage or cartilage and cartilage. Degradation of matrix components carries on and, as the cartilage thins and breaks with deeper fissures, fibrillation and so on, adhesions may also form.

Together with cartilage thinning, two phenomena can occur at the cartilage-subchondral bone interface: there may be an osteoclast activation (*regressive remodeling*) followed by trabecular rearrangement, and there may be new bone apposition (*progressive remodeling*). Apposition and resorption will follow the joint's stress lines, in an attempt to restore articular surface where necessary (Embree et al. 2011).

Progressive and Regressive Remodeling Bone apposition is allowed by the calcified cartilage layer which produces chondroid or fibrous bone, therefore making this a separate ossification

mechanism which, according to some, is responsible for osteophytes formation and subchondral sclerosis (Katakami et al. 2008; Wu et al. 2015).

Progressive remodeling is part of functional remodeling and it can be translated into a cortical thickening of the condylar head profile. Functional remodeling is considered an adaptation mechanism to loads and as an intermediate phase prior to OA (Mercuri 2008; Tanaka et al. 2008; Ahmad et al. 2009; Ok et al. 2014; Seo et al. 2015).

As outlined by Schellhas, an almost uniquely regressive remodeling may follow the functional remodeling stage (Schellhas 1989). Such regressive remodeling might come as a confined phenomenon and lead to an overall reduction in condyle size, or be associated with the development of OA more common features such as osteophytes and subchondral sclerosis, or even come together with erosive alterations and intense bone edema.

8.1.4 Presentation and Diagnosis

OA signs and symptoms usually include joint noises such as crepitus, gritting, crackling, popping or snapping during jaw movement, chronic limitation of jaw movements and pain. Sometimes a history of clicking sounds, which at some point disappeared, is reported by patients. According to severity and involvement of one or both joints, deviations or deflections of the incisal path may be present during opening and protrusion (Fig. 8.1a, b). Lateral movements might be very limited, blocked or with asymmetrical extents. Deflection to one side during opening and protrusion may be due to loss of translational capacity in the damaged joint, and it will not necessarily be apparent in bilateral OA with similar-stage disease. In general, late-stage DJD may show painless mild residual symptoms, such as crepitus and chronic restriction of motion.

Whenever regressive remodeling prevails, a decrease in condylar height can occur along with a mandibular ipsilateral shift, facial asymmetry and a dental malocclusion (Schellhas 1989).



Fig. 8.1 30-year-old female—Right TMJ OA. Closed mouth position (a) and deflection toward the affected side during mouth opening (b)

Preliminary diagnosis is often based on clinical suspicion. A history of crepitus during movements, with pain or without, and changes or limitations in jaw movements should lead to the hypothesis of some degree of OA. Before even getting to the so-called 'chief complaint', patients may also have undergone some sort of x-ray investigation showing signs of OA.

The differential diagnosis between DJD and rheumatic or other diseases (idiopathic resorption and so forth) can be difficult when erosive patterns and bone resorption are the dominant aspects.

In some cases, joints undergoing degenerative changes may not exhibit typical clinical characteristics that are indicative of disease, hence the importance of diagnosing TMJ OA through imaging techniques (Peterson 2010; Dias et al. 2016).

In conclusion TMJ OA is to be considered as a later manifestation of an ongoing process in which TMJ structural damage should be traced back to specific causes by the clinician. Understanding the possible explanation for joint deterioration is essential for proper patientdoctor communication, treatment, prognosis, and for possibly delaying disease progression.

8.1.5 Imaging

Radiological diagnosis and OA staging for other joints can count on a considerable number of classifications. The most common grading systems encompass narrowing of joint space (grade I), presence of osteophytes (grade II), subchondral bone sclerosis and bone contour deformity (grade III and IV), according to the Kellgreen-Lawrence and the International Knee Documentation Committee (IKDC) scores (Wright 2014).

These classifications may, in theory, hold true for the TMJ too, however this joint displays functional and morphological peculiarities that other joints do not exhibit such as the aforementioned remodeling capacity (Tanaka et al. 2008).

In general, TMJ OA radiological assessment first of all requires an accurate evaluation of bone

remodeling, especially of the condyle, followed by an analytical search for the following degenerative disease-related radiographic signs (Ahmad et al. 2009):

- Osteophyte
- Subchondral sclerosis
- Generalized sclerosis
- Surface erosion
- Subcortical cyst
- Bone edema

For both traditional x-ray techniques (plain film radiography, conventional tomography, orthopantomography) and CT scans, identical diagnostic elements will be used. Clearly, CT will have a high spatial resolution and high diagnostic accuracy for degenerative TMJ alterations (Larheim et al. 2015).

In the case of MRI the same signs will have to be explored, albeit with a lower sensitivity for calcified components of joints, but soft tissues will be better assessed. MRI can therefore simultaneously detect disc displacements, joint effusion and bone marrow edema (Emshoff et al. 2003a, b, 2006; Emshoff and Rudisch 2007; Ahmad et al. 2009; Alkhader et al. 2010; Dias et al. 2012). Because of this additional knowledge, MRI has a higher specificity as opposed to CT, which still has a higher sensitivity in diagnosing OA and shows a better interobserver concordance (Ahmad et al. 2009).

Articular Surfaces Remodeling Joint surface remodeling is caused by a change in loading forces, followed by induced cartilage and subchondral bone responses which take place together with substantial histological changes (Wu et al. 2015; Zhang et al. 2015).

In particular, condylar remodeling is frequently associated with a thinning of the cartilage layer. It occurs along the joint's stress lines and it is therefore related to the position of the condyle within the fossa.

Because posterior positioning of the condyle is more frequently associated to disc disorders, regressive remodeling is found to happen more often here, at the posterior surface. This part therefore appears flattened in the sagittal view, whereas the anterior part is usually preserved or increased due to progressive remodeling (Fig. 8.2a–d). Flattening of the anterior surface is a less common finding (Fig. 8.2e).

In the coronal plane, remodeling is generally first seen at the lateral pole (Fig. 8.3a, b), with a reduction in size of this particular part (Kurita et al. 2003). At a later stage, remodeling can then spread to the whole condylar surface and result in a generally flattened contour (Fig. 8.3c). More advanced cases of regressive remodeling will exhibit an overall smaller condyle, with a decreased vertical height and a thinner anteroposterior diameter (Fig. 8.4a–d) (Gomes et al. 2015).

Not only does the condyle undergo remodeling, but also the glenoid fossa and the eminence may be subjected to DJD changes. The fossa commonly broadens and deepens and this leads to an increase in joint coverage (coverage index: the ratio between the width and the depth of the fossa in the coronal view). Because the osteoarthrotic condyle is relatively smaller, condylar stability within the fossa may be lost and, during radiological assessment, the small eccentric condyle will emerge more than the wider glenoid fossa (Dupuy-Bonafé et al. 2014). In these cases, the articular tubercle too will be more prominent because of the deeper fossa (Fig. 8.5a-c). On the other hand, some cases exhibit remodeling mostly at the tubercle. Flattening is a common feature and the glenoid fossa will look more shallow (Fig. 8.4a–c, Fig. 8.5d–e) (Kurita et al. 2000).

TMJ Bone Degenerative Changes As previously explained, CT guarantees a better accuracy in detecting traditional OA diagnostic signs (Ahmad et al. 2009; Alkhader et al. 2010). Slight subchondral sclerosis or small osteophytes may go undetected in MRI, even because osteosclerosis phenomena (osteophytes, subchondral sclerosis, diffuse trabecular sclerosis) give off constant signal hypointensity. DJD signs are summarized below.



Fig. 8.2 45-year-old female, asymptomatic right TMJ, painful left TMJ. On the right side the posterior surface of the condyle appears flatter (**a**) (*arrow*—sagittal SE PD), the condyle preserves regular bone marrow signal with no bone edema (**b**) (sagittal STIR). On the left side STIR sagittal images with closed mouth (**c**) and open

mouth (d) sequences show remodeling of the posterior surface of the condyle and subchondral bone edema (*arrow*). Remodeling of the anterior surface of the condyle is less frequently observed (*arrow* in (e)—sagittal SE PD)



Fig. 8.3 A condyle which is free from degenerative changes seen in coronal SE T2 (a). Remodeling starts from the lateral pole (*arrow*, b). Also in severe degenera-

tive forms (c) the articular profile is flattened but the lateral pole (*arrow*) is greatly remodeled and affected by marginal osteophytosis



Fig. 8.4 In more severe forms of condylar remodeling, the condylar head is reduced in size and the condylar neck is shortened (sagittal SE PD **a**–**c**) and appears thinned out

(arrow) in the axial plane (d). The tubercle also shows remodeling and appears flatter

Fig. 8.5 Remodeling of the glenoid cavity (*arrows* in **a**—sagittal SE PD) causes it to deepen and become wider with respect to the condyle. In more severe cases (sagittal SE PD closed mouth in (**b**) and open mouth in (**c**)) the tubercle also appears relatively larger (*arrows*) and its posterior slope becomes steeper. Remodeling may affect

the tubercle as well, thus giving it a flatter appearance, as seen in this early case of Idiopathic Condylar Resorption which is described in section 8.3 (d, e). Here, the slope disappears and tubercle and glenoid fossa appear on the same level



Osteophytes are sclerotic bony spurs found on the articular surface. Most of the time an osteophyte will lie on the anterior aspect of the condylar head. Due to its better spatial resolution, CT can detect small and initial osteophyte formation which would not be apparent in MR. In more advanced DJD, osteophytes increase in size and are well visible in MRI (Fig. 8.6a–d). Subchondral sclerosis is a sclerotic thickening of the subchondral bone. It occurs where joint loading forces are bigger and this usually is where the articular space is smaller. In this scenario as well, CT is more sensitive for small, initial subchondral sclerosis (Fig. 8.7a–b) areas as opposed to MR, although MR can show related alterations such as cartilage and discal changes,



Fig. 8.6 A small osteophyte can be visible in CT scans (*arrow*, **a**) whereas it is scarcely visible in MRI (**b**). When osteophytes become larger, it is possible to observe them

in MRI as well (closed mouth and open mouth, c and d). The tubercle is flattened out and the posterior surface of the condyle is flat due to remodeling

Fig. 8.7 Trabecular sclerosis of subchondral bone is one of the first signs of DJD and it is best seen in CT scan (**a**, reformatted coronal CT). The corresponding MR (**b**) does not allow to evaluate subchondral sclerosis, but there is an altered signal within the overlying cartilage due to chondropathy. The disc is not visible (*arrow*)





Fig. 8.8 This case shows extensive subchondral sclerosis of the condylar head. Its extent makes it visible in MR. This coronal T2 sequence shows hypointense trabecular bone due to degenerative sclerosis

as well as bone edema. Subchondral sclerosis can extend to large condylar portions (Fig. 8.8) and become apparent in MRI too.

Bone erosion is defined by a focal discontinuity of the osteocartilaginous structure. It is usually quite limited but it notably involves the subchondral bone with an ill-defined area without trabeculae (Fig. 8.9a–c). Bone erosion is a common feature of the whole OA spectrum, whether purely degenerative or due to rheumatological forms, and it is associated with bone edema in MRI.

Subchondral cysts are small, mostly round defects with sclerotic margins within the subchondral bone. They can be explained as pseudocysts, as there are no proper cyst walls and because the sclerotic margins are outlined by adjacent cancellous bone reaction. Sometimes a wider surrounding sclerotic halo can be better seen in CT (Fig. 8.10a, b). The appearance of subchondral cysts is related to late-stage modifications and because of this, they are usually accompanied by extensive remodeling, erosions and diffuse subchondral sclerosis, all of which are seen in MRI (Fig. 8.10c–e).

Bone marrow edema, or more simply bone edema, is the result of partial or complete replacement of yellow bone marrow with fluid. In MRI, this is marked by signal hyperintensity in fat-suppression sequences (Thiryayi et al. 2008). Bone edema signal appears in fact like water rather than fatty marrow. More in detail, edema shows hyperintensity in fat-suppressed sequences, hyperintensity in TR (T2 and PD), and hypointensity in T1 weighted sequences. Bone marrow edema signal is an MRI distinctive finding. It is however, a non-specific sign whose relevance depends on its cause. In traumas for instance, bone marrow hemorrhage occurs, in functional overloads bone marrow is replaced by inflammatory exudate, whereas in OA inflammatory infiltrate is mostly present in the case of rheumatological forms (Thiryayi et al. 2008; Meyers and Laor 2013; Vaid et al. 2014). As described by



Fig. 8.9 The anterior osteocartilaginous profile of the condyle is interrupted by a small bone erosion (*arrow*, **a**) which is surrounded by extensive bone marrow edema

(*arrow* in **b**—sagittal STIR sequence). The erosion (*arrow*) is also visible in coronal SE T2 view (**c**)

histological studies, remodeling-related edema is marked by inflammatory exudate that is associated with interstitial fibrosis and drop in the yellow marrow component in more advanced DJD (Larheim et al. 1999).

In case of functional overload, bone edema follows the joint's stress lines like in remodeling, with which it is often associated together with DD (Emshoff et al. 2003a, b). Since bone often responds to remodeling with subchondral sclerosis, bone edema is strongly linked to sclerosis as well. Besides, subchondral sclerosis' hypointensity may conceal edema hyperintensity in fatsuppression or long TR sequences.

In the case of OA, condylar edema can be diagnosed in nearly 60% of cases, but the literature does not agree on whether this element correlates with subjective pain perception or other painful conditions (DDwR, DDWoR, DJD) (Sano et al. 1999; Sano 2000; Emshoff et al. 2003a, b; Chiba et al. 2006). Nevertheless, in up to 37% of DJDs, bone edema can be found (Emshoff et al.



Fig. 8.10 Reformatted sagittal (**a**) and axial (**b**) CT—a subchondral cyst is visible. It is outlined by sclerotic margins and surrounded by degenerative trabecular sclerosis. The corresponding sagittal STIR in open mouth (**c**) and

closed mouth axial SE T1 (d), clearly show the subchondral cyst and its sclerotic margins. Coronal T2 (e) also shows the extent of the cyst

2003a, b; Takahara et al. 2017). Whenever bone edema is present, pain is 15-fold more likely, and disc disorders are three-fold more probable (DDWoR—nine-fold and DDWor plus effusion—more than six-fold more likely) (Emshoff et al. 2003a, b). Conversely, it was demonstrated that in up to 10% of bone edemas, there is no joint pain (Takahara et al. 2017; Wahaj et al. 2017). The most suitable sequences for revealing bone edema are SE T1 and fat-saturated SE T2 (rather than SE T2 and SE PD) (Morimoto et al. 2004; Wahaj et al. 2017).

The features outlined above do not belong to the condyle only: trabecular sclerosis, small erosions and subcortical cysts may be present at the articular tubercle as well, especially when there is some degree of remodeling in association.

As proposed in the Diagnostic Criteria (DC), osteoarthrosis and osteoarthritis are two separate subgroups within DJD (Schiffman et al. 2014). Osteoarthritis is a degenerative condition accompanied by arthralgia and signs of inflammation, although these are less remarkable than those related to rheumatologic origin (Mercuri 2008; Schiffman et al. 2014). Differently from osteoarthrosis, osteoarthritis therefore shows CT and MR signs of inflammation of soft tissue and bone (erosions, bone edema, articular effusion, sinovitis, capsuloligamentous edema) (Fig. 8.11a–f).

TMJSoftTissuesDegenerativeChangesNowadays, a correlation between DDand DJD has been pointed out extensively (for adetailed description of Disc Disorders, see Chap.7). For instance, Wilkes' classification and thework of other Authors illustrate that DD (DDWoRespecially)precedesDJD (Schellhas1989;Dimitroulis2005;Dias et al.2016).With this respect, MRI offersthe best diagnostic accuracy and highest concordance for DD and its characteristics have beenpreviously explained (Ahmad et al.2009).

Similarly to bone edema, the presence of *joint effusion* does not seem to necessarily correlate with arthralgia although, according to many, this

is generally the distinguishing sign presented by DD or DJD patients which complain about joint pain (Larheim et al. 2001; Güler et al. 2005). In this perspective, effusion is deemed responsible for pain as it conveys pro-inflammatory mediators from the inflamed synovium, and this could be the reason why joint effusion patients benefit from intra-articular lavage, although a clear correlation has not been shown yet and studies have reached conflicting conclusions (Emshoff and Rudisch 2007; Thomas et al. 2018). Nevertheless, a higher count of pro-inflammatory cytokines and proteins can actually be found in considerably effused joints, and MRI signs of synovitis are more frequently associated (Segami et al. 2001, 2002). Effusion can be seen as a presence of extra fluid within the joint space that is hyperintense in T2-weighted sequences. It is most typically found at the upper joint compartment and, although MRI-based quantitative assessment of synovitis and effusion are possible for other joints, there is no such evaluation routinely performed for the TMJ.

8.1.6 Management

As DJD is the shared final pathway for a number of conditions, it is clearly mandatory to identify and address the underlying cause, whenever possible and if known. DJDs are the wellestablished consequences of many circumstances and, although restoration of any given structural alteration is not always required to successfully treat the patient, it remains an important aspect of treatment for some cases (Israel 2016). This is especially true for inflammatory diseases which are described in the next section.

Provided that a DJD diagnosis has been formulated and morphological alterations have all been described with imaging, it is a matter of superimposing this knowledge to the main concern of the patient which may actually vary quite considerably. Pain management is the first element to consider regardless of the underlying features. Restriction of motion and its subjective relevance then need to be evaluated.



Fig. 8.11 Osteoarthritis (**a**-**c**)—effusion (*black arrow*), edema (*white arrow* in **a**) and slight erosion (*white arrow* in **b**) are well recognizable. Coronal T2 (**c**) shows effusion (*black arrow*). These findings are similar to those found in

osteoarthrosis (**d–f**) (remodeling*—black arrow*, osteophytosis*—white arrow*) on the contralateral side, but effusion and edema are not visible in this case

In clear, defined cases of DJD different scenarios can therefore be encountered, with variable degrees of joint sounds:

- patients with pain and satisfactory mouth opening;
- patients with pain and severe restriction of motion;
- painless patients with satisfactory mouth opening;
- patients with chronic restriction of motion and variable pain.

This type of distinction was observed and classified by Wilkes. In his staging system, he assumed that there was a progression of the disease and he subdivided DD/DJD patients according to symptoms and radiological findings, in order to outline indications for treatment as specifically as possible, as it is clear that not all DJD patients are alike (Wilkes 1989). Still, this classification conceals a weakness, in that clinical and radiological patterns do not necessarily evolve in an intertwined way, and one severe radiological stage does not necessarily fit with the same clinical severity (Nitzan et al. 2017).

For this reason, a wide range of options may be appropriate and has been described, including no treatment, conservative non-invasive therapies, minimally invasive surgical procedures (arthrocentesis, arthroscopy), arthroplasty (repair and harmonization of intra-articular tissues), discectomy, and total joint replacement.

Non-invasive therapies are usually advocated for early OA and in patients who do not want or cannot undergo other types of treatment. Non-invasive therapies are also used as adjunctive modalities in more invasive treatments where combined approaches are often needed. NSAIDs (non-steroidal anti-inflammatory drugs), occlusal splints, physical therapy, transcutaneous electrical nerve stimulation (TENS) and modification of dietary habits are well known. Their scope is to relieve pain, improve muscle relaxation, decrease joint load and provide occlusal stability (McNeely et al. 2006; Murphy et al. 2013). More invasive treatments are appropriate in selected patients who maintain a restricted range of motion and who do not respond to conservative treatment for at least 6 months and suffer because of the severe TMJ functional impairment (Wänman et al. 2016; List and Jensen 2017).

In these cases, arthrocentesis/arthroscopy may be indicated, especially if imaging and clinical findings suggest that there is a mechanical cause (major adhesion, osteophytes, disc rupture) which is preventing a wider mouth opening and that cannot be otherwise addressed. Isolated features alone, such as presence of joint effusion, cannot be considered a sufficient indication for intra-articular lavage at present time (Thomas et al. 2018).

Arthrocentesis can restore roto-translational qualities in early adhesion, because of the forceful separation of joint surfaces due to the injection. It is used for wash-out of inflammatory byproducts and it can be performed together with hyaluronic acid injection for viscosupplementation. Operative arthroscopy is instead carried out under general anesthesia and it allows direct visualization and treatment for more compromised joints (Nitzan et al. 1991; Fridrich et al. 1996; Carvajal and Laskin 2000; Goudot et al. 2000; Nishimura et al. 2001; Alpaslan et al. 2003; Al-Belasy and Dolwick 2007; Monje-Gil et al. 2012; Israel 2016; Nitzan et al. 2017). Again, these two procedures must fit into a programmed treatment plan and must not be looked at as standalone interventions.

Disc repositioning surgeries are not here discussed as they rarely find indication in severely damaged joints, however other arthroscopic procedures such as disc release, retrodiscal cauterization, arthroplasty, laser surgery and discectomies have all been described as viable arthroscopic DJD procedures (Monje-Gil 2014).

Open surgeries with arthroplasty and costochondral grafts have been a workhorse for decades but their role in adult DJD patients is currently decreasing. These techniques may in fact not be suitable in dentate patients who would encounter all kinds of discomforts associated with a probable postoperative malocclusion or constant functional impairment. Fortunately, it is by now a sound knowledge that TMJ alloplastic replacement is a safe, predictable and reliable option and it may spare unfruitful unnecessary surgeries in severely damaged joints (Mercuri et al. 2007; Dela Coleta et al. 2009; Mehra et al. 2009; Gerbino et al. 2017). Besides, end-stage DJD may not benefit from merciful non-prosthetic surgeries which only serve as intermediate options before surrendering to TMJ replacement. This is however an expensive option and patient-specific prostheses are still undergoing medical devices' approval procedures in some countries (Gerbino et al. 2017).

8.2 Inflammatory TMJ Rheumatic Diseases

Inflammatory rheumatic diseases such as Rheumatoid Arthritis (RA), Juvenile Idiopathic Arthritis (JIA) and Spondyloarthritis (SpA) can involve the TMJ. Symptoms however, will be present in only a minority of these patients (Scrivani et al. 2008). The incidence of TMJ involvement in such diseases is often underestimated, and rheumatologists may lack the experience in managing and treating the TMJ in case it is affected (O'Connor et al. 2017). Tenderness and stiffness are frequent in TMJs that are subjected to rheumatologic processes, destruction of the joint surfaces may occur in more severe forms, with deformity and malocclusion as possible outcomes (Gray et al. 1995). Management is mostly non-surgical, but occasionally joint replacement is required.

Data about prevalence of TMJ involvement can be quite striking: a rather high prevalence has in fact been described in around 40–93% of JIAs, and in 45–92% of RA patients (Pantoja et al. 2018). For Psoriatic Arthritis (PsA), prevalence of TMJ involvement has been reported to range from 35 to 80% (Kononen 1987; Dervis and Dervis 2005; Crincoli et al. 2015). In Ankylosing Spondylitis, prevalence varies from 4 to 35% (Arora et al. 2013).

Common upregulation of Matrix Metalloproteinases (MMPs) in Rheumatoid Arthritis (RA), Psoriatic Arthritis (PsA), and other spondyloarthropathies, demonstrates that it is likely that the immunopathogenic mechanisms behind cartilage destruction are shared among inflammatory arthritides (Ribbens 2002).

8.2.1 Rheumatoid Arthritis

Rheumatoid Arthritis (RA) is a chronic autoimmune inflammatory disease of unknown etiology characterized by symmetrical polyarthritis (Smolen et al. 2016).

Epidemiology RA has a prevalence of 0.5–1%, which decreases from North to South (in the Northern Hemisphere), and from urban to rural areas (Silman and Pearson 2002; Alamanos et al. 2006). A positive family history increases the risk of RA by about three to five times. As twins have an increased risk as well, genetic factors are believed to be connected with pathogenesis (Silman et al. 1993).

TMJ involvement in RA is variable, it appears to be quite low in the early stages of disease and it can be bilateral (Atzeni et al. 2015; Sodhi et al. 2015). Natural progression of this disease leads to worsening of overall joint damage, development of disability and increase in mortality (Smolen et al. 2016).

Etiology and Pathogenesis Genome-wide association studies have characterized about 100 loci associated with RA, most of which are related to immune mechanisms and that are also in common with other chronic inflammatory diseases (Roberson and Bowcock 2010; Okada et al. 2014). Moreover, some Human Leukocyte Antigen (HLA) genotypes correlate with more aggressive erosive disease types which also possess higher mortality rates (Gregersen et al. 1987; Klein and Gay 2015).

Anti-Citrullinated Protein Antibody (ACPA) is highly specific to RA and its target is expressed in the synovia. ACPA may be involved in the

pathogenesis of the disease, and it has been shown to be a more specific marker for RA than Rheumatoid Factor (RF), especially for earlydisease subjects (Kuhn et al. 2006; Liao et al. 2008). In early RA, specificity of ACPA ranges from 94 to 100% as opposed to RF, in which specificity ranges from 23 to 96%. Sensitivity of RF and ACPA are equivalent in both early and late-stage forms (Silman 1988; Rantapää-Dahlqvist et al. 2003; Nielen et al. 2005). Since the inclusion of ACPA into the 1987 American College of Rheumatology (ACR) criteria to classify early RA, there has been an increase in earlydetection sensitivity from 25 to 44%, with no change in specificity, which is 86% (Table 8.1) (Nielen et al. 2005; Aletaha et al. 2010).

Clinical Presentation In most cases, RA manifests with painful, swollen, hot and reddened joints, associated with prolonged morning stiffness (lasting longer than 30 min). In most cases,

Table 8.1 2010 ACR/EULAR (European LeagueAgainst Rheumatism) classification criteria for rheumatoid arthritis

Criterion	Score	
Joint involvement		
1 large joint	0	
2–10 large joints	1	
1–3 small joints (with or without involvement of large joints)	2	
4–10 small joints (with or without involvement of large joints)	3	
>10 joints (at least 1 small joint)	5	
Serology (at least 1 test result is needed for classification)		
Negative RF and ACPA	0	
Low positive RF and ACPA	2	
High positive RF and ACPA	3	
Acute phase reactants (at least 1 test result is needed for classification)		
Normal CRP and ESR	0	
Abnormal CRP and ESR	1	
Duration of symptoms		
< 6 weeks	0	
≥ 6 weeks	1	

A score \geq 6/10 is needed for classification of a patient as having definite RA

RF Rheumatoid factor, *ACPA* Anti citrullinated protein antibody, *CRP* C-Reactive Protein, *ESR* Erythrocyte sedimentation rate the disease begins with joint pain, followed by inflammation of the involved joints within weeks or months. More rarely, the onset is acute and sudden, with a rapid appearance of arthritis. The first joints to be involved are usually at the feet and wrists, but larger joints such as shoulders, knees and elbows can also be affected (Nielen et al. 2005).

Joint involvement can be preceded by or associated with systemic manifestations, such as fever, fatigue, weight loss, muscle pain and skin redness. A number of joints is usually affected by the disease, with a symmetrical distribution (*polyarticular* onset, such as hands and feet bilaterally), as opposed to one or few joints as in the less frequent case of mono- or *oligoarticular* onset (Nielen et al. 2005; Aletaha et al. 2010).

If RA is not properly treated or does not respond to treatment, chronic inflammation can lead to cartilage destruction, bone erosion and deformity. In patients with severe RA, other organs may also be affected, with an increased risk of cardiovascular, infective, hematological, gastrointestinal and respiratory complications, leading to an increase in mortality rates by at least two times compared to the general population. Such comorbidities not only do affect the quality of life, but also the efficacy of treatment and prognosis (Smolen et al. 2016).

The TMJ can at times be affected in the early stages of the disease. Clinical signs are pain, morning stiffness and progressive functional impotence. In contrast to other joints affected by RA, the finding of a clinically palpable swelling is rare. Occlusal changes might be reported and they typically include a progressive anterior open bite due to the involvement of both condyles. When present, synovitis shows the same characteristics seen in other joints (Atzeni et al. 2015). Clinical examination reveals a decreased mandibular mobility, a stiff end feel on passive opening and tenderness upon pressure. Only in some cases, a swelling might be found during palpation of the condyle in the preauricular area. More severe consequences, such as mandibular hypoplasia, may occur if mandibular growth is not yet complete like in the case of children (Sodhi et al. 2015).

8.2.2 Spondyloarthritis

The term Spondyloarthritis (SpA) refers to a set of inflammatory conditions with some common characteristics and typical extra-articular manifestations that may occur at first presentation or later. Ankylosing Spondylitis (AS), perhaps the best known among SpAs, is a chronic inflammatory disease affecting the axial skeleton, the entheses, and occasionally the peripheral joints (Ramos-Remus et al. 1997). Psoriatic Arthritis (PsA) is a unique type of inflammatory joint disease that has a peripheral predominance and is associated with cutaneous psoriasis (Fig. 8.12).

8.2.2.1 Ankylosing Spondylitis

Epidemiology The prevalence of AS has been reported from population studies to be between 0.1% in the Netherlands and 1.1-1.4% in Norway (van der Linden et al. 1984; Manemi et al. 2009). The literature reports the male-to-female ratio to range between 2.4:1 and 18:1 (Gran et al. 1985; Locher et al. 1996; Braun et al. 1998). The incidence and prevalence of AS generally mirrors the frequency of HLA-B27 in the population, which explains the virtual absence of AS in Southern Africa, low rates in Japan, higher rates in Norway as compared with other European countries, and very high rates among the native peoples of arctic

and subarctic regions of Eurasia and North America (Gabriel and Michaud 2009).

Etiology and **Pathogenesis** Ankylosing Spondylitis affects both synovial and cartilaginous joints, as well as the attachment sites of tendons and ligaments. The current diagnostic criteria include axial symptoms, limitation of spinal movement and radiological evidence of sacroiliitis (Table 8.2). Immunological activity is

 Table 8.2
 The Assessment of spondyloarthritis interna tional Society (ASAS) classification criteria for axial SpA (in patients with back pain ≥ 3 months and age at onset <45 years)

Sacroiliitis on imaging plus \geq	HLAB27 plus ≥ 2 other
1 SpA feature	SpA features
SpA features:	Sacroiliitis on
 Inflammatory back pain 	imaging:
Arthritis	Active (acute)
• Enthesitis	inflammation on MRI
• Uveitis	highly suggestive of
Dactylitis	sacroiliitis associated
Psoriasis	with SpA or
Crohn's disease/ulcerative	• Definite radiographic
colitis	sacroiliitis according
Good response to NSAIDs	to mod. New York
 Family history for SpA 	criteria
• HLA-B27	
Elevated CRP	

NSAIDs non-steroidal anti-inflammatory drugs, HLA Human leukocyte antigen, CRP C-Reactive Protein, ESR Erythrocyte sedimentation rate



SpA: Spondyloarthritis AS: Ankylosing Spondylitis PsA: Psoriatic Arthritis ReA: Reactive Arthritis IBD-A: Inflammatory Bowel Disease associated Arthritis

suggested by the presence of HLA-B27 in over 90% of patients with AS (Dougados et al. 1991). In the case of TMJ involvement, the most common clinical features are pain and tenderness with limited mouth opening. Suspicion of TMJ arthritic involvement is nevertheless related to radiological findings, such as condylar erosions, in combination with TMJ symptoms (Locher et al. 1996). In addition to axial, entheseal, and appendicular skeletal involvement, AS can also be associated with extra-articular manifestations, especially uveitis and, less commonly, heart and lung disease. AS can occur as a primary disorder or it can complicate other types of SpAs, especially in the subset of Psoriasis and Inflammatory Bowel Disease (IBD) (Dougados et al. 1991). Whether mechanisms contributing to mucosal inflammation in IBD, and potentially in SpA, can also affect musculoskeletal disease in SpA is still to be verified. Although bacterial exposure of joint and musculoskeletal tissues is minimal in comparison with intestine and skin, they can be exposed to great biomechanical stress, which is especially pronounced at the entheses. The pathogenesis of joint inflammation in SpA can be understood from biopsy studies comparing synovial membrane characteristics before and after treatment with anti-Tumor Necrosis Factor (TNF) agents. The number of inflammatory cells, contributing to signs and symptoms of musculoskeletal inflammation, especially macrophages, T cells and neutrophils, decreases after treatment with anti-TNF agents such as etanercept or infliximab (Baeten et al. 2001; Rudwaleit et al. 2001).

Tissue damage and inflammatory response may arise in different ways. The inflammatory response can be mediated by innate immune receptors such as biglycan, a prominent extracellular matrix molecule in tendons. Biglycan is released when tissue is damaged with consequent direct activation of Toll-like Receptor-2 (TLR-2) and TLR-4 (May et al. 2000).

As an alternative, a mechanism for damage could be due to the direct effect that biomechanical stress carries out on structural cells. High biomechanical stress induces nuclear translocation of Nuclear Factor kappa-light-chain-enhancer of activated B cells (NF- κ B) and transcription of a proinflammatory gene (Kruithof et al. 2005). Chondrocytes and tendon fibroblasts then begin to release proinflammatory cytokines, chemokines, and leukocyte growth factors, which, in turn, can attract and activate T and B lymphocytes, neutrophils and macrophages which will be responsible for the inflammatory lesions (Kruithof et al. 2005).

Clinical Presentation The distinctive hallmark of AS is inflammatory back pain associated with radiographic signs of sacroiliitis and often spondylitis. Back pain is an insidious, persistent lower back pain which usually appears before the age of 50 (Gladman 1998; Gran and Husby 1998; Van Der Linden and Van Der Heijde 1998; Sieper et al. 2002; Rudwaleit et al. 2004). Symptoms tend to worsen at night, and morning stiffness is relieved with exercise or movement (Gladman 1998: Rudwaleit et al. 2004). Prompt response to non-steroidal antiinflammatory drugs (NSAIDs) within 48 h is a good predictor of the presence of inflammatory back pain and of axial SpA (Amor et al. 1990; Sieper and Rudwaleit 2005).

Quite common examples of enthesitis in AS are that of the Achilles tendon and of the plantar fascia onto the calcaneus (Gladman 1998; McGonagle et al. 1998; Francois et al. 2001; Rudwaleit et al. 2004).

8.2.2.2 Psoriatic Arthritis

Epidemiology Although the exact prevalence of PsA is unknown, reports indicate it in 7–42% of patients affected by Psoriasis (Gelfand et al. 2005; Gladman et al. 2007). PsA can develop at any age however, in the majority of cases, it appears between age 30 and 50. Unlike other types of inflammatory arthritis that have a typically pronounced female predominance, PsA seems to affect men equally or even at a slightly higher rate (Gladman et al. 2007).

Etiology and Pathogenesis Immunopathogenesis of PsA is still poorly clarified. The discovery of TNF overexpression, of the interleukin (IL-23)/T helper 17 (Th17) axis and of the significance of Receptor Activator of Nuclear Factor κ B (RANK)/RANKL, helped to better understand mechanisms of all spondyloarthritis, PsA included, however, a true autoimmune response has not been yet identified in PsA (Lam et al. 2000; Sato et al. 2006; Kikuta et al. 2013). Moreover, a connection between infections and spondyloarthritis increases the possibility that the microbiome of gut and skin plays a role in skin and joint inflammation (Barnas and Ritchlin 2015).

Clinical Presentation Common features of PsA include dactylitis and enthesitis. PsA can also be accompanied by scalp and nails signs, such as onycholysis (Chandran et al. 2008).

Diagnosis of PsA can be complex because even standard rheumatologic laboratory tests such as RF and ACPA are not specific compared to RA for instance. PsA is mainly established by the presence of characteristic signs and symptoms associated in both skin and joints, although symptoms of PsA can appear before the onset of Psoriasis (*psoriatic arthritis sine psoriasis*), and by the exclusion of other more common inflammatory arthritides.

Classification for Psoriatic Arthritis (CASPAR) criteria are the ones currently used for diagnosis (Table 8.3) (Chandran et al. 2008).

Table 8.3 CASPAR classification criteria for PsA

Criterion	Score
Evidence of psoriasis (one of a, b, c)	
(a) Current psoriasis	2
(b) Personal history of psoriasis	1
(c) Family history of psoriasis	1
Psoriatic nail dystrophy	1
Negative test result for RF	1
Dactylitis (one of a, b)	
(a) Current	1
(b) History recorded by a rheumatologist	1
Radiological evidence of juxta-articular new	1
bone formation	

A score \geq 3 is needed for classification of a patient as having definite PsA

RF Rheumatoid Factor

A careful medical history, physical examination, blood tests, imaging of the involved joints along with a dermatologic evaluation are therefore all used to diagnose PsA.

PsA can affect joints early, with irreversible and progressive joint damage and it can severely affect a patient's quality of life (Congi and Roussou 2010). Typically, PsA presents as a mild, oligoarticular disease but can become polyarticular with time and progress to a severe, erosive condition (Jones et al. 1994).

8.2.3 Juvenile Idiopathic Arthritis

Juvenile Idiopathic Arthritis (JIA) is a childhood inflammatory disease characterized by arthritis of unknown origin with onset before 16 years of age which can be preceded by fever and skin rash (Prakken et al. 2011). Seven subtypes of JIA have been described and they are characterized by distinct clinical and laboratory variables (Table 8.4) (Petty et al. 2004).

Epidemiology JIA has an estimated global incidence between 1.6 and 23 cases per 100,000 children (Prieur et al. 1987; Savolainen et al. 2003). No geographical predominance is known, however, most populations studied are within Europe and North America.

Etiology and Pathogenesis Etiopathogenesis of the disease is still unclear. The most accepted theory supports the influence of immunogenic mechanisms secondary to genetic and environmental factors (Barut et al. 2017). According to recent studies, gut microbiome is also emerging as a contributing element for autoimmune diseases, JIA included (Verwoerd et al. 2016). The increased frequency of autoimmune diseases among JIA patients suggests a genetic basis, besides, HLA-B27 is also associated (Prahalad et al. 2002; Ferucci et al. 2005; Aggarwal and Misra 2015; Adrovic et al. 2016; Zhou et al. 2016). Infections with stress and trauma, are considered to be among the key determinants: enteric infections, rubella virus, parvovirus B19, mumps virus, Epstein-Barr virus, hepatitis

General Criteria		
Arthritis in ≥ 1 joints (swelling or effusion or the presence of 2 or more of the following signs: limitation of range of		
motion, pain or ter	nderness on motion, increased heat)	
Age of onset—before 16 years		
Duration ≥6 weeks		
Exclusion of other forms of juvenile arthritis		
Category	Definition	
Systemic	Arthritis in one or more joints with or preceded by fever of at least 2 weeks duration	
arthritis	documented to be daily for at least 3 days, and accompanied by one or more of:	
	1. evanescent erythematous rash	
	2. lymphadenopathy	
	3. hepatomegaly and/or splenomegaly	
	4. serositis	
Oligoarthritis	Arthritis in one to four joints in the first 6 months.	
(persistent or	Persistent: Arthritis affecting no more than four joints throughout the disease course	
extended)	• Extended: Arthritis affecting a total of more than four joints after the first 6 months of disease	
Polyarthritis	Arthritis affecting more than four joints during the first 6 months of disease; tests for RF are	
(RF negative)	negative	
Polyarthritis	Arthritis affecting more than four joints during the first 6 months of disease; tests for RF are	
(RF positive)	positive (on at least two occasions more than 3 months apart)	
Psoriatic	Arthritis and psoriasis, or arthritis and at least two of:	
arthritis	1. Dactylitis	
	2. Nail abnormalities (pitting or onycholysis)	
	3. Psoriasis in a first-degree relative	
Enthesitis	Enthesitis with at least two of:	
related arthritis	1. Presence or a history of sacroiliac joint tenderness and/or inflammatory lumbosacral pain	
	2. Presence of HLA-B27	
	3. Onset of arthritis in a male older than 6 years of age	
	4. Acute (symptomatic) anterior uveitis	
	5. History of ankylosing spondylitis, enthesitis-related arthritis, sacroiliitis with inflammatory	
	bowel disease, Reiter's syndrome, or acute anterior uveitis in a first-degree relative	
Undifferentiated arthritis	Arthritis that fulfills criteria in no category or in two or more of the above categories	

Table 8.4 International league of associations for rheumatology (ILAR) classification for juvenile idiopathic arthritis

RF rheumatoid factor, HLA Human leukocyte antigen

B, mycoplasma and chlamydia have been linked to JIA (Weiss and Ilowite 2005; Gonzalez et al. 2007; Hinks et al. 2017).

T-lymphocytes cytokines and secreted are responsible for joint destruction in JIA. Macrophages produce pro-inflammatory cytokines such as IL-1, IL-6 and TNF- α . Thus, typical acute phase markers such as C-Reactive Protein (CRP) and Erythrocyte Sedimentation Rate (ESR), usually increase during acute inflammation of joints. Synovial inflammation (synovitis) is characterized by villous hypertrophy and hyperemia of the synovial tissue. The T-lymphocyte percentage in synovial fluids can be different among various JIA subtypes, possibly explaining the difference that can be observed in treatment response among the different JIA subgroups (Prakken et al. 2011; Zhou et al. 2016).

Clinical Presentation Approximately 50% of children with systemic JIA develop polyarthritis within 3–12 months from the onset of the fever. Wrists, knees, and ankles are most commonly involved, with the cervical spine, temporomandibular joints, hands and hips also being affected. Cricoarytenoid arthritis may result in hoarseness and laryngeal stricture (Lomater et al. 2000).

Oligoarticular JIA occurs more frequently in girls, ANA positivity is present in 40–85% of children with this onset, and this form is widely

associated with chronic anterior uveitis. As uveitis is often asymptomatic, it is essential for an ophthalmologist to perform a routine eye examination (Skarin et al. 2009).

TMJ involvement is often undiagnosed because small children may have difficulties in describing subjective symptoms from the area and deviations/deflections and TMJ sounds may be unnoticed by pediatricians (Twilt et al. 2004; Stoll et al. 2015). Mandibular hypoplasia due to inflammation and destruction may be a possible outcome of JIA.

As a consequence of the above-described TMJ involvement, micro/retrognathia is reported to develop in 30% of affected children, with consequent malocclusion and associated facial changes (Karhulahti et al. 1993; Pedersen et al. 2001; Ringold and Cron 2009). Adequate evaluation by an orthodontist or pedodontist of all children affected by JIA is highly recommended for early diagnosis and treatment planning (Stoll et al. 2015).

8.2.4 Imaging

For most inflammatory TMJ rheumatic diseases, especially in the case of JIA, clinical examination and its findings are insufficient for early detection of joint inflammation (Weiss et al. 2008; Keller et al. 2015). Up to 70% of JIA patients with active early TMJ involvement are in fact symptom-free (Weiss et al. 2008). The opposite situation may also happen, as when rheumatic patients complain about TMJ pain due to reasons other than their rheumatic disease. With this respect, erroneous diagnosis of rheumatic TMJ involvement may lead to overdiagnosis and overtreatment, and this explains why imaging is crucial (Koos et al. 2014).

Early features in case of TMJ involvement commonly include synovial alterations whereas bone is found to be affected at a later stage, even after years, as shown on orthopantomograms, plain film radiography and CT (Navallas et al. 2017).

Although orthopantomograms are not used routinely for diagnosis, they are helpful in high-

lighting mandibular asymmetries which normally follow unilateral condylar resorption (Fig. 8.13a). Some peculiar condylar deformity features, such as JIA's flat condyle, can be shown with this technique, albeit belatedly (Navallas et al. 2017). Plain film radiography and tomography (Fig. 8.13b, c) can also detect erosive patterns of condyles, but later than CT would. Because of its high spatial resolution, CT has the power to outline bone alterations which are typically found in inflammatory TMJ rheumatic diseases (osteoporosis, RA erosions, PsA syndesmophytes), but since these are bony changes, CT diagnosis is a late one (Navallas et al. 2017).

Ultrasonography (US) is a simple and straightforward technique which can be deployed in early inflammatory TMJ rheumatic diseases because it shows joint effusion, synovitis and rough bone erosions, which still are present only at a later stage (Weiss et al. 2008; Navallas et al. 2017) (Fig. 8.14).

Unlike the previous imaging techniques, contrast MRI can visualize bone marrow and define the extent of the synovial pannus. Joint erosions and lesions of capsule, disc and ligaments can be detected and are highly correlated with arthroscopic findings (Miller et al. 2018; Leschied et al. 2019).

Early arthritis diagnosis is of uttermost significance especially in the case of JIA. Vertical growth depends on endochondral ossification of condylar subchondral bone and, because inflammatory arthritis affects subchondral bone first, undiagnosed and unaddressed TMJ JIA may result in mandibular growth anomalies and undesirable facial features (Meyers and Laor 2013; Vaid et al. 2014; Navallas et al. 2017; Tolend et al. 2018).

In a 2014 review, Munir explained that there is fair evidence to recommend TMJ MRI for the purpose of JIA early diagnosis. The same cannot be said for axial joints such as the spine and sacroiliac joints as there is not enough evidence in the literature yet (Munir et al. 2014). In the aforementioned review, signs to evaluate for early diagnosis were presence of synovial pannus, joint effusion and synovial contrast-enhancement.



Fig. 8.13 RA—orthopantomogram (**a**) shows massive condylar deformity, on the right side in particular, with joint surface erosion (*arrows*). RA—closed-mouth (**b**) and open-mouth (**c**) tomography images show condylar erosion



Fig. 8.14 US shows joint effusion and synovitis (white arrow)

Common early arthritis signs are therefore (Fig. 8.15a–d):

- bone marrow edema
- bone marrow contrast-enhancement in T1-weighted fat-suppressed images
- joint effusion (mild >1 mm, severe >2 mm)
- synovial thickening (mild >1 mm, severe >2 mm)
- · marked synovial contrast-enhancement

These features have been used to define scoring systems aimed at early detection of JIA (Meyers and Laor 2013; Vaid et al. 2014; Tolend et al. 2018).



Fig. 8.15 Early RA—bone marrow edema (**a**) and mild synovitis (**b**). Examination 1 year later shows edema (**c** *arrow*) and synovitis worsening, which are now associated with joint effusion (**d**)

Joint effusion in MRI has been addressed previously in Chap. 8. It is defined by signal hyperintensity in fat-suppression and in long TR sequences (T2 and PD), and by hypointensity in T1-weighted sequences. Bone marrow edema is instead a non-specific sign, and it is usually found in remodeled joints such as in DJD and trauma.

Synovitis is exceptionally pronounced in RA and JIA, where it creates a pannus with membrane thickening (<1–2 mm) and jutting into joint spaces. Its signal characteristics change according to time from onset. In recent synovitis,

hyperintensity is similar to that of fluids in long TR sequences, chronic synovitis is hypointense instead (Navallas et al. 2017).

Moreover, synovitis shows quite early contrastenhancement, because of synovial high vascularity and vessels permeability, that is particularly persistent at 7–10 min from intravenous injection (Caruso et al. 2017). Some authors attempted at quantifying contrast-enhancement and allocating threshold values for the ratio between synovial enhancement and adjacent muscle enhancement. These reports are difficult to translate to common MRI settings and have to be looked at wisely because of the issues concerning gadolinium deposition in the brain (Peacock et al. 2016; Resnick et al. 2016; Caruso et al. 2017; Elbeshlawi and AbdelBaki 2018).

Synovitis seems to be present in nonrheumatological conditions too, such as disc dislocation and osteoarthritis, although there is not much debate over this. Signs of inflammation like joint effusion, bone marrow edema and synovial contrast enhancement, are quantitatively and qualitatively comparable to those observed in disc dislocation cases, however there are actually few studies that correlate MRI findings of inflammatory arthritis with the ones of non-rheumatological conditions (Fig. 8.16a, b) (Farina et al. 2009; Kellenberger et al. 2015, 2018a, b, 2019).

This lack of specificity can lead to dilemmas in differential diagnosis, for instance Oligoarticular JIA affecting the knee may present with synovial features due to a discal dislocation rather than TMJ JIA (Kellenberger et al. 2015). Besides, both aspects can coexist as JIA discs are thin and dislocated, at times with perforations and fragmentations due to the aggressive action of the synovial pannus (Kellenberger et al. 2018b). MRI signs of effusion and, to a lesser extent synovial hyperplasia, can also be detected in the general population (up to 10% of the pediatric population), in pain-free subjects with no history of rheumatologic disease (Tzaribachev et al. 2009; Stoll et al. 2018). Ultimately, rheumatological forms can only be distinguished because of later typical bone alterations.

Imaging features become more specific as diseases evolve (and are of course easier to diagnose), and inflammation spreads beyond synovia, disc and capsule, to the bone.

RA typically shows osteoporosis and erosions, the latter being present in more than 80% of TMJ RA patients. Later-stage RA has a decreased condylar volume (pencil head defor*mation*), with bone edema whose marrow does not show fibrosclerotic involution (Fig. 8.17ad). Less than 10% of RA subjects shows erosions of the tuberculum and the glenoid fossa. Disc instability with subsequent dislocation is present in about 95% of patients, and it is due to early formation of synovial pannus that affects up to 85% of patients and which then causes capsulo-ligamentous alterations. In chronically inflamed joints, the disc is often small and ruptured (Fig. 8.18a-c). Quite commonly, reactive enlarged mandibular angle lymph nodes are detected (Hirahara et al. 2017).



Fig. 8.16 Asymptomatic patient with no signs of inflammatory arthropathy showed anterior disc dislocation (**a**), and synovitis (**b** *arrows*) which appears like hypointense spots in T2 weighted-sequence



Fig. 8.17 Late-stage RA with no signs of ongoing disease activity—Sagittal Oblique PD (**a**), STIR (**b**), axial view (**c**). The right condyle is small, with central erosive

Apart from condylar alterations, advanced JIA is often associated with structural changes of the ramus. Inflammation is responsible for affecting the subchondral bone, which is where endochondral ossification takes place, and it so influences the vertical height of the ramus (Fig. 8.19a, b). Typical signs for JIA are:

 first partial and then complete flattening of the condylar surface, which shows a 'truncated' appearance, with a flat broad end;

lacunae (*arrow*) and no effusion is visible. T2 coronal view (**d**) shows that the condyle has lost its physiological shape

- shortening of the condylar process and of the ramus with mandibular angle opening and a pronounced antegonial notch;
- faint condylar erosions;
- degenerative discal changes, usually displacement.

These signs are useful for diagnosis using a scoring system (not in early forms), and for overall JIA staging (Vaid et al. 2014; Tolend et al. 2018). Assessment of ramus and condylar



Fig. 8.18 Late-stage RA with signs of ongoing disease activity—closed mouth Sagittal Oblique STIR (**a**) shows a small, eroded condyle with intense bone marrow edema (*star*). The articular eminence also shows erosions with

structural changes in the growing patient can be troublesome due to the constantly changing morphology of the mandible. In children, the articular eminence is quite flat and the fossa is wide. As growth continues, the eminence gets steeper and the fossa becomes deeper. Threedimensionally, the condyle is round at first, and then it gradually acquires a flatter outline (in the coronal view) and a larger transversal diameter (in the axial plane). The short ramus and neck are

bone edema (*arrow*). Open-mouth images (**b**) show joint effusion and anterior disc dislocation (*arrow*) with ruptured retrodiscal laminae (*star*). Axial (**c**) image shows a left eroded condyle

straight at the beginning, and then they grow into the typical vertically-developed, angled appearance of the adult mandible (Fig. 8.20) (Karlo et al. 2010; Angenete et al. 2018; Kellenberger et al. 2018a). Bone marrow MR intensity changes during development as children have a prevalent hematopoietic bone marrow which is mildly hypointense in T1 weighted sequences, with a chance of mottled areas, and hyperintense in fatsuppression sequences.



Fig. 8.19 Late-stage JIA—closed-mouth Sagittal PD (a) displays a short condylar neck and a flat condyle. Sagittal STIR (b) shows joint effusion (*arrow*), with synovitis

(*star*), and an enlarged lymph node is visible at the mandibular angle (*arrow*)



Fig. 8.20 (a-d) Ramus, condylar neck and head changes during growth, from a mixed-dentition stage to adulthood

Yellow marrow replaces red marrow as the subject matures, and this gives off a typical hyperintensity of cancellous bone in T1 and T2 with full signal saturation in fat-suppressed sequences.

MRI signs of effusion and synovitis are seen in SpA too, however AS and PsA display a highly vascular but less hyperplastic synovia. Pannus thickness is finer than in RA and JIA, however it is not possible to make a distinction between SpA and RA/JIA based solely on signal features of synovitis (Lee et al. 2008; Skármeta et al. 2018).

Bone marrow edema is often visible in SpA, however in the case of PsA it is uneven and it involves extra-articular entheses, especially in children (JPsA) (Lee et al. 2008).

Erosions are typically detectable, however they show less aggressiveness than those of RA and JIA. Subchondral bone reaction in SpA includes osteoclastic activation as well as new bone formation, yet it is rare to observe TMJ enthesophytes/syndesmophytes. Both CT and MRI are therefore not specific enough to differentiate among the diverse forms of inflammatory TMJ rheumatic diseases (Atsü and Ayhan-Ardic 2006; Aliko et al. 2011; Skarmeta 2018).

8.2.5 Management

The therapeutic spectrum available to the rheumatologist for the treatment of inflammatory rheumatism has grown enormously during the last decade.

Glucocorticoids and NSAIDs still play an important role in the early stages but are being prescribed less and at lower doses because of long-term adverse effects. The role of conventional Disease Modifying Anti Rheumatic Drugs (DMARD) as drugs which can affect the history of the disease, is still valid and recognized. This is especially true for Methotrexate which is widely used for RA and peripheral PsA. Biologic DMARDs such as anti-TNF, anti-IL6 and anti-CTLA4, and target synthetic DMARDs such as Janus Kinase Inhibitors, are showing promising results in long-term disease control and prevention of joint damage. The most recent literature has shown that early and aggressive therapy is able to limit enormously and in some cases to stop the disease therefore, the most advanced therapies may become indicated already 3 months after the failure of conventional therapy (Emery and Salmon 1995; Smolen et al. 2007; Nam et al. 2010: Daïen et al. 2017).

Intra-articular steroids or lavage plus hyaluronic acid injection may be chosen to improve joint symptoms (in association with DMARDs) especially in patients with early arthritis (Stoustrup et al. 2013; Olsen-Bergem and Bjørnland 2014; Daïen et al.2017).

More severe cases may require surgical treatment and, when anatomy and function are lost, TMJ prosthesis may be necessary (Nørholt et al. 2015). It is always useful to combine physical therapies; there are various forms of treatment, all with the aim of achieving functional recovery. Prompt diagnosis and early treatment can alleviate both pain and inflammation, and help to prevent progressive joint involvement and damage (Congi and Roussou 2010).

8.3 Idiopathic Condylar Resorption and other Joint Diseases

Idiophatic Condylar Resorption (ICR) is a condition defined by a severe and progressive resorption of condylar heads. During the past years various names have been used for this inexplicable resorption (idiopathic condylysis, condylar atrophy, aggressive condylar resorption, cheerleader syndrome), all of which aimed at identifying an aggressive, irreversible low-inflammatory condition mostly diagnosed in young females and not related to other causes, most notably rheumatic diseases (Wolford and Cardenas 1999 and Wolford 2001; Sansare et al. 2015).

8.3.1 Epidemiology

ICR is a rare condition. Females are more affected than males (ratio 9:1) and the usual onset is at around 20 years of age (Arnett et al. 1996a).

8.3.2 Etiology and Pathogenesis

Theories about ICR causes can only partially explain this condition. Hormonal imbalances in adolescent girls are thought to play a role because of estrogen receptors that are present in the TMJ (Abubaker et al. 1996; Wolford 2001). Orthodontics-induced changes in occlusion and orthognathic surgery (particularly in the case of counter clockwise rotations of the maxillomandibular complex) are also believed to affect TMJ load and lead to unexpected condylar resorption (Arnett et al. 1996a,b; Mehra et al. 2016). Some skeletal features seem to be associated with ICR: skeletal class II with steep mandibular plane, anterior open bite and posteriorly inclined condyles (Wolford et al. 1993; Hwang et al. 2004; Troulis et al. 2008; Mehra et al. 2016).

8.3.3 Clinical Presentations

Patients typically present with anterior open bite, skeletal class II, steep lower occlusal plane, mandibular post-rotation and loss of posterior facial height. This however only suggests that the set of characteristics that have been identified as associated factors are perhaps an effect of ICR rather than its cause. Occlusal wear may be detected mostly posteriorly, or it could also be present on more mesial teeth that are no longer in contact. Joint noises are often described and facial deformity is more visible in case of severe, bilateral ICR (Wolford and Cardenas 1999; Wolford 2001; Mercuri 2008; Young 2017).

8.3.4 Imaging

Idiopathic Condylar Resorption requires a careful clinical assessment due to the fact that radiological features of this condition are shared to a certain extent by a number of other low- and high-inflammatory disorders (osteoarthritic phase of DJD and RA/JIA respectively) (Mercuri 2008; Sansare et al. 2013; Sansare et al. 2015; Kristensen et al. 2017). The peculiar sign of ICR is an erosive process that is common to the aforementioned forms, accompanied by areas of bone resorption (Kristensen et al. 2017). Resorption here starts antero-superiorly and then spreads to the whole condyle, thus inducing complete resorption (condylysis). Resorption outweighs remodeling and this distinguishes IRC from purely degenerative processes which normally have flattened out condyles in load-bearing areas. ICR arises toward the end of mandibular growth and it can also be unilateral, unlike JIA for instance. As the rapidly progressing condylar bone resorption phase burns out, the inflammatory process comes to a halt. The outcome is a typical pencil-shaped condyle in all three dimensions.

Again, ICR lacks pathognomic imaging signs and it is mandatory to rule out secondary resorptions due to trauma, previous cranio-maxillofacial surgical procedures and congenital forms (Papadaki et al. 2007; Mitsimponas et al. 2018). Outstanding resorption and bone edema will involve the whole condyle quite rapidly. The condylar surface will become irregular and an overall shortening will occur (Fig. 8.21a–g). Effusion is usually limited and presence of synovitis can be variable and surely more limited than that of JIA (Wolford 2001; Chouinard et al. 2018).

Erosion is rapidly progressive but it might just as easily shift toward remission. In case osteoarthritis phenomena burn out, edema will also fade and the condylar contour can be restored, with an overall smaller condylar head (Posnick and Fantuzzo 2007). The affected condyle will show reduced height and width however its thickness is like that of degenerative remodeling (Kristensen et al. 2017). Similarly to JIA, a posterior inclination of the ramus and condylar neck may be visible, as well as a marked antegonial notch (Papadaki et al. 2007; Kristensen et al. 2017; Kajii et al. 2018). After the inflammatory process has stopped the condyle may look smaller but with some reestablishment of joint morphology, whereas in JIA, condyles-which are not the only localization of disease-will maintain their 'truncated' appearance (Chouinard et al. 2018).

8.3.5 Management

Treatment of ICR depends on the extent of the joint damage and on the associated facial deformity (Mehra et al. 2016). The resorption status has to be known before irreversible procedures are carried out. Once the process has ceased, treatment will address the probable outcomes which may be various degrees of TMJ impairment, malocclusion and facial deformity (Young 2017). Treatment options might therefore encompass: orthodontic treatment only, orthognathic surgery and a combination of orthognathic surgery and TMJ prosthetic replacement (Mercuri 2007; Papadaki et al. 2007; Wolford et al. 2015; Wolford and Gonçalves 2015). Esthetic concerns may also be addressed by means of ancillary procedures such as genioplasty.

Customized TMJ prostheses have precise indications and they may be used in association with orthognathic surgery. Computer-planned



Fig. 8.21 15-year-old patient with ICR—TMJ pain since 4 years in the medical history. Dixon PD images (a, b), coronal SE T2 (c) and axial SE T1 (d) show condylolysis with a reduction in size and bone sclerosis. Effusion and

synovitis cannot be detected. Orthopantomogram (e) displays condylolysis too. The same condyle already showed a decreased size (f) with respect to the contralateral (g) 3 years earlier



Fig. 8.21 (continued)

prosthetic joint replacement can match the patient's final anatomy and fit in the virtually programmed counterclockwise rotation of the maxillomandibular complex. This allows to improve the esthetic outcome by restoring severely decreased posterior facial height, to reestablish function and occlusion and to move the chin and mandible forward. This integrated surgical approach shows a predictable and stable result (Mehra et al. 2016; Gerbino et al. 2017).

8.3.6 Other Joint Diseases

Osteochondritis Dissecans Osteochondritis Dissecans (OCD) is a condition which rarely involves the TMJ and is more commonly found in young patients' knee (Olley and Leopard 1978; Carls et al. 1995; Campos et al. 2005; Orhan et al. 2006; Misirlioglu et al. 2014; Smolka et al. 2016). Diagnosis is possible with imaging, as clinical signs and symptoms (pain, sounds and limited mandibular movements) are not disease-specific.

The condyle shows resorption, surface erosions and is surrounded by a limited number of loose bodies (usually one or two) that are visible on traditional x-rays and CT scan. Loose bodies detach from the articular surface and they are made of cartilage and subchondral bone. MRI may detect the loose bodies as well as associated signs of Disc Disorders and help in differential diagnosis with Synovial Chondromatosis.

The management of OCD mostly depends on the number and location of loose bodies as well as presence of functional impairment. Treatments option ranges from conservative modalities aimed at controlling pain, to removal and histopathologic examination of loose bodies through arthroscopy or open surgery (Olley and Leopard 1978; Schellhas et al. 1989; Carls et al. 1995; Orhan et al. 2006).

References

- Abubaker AO, Hebda PC, Gunsolley JN. Effects of sex hormones on protein and collagen content of the temporomandibular joint disc of the rat. Oral Maxillofac Surg. 1996;54:721–7. https://doi.org/10.1016/ S0278-2391(96)90690-4.
- Adrovic A, Barut K, Sahin S, Kasapcopur O. Juvenile Spondyloarthropathies. Curr Rheumatol Rep. 2016;18:55. https://doi.org/10.1007/ s11926-016-0603-y.
- Aggarwal A, Misra DP. Enthesitis-related arthritis. Clin Rheumatol. 2015;34:1839–46. https://doi. org/10.1007/s10067-015-3029-4.
- Ahmad M, Hollender L, Anderson Q, Kartha K, Ohrbach R, Truelove EL, et al. Research diagnostic criteria for temporomandibular disorders (RDC/TMD): development of image analysis criteria and examiner reliability for image analysis. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2009;107:844–60. https://doi.org/10.1016/j.tripleo.2009.02.023.
- Alamanos Y, Voulgari PV, Drosos AA. Incidence and prevalence of rheumatoid arthritis, based on the 1987 American College of Rheumatology criteria: a systematic review. Semin Arthritis Rheum. 2006;36:182–8. https://doi.org/10.1016/j. semarthrit.2006.08.006.
- Aletaha D, Neogi T, Silman AJ, Funovits J, Felson DT, Bingham CO 3rd, et al. 2010 rheumatoid arthritis classification criteria: an American College of Rheumatology/European League Against Rheumatism collaborative initiative. Ann Rheum Dis. 2010;69:1580–8. https://doi.org/10.1136/ ard.2010.138461.
- Aliko A, Ciancaglini R, Alushi A, Tafaj A, Ruci D. Temporomandibular joint involvement in rheumatoid arthritis, systemic lupus erythematosus and systemic sclerosis. Int J Oral Maxillofac Surg. 2011;40:704–9. https://doi.org/10.1016/j. ijom.2011.02.026.
- Alkhader M, Ohbayashi N, Tetsumura A, Nakamura S, Okochi K, Momin MA, et al. Diagnostic performance of magnetic resonance imaging for detecting osseous abnormalities of the temporomandibular joint and its correlation with cone beam computed tomography. Dentomaxillofac Radiol. 2010;39:270–6. https://doi. org/10.1259/dmfr/25151578.
- Alpaslan C, Dolwick MF, Heft MW. Five-year retrospective evaluation of temporomandibular joint arthrocentesis. Int J Oral Maxillofac Surg. 2003;32:263–7. https://doi.org/10.1054/ijom.2003.0371.
- Al-Belasy FA, Dolwick MF. Arthrocentesis for the treatment of temporomandibular joint closed lock: a review article. Int J Oral Maxillofac Surg. 2007;36:773–82. https://doi.org/10.1016/j.ijom.2007.04.005.
- Amor B, Dougados M, Mijiyawa M. Criteria of the classification of spondylarthropathies. Rev Rhum Mal Osteoartic. 1990;57:85–9.
- Angenete OW, Augdal TA, Jellestad S, Rygg M, Rosendahl K. Normal magnetic resonance appearances of the temporomandibular joints in children and young adults aged 2–18 years. Pediatr Radiol. 2018;48:341– 9. https://doi.org/10.1007/s00247-017-4048-x.
- Arnett GW, Milam SB, Gottesman L. Progressive mandibular retrusion—idiopathic condylar resorption. Part I. Am J Orthod Dentofac Orthop. 1996a;110:8–15. https://doi.org/10.1016/S0889-5406(96)70081-1.
- Arnett GW, Milam SB, Gottesman L. Progressive mandibular retrusion—idiopathic condylar resorption. Part II. Am J Orthod Dentofac Orthop. 1996b;110:117–27. https://doi.org/10.1016/ S0889-5406(96)70099-9.
- Arora P, Amarnath J, Ravindra SV, Rallan M. Temporomandibular joint involvement in ankylosing spondylitis. BMJ Case Rep. 2013; 2013:bcr2013009386. https://doi.org/10.1136/ bcr-2013-009386.
- Atsü SS, Ayhan-Ardic F. Temporomandibular disorders seen in rheumatology practices: a review. Rheumatol Int. 2006;26:781–7. https://doi.org/10.1007/ s00296-006-0110-y.
- Atzeni F, Govoni M, Salaffi F, Sarzi PP. Semeiotica Reumatologica. Parte II - Colonna Vertebrale e Articolazione Temporo-Mandibolare. 2015 ed. Fidenza, Parma: Mattioli; 2015. p. 26–7.
- Baeten D, Van Damme N, Van den Bosch F, Kruithof E, De Vos M, Mielants H, et al. Impaired Th1 cytokine production in spondyloarthropathy is restored by anti-TNFalpha. Ann Rheum Dis. 2001;60:750–5. https:// doi.org/10.1136/ard.60.8.750.
- Bansal M. Prevalence and diagnostic features of osteoarthrosis of the temporomandibular joint: a review. Int J Res Orthop. 2016;2:1–4. https://doi.org/10.18203/ issn.2455-4510.IntJResOrthop20160708.
- Barnas JL, Ritchlin CT. Etiology and pathogenesis of psoriatic arthritis. Rheum Dis Clin N Am. 2015;41:643– 63. https://doi.org/10.1016/j.rdc.2015.07.006.
- Barut K, Adrovic A, Şahin S, Kasapçopur Ö. Juvenile idiopathic arthritis. Balkan Med J. 2017;34:90–101. https://doi.org/10.4274/balkanmedj.2017.0111.

- Bernhardt O, Biffar R, Kocher T, Meyer G. Prevalence and clinical signs of degenerative temporomandibular joint changes validated by magnetic resonance imaging in a non-patient group. Ann Anat. 2007;189:342–6. https:// doi.org/10.1016/j.aanat.2007.02.008.
- Braun J, Bollow M, Remlinger G, Eggens U, Rudwaleit M, Distler A, et al. Prevalence of spondylarthropathies in HLA-B27 positive and negative blood donors. Arthritis Rheum. 1998;41:58–67. https:// doi.org/10.1002/1529-0131(199801)41:1<58::AID-ART8>3.0.CO;2-G.
- Campos PS, Freitas CE, Pena N, Gonzalez MO, Almeida SM, Mariz AC, et al. Osteochondritis dissecans of the temporomandibular joint. Dentomaxillofac Radiol. 2005;34:193–7. https://doi.org/10.1259/ dmfr/59267138.
- Campos PS, Macedo Sobrinho JB, Crusoé-Rebello IM, Pena N, Dantas JA, Mariz AC, et al. Temporomandibular joint disc adhesion without mouth-opening limitation. J Oral Maxillofac Surg. 2008;66:551–4. https://doi.org/10.1016/j. joms.2006.11.006.
- Carls FR, von Hochstetter A, Engelke W, Sailer HF. Loose bodies in the temporomandibular joint. The advantages of arthroscopy. J Craniomaxillofac Surg. 1995;23:215–21. https://doi.org/10.1016/ S1010-5182(05)80210-0.
- Caruso P, Buch K, Rincon S, Hakimelahi R, Peacock ZS, Resnick CM, et al. Optimization of quantitative dynamic postgadolinium MRI technique using normalized ratios for the evaluation of temporomandibular joint synovitis in patients with juvenile idiopathic arthritis. AJNR Am J Neuroradiol. 2017;38:2344–50. https://doi.org/10.3174/ajnr.A5424.
- Carvajal WA, Laskin DM. Long-term evaluation of arthrocentesis for the treatment of internal derangements of the temporomandibular joint. J Oral Maxillofac Surg. 2000;58:852–5. https://doi.org/10.1053/ joms.2000.8201.
- Catherine Z, Breton P, Bouletreau P. Condylar resorption after orthognathic surgery: a systematic review. Rev Stomatol Chir Maxillofac Chir Orale. 2016;117:3–10. https://doi.org/10.1016/j.revsto.2015.11.002.
- Chandran V, Schentag CT, Gladman DD. Sensitivity and specificity of the CASPAR criteria for psoriatic arthritis in a family medicine clinic setting. J Rheumatol. 2008;35:2069–70.
- Chen K, Man C, Zhang B, Hu J, Zhu SS. Effect of in vitro chondrogenic differentiation of autologous mesenchymal stem cells on cartilage and subchondral cancellous bone repair in osteoarthritis of temporomandibular joint. Int J Oral Maxillofac Surg. 2013;42:240–8. https://doi.org/10.1016/j.ijom.2012.05.030.
- Chiba M, Kumagai M, Fukui N, Echigo S. The relationship of bone marrow edema pattern in the mandibular condyle with joint pain in patients with temporomandibular joint disorders: longitudinal study with MR imaging. Int J Oral Maxillofac Surg. 2006;35:55–9. https://doi.org/10.1016/j. ijom.2005.04.020.

- Chisnoiu AM, Picos AM, Popa S, Chisnoiu PD, Lascu L, Picos A, et al. Factors involved in the etiology of temporomandibular disorders - a literature review. Clujul Med. 2015;88:473–8. https://doi.org/10.15386/ cjmed-485.
- Chouinard AF, Kaban LB, Peacock ZS. Acquired abnormalities of the temporomandibular joint. Oral Maxillofac Surg Clin North Am. 2018;30:83–96. https://doi.org/10.1016/j.coms.2017.08.005.
- Congi L, Roussou E. Clinical application of the CASPAR criteria for psoriatic arthritis compared to other existing criteria. Clin Exp Rheumatol. 2010;28:304–10.
- Cortés D, Exss E, Marholz C, Millas R, Moncada G. Association between disk position and degenerative bone changes of the temporomandibular joints: an imaging study in subjects with TMD. Cranio. 2011;29:117– 26. https://doi.org/10.1179/crn.2011.020.
- Crincoli V, Di Comite M, Di Bisceglie MB, Fatone L, Favia G. Temporomandibular disorders in psoriasis patients with and without psoriatic arthritis: an observational study. Int J Med Sci. 2015;12:341–8. https:// doi.org/10.7150/ijms.11288.
- Daïen CI, Hua C, Combe B, Landewe R. Pharmacologic and non-pharmacologic therapies in early arthritis: results of a systematic literature informing the 2016 update of the EULAR recommendations for the management of early arthritis. RMD Open. 2017;3:e000404. https://doi.org/10.1136/ rmdopen-2016-000404.
- Das SK. TMJ osteoarthritis and early diagnosis. J Oral Biol Craniofac Res. 2013;3:109–10. https://doi. org/10.1016/j.jobcr.2013.10.003.
- Dela Coleta KE, Wolford LM, Goncalves JR, Pinto Ados S, Pinto LP, Cassano DS. Maxillo-mandibular counter-clockwise rotation and mandibular advancement with TMJ concepts total joint prostheses: part I—skeletal and dental stability. Int J Oral Maxillofac Surg. 2009;38:126–38. https://doi.org/10.1016/j. ijom.2008.11.024.
- de Melo DP, Sousa Melo SL, de Andrade Freitas Oliveira LS, Ramos-Perez FM, Campos PS. Evaluation of temporomandibular joint disk displacement and its correlation with pain and osseous abnormalities in symptomatic young patients with magnetic resonance imaging. Oral Surg Oral Med Oral Pathol Oral Radiol. 2015;119:107–12. https://doi.org/10.1016/j. 0000.2014.09.022.
- Dervis E, Dervis E. The prevalence of temporomandibular disorders in patients with psoriasis with or without psoriatic arthritis. J Oral Rehabil. 2005;32:786–93. doi.org/10.1111/j.1365-2842.2005.01521.x
- Dias IM, Coelho PR, Picorelli Assis NM, Pereira Leite FP, Devito KL. Evaluation of the correlation between disc displacements and degenerative bone changes of the temporomandibular joint by means of magnetic resonance images. Int J Oral Maxillofac Surg. 2012;41:1051–7. https://doi.org/10.1016/j. ijom.2012.03.005.
- Dias IM, Cordeiro PC, Devito KL, Tavares ML, Leite IC, Tesch RS. Evaluation of temporomandibular joint disc

displacement as a risk factor for osteoarthrosis. Int J Oral Maxillofac Surg. 2016;45:313–7. https://doi. org/10.1016/j.ijom.2015.09.016.

- Dimitroulis G. The prevalence of osteoarthrosis in cases of advanced internal derangement of the temporomandibular joint: a clinical, surgical and histological study. Int J Oral Maxillofac Surg. 2005;34:345–9. https://doi. org/10.1016/j.ijom.2004.10.013.
- Dougados M, van der Linden S, Juhlin R, Huitfeldt B, Amor B, Calin A, et al. The European Spondylarthropathy study group preliminary criteria for the classification of spondylarthropathy. Arthritis Rheum. 1991;34:1218–27. https://doi.org/10.1002/ art.1780341003.
- Dupuy-Bonafé I, Otal P, Montal S, Bonafé A, Maldonado IL. Biometry of the temporomandibular joint using computerized tomography. Surg Radiol Anat. 2014;36:933–9. https://doi.org/10.1007/ s00276-014-1277-7.
- Elbeshlawi I, AbdelBaki MS. Safety of gadolinium administration in children. Pediatr Neurol. 2018;86:27–32. https://doi.org/10.1016/j. pediatrneurol.2018.07.010.
- Embree M, Ono M, Kilts T, Walker D, Langguth J, Mao J, et al. Role of subchondral bone during early-stage experimental TMJ osteoarthritis. J Dent Res. 2011;90:1331–8. https://doi.org/10.1177/0022034511421930.
- Emery R, Salmon M. Early rheumatoid arthritis: time to aim for remission? Ann Rheum Dis. 1995;54:944–7. https://doi.org/10.1136/ard.54.12.944.
- Emshoff R, Brandlmaier I, Schmid C, Bertram S, Rudisch A. Bone marrow edema of the mandibular condyle related to internal derangement, osteoarthrosis, and joint effusion. J Oral Maxillofac Surg. 2003a;61:35–40. https://doi.org/10.1053/ joms.2003.50006.
- Emshoff R, Brandlmaier I, Gerhard S, Strobl H, Bertram S, Rudisch A. Magnetic resonance imaging predictors of temporomandibular joint pain. J Am Dent Assoc. 2003b;134:705–14. https://doi.org/10.14219/jada. archive.2003.0256.
- Emshoff R, Gerhard S, Ennemoser T, Rudisch A. Magnetic resonance imaging findings of internal derangement, osteoarthrosis, effusion, and bone marrow edema before and after performance of arthrocentesis and hydraulic distension of the temporomandibular joint. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2006;101:784–90. https://doi.org/10.1016/j. tripleo.2005.09.005.
- Emshoff R, Rudisch A. Temporomandibular joint internal derangement and osteoarthrosis: are effusion and bone marrow edema prognostic indicators for arthrocentesis and hydraulic distention? J Oral Maxillofac Surg. 2007;65:66–73. https://doi.org/10.1016/j. joms.2005.11.113.
- Ernberg M. The role of molecular pain biomarkers in temporomandibular joint internal derangement. J Oral Rehabil. 2017;44:481–91. https://doi.org/10.1111/ joor.12480.

- Farina D, Bodin C, Gandolfi S, De Gasperi W, Borghesi A, Maroldi R. TMJ disorders and pain: assessment by contrast enhanced MRI. Eur J Radiol. 2009;70:25–30. https://doi.org/10.1016/j.ejrad.2008.01.014.
- Ferucci ED, Majka DS, Parrish LA, Moroldo MB, Ryan M, Passo M, et al. Antibodies against cyclic citrullinated peptide are associated with HLA-DR4 in simplex and multiplex polyarticular-onset juvenile rheumatoid arthritis. Arthritis Rheum. 2005;52:239– 46. https://doi.org/10.1002/art.20773.
- Francois RJ, Braun J, Khan MA. Entheses and enthesitis: a histopathologic review and relevance to spondyloarthritides. Curr Opin Rheumatol. 2001;13:255–64. https://doi.org/10.1097/00002281-200107000-00003.
- Fridrich KL, Wise JM, Zeitler DL. Prospective comparison of arthroscopy and arthrocentesis for temporomandibular joint disorders. J Oral Maxillofac Surg. 1996;54:816–20. https://doi.org/10.1016/ S0278-2391(96)90526-1.
- Gabriel SE, Michaud K. Epidemiological studies in incidence, prevalence, mortality and comorbidity of the rheumatic disease. Arthritis Res Ther. 2009;11:229. https://doi.org/10.1186/ar2669.
- Gelfand JM, Gladman DD, Mease PJ, Smith N, Margolis DJ, Nijsten T, et al. Epidemiology of psoriatic arthritis in the population of the United States. J Am Acad Dermatol. 2005;53:573. https://doi.org/10.1016/j. jaad.2005.03.046.
- Gerbino G, Zavattero E, Bosco G, Berrone S, Ramieri G. Temporomandibular joint reconstruction with stock and custom-made devices: indications and results of a 14-year experience. J Craniomaxillofac Surg. 2017;45:1710–5. https://doi.org/10.1016/j. jcms.2017.07.011.
- Gregersen PK, Silver J, Winchester RJ. The shared epitope hypothesis: an approach to understanding the molecular genetics of susceptibility to rheumatoid arthritis. Arthritis Rheum. 1987;30:1205–13. https:// doi.org/10.1002/art.1780301102.
- Gil C, Santos KC, Dutra ME, Kodaira SK, Oliveira JX. MRI analysis of the relationship between bone changes in the temporomandibular joint and articular disc position in symptomatic patients. Dentomaxillofac Radiol. 2012;41:367–72. https://doi. org/10.1259/dmfr/79317853.
- Gill DS, El Maaytah M, Naini FB. Risk factors for postorthognathic condylar resorption: a review. World J Orthod. 2008;9:21–5.
- Gladman DD. Clinical aspects of the spondyloarthropathies. Am J Med Sci. 1998;316:234–8. https://doi. org/10.1016/S0002-9629(15)40413-6.
- Gladman DD, Mease PJ, Strand V, Healy P, Helliwell PS, Fitzgerald O, et al. Consensus on a core set of domains for psoriatic arthritis. J Rheumatol. 2007;34:1167–70.
- Gomes LR, Gomes M, Jung B, Paniagua B, Ruellas AC, Goncalves JR, et al. Diagnostic index of three-dimensional osteoarthritic changes in temporomandibular joint condylar morphology. J Med Imaging (Bellingham). 2015;2:034501. https://doi. org/10.1117/1.JMI.2.3.034501.

- Gonzalez B, Larrañaga C, León O, Díaz P, Miranda M, Barría M, et al. Parvovirus B19 may have a role in the pathogenesis of juvenile idiopathic arthritis. J Rheumatol. 2007;34:1336–40.
- Goudot P, Jaquinet AR, Hugonnet S, Haefliger W, Richter M. Improvement of pain and function after arthroscopy and arthrocentesis of the temporomandibular joint: a comparative study. J Craniomaxillofac Surg. 2000;28:39–43. https://doi.org/10.1054/ jcms.1999.0103.
- Gran JT, Husby G, Hordvik M. Prevalence of ankylosing spondylitis in males and females in a young middleaged population of Tromsø, northern Norway. Ann Rheum Dis. 1985;44:359–67. https://doi.org/10.1136/ ard.44.6.359.
- Gran JT, Husby G. Clinical, epidemiologic and therapeutic aspects of ankylosing spondylitis. Curr Opin Rheumatol. 1998;10:292–8.
- Gray RJM, Davies SJ, Quayle AA. A clinical guide to temporomandibular disorders. London: British Dental Journal Books; 1995. p. 16–7.
- Güler N, Uçkan S, Imirzalioğlu P, Açikgözoğlu S. Temporomandibular joint internal derangement: relationship between joint pain and MR grading of effusion and total protein concentration in the joint fluid. Dentomaxillofac Radiol. 2005;34:175–81. https://doi.org/10.1259/dmfr/49181266.
- Hinks A, Bowes J, Cobb J, Ainsworth HC, Marion MC, Comeau ME, et al. Fine-mapping the MHC locus in juvenile idiopathic arthritis (JIA) reveals genetic heterogeneity corresponding to distinct adult inflammatory arthritic diseases. Ann Rheum Dis. 2017;76:765–72. https://doi.org/10.1136/ annrheumdis-2016-210025.
- Hirahara N, Kaneda T, Muraoka H, Fukuda T, Ito K, Kawashima Y. Characteristic magnetic resonance imaging findings in rheumatoid arthritis of the temporomandibular joint: focus on abnormal bone marrow signal of the mandibular condyle, pannus, and lymph node swelling in the parotid glands. J Oral Maxillofac Surg. 2017;75:735–41. https://doi.org/10.1016/j. joms.2016.09.051.
- Honda K, Natsumi Y, Urade M. Correlation between MRI evidence of degenerative condylar surface changes, induction of articular disc displacement and pathological joint sounds in the temporomandibular joint. Gerodontology. 2008;25:251–7. https://doi. org/10.1111/j.1741-2358.2008.00219.x.
- Hoppenreijs TJ, Stoelinga PJ, Grace KL, Robben CM. Long-term evaluation of patients with progressive condylar resorption following orthognathic surgery. Int J Oral Maxillofac Surg. 1999;28:411–8. https://doi.org/10.1016/ S0901-5027(99)80052-6.
- Horton LM, John RM, Karibe H, Rudd P. Jaw disorders in the pediatric population. J Am Assoc Nurse Pract. 2016;28:294–303. https://doi. org/10.1002/2327-6924.12322.
- Hwang SJ, Haers PE, Seifert B, Sailer HF. Non-surgical risk factors for condylar resorption after orthognathic

surgery. J Craniomaxillofac Surg. 2004;32:103–11. https://doi.org/10.1016/j.jcms.2003.09.007.

- Israel HA. Internal derangement of the temporomandibular joint: new perspectives on an old problem. Oral Maxillofac Surg Clin North Am. 2016;28:313–33. https://doi.org/10.1016/j.coms.2016.03.009.
- Jones SM, Armas JB, Cohen MG, Lovell CR, Evison G, McHugh NJ. Psoriatic arthritis: outcome of disease subsets and relationship of joint disease to nail and skin disease. Br J Rheumatol. 1994;33:834–9. https:// doi.org/10.1093/rheumatology/33.9.834.
- Kajii TS, Fujita T, Sakaguchi Y, Shimada K. Osseous changes of the mandibular condyle affect backwardrotation of the mandibular ramus in angle class II orthodontic patients with idiopathic condylar resorption of the temporomandibular joint. Cranio. 2018;23:1–8. https://doi.org/10.1080/08869634.2017.1421446.
- Kalladka M, Quek S, Heir G, Eliav E, Mupparapu M, Viswanath A. Temporomandibular joint osteoarthritis: diagnosis and long-term conservative management: a topic review. J Indian Prosthodont Soc. 2014;14:6–15. https://doi.org/10.1007/s13191-013-0321-3.
- Karhulahti T, Ylijoki H, Rönning O. Mandibular condyle lesions related to age at onset and subtypes of juvenile rheumatoid arthritis in 15-year-old children. Scand J Dent Res. 1993;101(5):332–8.
- Karlo CA, Stolzmann P, Habernig S, Müller L, Saurenmann T, Kellenberger CJ. Size, shape and agerelated changes of the mandibular condyle during childhood. Eur Radiol. 2010;20:2512–7. https://doi. org/10.1007/s00330-010-1828-1.
- Katakami K, Shimoda S, Kobayashi K, Kawasaki K. Histological investigation of osseous changes of mandibular condyles with backscattered electron images. Dentomaxillofac Radiol. 2008;37:330–9. https://doi.org/10.1259/dmfr/93169617.
- Kellenberger CJ, Arvidsson LZ, Larheim TA. Magnetic resonance imaging of temporomandibular joints in juvenile idiopathic arthritis. Semin Orthod. 2015;21:111–20. https://doi.org/10.1053/j. sodo.2015.02.007.
- Kellenberger CJ, Abramowicz S, Arvidsson LZ, Kirkhus E, Tzaribachev N, Larheim TA. Recommendations for a standard magnetic resonance imaging protocol of temporomandibular joints in juvenile idiopathic arthritis. J Oral Maxillofac Surg. 2018a;76:2463–5. https:// doi.org/10.1016/j.joms.2018.06.027.
- Kellenberger CJ, Junhasavasdikul T, Tolend M, Doria AS. Temporomandibular joint atlas for detection and grading of juvenile idiopathic arthritis involvement by magnetic resonance imaging. Pediatr Radiol. 2018b;48:411–26. https://doi.org/10.1007/ s00247-017-4000-0.
- Kellenberger CJ, Bucheli J, Schroeder-Kohler S, Saurenmann RK, Colombo V, Ettlin DA. Temporomandibular joint magnetic resonance imaging findings in adolescents with anterior disk displacement compared to those with juvenile idiopathic arthritis. J Oral Rehabil. 2019;46:14–22. https://doi. org/10.1111/joor.12720.

- Keller H, Müller LM, Markic G, Schraner T, Kellenberger CJ, Saurenmann RK. Is early TMJ involvement in children with juvenile idiopathic arthritis clinically detectable? Clinical examination of the TMJ in comparison with contrast enhanced MRI in patients with juvenile idiopathic arthritis. Pediatr Rheumatol Online J. 2015;13:56. https://doi.org/10.1186/ s12969-015-0056-2.
- Kikuta J, Wada Y, Kowada T, Wang Z, Sun-Wada GH, Nishiyama I, et al. Dynamic visualization of RANKL and Th17-mediated osteoclast function. J Clin Invest. 2013;123:866–73. https://doi.org/10.1172/JCI65054.
- Klein K, Gay S. Epigenetics in rheumatoid arthritis. Curr Opin Rheumatol. 2015;27:76–82. https://doi. org/10.1097/BOR.00000000000128.
- Köhler AA, Helkimo AN, Magnusson T, Hugoson A. Prevalence of symptoms and signs indicative of temporomandibular disorders in children and adolescents. A cross-sectional epidemiological investigation covering two decades. Eur Arch Paediatr Dent. 2009;10(Suppl 1):16–25. https://doi.org/10.1007/ BF03262695.
- Kononen M. Clinical signs of craniomandibular disorders patients with psoriatic arthritis. Scand J Dent Res. 1987;95:340–6. https://doi. org/10.1111/j.1600-0722.1987.tb01851.x.
- Koos B, Twilt M, Kyank U, Fischer-Brandies H, Gassling V, Tzaribachev N. Reliability of clinical symptoms in diagnosing temporomandibular joint arthritis in juvenile idiopathic arthritis. J Rheumatol. 2014;41:1871– 7. https://doi.org/10.3899/jrheum.131337.
- Kristensen KD, Schmidt B, Stoustrup P, Pedersen TK. Idiopathic condylar resorptions: 3-dimensional condylar bony deformation, signs and symptoms. Am J Orthod Dentofac Orthop. 2017;152:214–23. https:// doi.org/10.1016/j.ajodo.2016.12.020.
- Kruithof E, Baeten D, Van den Bosch F, Mielants H, Veys EM, De Keyser F. Histological evidence that infliximab treatment leads to downregulation of inflammation and tissue remodelling of the synovial membrane in spondyloarthropathy. Ann Rheum Dis. 2005;64:529– 36. https://doi.org/10.1136/ard.2003.018549.
- Kuhn KA, Kulik L, Tomooka B, Braschler KJ, Arend WP, Robinson WH, et al. Antibodies against citrullinated proteins enhance tissue injury in experimental autoimmune arthritis. J Clin Invest. 2006;116:961–73. https://doi.org/10.1172/JCI25422.
- Kurita H, Ohtsuka A, Kobayashi H, Kurashina K. Flattening of the articular eminence correlates with progressive internal derangement of the temporomandibular joint. Dentomaxillofac Radiol. 2000;29:277– 9. https://doi.org/10.1038/sj/dmfr/4600542.
- Kurita H, Ohtsuka A, Kobayashi H, Kurashina K. Resorption of the postero-superior corner of the lateral part of the mandibular condyle correlates with progressive TMJ internal derangement. Int J Oral Maxillofac Surg. 2003;32:363–7. https://doi.org/10.1054/ijom.2002.0361.
- Lam J, Takeshita S, Barker JE, Kanagawa O, Ross FP, Teitelbaum SL. TNF-alpha induces osteoclastogen-

esis by direct stimulation of macrophages exposed to permissive levels of RANK ligand. J Clin Invest. 2000;106:1481–8. https://doi.org/10.1172/JCI11176.

- Larheim TA, Westesson PL, Hicks DG, Eriksson L, Brown DA. Osteonecrosis of the temporomandibular joint: correlation of magnetic resonance imaging and histology. J Oral Maxillofac Surg. 1999;57:888–98. https://doi.org/10.1016/S0278-2391(99)90001-0.
- Larheim TA, Westesson PL, Sano T. MR grading of temporomandibular joint fluid: association with disk displacement categories, condyle marrow abnormalities and pain. Int J Oral Maxillofac Surg. 2001;30:104–12. https://doi.org/10.1054/ijom.2000.0017.
- Larheim TA, Abrahamsson AK, Kristensen M, Arvidsson LZ. Temporomandibular joint diagnostics using CBCT. Dentomaxillofac Radiol. 2015;44:20140235. https://doi.org/10.1259/dmfr.20140235.
- Lee EY, Sundel RP, Kim S, Zurakowski D, Kleinman PK. MRI findings of juvenile psoriatic arthritis. Skelet Radiol. 2008;37:987–96. https://doi.org/10.1007/ s00256-008-0537-1.
- Lei J, Han J, Liu M, Zhang Y, Yap AU, Fu KY. Degenerative temporomandibular joint changes associated with recent-onset disc displacement without reduction in adolescents and young adults. J Craniomaxillofac Surg. 2017;45:408–13. https://doi.org/10.1016/j. jcms.2016.12.017.
- Leschied JR, Smith EA, Baker S, Khalatbari S, Aronovich S. Contrast-enhanced MRI compared to direct joint visualization at arthroscopy in pediatric patients with suspected temporomandibular joint synovitis. Pediatr Radiol. 2019;49:196–202. https://doi.org/10.1007/ s00247-018-4291-9.
- Liao KP, Batra KL, Chibnik L, Schur PH, Costenbader KH. Anti-cyclic citrullinated peptide revised criteria for the classification of rheumatoid arthritis. Ann Rheum Dis. 2008;67:1557–61. https://doi.org/10.1136/ ard.2007.082339.
- List T, Jensen RH. Temporomandibular disorders: old ideas and new concepts. Cephalalgia. 2017;37: 692–704. https://doi.org/10.1177/0333102416686302.
- Locher MC, Felder M, Sailer HF. Involvement of the temporomandibular joints in ankylosing spondylitis (Bechterew's disease). J Craniofac Surg. 1996;24:205– 13. https://doi.org/10.1016/S1010-5182(96)80003-5.
- Lomater C, Gerloni V, Gattinara M, Mazzotti J, Cimaz R, Fantini F. Systemic onset juvenile idiopathic arthritis: a retrospective study of 80 consecutive patients followed for 10 years. J Rheumatol. 2000;27: 491–6.
- Manemi RV, Fasanmade A, Revington PJ. Bilateral ankylosis of the jaw treated with total alloplastic replacement using the TMJ concepts system in a patient with ankylosing spondylitis. Br J Oral Maxillofac Surg. 2009;47:159–61. https://doi.org/10.1016/j. bjoms.2008.08.020.
- Manfredini D, Segù M, Arveda N, Lombardo L, Siciliani G, Alessandro R. Temporomandibular joint disorders in patients with different facial morphology. A systematic review of the literature. J Oral Maxillofac

Surg. 2016;74:29–46. https://doi.org/10.1016/j. joms.2015.07.006.

- May E, Märker-Hermann E, Wittig BM, Zeitz M, Meyer zum Büschenfelde KH, Duchmann R. Identical T-cell expansions in the colon mucosa and the synovium of a patient with enterogenic spondyloarthropathy. Gastroenterology. 2000;119:1745–55. https://doi. org/10.1053/gast.2000.20173.
- Maydana AV, de Sousa TR, Denardin OV, da Silva Ursi WJ, Dworkin SF. Possible etiological factors in temporomandibular disorders of articular origin with implications for diagnosis and treatment. Dental Press J Orthod. 2010;15:78–86. https://doi.org/10.1590/ S2176-94512010000300010.
- McGonagle D, Gibbon W, Emery P. Classification of inflammatory arthritis by enthesitis. Lancet. 1998;352:1137–40. https://doi.org/10.1016/ S0140-6736(97)12004-9.
- McNeely ML, Armijo Olivo S, Magee DJ. A systematic review of the effectiveness of physical therapy interventions for temporomandibular disorders. Phys Ther. 2006;86:710–25. https://doi.org/10.1093/ ptj/86.5.710.
- Mehra P, Wolford LM, Baran S, Cassano DS. Singlestage comprehensive surgical treatment of the rheumatoid arthritis temporomandibular joint patient. J Oral Maxillofac Surg. 2009;67:1859–72. https://doi. org/10.1016/j.joms.2009.04.035.
- Mehra P, Nadershah M, Chigurupati R. Is alloplastic temporomandibular joint reconstruction a viable option in the surgical management of adult patients with idiopathic condylar resorption? J Oral Maxillofac Surg. 2016;74:2044–54. https://doi.org/10.1016/j. joms.2016.04.012.
- Mercuri LG. A rationale for total alloplastic temporomandibular joint reconstruction in the management of idiopathic/progressive condylar resorption. J Oral Maxillofac Surg. 2007;65:1600–9. https://doi. org/10.1016/j.joms.2006.03.056.
- Mercuri LG, Edibam NR, Giobbie-Hurder A. Fourteenyear follow-up of a patient-fitted total temporomandibular joint reconstruction system. J Oral Maxillofac Surg. 2007;65:1140–8. https://doi.org/10.1016/j. joms.2006.10.006.
- Mercuri LG. Osteoarthritis, osteoarthrosis, and idiopathic condylar resorption. Oral Maxillofac Surg Clin North Am. 2008;20:169–83. https://doi.org/10.1016/j. coms.2007.12.007.
- Meyers AB, Laor T. Magnetic resonance imaging of the temporomandibular joint in children with juvenile idiopathic arthritis. Pediatr Radiol. 2013;43:1632–41. https://doi.org/10.1007/s00247-013-2769-z.
- Miller E, Inarejos Clemente EJ, Tzaribachev N, Guleria S, Tolend M, Meyers AB. Imaging of temporomandibular joint abnormalities in juvenile idiopathic arthritis with a focus on developing a magnetic resonance imaging protocol. Pediatr Radiol. 2018;48:792–800. https://doi.org/10.1007/s00247-017-4005-8.
- Misirlioglu M, Adisen MZ, Yilmaz S. Radiographic diagnosis of osteochondritis dissecans of the

temporomandibular joint: two cases. Med Princ Pract. 2014;23:580–3. https://doi.org/10.1159/000363572.

- Mitsimponas K, Mehmet S, Kennedy R, Shakib K. Idiopathic condylar resorption. Br J Oral Maxillofac Surg. 2018;56:249–55. https://doi.org/10.1016/j. bjoms.2018.02.016.
- Monje-Gil F, Nitzan D, Gonzalez-Garcia R. Temporomandibular joint arthrocentesis. Review of the literature. Med Oral Patol Oral Cir Bucal. 2012;17(4):e575–81. https://doi.org/10.4317/ medoral.17670.
- Monje-Gil F. Surgical management of temporomandibular joint: vol.1 arthroscopy. ebook; 2014. p. 163–256.
- Morimoto Y, Tanaka T, Masumi S, Tominaga K, Shibuya T, Kito S, et al. Significance of frequency-selective fat saturation T2-weighted MR images for the detection of bone marrow edema in the mandibular condyle. Cranio. 2004;22:115–23. https://doi.org/10.1179/crn.2004.015.
- Munir S, Patil K, Miller E, Uleryk E, Twilt M, Spiegel L, et al. Juvenile idiopathic arthritis of the axial joints: a systematic review of the diagnostic accuracy and predictive value of conventional MRI. AJR Am J Roentgenol. 2014;202:199–210. https://doi. org/10.2214/AJR.12.10475.
- Murphy MK, MacBarb RF, Wong ME, Athanasiou KA. Temporomandibular disorders: a review of etiology, clinical management, and tissue engineering strategies. Int J Oral Maxillofac Implants. 2013;28:e393– 414. https://doi.org/10.11607/jomi.te20.
- Nam JL, Winthrop KL, van Vollenhoven RF, Pavelka K, Valesini G, Hensor EM, et al. Current evidence for the management of rheumatoid arthritis with biological disease-modifying antirheumatic drugs: a systematic literature review informing the EULAR recommendations for the management of RA. Ann Rheum Dis. 2010;69:976–86. https://doi.org/10.1136/ ard.2009.126573.
- Navallas M, Inarejos EJ, Iglesias E, Cho Lee GY, Rodríguez N, Antón J. MR imaging of the temporomandibular joint in juvenile idiopathic arthritis: technique and findings. Radiographics. 2017;37: 595–612. https://doi.org/10.1148/rg.2017160078.
- Nielen MM, van der Horst AR, van Schaardenburg D, van der Horst-Bruinsma IE, van de Stadt RJ, Aarden L, et al. Antibodies to citrullinated human fibrinogen (ACF) have diagnostic and prognostic value in early arthritis. Ann Rheum Dis. 2005;64:1199–204. https:// doi.org/10.1136/ard.2004.029389.
- Nishimura M, Segami N, Kaneyama K, Suzuki T. Prognostic factors in arthrocentesis of the temporomandibular joint: evaluation of 100 patients with internal derangement. J Oral Maxillofac. 2001;59:874–7. https://doi.org/10.1053/joms.2001.25019.
- Nitzan DW, Dolwick MF, Martinez GA. Temporomandibular joint arthrocentesis: a simplified treatment for severe, limited mouth opening. J Oral Maxillofac Surg. 1991;49:1163–7. https://doi. org/10.1016/0278-2391(91)90409-F.

- Nitzan DW, Svidovsky J, Zini A, Zadik Y. Effect of arthrocentesis on symptomatic osteoarthritis of the temporomandibular joint and analysis of the effect of preoperative clinical and radiologic features. J Oral Maxillofac Surg. 2017;75:260–7. https://doi. org/10.1016/j.joms.2016.08.017.
- Nørholt SE, Bjørnland T, Pedersen TK. Jaw surgery for correction of dentofacial anomalies caused by JIA. Semin Orthod. 2015;21:140–7. https://doi. org/10.1053/j.sodo.2015.02.011.
- O'Connor RC, Fawthrop F, Salha R, Sidebottom AJ. Management of the temporomandibular joint in inflammatory arthritis: involvement of surgical procedures. Eur J Rheumatol. 2017;4:151–6. https://doi. org/10.5152/eurjrheum.2016.035.
- Ok SM, Lee J, Kim YI, Lee JY, Kim KB, Jeong SH. Anterior condylar remodeling observed in stabilization splint therapy for temporomandibular joint osteoarthritis. Oral Surg Oral Med Oral Pathol Oral Radiol. 2014;118:363–70. https://doi.org/10.1016/j. 0000.2014.05.022.
- Okada Y, Wu D, Trynka G, Raj T, Terao C, Ikari K, et al. Genetics of rheumatoid arthritis contributes to biology and drug discovery. Nature. 2014;506(7488):376–81. https://doi.org/10.1038/nature12873.
- Olley SF, Leopard PJ. Osteochondritis dissecans affecting the temporo-mandibular joint. Br J Oral Surg. 1978;16:21–5. https://doi.org/10.1016/ S0007-117X(78)80051-1.
- Olsen-Bergem H, Bjørnland T. A cohort study of patients with juvenile idiopathic arthritis and arthritis of the temporomandibular joint: outcome of arthrocentesis with and without the use of steroids. Int J Oral Maxillofac Surg. 2014;43:990–5. https://doi. org/10.1016/j.ijom.2014.03.018.
- Orhan K, Arslan A, Kocyigit D. Temporomandibular joint osteochondritis dissecans: case report. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2006;102(4):e41–6. https://doi.org/10.1016/j. tripleo.2006.01.002.
- Pantoja LLQ, de Toledo IP, Pupo YM, Porporatti AL, De Luca CG, Zwir LF, et al. Prevalence of degenerative joint disease of the temporomandibular joint: a systematic review. Clin Oral Investig. 2018;23:2475–88. https://doi.org/10.1007/s00784-018-2664-y.
- Papadaki ME, Tayebaty F, Kaban LB, Troulis MJ. Condylar resorption. Oral Maxillofac Surg Clin North Am. 2007;19:223–34. https://doi.org/10.1016/j. coms.2007.01.002.
- Peacock ZS, Vakilian P, Caruso P, Resnick CM, Vangel M, Kaban LB. Quantifying synovial enhancement of the pediatric temporomandibular joint. J Oral Maxillofac Surg. 2016;74:1937–45. https://doi.org/10.1016/j. joms.2016.03.010.
- Peck CC, Goulet JP, Lobbezoo F, Schiffman EL, Alstergren P, Anderson GC, et al. Expanding the taxonomy of the diagnostic criteria for temporomandibular disorders. J Oral Rehabil. 2014;41:2–23. https://doi.org/10.1111/joor.12132.

- Pedersen TK, Jensen JJ, Melsen B, Herlin T. Resorption of the temporomandibular condylar bone according to subtypes of juvenile chronic arthritis. J Rheumatol. 2001;28(9):2109–15.
- Peterson A. What you can and cannot see in TMJ imaging-an overview related to the RDC/TMD diagnostic system. J Oral Rehabil. 2010;37:771–8. https://doi. org/10.1111/j.1365-2842.2010.02108.x.
- Petty RE, Southwood TR, Manners P, Baum J, Glass DN, Goldenberg J, et al. International league of associations for rheumatology classification of juvenile idiopathic arthritis: second revision, Edmonton, 2001. J Rheumatol. 2004;31:390–2.
- Posnick JC, Fantuzzo JJ. Idiopathic condylar resorption: current clinical perspectives. J Oral Maxillofac Surg. 2007;65:1617–23. https://doi.org/10.1016/j. joms.2007.03.026.
- Prahalad S, Shear ES, Thompson SD, Giannini EH, Glass DN. Increased prevalence of familial autoimmunity in simplex and multiplex families with juvenile rheumatoid arthritis. Arthritis Rheum. 2002;46:1851–6. https://doi.org/10.1002/art.10370.
- Prakken B, Albani S, Martini A. Juvenile idiopathic arthritis. Lancet. 2011;377:2138–49. https://doi. org/10.1016/S0140-6736(11)60244-4.
- Prieur AM, Le Gall E, Karman F, Edan C, Lasserre O, Goujard J. Epidemiologic survey of juvenile chronic arthritis in France. Comparison of data obtained from two different regions. Clin Exp Rheumatol. 1987;5:217–23.
- Ramos-Remus C, Major P, Gomez-Vargas A, Petrikowski G, Hernandez-Chavez A, Gonzalez-Marin E, et al. Temporomandibular joint osseous morphology in a consecutive sample of ankylosing spondylitis patients. Ann Rheum Dis. 1997;56:103–7. https://doi. org/10.1136/ard.56.2.103.
- Rantapää-Dahlqvist S, de Jong BA, Berglin E, Hallmans G, Wadell G, Stenlund H, et al. Antibodies against cyclic citrullinated peptide and IgA rheumatoid factor predict the development of rheumatoid arthritis. Arthritis Rheum. 2003;48:2741–9. https://doi. org/10.1002/art.11223.
- Resnick CM, Vakilian PM, Breen M, Zurakowski D, Caruso P, Henderson L, et al. Quantifying temporomandibular joint synovitis in children with juvenile idiopathic arthritis. Arthritis Care Res (Hoboken). 2016;68:1795–802. https://doi.org/10.1002/acr.22911.
- Ribbens C, Martin y Porras M, Franchimont N, Kaiser MJ, Jaspar JM, Damas P. Increased matrix metalloproteinase-3 serum levels in rheumatic diseases: relationship with synovitis and steroid treatment. Ann Rheum Dis. 2002;61:161–6. https://doi.org/10.1136/ard.61.2.161.
- Ringold S, Cron RQ. The temporomandibular joint in juvenile idiopathic arthritis: frequently used and frequently arthritic. Pediatr Rheumatol Online J. 2009;7:11. https://doi.org/10.1186/1546-0096-7-11.
- Roberson ED, Bowcock AM. Psoriasis genetics: breaking the barrier. Trends Genet. 2010;26:415–23. https://doi. org/10.1016/j.tig.2010.06.006.

- Roh HS, Kim W, Kim YK, Lee JY. Relationships between disk displacement, joint effusion, and degenerative changes of the TMJ in TMD patients based on MRI findings. J Craniomaxillofac Surg. 2012;40:283–6. https://doi.org/10.1016/j.jcms.2011.04.006.
- Rudwaleit M, Siegert S, Yin Z, Eick J, Thiel A, Radbruch A, et al. Low T cell production of TNFalpha and IFNgamma in ankylosing spondylitis: its relation to HLA-B27 and influence of the TNF-308 gene polymorphism. Ann Rheum Dis. 2001;60:36–42. https:// doi.org/10.1136/ard.60.1.36.
- Rudwaleit M, van der Heijde D, Khan MA, Braun J, Sieper J. How to diagnose axial spondyloarthritis early. Ann Rheum Dis. 2004;63:535–43. https://doi. org/10.1136/ard.2003.011247.
- Sano T, Westesson PL, Larheim TA, Rubin SJ, Tallents RH. Osteoarthritis and abnormal bone marrow of the mandibular condyle. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1999;87:243–52. https://doi. org/10.1016/S1079-2104(99)70280-5.
- Sano T. Recent developments in understanding temporomandibular joint disorders. Part 1: bone marrow abnormalities of the mandibular condyle. Dentomaxillofac Radiol. 2000;29:7–10. https://doi.org/10.1038/sj/ dmfr/4600492.
- Sansare K, Raghav M, Mallya S, Mundada N, Karjodkar F, Randive P, et al. Aggressive condylar resorption. J Craniofac Surg. 2013;24:e95–6. https://doi. org/10.1097/SCS.0b013e3182798eff.
- Sansare K, Raghav M, Mallya SM, Karjodkar F. Management-related outcomes and radiographic findings of idiopathic condylar resorption: a systematic review. Int J Oral Maxillofac Surg. 2015;44:209– 16. https://doi.org/10.1016/j.ijom.2014.09.005.
- Sato K, Suematsu A, Okamoto K, Yamaguchi A, Morishita Y, Kadono Y, et al. Th17 functions as an osteoclastogenic helper T cell subset that links T cell activation and bone destruction. J Exp Med. 2006;203:2673–82. https://doi.org/10.1084/jem.20061775.
- Savolainen E, Kaipiainen-Seppänen O, Kröger L, Luosujärvi R. Total incidence and distribution of inflammatory joint diseases in a defined population: results from the Kuopio 2000 arthritis survey. J Rheumatol. 2003;30:2460–8.
- Schellhas KP. Internal derangement of the temporomandibular joint: radiologic staging with clinical, surgical, and pathologic correlation. Magn Reson Imaging. 1989;7:495–515. https://doi. org/10.1016/0730-725X(89)90404-9.
- Schellhas KP, Wilkes CH, Fritts HM, Omlie MR, Lagrotteria LB. MR of osteochondritis dissecans and avascular necrosis of the mandibular condyle. AJR Am J Roentgenol. 1989;152:551–60. https://doi. org/10.2214/ajr.152.3.551.
- Schiffman EL, Ohrbach R, Truelove E, Look J, Anderson G, Goulet JP, 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;28:6–27. https://doi.org/10.11607/jop.1151.

- Schmitter M, Essig M, Seneadza V, Balke Z, Schroder J, Rammelsberg P. Prevalence of clinical and radiographic signs of osteoarthrosis of the temporomandibular joint in an older persons community. Dentomaxillofac Radiol. 2010;39:231–4. https://doi. org/10.1259/dmfr/16270943.
- Scrivani SJ, Keith DA, Kaban LB. Temporomandibular disorders. N Engl J Med. 2008;359:2693–705. https:// doi.org/10.1056/NEJMra0802472.
- Segami N, Nishimura M, Kaneyama K, Miyamaru M, Sato J, Murakami KI. Does joint effusion on T2 magnetic resonance images reflect synovitis? Comparison of arthroscopic findings in internal derangements of the temporomandibular joint. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2001;92:341–5. https://doi. org/10.1067/moe.2001.117808.
- Segami N, Miyamaru M, Nishimura M, Suzuki T, Kaneyama K, Murakami K. Does joint effusion on T2 magnetic resonance images reflect synovitis? Part 2. Comparison of concentration levels of proinflammatory cytokines and total protein in synovial fluid of the temporomandibular joint with internal derangements and osteoarthrosis. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2002;94:515–21. https://doi. org/10.1067/moe.2002.126697.
- Seo YJ, Park SB, Kim YI, Ok SM, Kim SS, Son WS. Effects of condylar head surface changes on mandibular position in patients with temporomandibular joint osteoarthritis. J Craniomaxillofac Surg. 2015;43:1380–3. https://doi.org/10.1016/j. jcms.2015.06.031.
- Sieper J, Braun J, Rudwaleit M, Boonen A, Zink A. Ankylosing spondylitis: an overview. Ann Rheum Dis. 2002;61(Suppl 3):iii8–iii18. https://doi. org/10.1136/ard.61.suppl_3.iii8.
- Sieper J, Rudwaleit M. Early referral recommendations for ankylosing spondylitis (including pre-radiographic and radiographic forms) in primary care. Ann Rheum Dis. 2005;64:659–63. https://doi.org/10.1136/ ard.2004.028753.
- Silman AJ. Has the incidence of rheumatoid arthritis declined in the United Kingdom? Br J Rheumatol. 1988;27:77–9. https://doi.org/10.1093/ rheumatology/27.1.77.
- Silman AJ, MacGregor AJ, Thomson W, Holligan S, Carthy D, Farhan A, et al. Twin concordance rates for rheumatoid arthritis: results from a nationwide study. Br J Rheumatol. 1993;32:903–7. https://doi. org/10.1093/rheumatology/32.10.903.
- Silman AJ, Pearson JE. Epidemiology and genetics of rheumatoid arthritis. Arthritis Res. 2002;4(Suppl 3):S265–72. https://doi.org/10.1186/ar578.
- Skarin A, Elborgh R, Edlund E, Bengtsson-Stigmar E. Long-term follow-up of patients with uveitis associated with juvenile idiopathic arthritis: a cohort study. Ocul Immunol Inflamm. 2009;17:104–8. https://doi. org/10.1080/09273940802650398.

- Skármeta NP, Araneda L, Araya C. Destructive psoriatic arthritis of the temporomandibular joint: a clinical case, an overview of the pathophysiology and its differential diagnoses. Cranio. 2018:1–7. https://doi.org/ 10.1080/08869634.2018.1484575.
- Smolen JS, Aletaha D, Koeller M, Weisman MH, Emery P. New therapies for the treatment of rheumatoid arthritis. Lancet. 2007;370:1861–74. https://doi. org/10.1016/S0140-6736(07)60784-3.
- Smolen JS, Aletaha D, McInnes IB. Rheumatoid arthritis. Lancet. 2016;388:2023–38. https://doi.org/10.1016/ S0140-6736(16)30173-8.
- Smolka W, Sotlar K, Weiss M, Müller-Lisse U. Osteochondrosis dissecans of the temporomandibular joint: a case report and a review of literature. Oral Maxillofac Surg. 2016;20:321–5. https://doi. org/10.1007/s10006-016-0557-2.
- Sodhi A, Naik S, Pai A, Anuradha A. Rheumatoid arthritis affecting temporomandibular joint. Contemp Clin Dent. 2015;6:124–7. https://doi. org/10.4103/0976-237X.149308.
- Stegenga B, de Bont LG, Boering G. Osteoarthrosis as the cause of craniomandibular pain and dysfunction: a unifying concept. J Oral Maxillofac Surg. 1989;47:249– 56. https://doi.org/10.1016/0278-2391(89)90227-9.
- Stoll ML, Cron RQ, Saurenmann RK. Systemic and intra-articular antiinflammatory therapy of temporomandibular joint arthritis in children with juvenile idiopathic arthritis. Semin Orthod. 2015;21:125–33. doi.org/10.1053/j.sodo.2015.02.009
- Stoll ML, Guleria S, Mannion ML, Young DW, Royal SA, Cron RQ, et al. Defining the normal appearance of the temporomandibular joints by magnetic resonance imaging with contrast: a comparative study of children with and without juvenile idiopathic arthritis. Pediatr Rheumatol Online J. 2018;16:8. https://doi. org/10.1186/s12969-018-0223-3.
- Stoustrup P, Kristensen KD, Verna C, Küseler A, Pedersen TK, Herlin T. Intra-articular steroid injection for temporomandibular joint arthritis in juvenile idiopathic arthritis: a systematic review on efficacy and safety. Semin Arthritis Rheum. 2013;43:63–70. https://doi. org/10.1016/j.semarthrit.2012.11.003.
- Takahara N, Nakagawa S, Sumikura K, Kabasawa Y, Sakamoto I, Harada H. Association of Temporomandibular Joint Pain According to magnetic resonance imaging findings in Temporomandibular disorder patients. J Oral Maxillofac Surg. 2017;75:1848– 55. https://doi.org/10.1016/j.joms.2017.03.026.
- Takatsuka S, Yoshida K, Ueki K, Marukawa K, Nakagawa K, Yamamoto E. Disc and condyle translation in patients with temporomandibular disorder. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2005;99:614–21. https://doi.org/10.1016/j. tripleo.2004.08.024.
- Tanaka E, Detamore MS, Mercuri LG. Degenerative disorders of the temporomandibular joint: etiology, diagnosis, and treatment. J Dent Res. 2008;87:296–307. https://doi.org/10.1177/154405910808700406.

- Thilander B, Rubio G, Pena L, de Mayorga C. Prevalence of temporomandibular dysfunction and its association with malocclusion in children and adolescents: an epidemiologic study related to specified stages of dental development. Angle Orthod. 2002;72:146–54. https:// doi.org/10.1043/0003-3219(2002)072<0146:POTDA I>2.0.CO;2.
- Thiryayi WA, Thiryayi SA, Freemont AJ. Histopathological perspective on bone marrow oedema, reactive bone change and haemorrhage. Eur J Radiol. 2008;67:62–7. https://doi.org/10.1016/j. ejrad.2008.01.056.
- Thomas N, Harper DE, Aronovich S. Do signs of an effusion of the temporomandibular joint on magnetic resonance imaging correlate with signs and symptoms of temporomandibular joint disease? Br J Oral Maxillofac Surg. 2018;56:96–100. https://doi. org/10.1016/j.bjoms.2017.11.011.
- Tolend MA, Twilt M, Cron RQ, Tzaribachev N, Guleria S, von Kalle T, et al. Toward establishing a standardized magnetic resonance imaging scoring system for temporomandibular joints in juvenile idiopathic arthritis. Arthritis Care Res (Hoboken). 2018;70:758–67. https://doi.org/10.1002/acr.23340.
- Toller PA. Osteoarthrosis of the mandibular condyle. Br Dent J. 1973;134:223–31.
- Troulis MJ, Tayebaty FT, Papadaki M, Williams WB, Kaban LB. Condylectomy and costochondral graft reconstruction for treatment of active idiopathic condylar resorption. J Oral Maxillofac Surg. 2008;66:65– 72. https://doi.org/10.1016/j.joms.2007.08.030.
- Twilt M, Mobers SM, Arends LR, ten Cate R, van Suijlekom-Smit L. Temporomandibular involvement in juvenile idiopathic arthritis. J Rheumatol. 2004;31:1418–22.
- Tzaribachev N, Fritz J, Horger M. Spectrum of magnetic resonance imaging appearances of juvenile temporomandibular joints (TMJ) in non-rheumatic children. Acta Radiol. 2009;50:1182–6. https://doi. org/10.3109/02841850903271341.
- Vaid YN, Dunnavant FD, Royal SA, Beukelman T, Stoll ML, Cron RQ. Imaging of the temporomandibular joint in juvenile idiopathic arthritis. Arthritis Care Res (Hoboken). 2014;66:47–54. https://doi.org/10.1002/ acr.22177.
- van der Linden SM, Valkenburg HA, de Jongh BM, Cats A. The risk of developing ankylosing spondylitis in HLA-B27 positive individuals: a comparison of relatives of spondylitis patients with the general population. Arthritis Rheum. 1984;27:241–9. https://doi. org/10.1002/art.1780270301.
- Van Der Linden S, Van Der Heijde D. Ankylosing spondylitis: clinical features. Rheum Dis Clin N Am. 1998;24:663–676, vii. https://doi.org/10.1016/ S0889-857X(05)70036-3.
- Verwoerd A, Ter Haar NM, de Roock S, Vastert SJ, Bogaert D. The human microbiome and juvenile idiopathic arthritis. Pediatr Rheumatol

Online J. 2016;14:55. https://doi.org/10.1186/ s12969-016-0114-4.

- Wahaj A, Hafeez K, Zafar MS. Association of bone marrow edema with temporomandibular joint (TMJ) osteoarthritis and internal derangements. Cranio. 2017;35:4–9. https://doi.org/10.1080/08869634.2016 .1156282.
- Wänman A, Ernberg M, List T. Guidelines in the management of orofacial pain/TMD. An evidence-based approach. Nor Tannlegeforen Tid. 2016;126:104–12.
- Weiss JE, Ilowite NT. Juvenile idiopathic arthritis. Pediatr Clin N Am. 2005;52:413–42. https://doi.org/10.1016/j. pcl.2005.01.007.
- Weiss PF, Arabshahi B, Johnson A, Bilaniuk LT, Zarnow D, Cahill AM, et al. High prevalence of temporomandibular joint arthritis at disease onset in children with juvenile idiopathic arthritis, as detected by magnetic resonance imaging but not by ultrasound. Arthritis Rheum. 2008;58:1189–96. https://doi. org/10.1002/art.23401.
- Wilkes CH. Internal derangement of the temporomandibular joint: pathologic variations. Arch Otolaryngol Head Neck Surg. 1989;115:469–77. https://doi. org/10.1001/archotol.1989.01860280067019.
- Winocur E, Steinkeller-Dekel M, Reiter S, Eli I. A retrospective analysis of temporomandibular findings among Israeli-born patients based on the RDC/ TMD. J Oral Rehabil. 2009;36:11–7. https://doi. org/10.1111/j.1365-2842.2008.01898.x.
- Wolford LM, Chemallo PD, Hilliard FW. Occlusal plane alteration in orthognathic surgery. J Oral Maxillofac Surg. 1993;51:730–40. https://doi. org/10.1016/S0278-2391(10)80410-0.
- Wolford LM, Cardenas L. Idiopathic condylar resorption: diagnosis, treatment protocol, and outcomes. Am J Orthod Dentofac Orthop. 1999;116:667–77. https:// doi.org/10.1016/S0889-5406(99)70203-9.
- Wolford LM. Idiopathic condylar resorption of the temporomandibular joint in teenage girls (cheerleaders syndrome). Proc (Bayl Univ Med Cent). 2001;14:246–52. https://doi.org/10.1080/08998280.2 001.11927772.
- Wolford LM, Mercuri LG, Schneiderman ED, Movahed R, Allen W. Twenty-year follow-up study on a patient-fitted temporomandibular joint prosthesis: the Techmedica/TMJ concepts device. J Oral Maxillofac Surg. 2015;73:952–60. https://doi.org/10.1016/j. joms.2014.10.032.
- Wolford LM, Gonçalves JR. Condylar resorption of the temporomandibular joint: how do we treat it? Oral Maxillofac Surg Clin North Am. 2015;27:47–67.
- Wright RW. MARS group. Osteoarthritis classification scales: interobserver reliability and arthroscopic correlation. J Bone Joint Surg Am. 2014;16(96):1145–51. https://doi.org/10.2106/JBJS.M.00929.
- Wu G, Zhu S, Sun X, Hu J. Subchondral bone changes and chondrogenic capacity of progenitor cells from subchondral bone in the collagenase-induced tem-

poromandibular joints osteoarthritis rabbit model. Int J Clin Exp Pathol. 2015;8:9782–9.

- Yap AU, Dworkin SF, Chua EK, List T, Tan KB, Tan HH. Prevalence of temporomandibular disorder subtypes, psychologic distress, and psychosocial dysfunction in Asian patients. J Orofac Pain. 2003;17:21–8.
- Young A. Idiopathic condylar resorption: the current understanding in diagnosis and treatment. J Indian Prosthodont Soc. 2017;17:128–35. https://doi. org/10.4103/jips_jips_60_17.
- Zhao YP, Zhang ZY, Wu YT, Zhang WL, Ma XC. Investigation of the clinical and radiographic features of osteoarthrosis of the temporomandibular joints in adolescents and young adults. Oral Surg Oral

Med Oral Pathol Oral Radiol Endod. 2011;111:e27–34. https://doi.org/10.1016/j.tripleo.2010.09.076.

- Zhang C, Xu Y, Cheng Y, Wu T, Li H. Effect of asymmetric force on the condylar cartilage, subchondral bone and collagens in the temporomandibular joints. Arch Oral Biol. 2015;60:650–63. https://doi.org/10.1016/j. archoralbio.2015.01.008.
- Zhou J, Ding Y, Zhang Y, Feng Y, Tang X, Zhao X. CD3+CD56+ natural killer T cell activity in children with different forms of juvenile idiopathic arthritis and the influence of etanercept treatment on polyarticular subgroup. Clin Immunol. 2016;176: 1–11. https://doi.org/10.1016/j.clim.2016.12.001.

Paolo Tosco, Vito Chianca, and Guglielmo Ramieri

Key Points

- · Growing masses, benign and malignant, can alter TMJ functions and mimic symptoms that are also encountered in the most common temporomandibular disorders.
- TMJ tumors are uncommon. They can be primary tumors, secondary tumors, and metastases.
- Masses detected on CBCT and TMJ MRI will need further assessment. In the case of malignancies CT and MRI have to be extended within the head and neck. Contrast is mandatory for malignant tumors and imaging modalities are performed differently, with the purpose of locoregional staging and surgical planning.
- Benign tumors have a slow growth, nonspecific symptoms and may undergo a significant delay in diagnosis because of misinterpretation or overlooking.
- Malignant tumors generally show a rather rapidly grow mass, perhaps involving surrounding anatomical regions (ears, parotid, medial

P. Tosco (\boxtimes) Clinica Fornaca di Sessant, Turin, Italy

V. Chianca IRCCS Istituto Ortopedico Galeazzi, Milan, Italy

G. Ramieri Department of Surgical Sciences, Division of Maxillofacial Surgery, University of Turin, Turin, Italy e-mail: guglielmo.ramieri@unito.it

© Springer Nature Switzerland AG 2020

https://doi.org/10.1007/978-3-030-25421-6_9

T. Robba et al. (eds.), MRI of the Temporomandibular Joint,

cranial fossa, infratemporal fossa, zygomatic and temporal bone).

Primary malignancies from breast, lung, prostate, kidney, rectum, and stomach may spread to this region. TMJ metatases are extremely rare.

- Management depends on patient age, tumor extension, and behavior, ranging from simple curettage to resection with total TMJ reconstruction. Final diagnosis is histological after surgical intervention.
- Prognosis of benign tumors is very good, on the other hand, malignant tumors have poor survival rates.
- Tumor-like lesions are condylar or intraarticular non-neoplastic enlargements which behave like space-occupying masses.

The purpose of this chapter is to provide knowledge about tumors that affect temporomandibular joint (TMJ) function. Because of this, details on tumor biology, behavior, and treatment will not be discussed. Tumors and pseudotumors can mimic more common non-neoplastic conditions given that pain and impairment are common features. Focus will therefore be on these aspects and on imaging of the most prevalent, although rare, TMJ tumors.

TMJ tumors can be primary, secondary (spreading from adjacent structures) or they can be metastases from a distant primary malignant tumor. Primary tumors, benign and malignant,

Tumors and Tumor-Like Lesions



²¹⁹

can originate from bone, cartilage, or soft tissue and progressively enlarge, thus occupying the joint space to various degrees. Tumor masses commonly remain silent until their size increases and symptoms appear, and they can be incidentally detected when imaging is carried out for other reasons (Mostafapour and Futran 2000; Al-Jamali et al. 2013).

9.1 Benign Tumors

9.1.1 Imaging of Benign Tumors

Presence of benign tumors and pseudotumors is often an incidental finding on orthopantomogram, CBCT, or MRI. In such cases the patient will be referred for further evaluation, biopsy procedures, and treatment planning.

Imaging signs for osteolytic and osteoblastic lesions hold true for bone lesions in general and for the jaws (Harmon et al. 2015). Aggressiveness can be inferred from radiological assessment. For instance, a sclerotic, well-defined lesions with no periosteal reaction (like in the case of osteoma) will not show an aggressive behavior which is instead to be expected if new bone tissue with an osteoid matrix and triangular spicule-like periosteal reaction is found, like in the case of osteosarcoma.

Radiological features need to be associated with patient age and location so that differential diagnosis can be processed down to a few, perhaps more frequent, histotypes (Kaplan et al. 1994). Small intra-articular loose calcified bodies may therefore reveal pigmented villonodular synovitis (PVNS), pseudogout, or rice-body rheumatoid arthritis (Romañach et al. 2011).

Non-aggressive benign lesions will require CT scan which may be carried out as CBCT, as long as the Field of View is sufficient. MRI may further be required to have an overview on soft tissues and joint function whenever the expanding mass is thought to have an effect on joint mechanics. In such an instance, MRI might as well be of the TMJ only, with no intravenous contrast (Boeddinghaus and Whyte 2008).

The most significant features of benign tumors are outlined below however, as a general rule, osteochondroma and osteoma show regular cancellous bone or compact bone (*osteosclerosis*), whereas ossifying fibroma, chondroblastoma, and osteoblastoma have radiotransparent areas (*osteolysis*) within which osteosclerosis can also be found.

9.1.2 Osteochondroma

Osteochondroma (OC) is a benign, slowly growing tumor also known as osteocartilaginous exostosis (Koga et al. 2006). It presents like an exophytic mass with a broad base, usually extending anteromedially in the direction of the upper belly of the lateral pterygoid muscle. It causes condylar enlargement and deformity (Fig. 9.1a-e). Cortical bone is usually macroscopically irregular but preserved, and cancellous bone may show some trabecular thinning. As a rule, a cartilaginous cap is covering this bony outgrowth and this helps in the differential diagnosis with osteoma (Unni 2010; Sekhar and Loganathan 2015; Mehra et al. 2016). Other conditions may be confused with this benign tumor: smaller osteochondromas may at times resemble an osteophyte (Fig. 9.2a-c) or a healed condylar fracture. Non-ossifying fibroma, much like other expanding fibrous bone lesions, can also cause condylar enlargement, however it differs from osteochondroma in that it displays newly formed bone with partial ossification.

9.1.3 Osteoma

TMJ osteoma is a rare, benign, slowly growing tumor composed of either cortical or cancellous bone (Almeida and de Oliveira Filho 2011; de Souza et al. 2017). It can arise as a single mass, and as such it is less frequent than osteochondroma, or more rarely, it is part of Gardner syndrome (familial adenomatous polyposis), as patients affected may present multiple craniofacial osteomas.



Fig. 9.1 Voluminous osteochondroma (*white arrow*) of the left mandibular condyle, which has caused deformity and an increase in size of the condyle with respect to the contralateral in sagittal SE PD (\mathbf{a}), axial SE T1 (\mathbf{b}), and coronal SE T2 (\mathbf{c}) images. Condylar deformity is more prominent at the medial pole as seen on the coronal plane both in MR (\mathbf{c}) and in CBCT (\mathbf{d}). The pars intermedia

shows a wide perforation causing the disc to be ringshaped. The disc surrounds the condyle (*black arrows*), and movement is preserved, as demonstrated by the openmouth sequence (e). The articular surfaces of condyle and glenoid cavity are irregular. The fossa appears reshaped and it shows slight subchondral sclerosis



Fig. 9.2 Small osteochondroma (*white arrows*) of the medial pole of the left condyle. The lesion develops anterocranially (sagittal SE PD **a**-axial SE T1 **b**) along the

It presents as a well-defined mass made of compact bone, with round margins originating from the condylar surface. It is easily distinguishable from osteochondroma because of its sclerotic, well-defined, and radiopaque appearance (Bessho et al. 1987; Nah 2011).

9.1.4 Ossifying Fibroma

Ossifying Fibroma (OF) is defined as a benign tumor, with defined margins, made up of a fibrous

superior head of the lateral pterygoid muscle. In a SE T2 coronal sequence (c) the small osteochondroma is similar in appearance to an osteophyte of the medial pole

tissue and variably calcified areas (Barnes et al. 2005; Zavattero et al. 2013). Within the head and neck, this tumor is mostly found in the molar region of the mandibular body as differentiation of bone and cementum has an odontogenic significance here and in tooth-bearing areas. Cementum-like elements may actually be present among the mineralized component of OF and this led in the past to the name *cemento-ossifying fibroma*. OFs are diagnosed between the second and the fourth decade of life, with a female predominance. In the TMJ, OF is rare, slow-growing,

and composed of bone at different maturation stages together with a fibrous component (Brannon and Fowler 2001). The growth pattern has a tendency to cortical expansion as well as a local aggressiveness.

Differential diagnosis includes fibro-osseous conditions such as fibrous dysplasia.

9.1.5 Chondroblastoma

Chondroblastoma with TMJ involvement has been described, but it represents an extremely rare occurrence. Origin from within the TMJ is even more rare (Bui et al. 2009).

This tumor has a cartilaginous origin, it contains multinucleated giant cells, and it presents as an oval radiotransparent mass with thin defined margins (Toro et al. 2005). Its behavior is nonaggressive, however, it may recur after resection. More aggressive behavior with bone resorption has also been reported in the literature (Kim et al. 2015).

Extracranial localizations of this tumor are known to be particularly painful and occurring at a young age, whereas the few reports of TMJ chondroblastoma usually indicate painless, older patients (Toro et al. 2005).

9.2 Malignant Tumors

Signs and symptoms of TMJ malignant tumors show a rapid onset swelling and pain in the preauricular area combined with lower jaw hypomobility, trismus, and changes in occlusion. Facial asymmetry is a common sign, however other features, such as neurological motor or sensory impairment, may arise as well and they indicate involvement of adjacent structures.

9.2.1 Imaging of Malignant Tumors

When CBCT or TMJ MRI reveal aggressive bone lesions, further contrast CT or contrast MRI must be carried out according to head and neck tumors protocols (Juliano and Moonis 2018). Imaging techniques must be able to deliver necessary information regarding biopsy procedures, staging, and surgical resection (Boeddinghaus and Whyte 2008).

9.2.2 Chondrosarcoma

Chondrosarcoma (CHS) is a rare malignant mesenchymal tumor derived from cartilage cells. Head and neck chondrosarcomas represent up to 12% of all chondrosarcomas (Oh et al. 2016).

The tumor mass is generally slow-growing, it can cause condylar erosion and affect adjacent structures because of tumor growth.

CHS may be classified as either primitive or secondary, depending on whether it develops from otherwise healthy bone or from pre-existing lesions (enchondroma, osteochondroma) (Garzino-Demo et al. 2010; Giorgione et al. 2015; Macintosh et al. 2015).

Depending on type (endosseous or peripheral CHS) and grading, CT and MRI appearance may vary sensibly. Grade I CHS will show areas of cartilage matrix with calcifications, from Grade II to Grade III such areas extend and their signal characteristics get more and more different from those of cartilage. Contrast-enhancement in grade I CHS is peripheral and ring-and-arc-like, whereas it spreads out and intensifies in higher, more aggressive grades.

Concerning TMJ MRI, it is important to distinguish cartilage of synovial chondromatosis, for instance, from newly formed cartilage of a grade I CHS. Synovial chondromatosis occupies the joint space and may cause joint surface erosion. Differential diagnosis of CHS is therefore essentially made with synovial chondromatosis, chondroblastic sarcoma, osteogenic sarcoma, and metastases (Angiero et al. 2007; Oliveira et al. 2009; Macintosh et al. 2015).

9.2.3 Osteosarcoma

Osteosarcoma is a highly malignant tumor which most commonly affects the long bones (Bennett et al. 2000; dos Santos and Cavalcanti 2002). Only 5–6% of all osteosarcomas arise in the jaws, making TMJ osteosarcoma an extremely rare occurrence (Schwartz and Alpert 1963; Mardinger et al. 2001; Zorzan et al. 2001). It is a mesenchymal stem cell tumor and it produces immature bone. Predisposing factors associated with the tumor are: pre-existing lesions (bone cysts), fibrous dysplasia, previous radiation therapy, osteogenesis imperfecta, genetic factors, and viral agents (August et al. 1997; Van Es et al. 1997; Mardinger et al. 2001; Zorzan et al. 2001; dos Santos and Cavalcanti 2002).

The mass grows rather rapidly and it easily involves the surrounding anatomical regions (ears, salivary glands, medial cranial fossa, infratemporal fossa, zygomatic and temporal bone). TMJ osteosarcomas tend to metastasize later compared to those of other bones (Wang et al. 2012).

Differential diagnosis includes metastatic disease, chondrosarcoma, aneurysmal bone cyst.

Imaging features in both CT and MRI depend upon histotype. Osteosarcoma has been differentiated into chondroblastic, fibroblastic, osteoblastic, and telangiectatic osteosarcoma. Rare telangiectatic forms will show a purely lytic lesion with fluid levels, while osteoblastic osteosarcoma exhibits an osteoblastic ivory area with intense calcified spicule-like periosteal reaction.

9.2.4 Metastasis

Metastatic disease of the TMJ often presents with nonspecific symptoms. There may be pain, swelling, pathological fracture, some degree of TMJ dysfunction, occlusal changes, hearing loss, facial asymmetry, trismus, and trigeminal paresthesia (Kruse et al. 2010). Hematogenous dissemination of tumors to this area is indeed very rare. In most cases there is a well-known medical history of tumor, however in a limited number of cases, joint disease will lead to the discovery of an unknown primary disease (Qiu et al. 2013; Guarda-Nardini et al. 2017).

Most common primary malignancies metastasizing to TMJ include breast, lung, prostate, kidneys, rectum, and stomach (Smolka et al. 2004; Miles et al. 2006; Katsnelson et al. 2010; Freudlsperger et al. 2012). Rareness may be explained by a relatively poor quantity of hematopoietic marrow as opposed to other parts of the jaws (such as the mandibular molar area) which are instead more commonly affected by metastatic tumors (Porter et al. 1996; Kruse et al. 2010; Matsuda et al. 2017).

9.3 Tumor-Like Lesions

Tumor-like lesions of the TMJ can potentially occupy the joint space because of their expansive behavior (Clayman 2006). Due to their prognosis as well as signs and symptoms, they can be compared to benign tumors.

9.3.1 Imaging of Tumor-Like Lesions

Cystic lesions of the condyle appear radiotransparent in orthopantomograms and hypodense in CT. Because of their nature, they will appear hyperintense in T2 and STIR MRI sequences, and hypointense in T1. In general, tumor-like lesions follow the same imaging principles of benign tumors and differential diagnosis will often take these into consideration. Cystic lesions are also to be distinguished from subchondral bone cysts that instead belong to degenerative joint disease or rheumatological conditions. True cystic lesions most probably have distinct bone walls that can better be described by CT scan rather than MRI. Among these, aneurysmal bone cyst expands like a cystic void whose cortex remarkably thins out as growth continues centrifugally, leading to a discontinuous aspect of the cortical bone itself. Fluid levels can be detected in MRI.

9.3.2 Synovial Chondromatosis

Synovial chondromatosis (SC) is a rare, benign and chronic cartilaginous metaplasia of the mesenchymal remnants of the synovial tissue. Cartilaginous nodules are formed and they are either pedunculated or detached from the synovial membrane, thus becoming loose bodies within the joint space (Milgram 1977). SC usually affects other synovial joints like knee and elbow, but it has been described at the TMJ in many instances (almost 200 cases), with a slightly increased female-to-male ratio and a mean age of 46 years (Lieger et al. 2007; Pinto Jr et al. 2015). Most cases are unilateral but a few bilateral cases have also been reported (Keogh et al. 2002; von Lindern et al. 2002; Guarda-Nardini et al. 2010, Guijarro-Martinez et al. 2011; Shah et al. 2011; Pau et al. 2014).

Etiology of SC is not clearly understood. SC is subdivided into primary SC (not associated with causal factors and considered as more aggressive) and secondary SC (related to a previous trauma/microtrauma or degenerative disease) (Holmlund et al. 2003; Guarda-Nardini et al. 2010). Three phases describe the evolution pattern of SC: in the first stage a metaplasia of the synovial membrane takes place with a proliferation of undifferentiated cells and without free bodies, in the intermediate phase there is a presence of metaplastic nodules with loose bodies, and the third stage is characterized by multiple loose calcified bodies without synovial activity (Milgram 1977; Blankestijn et al. 1985).

The most important imaging finding for SC is the presence of cartilage nodules both in orthopantomogram and CT scan. Calcifications are numerous within the nodules' outer parts (Fig. 9.3a–d). They may present in various sizes and they will cause capsule proliferation and growth. MRI will show the typical cartilage hyperintensity in T2 and hypointensity in T1 if their size is relevant and if they are not largely calcified (Levine et al. 2016). Joint effusion is usually present. As surgical resection is mandatory, MRI allows to thoroughly carry out preoperative assessment of the involvement of adjacent structures such as the middle cranial fossa (Lim et al. 2011). Differential diagnosis includes osteoarthritis, osteochondroma, chondrocalcinosis, pigmented villonodular synovitis, and osteochondritis dissecans (Balasundaram et al. 2009).

9.3.3 Pigmented Villonodular Synovitis

Pigmented villonodular synovitis (PVNS) is a rare, benign but locally aggressive mass which derives from TMJ synovial membrane. Around 100 cases of PVNS affecting TMJ have been reported (Verspoor et al. 2018).

This condition seems to be due to a chronic inflammatory response to an unknown stimulus, or to repetitive joint hemorrhage following trauma (Granowitz et al. 1976; Song et al. 1999; Herman et al. 2009; Damodar et al. 2015).The tumor develops in tendons and joints and it is characterized by a thick synovial membrane with villi and folds or pedunculated nodules (Nomura et al. 2018). Its name is also due to a peculiar hemosiderin deposition which gives a redbrownish color.

As the tumor grows, it can involve surrounding TMJ structures such as the cranial base and the ear canal.

MRI gradient echo sequences will show ferromagnetic artifacts due to hemosiderin deposits. Because of these and because of fibrous tissue and increased cellularity, an overall hypointensity will be found in all sequences, T2 and fatsuppression included.

Differential diagnosis includes tuberculosis septic arthritis, malignant synovioma, hemophilia, rheumatoid arthritis, gout, synovial chondromatosis, parotid tumor, and carcinoma of the external and middle ear.

9.3.4 Aneurysmal Bone Cyst

Aneurysmal bone cyst (ABC) is a rare, osteolytic, pseudocystic, osseous lesion that rarely affects the condyle (unilaterally). Very few cases of condylar ABC have been reported, and other also uncommon oral-maxillofacial cases have been described in young patients under 20 years of age (Kransdorf and Sweet 1995; Motamedi et al. 2008).

Etiology and pathogenesis are still unclear although a history of trauma is often reported (Motamedi 2002; Motamedi et al. 2008). Theories include reactive vascular malformation, vascular



Fig. 9.3 Synovial chondromatosis of the right TMJ. Orthopantomogram (**a**) shows multiple small radiotransparent areas in the joint cavity, which are surrounded by calcific margins (*white arrow*). Reformatted axial (**b**) and coronal (**c**) CT scans also display the same globular struc-

tures (*white arrow*) with calcified walls which almost entirely occupy the articular space. Gradient Echo coronal sequence (**d**) shows the calcifications as clearly hypointense punctiform images (*arrow*). Courtesy of Prof Ambrosina Michelotti

incidents within an existing lesion, and genetic predisposition (Cottalorda and Bourelle 2007; Motamedi et al. 2008).

Although benign in nature (they commonly show a slow growth), the lesion can expand rapidly and can be locally both destructive and expansive (Mankin et al. 2005; Liu et al. 2017).

The lesion may appear as a unicystic or multilocular, with an expanding behavior which sometimes extends to soft tissues. Perforation of cortical bone may accompany ABC growth (Motamedi et al. 2008).

9.3.5 Ganglion Cyst

Ganglion cyst is a benign lesion that occurs near joints (wrist, foot, shoulder, and knee are the most affected sites) and rarely affects the TMJ. Unlike synovial cysts, which are true cysts lined with endothelial cells and filled with synovial fluid, ganglion cysts are not related to the joint space and they contain a highly viscous gelatinous protein material with an acellular fibrous tissue inner lining (Algharib et al. 2015).

Etiopathogenetic explanation for this pseudocyst is that it develops from a myxoid degeneration of the collagenous tissue of the joint capsule (Goudot et al. 1999; Silva et al. 2005; Ali et al. 2006; Lee et al. 2014; Algharib et al. 2015; Levarek and Nolan 2016).

Most cases are unilateral and unilocular. Differential diagnosis includes synovial cysts, salivary gland tumor, sebaceous cysts, and condyle malignancies (Yanagi et al. 2003; Silva et al. 2005; Solomon et al. 2008; Algharib et al. 2015).

9.3.6 Calcium Pyrophosphate Dihydrate Deposition Disease

Calcium pyrophosphate dihydrate deposition disease (CPDD), also known as *pseudogout*, is a rare crystalline arthropathy caused by the deposition of calcium pyrophosphate crystals in the joints (Grant et al. 1999; Steinbach and Resnick 1996). Abnormal deposition of pyrophosphate on collagen fibers activates the release of inflammatory mediators, which cause an articular damage known as *chondrocalcinosis* (Marsot-Dupuch et al. 2004).

CPDD arthropathy is often associated with other medical conditions including trauma, gout, hemochromatosis, hypomagnesemia, hypophosphatemia, hypercalcemia, hyperparathyroidism, Wilson's disease, and alkaptonuria (Steinbach and Resnick 1996). It may sporadically occur on a hereditary basis too (Abhishek and Doherty 2016).

Causes of chondrocalcinosis are not currently known. The fact that pseudogout is frequently associated with other pathological conditions and with an advanced age (about 50% of subjects over 85 years of age are affected by chondrocalcinosis) suggests that the deposition of calcium pyrophosphate crystals are secondary to the degenerative or metabolic phenomena of the affected tissues (Steinbach and Resnick 1996; Marsot-Dupuch et al. 2004; Reynolds et al. 2008).

CPPD may be associated with various chronic and acute symptoms or it may be unnoticed (McCarty and Hollander 1961; Molloy and McCarthy 2006). The joints most commonly affected are the shoulder, knee, hip, elbow, and metacarpophalangeal joints, while axial skeleton involvement (both spinal and cranial) is less common (Grant et al. 1999; Steinbach and Resnick 1996; Marsot-Dupuch et al. 2004; Reynolds et al. 2008). TMJ involvement has been reported to present with pain, swelling, and hearing loss (Marsot-Dupuch et al. 2004; Scavarda et al. 2007).

Several clinical presentations of CPPD in the joints exist, which include totally asymptomatic to acute inflammatory arthritis (pseudogout), chronic degenerative arthritis (pseudo-osteoarthritis), or chronic symmetric inflammatory polyarthritis (pseudorheumatoid arthritis) (Reynolds et al. 2008). Presentation of CPDD in the form of a "tumor-like" mass has been indicated with the term tophaceous pseudogout. This is a clinical variant of CPPD, associated with swelling and pain of involved joints. Tophaceous pseudogout is more frequently found in large joints. Small joints such as the TMJ can be affected too, albeit more rarely (McCarty and Hollander 1961; Scott 1978; Resnick and Niwayama 1988; Ishida et al. 1995; Kurihara et al. 1997; Reynolds et al. 2008; Aoyama et al. 2000). CPPD of the TMJ may mimic tumors, gout, and synovial chondromatosis both clinically and radiographically and an open biopsy is usually recommended to make a correct diagnosis (Kurihara et al. 1997; Aoyama et al. 2000; Barthelemy et al. 2001; Marsot-Dupuch et al. 2004).

Highest specificity imaging for calcium pyrophosphate dihydrate deposition disease (CPDD) is provided by CT, which is capable of showing a large fossa-occupying mass with small synovial calcifications made of calcium pyrophosphate dihydrate (CPP) crystals. Again, because of these crystals, an inflammatory response is triggered with activation of neutrophils and macrophages and release of inflammatory mediators (Srinivasan 2012). Special aggressiveness of this tissue can lead to erosion of the temporal bone, of the greater wing of the sphenoid bone and of the mastoid air cells (Fig. 9.4a–c) (Srinivasan 2012; Kudoh et al. 2017).

Although MRI can well define CPDD TMJ synovitis, it cannot show the small synovial calcifications that expand the joint and erode bone nearby as precisely as CT scan (Fig. 9.4a-e). As the whole joint is occupied by an expanding mass, chondrocalcinosis of the TMJ is usually to be differentially diagnosed from synovial chondromatosis and chondrosarcoma (Kudoh et al. 2017). Synovial chondromatosis is characterized by a diffusely hypointense synovial tissue (in T1-T2 weighted images), that occupies the joints space, or by globular formations (Murphey et al. 2007). The latter have different shape and size according to the degree of synovial cartilage metaplasia and synovial ossification. Chondromatosis free bodies usually present with larger calcifications than those seen in CPDD (Abdelsayed et al. 2014).

9.4 Clinical Presentation and Management

TMJ tumors show nonspecific symptoms and may mimic other TMJ disorders. Signs and symptoms are related to tumor dimension and its main vector of growth. As the tumor enlarges, subjects can go from being asymptomatic to complaining about limitation during jaw movements, pain, ipsilateral open-bite, chin and midline deviation toward the non-affected side, swelling in the preauricular region and joint sounds (Siar et al. 2004, Dhirawani et al. 2014). In larger tumors, an increase in vertical dimension of the condyle may also lead to remarkable progressive facial asymmetry. Because of this, clinical suspicion of more common conditions such as condylar hyperplasia is justified (Wolford et al. 2014).

In locally more aggressive histotypes, the mass can cause swelling, bone erosion, hearing impairment, skin auricular symptoms (otalgia, hearing loss, tinnitus, otitis media, otorrhea), preauricular paresthesia, and facial nerve palsy (Albright et al. 2000; Ali et al. 2006; Mumert et al. 2012; Roman-Ramos et al. 2017).

Pathological fractures of the condyle may occur and they have been described mostly in case of malignancies and cystic lesions (Motamedi 2002; Jia et al. 2006).

Treatment choice depends on histotype, tumor extension, local aggressiveness, and on patient characteristics such as degree of impairment and age. Surgical therapy allows for final diagnostic histopathology and it may range from simple curettage to resection and TMJ reconstruction (Wu et al. 2011; Savolainen and Kellokoski 2013; Liu et al. 2017).

For less aggressive, slow-growing tumors, surgical excision is generally the most common approach, although for small indolent lesions a wait-and-see strategy may be used (Chandu et al. 1997; do Egito Vasconcelos et al. 2007).

For particular tumor-like lesions, arthroscopy may prove useful both for diagnosis and treatment. However, if mass growth is significant or the number and size of loose bodies are unfavorable, open surgery is the best option (Guarda-Nardini et al. 2010).

For more aggressive, larger tumors, resective surgery is required followed by TMJ reconstruction (Zachariades 1989; Zavattero et al. 2013; Jiao et al. 2015; Mehra et al. 2016). When indicated, resection has to be wide enough to improve prognosis and avoid recurrences (Garzino-Demo et al. 2010). Nowadays wide resections are not as disabling as they used to be, given that reconstructive techniques have evolved and have been implemented. Adjuvant therapies (radio- immuno- and chemotherapy) may also be needed.

Fig. 9.4 CT images showing deposits of calcium pyrophosphate crystals within the synovial tissue which occupy the joint (**a**, *b*, *star*). Erosions involve the condyle (**a**, *arrow*), the temporal bone (**b**, **c**, *arrows*), and the middle ear (**b**, *star*). Sagittal Gradient Echo T2 fat sat

(d, arrow) and coronal SE T2 (e, arrow) both show synovitis within the joint space. Calcifications, that are typically well visible in CT scans, can be seen as hypointense bodies in Gradient Echo sequences (d)



References

- Abdelsayed RA, Said-Al-Naief N, Salguerio M, Holmes J, El-Mofty SK. Tophaceous pseudogout of the temporomandibular joint: a series of 3 cases. Oral Surg Oral Med Oral Pathol Oral Radiol. 2014;117:369–75. https://doi.org/10.1016/j.oooo.2013.12.007.
- Abhishek A, Doherty M. Update on calcium pyrophosphate deposition. Clin Exp Rheumatol. 2016;34:32–8.
- Al-Jamali JM, Voss PJ, Bayazzed BA, Spanou A, Otten JE, Schmelzeisen M. Malignant tumors could be misinterpreted as temporomandibular joint disorders. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2013;116:e362–7. https://doi.org/10.1016/j. 0000.2012.01.039.
- Albright JT, Diecidue RJ, Amritpal J, Keane WM. Intraosseous ganglion of the temporomandibular joint presenting with otorrhea. Arch Otolaryngol Head Neck Surg. 2000;126:665–8. https://doi.org/10.1001/ archotol.126.5.665.
- Algharib A, Parekh J, Sultan A, Hopper C. A rare case of a ganglionic cyst of the TMJ—case report and review of the literature. J Oral Maxillofac Surg Med Pathol. 2015;27(3):385–9. https://doi.org/10.1016/j. ajoms.2014.06.002.
- Ali ZA, Busaidy KF, Wilson J. Unusual presentation of a ganglion cyst of the temporomandibular joint: case report and distinction from synovial cyst. J Oral Maxillofac Surg. 2006;64:1300–2. https://doi. org/10.1016/j.joms.2006.04.013.
- Almeida LE, de Oliveira Filho MA. Giant mandibular condyle osteoma. J Craniofac Surg. 2011;22:1147–9. https://doi.org/10.1097/SCS.0b013e318210baee.
- Angiero F, Vinci R, Sidoni A, Stefani M. Mesenchymal chondrosarcoma of the left coronoid process: report of a unique case with clinical, histopathologic, and immunohistochemical findings, and a review of the literature. Quintessence Int. 2007;38:349–55.
- Aoyama S, Kino K, Amagasa T, Kayano T, Ichinose S, Kimijima Y. Differential diagnosis of calcium pyrophosphate dihydrate deposition of the temporomandibular joint. Br J Oral Maxillofac Surg. 2000;38:550–3. https://doi.org/10.1054/bjom.2000.0313.
- August M, Magennis P, Dewitt D. Osteogenic sarcoma of the jaws: factors influencing prognosis. Int J Oral Maxillofac Surg. 1997;26:198–204. https://doi. org/10.1016/S0901-5027(97)80819-3.
- Balasundaram A, Geist JR, Gordon SC, Klasser GD. Radiographic diagnosis of synovial chondromatosis of the temporomandibular joint: a case report. J Can Dent Assoc. 2009;75:711–4.
- Barnes L, Eveson JW, Reichart P, Sidransky D, World Health Organization. Classification of tumours. Pathology and genetics of head and neck tumours. Lyon: IARC Press; 2005. p. 319.
- Barthelemy I, Karanas Y, Sannajust J-P, Emering C, Mondie J-M. Gout of the temporomandibular joint: pitfalls in diagnosis. J Craniomaxillofac Surg. 2001;29:307–10. https://doi.org/10.1054/jcms.2001.0244.

- Bennett JH, Thomas G, Evans AW, Speight PM. Osteosarcoma of the jaws: a 30-year retrospective review. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2000;90:323–32. https://doi.org/10.1067/ moe.2000.108274.
- Bessho K, Murakami KI, Iizuka T, Ono T. Osteoma in mandibular condyle. Int J Oral Maxillofac Surg. 1987;16:372–5. https://doi.org/10.1016/ S0901-5027(87)80162-5.
- Blankestijn J, Panders AK, Vermey A, Scherpbier AJ. Synovial chondromatosis of the temporomandibular joint. Report of three cases and a review of the literature. Cancer. 1985;55:479–85. https://doi. org/10.1002/1097-0142(19850115)55:2<479::AID-CNCR2820550232>3.0.CO;2-N.
- Boeddinghaus R, Whyte A. Current concepts in maxillofacial imaging. Eur J Radiol. 2008;66:396–418. https://doi.org/10.1016/j.ejrad.2007.11.019.
- Brannon RB, Fowler CB. Benign fibro-osseous lesions: a review of current concepts. Adv Anat Pathol. 2001;8:126–43. https://doi. org/10.1097/00125480-200105000-00002.
- Bui P, Ivan D, Oliver D, Busaidy KF, Wilson J. Chondroblastoma of the Temporomandibular joint: report of a case and literature review. J Oral Maxillofac Surg. 2009;67:405–9. https://doi.org/10.1016/j.joms.2008.01.043.
- Chandu A, Spencer JA, Dyson DP. Chondroma of the mandibular condyle: an example of a rare tumour. Dentomaxillofac Radiol. 1997;26:242–5. https://doi. org/10.1038/sj.dmfr.4600243.
- Clayman L. Surgical management of benign and malignant neoplasms. In: Laskin DM, Greene CS, Hylander WL, editors. TMDs. An evidence-based approach to diagnosis and treatment. Chicago: Quintessence Publishing; 2006. p. 509–32.
- Cottalorda J, Bourelle S. Modern concepts of primary aneurysmal bone cyst. Arch Orthop Trauma Surg. 2007;127:105–14. https://doi.org/10.1007/ s00402-006-0223-5.
- Damodar D, Chan N, Kokot N. Pigmented villonodular synovitis of the temporomandibular joint: case report and review of the literature. Head Neck. 2015;37:E194–9. https://doi.org/10.1002/ hed.24056.
- de Souza NT, Cavalcante RLC, de Albuquerque Cavalcante MA, Hespanhol W, de Oliveira MR, de Carvalho FD, de Carvalho Coutinho TM, Gonçalves LS. An unusual osteoma in the mandibular condyle and the successful replacement of the temporomandibular joint with a custom-made prosthesis: a case report. BMC Res Notes. 2017;10:727. https://doi. org/10.1186/s13104-017-3060-4.
- Dhirawani RB, Anand K, Lalwani G, Pathak S, Thakkar B. True chondroma of the mandibular condyle: a rare case. Ann Maxillofac Surg. 2014;4:220–3. https://doi.org/10.4103/2231-0746.147152.
- do Egito Vasconcelos BC, Porto GG, Bessa-Nogueira RV. Rare benign tumors of the mandibular condyle: report of 2 cases and literature review. J Oral

Maxillofac Surg. 2007;65:1830–5. https://doi. org/10.1016/j.joms.2006.06.262.

- dos Santos DT, Cavalcanti MG. Osteosarcoma of the temporomandibular joint: report of 2 cases. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2002;94:641–7. https://doi.org/10.1067/moe.2001.129181.
- Freudlsperger C, Kurth R, Werner MK, Hoffmann J, Reinert S. Condylar metastasis from prostatic carcinoma mimicking temporomandibular disorder: a case report. Oral Maxillofac Surg. 2012;16:79–82. https:// doi.org/10.1007/s10006-010-0256-3.
- Garzino-Demo P, Tanteri G, Boffano P, Ramieri G, Pacchioni D, Maletta F, Bianchi CC, Bianchi SD, Berrone S. Chondrosarcoma of the temporomandibular joint: a case report and review of the literature. J Oral Maxillofac Surg. 2010;68:2005–11. https://doi. org/10.1016/j.joms.2009.09.077.
- Giorgione C, Passali FM, Varakliotis T, Sibilia M, Ottaviani F. Temporo-mandibular joint chondrosarcoma: case report and review of the literature. Acta Otorhinolaryngol Ital. 2015;35:208–11.
- Goudot P, Jaquinet R, Richter M. Cysts of the temporomandibular joint. report of two cases. Int J Oral Maxillofac Surg. 1999;28:338–40. https://doi. org/10.1034/j.1399-0020.1999.285280504.x.
- Granowitz SP, D'Antonio J, Mankin HL. The pathogenesis and long-term and results of pigmented villonodular synovitis. Clin Orthop Relat Res. 1976;114:335–51.
- Grant GA, Wener MH, Yaziji H, Futran N, Bronner MP, Mandel N, et al. Destructive tophaceous calcium hydroxyapatite tumor of the infratemporal fossa. Case report and review of the literature. J Neurosurg. 1999;90:148–52. https://doi.org/10.3171/ jns.1999.90.1.0148.
- Guarda-Nardini L, Piccotti F, Ferronato G, Manfredini D. Synovial chondromatosis of the temporomandibular joint: a case description with systematic literature review. Int J Oral Maxillofac Surg. 2010;39:745–55. https://doi.org/10.1016/j.ijom.2010.03.028.
- Guarda-Nardini L, Stellini E, Di Fiore A, Manfredini DA. Rare case of misdiagnosed silent lung cancer with solitary metastasis to the temporomandibular joint condyle. J Oral Facial Pain Headache. 2017;2:180–5. https://doi.org/10.11607/ofph.1672.
- Guijarro-Martinez R, Puche Torres M, Marqués Mateo MM, Solìs Garcia I, Miragall Alba L, Iglesias Gimilio ME, et al. Bilateral synovial chondromatosis of the temporomandibular joint. J Craniomaxillofac Surg. 2011;39:261–5. https://doi.org/10.1016/j. jcms.2010.04.016.
- Harmon M, Arrigan M, Toner M, O'Keeffe SA. A radiological approach to benign and malignant lesions of the mandible. Clin Radiol. 2015;70:335–50. https:// doi.org/10.1016/j.crad.2014.10.011.
- Herman CR, Swift JQ, Schiffman EL. Pigmented villonodular synovitis of the temporomandibular joint with intracranial extension: a case and literature review. Int J Oral Maxillofac Surg. 2009;38:795–801. https://doi. org/10.1016/j.ijom.2009.02.013.

- Holmlund AB, Eriksson L, Reinholt FP. Synovial chondromatosis of the temporomandibular joint: clinical, surgical and histological aspects. Int J Oral Maxillofac Surg. 2003;32:143–7. https://doi.org/10.1054/ ijom.2002.0300.
- Ishida T, Dorfman HD, Bullough PG. Tophaceous pseudogout (tumoral calcium pyrophosphate dihydrate crystal deposition disease). Hum Pathol. 1995;26:587–93. https://doi.org/10.1016/0046-8177(95)90161-2.
- Jia J, Zhang WF, Liu B, Zhao YF. Pathological fracture of condyle from metastatic breast adenocarcinoma. Oral Oncol Extra. 2006;42(3):98–100. https://doi. org/10.1016/j.ooe.2005.09.009.
- Jiao Z, Abdelrehem A, Zhang SY. Ossifying fibroma in the temporomandibular joint: report of an unusual case and treatment perspectives. Int J Oral Maxillofac Surg. 2015;44:1362–7. https://doi.org/10.1016/j. ijom.2015.06.017.
- Juliano A, Moonis G. Computed tomography versus magnetic resonance in head and neck Cancer: when to use what and image optimization strategies. Magn Reson Imaging Clin N Am. 2018;26:63–84. https:// doi.org/10.1016/j.mric.2017.08.005.
- Kaplan I, Calderon S, Buchner A. Peripheral osteoma of the mandible: a study of 10 new cases and analysis of the literature. J Oral Maxillofac Surg. 1994;52:467– 70. https://doi.org/10.1016/0278-2391(94)90342-5.
- Katsnelson A, Tartakowsky JW, Miloro M. Review of the literature for mandibular metastasis illustrated by a case of lung metastasis to the temporomandibular joint in an HIV-positive patient. J Oral Maxillofac Surg. 2010;68:1960–4. https://doi.org/10.1016/j. joms.2009.07.075.
- Keogh CF, Torreggiani WC, Munk PL. Bilateral synovial chondromatosis of the temporomandibular joint. Clin Radiol. 2002;57:862. https://doi.org/10.1053/ crad.2002.0987.
- Kim SM, Hong SW, Ryu DJ, Huh JK. Chondroblastoma of the temporomandibular joint lateral capsule: a case report. Cranio. 2015;33:306–11. https://doi.org/10.10 80/08869634.2015.1097305.
- Koga M, Toyofuku S, Nakamura Y, Yoshiura K, Kusukawa J, Nakamura Y. Osteochondroma in the mandibular condyle that caused facial asymmetry: a case report. Cranio. 2006;24:67–70. https://doi.org/10.1179/ crn.2006.011.
- Kransdorf MJ, Sweet DE. Aneurysmal bone cyst: concept, controversy, clinical presentation, and imaging. AJR Am J Roentgenol. 1995;164:573–80. https://doi. org/10.2214/ajr.164.3.7863874.
- Kruse AL, Luebbers HT, Obwegeser JA, Edelmann L, Graetz KW. Temporomandibular disorders associated with metastases to the temporomandibular joint: a review of the literature and 3 additional cases. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2010;110:e21–8. https://doi.org/10.1016/j. tripleo.2010.02.031.
- Kudoh K, Kudoh T, Tsuru K, Miyamoto Y. A case of tophaceous pseudogout of the temporomandibular joint extending to the base of the skull. Int J

Oral Maxillofac Surg. 2017;46:355–9. https://doi. org/10.1016/j.ijom.2016.08.018.

- Kurihara K, Mizuseki K, Saiki T, Wakisaka H, Maruyama S, Sonobe J. Tophaceous pseudogout of the temporomandibular joint: report of a case. Pathol Int. 1997;47:578–80. https://doi. org/10.1111/j.1440-1827.1997.tb04544.x.
- Lee YT, Kwon SB, Cho SH, Eo S, Rhee SC. A ganglion cyst of the temporomandibular joint. Arch Plast Surg. 2014;41:777–80. https://doi.org/10.5999/ aps.2014.41.6.777.
- Levarek RE, Nolan PJ. Temporomandibular joint ganglion cyst: a unique case of complete resolution following subtotal excision. J Oral Maxillofac Surg. 2016;74:1783–91. https://doi.org/10.1016/j. joms.2016.02.022.
- Levine BD, Motamedi K, Seeger LL. Synovial tumors and proliferative diseases. Rheum Dis Clin N Am. 2016;42:753–68. https://doi.org/10.1016/j. rdc.2016.07.008.
- Lieger O, Zix J, Stauffer-Brauch EJ, Iizuka T. Synovial chondromatosis of the temporomandibular joint with cranial extension: a case report and literature review. J Oral Maxillofac Surg. 2007;65:2073–80. https://doi. org/10.1016/j.joms.2006.04.039.
- Lim SW, Jeon SJ, Choi SS, Choi KH. Synovial chondromatosis in the temporomandibular joint: a case with typical imaging features and pathological findings. Br J Radiol. 2011;84:e215–8. https://doi.org/10.1259/ bjr/69067316.
- Liu K, Guo C, Guo R, Meng J. A giant aneurysmal bone cyst in the mandibular condyle. J Craniofac Surg. 2017;28:e148–51. https://doi.org/10.1097/ SCS.0000000000003339.
- Macintosh RB, Khan F, Waligora BM. Chondrosarcoma of the temporomandibular disc: behavior over a 28-year observation period. J Oral Maxillofac Surg. 2015;73:465–74. https://doi.org/10.1016/j. joms.2014.09.020.
- McCarty DJ, Hollander JL. Identification of urate crystals in gouty synovial fluid. Ann Intern Med. 1961;54:452– 60. https://doi.org/10.7326/0003-4819-54-3-452.
- Mankin HJ, Hornicek FJ, Ortiz-Cruz E, Villafuerte J, Gebhardt MC. Aneurysmal bone cyst: a review of 150 patients. J Clin Oncol. 2005;23:6756–62. https://doi. org/10.1200/JCO.2005.15.255.
- Mardinger O, Givol N, Talmi YP, Taicher S. Osteosarcoma of the jaw. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2001;91:445–51. https://doi.org/10.1067/ moe.2001.112330.
- Marsot-Dupuch K, Smoker WRK, Gentry LR, Cooper KA. Massive calcium pyrophosphate dihydrate crystal deposition disease: a cause of pain of the temporomandibular joint. AJNR Am J Neuroradiol. 2004;25:876–9.
- Matsuda S, Yoshimura H, Kondo S, Sano K. Temporomandibular dislocation caused by pancreatic cancer metastasis: a case report. Oncol Lett. 2017;14:6053–8. https://doi.org/10.3892/ol.2017.6951.
- Mehra P, Arya V, Henry C. Temporomandibular joint condylar osteochondroma: complete condylectomy and

joint replacement versus low condylectomy and joint preservation. J Oral Maxillofac Surg. 2016;74:911–25. https://doi.org/10.1016/j.joms.2015.11.028.

- Miles BA, Schwartz-Dabney C, Sinn DP, Kessler HP. Bilateral metastatic breast adenocarcinoma within the temporomandibular joint: a case report. J Oral Maxillofac Surg. 2006;64:712–8. https://doi. org/10.1016/j.joms.2005.12.017.
- Milgram JW. The classification of loose bodies in human joints. Clin Orthop Relat Res. 1977;124:282–91. https://doi.org/10.1097/00003086-197705000-00039.
- Molloy ES, McCarthy GM. Calcium crystal deposition diseases: update on pathogenesis and manifestations. Rheum Dis Clin N Am. 2006;32:383–400. https://doi. org/10.1016/j.rdc.2006.02.001.
- Mostafapour SP, Futran ND. Tumors and tumorous masses presenting as Temporomandibular joint syndrome. Otolaryngol Head Neck Surg. 2000;123:459– 64. https://doi.org/10.1067/mhn.2000.109662.
- Motamedi MH. Destructive aneurysmal bone cyst of the mandibular condyle: report of the case and review of the literature. J Oral Maxillofac Surg. 2002;60:1357– 61. https://doi.org/10.1053/joms.2002.35744.
- Motamedi MH, Navi F, Eshkevari PS, Jafari SM, Shams MG, Taheri M, et al. Variable presentations of aneurysmal bone cysts of the jaws: 51 cases treated during a 30-year period. J Oral Maxillofac Surg. 2008;66:2098– 103. https://doi.org/10.1016/j.joms.2008.05.364.
- Mumert ML, Altay T, Shelton C, Hamsberger HR, Couldwell WT. Ganglion cyst of the temporomandibular joint with intracranial extension in a patient presenting with seventh nerve palsy. J Neurosurg. 2012;116:310–2. https://doi.org/10.3171/2011.10. JNS111247.
- Murphey MD, Vidal JA, Fanburg-Smith JC, Gajewski DA. Imaging of synovial chondromatosis with radiologic-pathologic correlation. Radiographics. 2007;27:1465–88. https://doi.org/10.1148/ rg.275075116.
- Nah KS. Osteomas of the craniofacial region. Imaging Sci Dent. 2011;41:107–13. https://doi.org/10.5624/ isd.2011.41.3.107.
- Nomura F, Ariizumi Y, Kiyokawa Y, Tasaki A, Tateishi Y, Koide N, et al. Pigmented villonodular synovitis occurring in the temporomandibular joint. Auris Nasus Larynx. 2018;46:609–17. https://doi.org/10.1016/j. anl.2018.10.021.
- Oh KY, Yoon HJ, Lee JI, Hong SP, Hong SD. Chondrosarcoma of the temporomandibular joint: a case report and review of the literature. Cranio. 2016;34:270–8. https://doi.org/10.1179/21510903 15Y.0000000016.
- Oliveira RC, Marques KD, Mendonca AR, Mendonca EF, Silva MR, Batista AC, et al. Chondrosarcoma of the temporomandibular joint: a case report in a child. J Orofac Pain. 2009;23:275–81.
- Pau M, Bicsak A, Reinbacher KE, Feichtinger M, Kärcher H. Surgical treatment of synovial chondromatosis of the temporomandibular joint with erosion of the skull base: a case report and review of the literature. Int J

Oral Maxillofac Surg. 2014;43:600–5. https://doi. org/10.1016/j.ijom.2013.10.019.

- Pinto AA Jr, Ferreira e Costa R, de Sousa SF, Chagas MR, do Carmo MA, de Lacerda JC. Synovial chondromatosis of the temporomandibular joint successfully treated by surgery. Head Neck Pathol. 2015;9:525–9. https://doi.org/10.1007/s12105-015-0626-0.
- Porter SR, Chaudhry Z, Griffiths MJ, Scully C, Kabala J, Whipp E. Bilateral metastatic spread of testicular teratoma to mandibular condyles. Eur J Cancer B Oral Oncol. 1996;32:359–61. https://doi. org/10.1016/0964-1955(96)00001-2.
- Qiu YT, Yang C, Chen MJ, Qiu WL. Metastatic spread to the mandibular condyle as initial clinical presentation: radiographic diagnosis and surgical experience. J Oral Maxillofac Surg. 2013;71:809–20. https://doi. org/10.1016/j.joms.2012.07.026.
- Resnick D, Niwayama G. Calcium pyrophosphate dihydrate (CPPD) crystal deposition disease. In: Diagnosis of bone and joint disorders. Philadelphia, PA: WB Saunders; 1988.
- Reynolds JL, Matthew IR, Chalmers A. Tophaceous calcium pyrophosphate dihydrate deposition disease of the temporomandibular joint. J Rheumatol. 2008;35:717–21.
- Romañach MJ, Brasileiro BF, León JE, Alves DB, de Almeida OP, Vargas PA. Pigmented villonodular synovitis of the temporomandibular joint: case report and review of the literature. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2011;111:e17–28. https:// doi.org/10.1016/j.tripleo.2010.11.019.
- Roman-Ramos M, Cariati P, Cabello-Serrano A, Garcia-Martin M, Garcia-Medina B. Arthroscopic approach for treating a pigmented villonodular synovitis of TMJ. J Clin Exp Dent. 2017;9:e312–4. https://doi. org/10.4317/jced.53114.
- Savolainen JJ, Kellokoski JK. Ganglion cyst of the temporomandibular joint. Int J Oral Maxillofac Surg. 2013;42:776–9. https://doi.org/10.1016/j. ijom.2012.10.028.
- Scavarda D, Litre CF, Froelich S, Srour R, Rousseaux P. Cervical tumoral calcium pyrophosphate dihydrate deposition disease 28 years after suboccipital craniotomy: case report. Neurosurgery. 2007;60:E1151. https://doi.org/10.1227/01.NEU.0000255477.06247. B8.
- Schwartz DT, Alpert M. The clinical course of mandibular osteogenic sarcoma. Oral Surg. 1963;16:769–76. https://doi.org/10.1016/0030-4220(63)90312-8.
- Scott JT, editor. Copeman's textbook of the rheumatic diseases. 5th ed. Edinburgh: Churchill Livingstone; 1978.
- Sekhar MM, Loganathan S. Giant Osteochondroma of the mandibular condyle. J Oral Maxillofac Pathol. 2015;19:407. https://doi. org/10.4103/0973-029X.174629.
- Shah SB, Ramanojam S, Gadre PK, Gadre KS. Synovial chondromatosis of temporomandibular joint: journey through 25 decades and a case report. J Oral Maxillofac Surg. 2011;69:2795–814. https://doi. org/10.1016/j.joms.2010.12.029.

- Siar CH, Jalil AA, Ram S, Ng KH. Osteoma of the condyle as the cause of limited-mouth opening: a case report. J Oral Sci. 2004;46:51–3. https://doi.org/10.2334/ josnusd.46.51.
- Silva EC, Guimaraes AL, Gomes CC, Gomez RS. Ganglion cyst of the temporomandibular joint. Br J Oral Maxillofac Surg. 2005;43:77–80. https://doi. org/10.1016/j.bjoms.2004.08.016.
- Smolka W, Brekenfeld C, Büchel P, Iizuka T. Metastatic adenocarcinoma of the temporomandibular joint from the cardia of the stomach: a case report. Int J Oral Maxillofac Surg. 2004;33:713–5. https://doi. org/10.1016/j.ijom.2003.10.013.
- Solomon LW, Frustino JL, Loree TR, Brecher ML, Alberico RA, Sullivan M. Ewing sarcoma of the mandibular condyle: multidisciplinary management optimizes outcome. Head Neck. 2008;30:405–10. https:// doi.org/10.1002/hed.20692.
- Song MY, Heo MS, Lee SS, Choi SC, Park TW, Lim CY, et al. Diagnostic imaging of pigmented villonodular synovitis of the temporomandibular joint associated with condylar expansion. Dentomaxillofac Radiol. 1999;28:386–90. https://doi.org/10.1038/sj/ dmfr/4600482.
- Srinivasan V, Wensel A, Dutcher P, Newlands S, Johnson M, Vates GE. Calcium pyrophosphate deposition disease of the temporomandibular joint. J Neurol Surg Rep. 2012 Oct;73(1):6–8. https://doi.org/10.105 5/s-0032-1329190.
- Steinbach LS, Resnick D. Calcium pyrophosphate dihydrate crystal deposition disease revisited. Radiology. 1996;200:1–9. https://doi.org/10.1148/ radiology.200.1.8657894.
- Toro C, Robiony M, Ferro D, Sembronio S, Zerman N, Politi M. Chondroblastoma of the mandibular condyle: case report of an extremely uncommon tumor. Oral Oncol Extra. 2005;41:132–6. https://doi.org/10.1016/j.ooe.2005.03.001.
- Unni KK, Inwards C. Dahlin's bone tumors: general aspects and data on 10,165 cases. 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2010.
- Van Es RJJ, Keus RB, Van Der Waal I, Koole R, Vermey A. Osteosarcoma of the jaw bones: long term follow up of 48 cases. Int J Oral Maxillofac Surg. 1997;26:191–7. https://doi.org/10.1016/ S0901-5027(97)80818-1.
- Verspoor FGM, Mastboom MJL, Weijs WLJ, Koetsveld AC, Schreuder HWB, Flucke U. Treatments of tenosynovial giant cell tumours of the temporomandibular joint: a report of three cases and a review of literature. Int J Oral Maxillofac Surg. 2018;47:1288–94. https:// doi.org/10.1016/j.ijom.2018.04.001.
- von Lindern JJ, Theuerkauf I, Niederhagen B, Bergé S, Appel T, Reich RH. Synovial chondromatosis of the temporomandibular joint: clinical, diagnostic, and histomorphologic findings. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2002;94:31–8. doi. org/10.1067/moe.2002.123498
- Wang S, Shi H, Yu Q. Osteosarcoma of the jaws: demographic and CT imaging features. Dentomaxillofac

Radiol. 2012;41:37–42. https://doi.org/10.1259/ dmfr/86834844.

- Wolford LM, Movahed R, Perez DE. A classification system for conditions causing condylar hyperplasia. J Oral Maxillofac Surg. 2014;72:567–95. https://doi. org/10.1016/j.joms.2013.09.002.
- Wu CI, Liu KW, Hsu YC, Chiang IP, Chang SCN. Treatment of temporomandibular joint ganglion cyst. J Craniofac Surg. 2011;22:1935–7. https://doi. org/10.1097/SCS.0b013e318211516d.
- Yanagi Y, Asaumi J, Maki Y, Murakami J, Hisatomi M, Matsuzaki H, et al. Incidentally found and unexpected tumors discovered by MRI examination for temporomandibular joint arthrosis. Eur

J Radiol. 2003;47:6–9. https://doi.org/10.1016/ S0720-048X(03)00095-0.

- Zachariades N. Neoplasms metastatic to the mouth, jaws and surrounding tissues. J Craniomaxillofac Surg. 1989;17:283–90. https://doi.org/10.1016/ S1010-5182(89)80098-8.
- Zavattero E, Garzino-Demo P, Berrone S. Ossifying fibroma affecting the mandibular condyle: report of an uncommon case. J Craniofac Surg. 2013;24:e351–3. https://doi.org/10.1097/SCS.0b013e3182902b23.
- Zorzan G, Tullio A, Bertolini F, Sesenna E. Osteosarcoma of the mandibular condyle: case report. J Oral Maxillofac Surg. 2001;59:574–7. https://doi. org/10.1053/joms.2001.22694.

Index

A

Adherences, 151 Adhesions, 150, 151, 177, 190 Aneurysmal bone cyst (ABC), 224, 225 Ankylosing spondylitis (AS), 193, 196, 203 Ankylosis, 27, 92, 97, 105, 109, 110, 113, 115, 119, 153, 154, 157, 158 Anterior band, 39, 42, 134, 145 Anterior displacements, 132, 133, 139 Anti-parallel orientation, 3, 4 Arthrosynovitis, 107, 119 Articular disc blastema, 38 Articulator, 67, 79, 83 Auriculotemporal nerve, 38 Axis of rotation, 59

B

B₀, 3–8, 16, 20 Bandwidth, 22 Basis images, 33 Benign tumors, 220, 224 Bilaminar zone, 44, 45, 63, 107, 133, 134, 140, 142, 149 Blastematic stage, 38 Border movements, 58, 68, 70, 75, 78, 82 Branchial arch, 91, 92 Bullet effect, 21

С

Calcium pyrophosphate dihydrate deposition disease (CPDD), 227 Capsular tear, 107–110, 120 Capsule(s), 38, 45, 66, 67, 84, 106, 107, 114, 119, 164 Cartilage, 37, 38, 58, 73, 92, 105, 176, 177, 192, 220, 223, 225 Cavitation stage, 38 Centric relation, 83, 85, 86, 126 Cephalogram, 95, 162 Chondrification of the condyle, 38 Chondroblastoma, 223 Chondroitin sulfate, 42 Chondrosarcoma, 223, 224 Click, 86 Closing movement, 49, 58, 68, 70, 81 Coils, 13, 18, 19 Collagen fibers, 40, 42, 43, 145, 146 Collateral ligaments, 39, 58, 107, 111 Computed tomography(CT), 2, 5, 18, 19, 25, 26, 30, 33, 67, 97, 98, 100, 105, 106, 113, 115, 117-119, 157, 160, 179, 184, 197, 223, 228 Condylar fracture (CF), 31, 105-107, 110, 113, 114, 117 Condylar head(s), 39, 63, 77, 105, 112-115, 120, 165, 177, 182, 185, 204, 205 Condylar hyperplasia, 98, 99, 102, 228 Condylar hypoplasia, 91-94, 98 Condyle(s), 26–28, 35, 39, 57, 59, 60, 63, 64, 67, 73, 75-77, 79, 83, 85, 86, 91, 94, 105, 108, 113, 116, 119, 121, 126, 127, 130, 134, 142, 151, 152, 186, 192, 224, 228 Condylography, 57, 66 Cone beam computed tomography (CBCT), 2, 26, 30-34, 98, 221, 223 Contrast(s), 1, 2, 5, 6, 8, 18, 19 Coronoid hyperplasia, 150, 160 Coronoid processes, 94, 160 Crepitus, 177, 178 Crossing signal, 86 Cryogenic gas, 20, 22

D

Deep masseter, 47 Deflection(s), 22, 105, 116, 130, 157, 160, 178, 197 Deflection forces, 22 Degenerative discal changes, 148, 201 Dental panoramic tomography (DPT), 162 Detector, 31 Deviation, 73, 97-99, 130, 197, 228 Diagnostic Criteria for Temporomandibular Disorders (DC/TMD), 83, 84 Diarthroses, 38 Dipole, 2 Disc, 39, 84, 126, 146 Disc displacement with reduction (DDwR), 34, 85, 86, 128, 131, 136, 139, 186 Disc displacement without reduction (DDWoR), 128, 130, 150-152, 176, 188

© Springer Nature Switzerland AG 2020 T. Robba et al. (eds.), *MRI of the Temporomandibular Joint*, https://doi.org/10.1007/978-3-030-25421-6 Disc perforation, 146, 148 Disc rupture, 107, 118, 119, 148 Disco-condylar ligament, 63 Disco-malleolar ligament, 38 Disco-temporal ligament, 44, 63 Dose, 26, 33, 34, 98 Dysostosis, 92

Е

Echo planar imaging (EPI), 15 Echo spacing (ESP), 15 Echo time (te), 17, 22 Echo train length (ETL), 23 Edema, 17, 107, 164 Effusion, 106, 108, 109, 188, 190, 203 Elastic fibers, 41, 44 Elastin, 40 Electromagnetism, 2 Embryology, 37, 38 Eminence, 38, 39, 60, 65, 68, 73, 76, 78, 81, 84, 119, 162, 202 Emitted radiation, 30 End feel, 72, 73, 75, 81, 82, 192 Endochondral ossification, 201 Erosion(s), 30, 32, 34, 185, 192, 197, 205, 228 Extracellular matrix, 40, 41, 145, 194

F

Fan x-ray beam, 30, 33 Faraday's law, 6 Fast acquisition techniques, 1, 15 Fast spin echo (TSE), 15, 32 Fat signal suppression, 1 Field of view (FOV), 14, 15, 22, 31–34 Fixed disc, 152 Flip angle (FA), 6, 11, 22 Free induction decay (FID), 8 Frequency encodings, 14 Functional movements, 58, 68, 72, 126

G

Gadolinium, 19
Ganglion cyst, 226, 227
Gantry, 13, 30
Ginglymoarthrodial synovial joint, 38
Glenoid fossa, 39, 41, 59, 60, 68, 76, 94, 115, 151, 152, 162, 164, 179, 182
Glycosaminoglycans (GAG), 40–42
Goldenhar syndrome, 94
Gradient echo (GE) sequences, 1, 8, 9, 12, 16, 108, 225, 226, 228
Gyromagnetic ratio, 3

H

Hemarthrosis, 106–108, 110, 119, 148 Hemifacial microsomia (HFM), 93, 94, 96, 98 Hemosiderin, 1, 11 Hinge axis, 58, 59, 66, 67, 70, 78, 82, 85 Hounsfield unit (HU), 30 Hyaluronic acid, 130, 145, 151, 190 Hydrogen nucleus, 2, 3 Hypermobility disorders, 162 Hypomobility disorders, 150 Hypoxia/reperfusion cycle, 145

I

Idiopathic condylar resorption (ICR), 182, 204–206 Image reconstruction, 11 Inferior retrodiscal lamina, 43 In-phase, 17 Intermittent locking, 127, 128, 142 Internal maxillary artery, 47, 49 Intramembranous ossification, 38 Isotropic voxel, 15

J

Joint disorders, 126–128, 130, 131, 133–136, 139, 140, 142, 144, 145, 147, 149–152, 154, 156, 158, 160, 162, 164, 165, 167
Joint sounds, 157, 190, 228
Juvenile idiopathic arthritis (JIA), 97, 191, 195–197, 203

K

Kinematic MRI, 18, 127, 135 *k*-space, 13, 15, 16

L

Larmor precession, 3 Lateral pterygoid muscle, 17, 38, 39, 44, 47, 48, 60, 107, 121, 127, 165, 220, 222 Laterotrusion, 64, 68, 70, 76, 127 Ligament laxity, 162, 176 Ligamentous laxity, 128 Limited opening, 128, 157 Lock, 88, 142, 152, 164, 165, 167 Longitudinal component Mz, 7 Lower compartment, 38, 58 Lubrication, 45, 130, 145, 150, 177 Luxation, 106, 162, 164, 166

Μ

Macroscopic magnetization, 3–5 Magnetic field, 1–4, 6–8, 18–21 Magnetic susceptibility, 1 Malignant tumors, 223 Mandibular intramembranous ossification, 37 Mandibular movement(s), 38, 57, 58, 64–66, 69, 70, 77, 78, 150 Mandibular nerve, 33, 38, 47 Mandibular ramus, 94, 116 Matrix, 22, 32, 145, 177 Maturation stage(s), 38, 223 Meckel's cartilage, 37, 38 Medial pterygoid muscle, 47, 63 Mediotrusion, 64, 68, 70, 86, 127 Metastasis, 224 MR scanner(s), 3, 5, 19, 21 Multichannel coil, 20 Multiplanar reconstructions, 33, 98, 118 Multislice computed tomography (MSCT), 26, 30, 34 Myofibroblasts, 40

Ν

Nager syndrome, 92 Nephrogenic systemic fibrosis, 19 Noise ratio, 15 Nonfracture injuries, 106 Nucleus, 2, 3 Number of excitation, 23

0

Open lock, 164, 165, 167 Opening movement, 49, 58, 60, 70, 81, 136 Orthopantomogram, 98, 100, 114, 118, 198, 226 Ossifying fibroma (OF), 220, 222–223 Osteoarthritis, 175, 189, 200, 225 Osteoarthrosis (OA), 105, 151, 175, 188 Osteochondritis dissecans, 207, 225 Osteochondroma, 160, 220, 222, 225 Osteoma, 160, 220 Osteophytes, 34, 178, 179, 184 Osteosarcoma(s), 220, 223, 224 Out-of-phase, 17

Р

Pannus, 197, 199, 200, 203 Parallel orientation, 3 Paramagnetic agent, 19 Pars intermedia, 39, 140 Peak kilovoltage (kVp), 33 Permanent magnet, 19, 20 Petrotympanic fissure, 39 Pharyngeal arch, 37 Phase encoding(s), 13, 14 Pierre Robin sequence (PRS), 92 Pigmented villonodular synovitis (PVNS), 220, 225 Pixel, 13, 15 Plain radiography, 25, 27 Planigraphy, 26, 27 Positional MRI, 18 Posselt diagram, 73, 77, 81, 82 Posterior attachment, 43 Posterior band, 11, 39, 42, 126, 146, 152 Posterior displacement, 132, 134 Prosthetic replacement (of the TMJ), 110, 205 Proteoglycans, 41, 42, 145 Proton(s), 1-3, 5, 16, 112 Proton density (PD), 6, 203

Protrusion, 44, 61, 63, 68, 70, 74, 75, 78, 86, 160 Psoriatic arthritis (PsA), 191, 193, 194, 196

Q

Qantum mechanics theory, 2 Quantum noise, 34 Quench, 20, 22

R

Radio frequency, 7, 13, 21 Raw data space, 13 Recapture (disc), 140 Receiving coil, 6 Reduction (disc), 127, 145 Region of interest (ROI), 31 Relaxation, 7, 9, 16, 19 Remodeling, 17, 102, 107, 177, 179, 181, 182 Resistive magnets, 20 Resonance phenomena, 3 Retraction, 63, 73 Retrodiscal laminae, 43, 107, 149, 202 Retrusion, 57, 61 Rheumatoid arthritis, 27, 32, 191, 192, 220, 225 Rotation, 2, 9, 26, 58, 60, 67, 70, 81 Row, 12, 14, 15, 22

\mathbf{S}

Safety, 1, 21 Sagittal axis, 39 Saturation bands, 17 Scanner, 20 Sequence(s), 1, 8, 9, 16, 18, 19, 70, 107, 109, 111, 146, 202 Shielding, 20 Short tau inversion recovery (STIR), 16 Signal to noise ratio (SNR), 5, 14 Slice selection, 13 Soft tissue injury, 106, 107 Spatial encoding, 13 Spatial resolution, 5, 14, 32 Specific absorption rate (SAR), 21 Sphenomandibular ligament, 38, 45 Spin angular momentum, 2, 3 Spin echo (se) sequences, 8, 9 Spin-lattice relaxation time, 7, 22 Spin precession frequency, 6, 12, 14 Spin relaxation, 6 Spin relaxation time, 8 Spin-spin relaxation time, 7 Spondyloarthritis (SpA), 191, 193 Squamous temporal bone, 39 Stickler syndrome, 92 Stylohyoid ligament, 45 Subchondral sclerosis, 177, 179, 185, 221 Subcortical cyst, 175, 179, 188 Subluxation, 162, 164 Superconductivity, 19, 20, 22 Superficial masseter, 47

Superficial temporal artery, 49 Superior retrodiscal lamina, 43, 107 Surface radiation dose, 34 Synovial chondromatosis, 26, 207, 223–228 Synovial thickening, 175, 198 Synovitis, 151, 188, 192, 196–200, 203, 205, 206, 228

Т

T1, 1, 12, 19, 202, 225 *T2*, 1, 8, 12, 109, 225 *T*_{2*} decay, 8 Temporal muscle, 46, 47 Temporal resolution, 5 3D acquisition, 15 Time of echo (TE), 8, 9, 17 Time of repetition (TR), 9, 15 Tomography, 25, 26, 30, 34, 101, 179, 198 Towne projection(s), 28, 116 Translation, 58, 60, 70, 80, 81, 138, 152, 162 Transmitted radiation, 30 Transmitting coil, 20 Transverse axis, 58 Transverse component M_{xy} , 7 Trauma, 29, 106, 107, 109, 115, 116, 176 Treacher Collins syndrome (TCS), 92, 93 Tumor-like lesions, 220, 222–225, 227, 228 Turbo spin echo (TSE), 15 2D acquisition, 15

U

Ultrasound (US), 34 Upper compartment, 60, 127, 152

V

Vertical axis, 14 Vertical ramus of the mandible, 39 Voxel, 13, 15, 30, 32

Х

X-ray tube, 31, 33