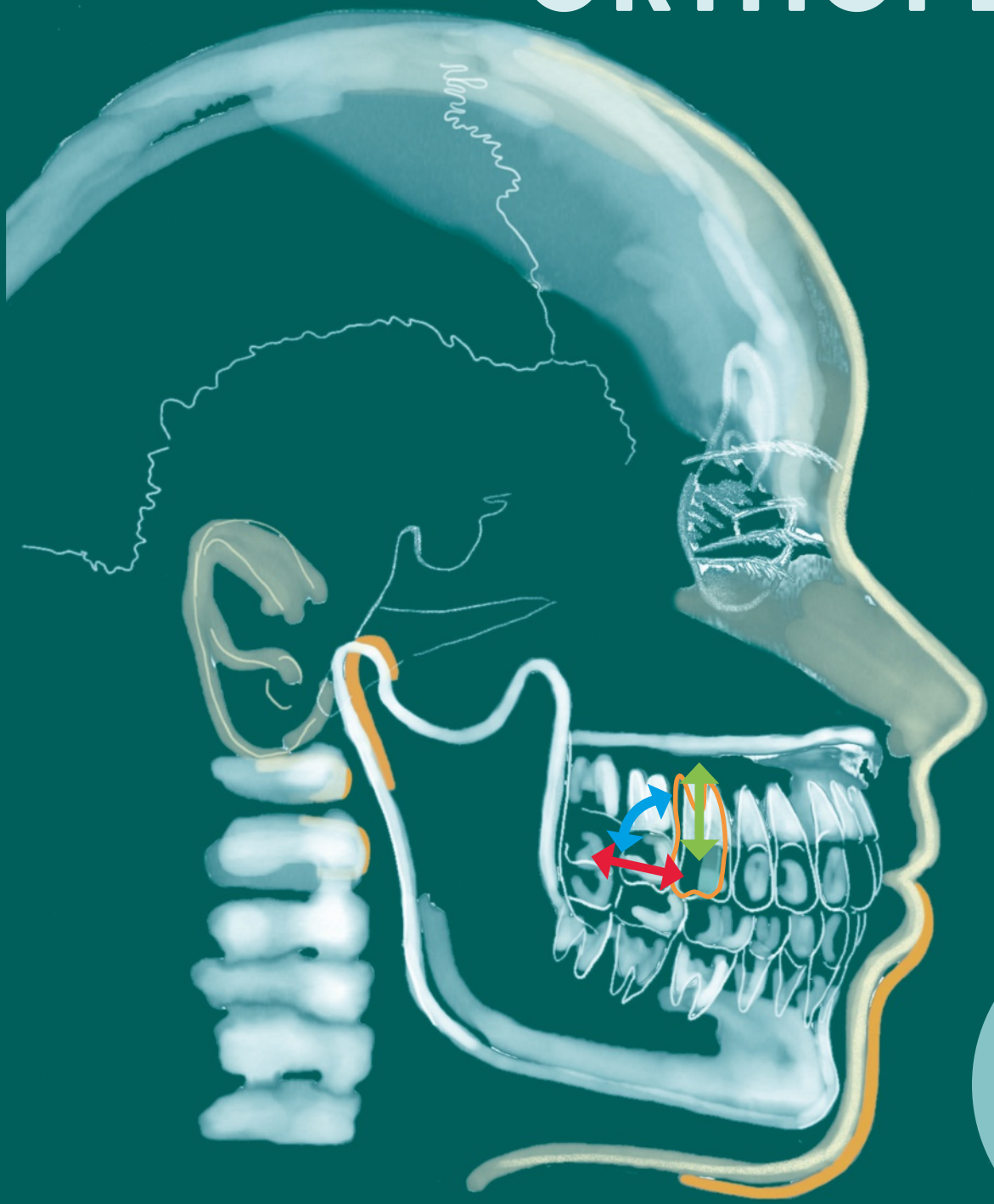
WERNER SCHUPP
JULIA HAUBRICH

ALIGNER ORTHODONTICS AND OROFACIAL ORTHOPEDICS



WITH CONTRIBUTIONS FROM:
YUXING BAI
WOLFGANG BOISSERÉE
FAYEZ ELKHOLY
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Table of Contents

INTRODUCTION VIII
ACKNOWLEDGMENT IX
FOREWORD XI
FOREWORD XII
FOREWORD OF THE FIRST EDITION XIII
ABBREVIATIONS XIV

CHAPTER 1

PHYSIOLOGY AND FUNCTIONAL NEUROANATOMY OF THE TEMPOROMANDIBULAR SYSTEM AND MUSCULOSKELETAL SYSTEM 1

- The function of the basal ganglia 2
- Brain neuroimaging 6
- The segmental dysfunction 7
- The influence of occlusion and the muscles onto the load in the temporomandibular joint and the periodontal ligament 8
- Composition of the mastication muscles and their pain process in muscles 13
- The trigeminus and its complex relationships 15

CHAPTER 2

BIOMECHANICAL ASPECTS OF TOOTH MOVEMENT WITH ALIGNERS 25

- Methodological principle of aligner therapy 26
- Importance of force and moment magnitudes for orthodontic tooth movement 27
- Determinants of force and moment magnitudes applied during aligner therapy 27
- Attachments and aligner modifications 33

CHAPTER 3

DIAGNOSTICS 41

- The orthodontic diagnosis sheet: part I 42
- The orthodontic diagnosis sheet: part II 45
- Virtual articulator 73

CHAPTER 4

TREATMENT PLANNING AND TREATMENT WITH ALIGNERS 81
The algorithm for diagnosis and treatment 82
Occlusion 83
Bone and periodontium 85
Esthetics 87
Basic rules 87
An example treatment plan 91
Course of therapy 93

CHAPTER 5

TREATMENT OF DIFFERENT MALOCCLUSIONS WITH DIFFERENT ALIGNER SYSTEMS 95
Topic 1.1 In-office aligner treatment 96
Topic 1.2 Flow Chart of the In-Office Aligner Technique 111
Topic 1.3 Attachments and bonding procedure 120
Topic 2 Derotation 125
Topic 3 Angulation/Deangulation 130
Topic 4 Crowding 139
Topic 5 Interproximal enamel reduction (IPR) 150
Topic 6 Extraction 153
Topic 7 Spacing 183
Topic 8 The transverse dimension 193
Topic 9 Anterior open bite 201
Topic 10 The deep bite 229
Topic 11 Class II non-extraction treatment in adults 235
Topic 12 Class III adult treatment 255

Topic 13.1 Treatment of children – single tooth movements 283
Topic 13.2 Treatment of children 289
Topic 13.3 Treatment of children – avoiding extractions 295
Topic 13.4 Treatment of children – management of missing teeth 311
Topic 13.5 Treatment of Class II in children 333
Topic 13.6 Treatment of Class III in children 383
Topic 14 Aligner orthodontics and orthognathic surgery 395
Topic 15 Management of gingiva height 412
Topic 16 Periodontology and orthodontic treatment 425
Topic 17 Impacted canines 452
Topic 18 Bone remodeling 471
Topic 19 Management of missing teeth in adults 477
Topic 20 Intrusion of a single tooth 495
Topic 21 Interdisciplinary dentistry 504
Topic 22 Treatment of patients with special needs 539
Topic 23 TMD treatment – initial therapy of occlusion with occlusal splint 552
Topic 24 Speed Up: Build up a "Speed Up" 622
Topic 25 Selective tooth grinding after aligner orthodontics 625
Topic 26 Retention after aligner orthodontics 632

CHAPTER 6
ADVANTAGES OF ALIGNER ORTHODONTICS 633

CHAPTER 7
AVOID GLOBAL WARMING AND PLASTIC WASTE 641

Introduction

Every scientific field, including medicine and orthodontics, is in continuous development and therefore subject to change. Some orthodontic inventions (eg, the Funktionsregler, named after Professor Fränkel) become an integral part of the orthodontic practice, while others cannot fulfill expectations or prove to be too complex and slip back into oblivion.

The movement of teeth using aligners was founded in 1926 by Remensnyder; Kesling popularized this method in 1945 and described it as a "tooth positioning appliance." Later, Sheridan invented the "Essix Tooth Moving System." Using the Essix technology is quite similar to the conventional fixed appliances, as the therapy can be constantly modified because of the multiple variables that arise during treatment. With the Essix aligners, mild to moderate crowding can be solved.

Align Technology was founded in 1997, being the first company to use the former aligner techniques and combine them with CAD/CAM (computer-aided design/computer-aided manufacture) technology. Advances and innovations in this technology have further improved and enhanced the Invisalign system. The Invisalign system was unique in that the clinician was able to plan the final result and the path of the treatment virtually with the ClinCheck software, even before the real treatment starts in the patient's mouth. In the past, Invisalign has been described as a successful tool for treating mild to moderate crowding, for the closure of naturally occurring spaces, as well as tipping movements. After several years of experience with the system, almost all tooth movements (see Chapter 5) can be performed with aligner therapy. Aligner orthodontics has become established worldwide and counts as being one of the most innovative orthodontic techniques. Meanwhile, other aligner systems have entered the market. Some work similar to the Invisalign system, while others allow the practitioner with adequate software to set up the virtual treatment goal all by himself. This edition will give insights into the In-Office Aligner Technique we have been using now for almost 5 years and believe will be a fixed component of the orthodontic office in the future. As with every orthodontic appliance, retention is crucial also in aligner orthodontics. In our office, patients are advised to come in for retention controls regularly after the orthodontic treatment. We have tried to document all patients for a longer period, offering also the possibility to show long-term result pictures. Unfortunately, this has not been possible for all of our patients.

As for every orthodontic appliance, no matter if removable or fixed, aligner orthodontics, too, need a high level of education, training, and experience. This book may give the beginner, as well as the experienced user, tips and techniques on how to integrate aligner orthodontics reasonably and successfully into the orthodontic office. The book gives an overview of Aligner Orthodontics since 2001, showing early and therefore now older treatment examples as well as formerly treated patients including developments of software systems and techniques over the years until today. Compared to our first book, different outsourced aligner systems are presented. We have put a main focus on the in-office aligner technique, as well as on the orthopedic part of our specialty.

Acknowledgment

This book would not have been possible without the cooperation and understanding of our patients, many friends, colleagues, practice staff, and of our families.

The initial idea for our first aligner book came from our friend and colleague Kenji Ojima. The book *Aligner Orthodontics* was first published in Japan.

This book does not claim to consider only the issues within aligner orthodontics as being purely scientific. It is rather supposed to give some assistance to the orthodontic practitioner with the diagnosis, treatment planning, and treatment with aligners. One main focus of this book is the function of the occlusion, the craniomandibular system, and the musculoskeletal system. For us, the treatment of the function, and the correction of the malocclusion come first. A perfect dentofacial esthetic is our second most important treatment goal. "Beautiful is what functions!" is a decisive idea of the Bauhaus art movement (1919–1933). So we focus our main attention on function, which according to the Bauhaus ethos leads to beauty and therefore takes priority. The treatment of complex functional disorders, as well as of esthetic deficits, is often only possible in the interdisciplinary concept. This is why we have described in some patients not only aligner orthodontics from functional and esthetical aspects, but also in singular cases the necessary supply with restoratives after orthodontic therapy. Compared with our first book, we have significantly expanded the orthopedic part and added the addition "orofacial orthopedics" to the title *Aligner Orthodontics*. For this reason, this book begins with a description of the complex craniomandibular system and its connection to the musculoskeletal system.

The significance of occlusion for the function of the craniomandibular system is described by Okeson quite insistently: "Occlusion has been an important consideration in orthodontics since the beginning of the discipline. Early emphasis was placed on the alignment of the teeth, the stability of the intercuspal position, and the esthetic value of proper tooth positioning. These factors remain important to orthodontists, but orthopedic principles associated with masticatory functions must also be considered. Orthopedic stability in the masticatory structures should be a routine treatment goal to help reduce risk factors associated with developing temporomandibular disorders."[1]

We were particularly pleased to have found co-authors that we were able to convince to support us in this book.

We were able to persuade Wolfgang Boisserée, with whom we have written the book *Kraniomandibuläres and Musculoskeletal System* (2012), to participate in the chapter "Diagnosis," which deliberately focuses on the theme of "function." He has also contributed to many writings on interdisciplinary dentistry showing the continued restorative treatment after orthodontic treatment.

The biomechanical aspects of tooth movement with aligners are described by Bernd Lapatki and Fayez Elkholy. In a clear and inspiring manner, they represent the basic biomechanical bases necessary for everyone, without which virtual planning in aligner orthodontics would not be possible.

Fayez Elkholy and Julia Funke deserve our thanks for their section of the chapter "In-office aligner treatment" with a perfect overview of different in-office systems, print, and aligner material.

We thank Xianju Xie for the support with the creation of the topic for the treatment of Chinese patients and their specialties. We also thank Yuxing Bai for the perfect illustration of the particular treatment of Chinese patients.

We thank our co-authors, as well as our friends and colleagues Stephen Chang for his graphics and Georg Meyer for the chapter "Short screening test", Margret Bäumer, and Carsten Appel for their tireless work and many professional discussions around the topic of aligner orthodontics and orofacial orthopedics, periodontology, and endodontology. We thank Kenji Ojima for the presentation of an extraction treatment for a Japanese patient. Petra Clauss and Gerd Christiansen we thank for the ideas in the chapter "Virtual articulator," which will surely improve our virtual treatment simulation (VTS) in aligner orthodontics.

Our thanks go also to our teachers: Ulrike Ehmer, Harold Gelb, Rainer-Reginald Miethke, Robert M. Ricketts, and Douglas Toll.

We would like to thank Mitra Derakshan, John Morton, Srini Kaza, and Bob Boyd, who were significantly involved with the development of the Invisalign technique. It has always been an enrichment to be able to participate with them in the development of aligner orthodontics in research and development.

We would also like to thank Toni Graf-Baumann, Rainer Heller, Stefan Kopp, Gerhard Marx, Dirk Polonius, and Peter Zernial, with whom we have developed diagnostic and therapeutic procedures in an interdisciplinary approach, between orthodontics and manual and osteopathic medicine.

We thank Christopher Lux, Chris Köbel, and Matthias Kern for providing additional perfect picture material. We would like to cordially thank our friend Anna Drexelius for the cover design.

We also thank the dental laboratory Läkamp, in particular Manfred Läkamp and Max Mainzer, for many innovative ideas and images, most notably with the technique Zirkonzahn.

In addition, we are grateful to Rainer-Reginald Miethke, who has not only written a foreword in the first edition but also has contributed many suggestions for improvement. For the foreword in this second edition, we cordially thank Vincenzo d'Anto and James Mah.

Tommaso Castroflorio and Francesco Garino, we thank you for sharing new thoughts and ideas concerning aligner treatment with us.

Almost all MRI scans of the temporomandibular joint (TMJ) are from the Media Park Clinic, Cologne. For this, we thank Magnus Andersson, Thomas Steimel, and colleagues.

We thank all patients and patients' parents for allowing us to treat them or their children. We would like to thank them deeply for the trust that they have placed in us.

Most treatments were only possible in a close interdisciplinary team. Our thanks here go to our colleagues Carsten Appel, Margret Bäumer, Wolfgang Boisserée, Frank Bröseler, Oliver Giers, Elisabeth Janson, Wolfgang Kater, Stefan Kopp, Sofia Krings Vogeler, Roland Mantsch, Pascal Marquardt, Ulrich Meyer, Ansgar Rademacher, Leslie Runkel, Ingolf Säckler, Jesko Schuppan, Christina Tietmann, and Marit Wendels.

Our sincere thanks go to the entire team of our orthodontic office and laboratory, particularly to Maria Harbrecht, for her meticulous assistance with the photo documentation, and our master technician Mario Klingberg, who have both contributed their cooperation and support to make this book possible.

1. Okeson JP. Evolution of occlusion and temporomandibular disorder in orthodontics: Past, present, and future. Am J Orthod Dentofacial Orthop 2015;147(5 Suppl):S216–S223.

Foreword

"No appliance exists which will allow an orthodontist to treat orthodontic problems without adding the necessary ingredient of Common Sense" (Thomas F. Mulligan).

In recent decades, there has been a significant increase in the use of clear aligners for the treatment of malocclusions in adults and children. However, despite the fact that orthodontic treatment with clear aligners is a quickly growing sector, there is still insufficient evidence with regard to the effectiveness and stability of the treatment compared with conventional therapies.

The authors Julia Haubrich and Werner Schupp have been early adopters of the technique and are among the most well-known clinicians who have contributed the most to the advancement of the fields. Their book is inspired by common sense and provides a thoughtful and pioneering vision for the future of the field.

The choice to open the textbook with a new chapter on physiology and functional neuroanatomy of the temporomandibular system and musculoskeletal system provides, at first glance, a clear idea of the authors' perspective on the importance of function in contrast with cosmetic and esthetically driven therapies. This section is completed by the revised section on diagnostic aspects, with a comprehensive overview of the different steps needed for a multidisciplinary and interdisciplinary treatment approach.

Meanwhile, the new Chapter 2, written by Prof. Bernd G. Lapatki and Fayez Elkholy, summarizes the biomechanical aspects underlying aligner therapies and allows every orthodontist to understand the key factors playing a critical role in obtaining a proper tooth–aligner interaction.

Every reader can take advantage of the wide list of clinical topics developed through well-documented case reports, providing the rationale and the mechanics for treating patients with different clinical conditions. The treatment of different malocclusions was performed with different aligner systems, with a special focus on in-office designed appliances, which are the future of the orthodontic discipline.

Aligners are being refined and will continue to improve with the passage of time. Thanks to the progress in artificial intelligence and to the improvement in material properties, new possibilities have emerged for their clinical evolution. Nevertheless, a deep knowledge of the key factors in aligner treatment still allows the common-sense orthodontist to have complete control of every treatment plan in order to overcome all the biomechanical limits of the appliance.

This book will help any practitioner interested in aligner therapy to gain a deeper insight into its biomechanical background, the essential steps in planning aligner therapy, and knowledge of detailed procedures in numerous clinical examples from an orthodontic and orthopedic point of view.

Vincenzo D'Antò, DDS, PhD, Specialist in Orthodontics
Associate Professor
University of Naples Federico II
Department of Neuroscience, Reproductive Sciences and Oral Sciences
Section of Orthodontics

Foreword

To date, over 14 million patients have been treated using aligners, and their popularity continues to rise as patients become more aware of these treatments and clinicians continue to expand their utilization. Despite the rich history of aligners, which spans several decades, there are less than a handful of books on this topic, one of which was from the current authors. Accordingly, this book comes at the right time, when clinicians are actively seeking organized and comprehensive information regarding aligners. Overall, the book is clearly written and accompanied by exquisite illustrations and abundant photographs. Related to the latter, the book is well over 600 pages but remains easy to read as the figures and illustrations greatly assist explanations and discussion. The book is enhanced with international collaborators, which include Yuxing Bai, Wolfgang Boisserée, Fayez Elkholy, Bernd Latatki, and Julia Funke.

The book begins with a chapter on physiology and functional neuroanatomy of the temporomandibular and musculoskeletal systems, respectively. This provides relevant basic information regarding the function of the masticatory system, which distinguishes this book from others. The foundations of the system provide essential parameters for understanding the etiology of malocclusions, diagnosis, establishing treatment goals, and treatment planning. The second chapter is also foundational and provides key information regarding the biomechanical aspects of an aligner system. For a relatively simple appliance, the biomechanical aspects are actually quite complex with variables such as material properties, gingival margin trim line, and the many attachment designs. Chapter 3 describes a systematic and detailed diagnostic approach and provides clinical examination forms to assist the clinician in conducting examinations. It includes information on occlusal analysis as well as an evaluation of the musculoskeletal system and temporomandibular joints with a virtual articulator. This is another area where this book sets itself apart. Chapter 4 provides the principles of treatment planning with aligners. An easy-to-follow planning algorithm for diagnosis and treatment planning is provided. This chapter is loaded with clinical content and tips such as the esthetic finishing checklist. This chapter is worth reading again and again as there are numerous concepts and principles that the authors have established from years of experience. Chapter 5 is the largest chapter, and it is not surprising as the types of malocclusions that are treated with aligners are as diverse as those with conventional fixed appliances. A flow chart of aligner technique is provided, which is invaluable for those beginning to provide aligner therapy. A unique benefit of this chapter is the advice on treatment staging. This aspect of aligner education is lacking or completely missing from other books. Numerous details, such as planning of attachments, limitations of movements, choice of materials, and the specifics of individual tooth movement directions are provided. Additional topics of software for treatment planning and in-office aligner treatments add to the comprehensiveness of this chapter. Chapter 6 discusses the advantages of aligner orthodontics. Historically, some clinicians were critical of the ability to treat malocclusions with aligners compared to contemporary fixed appliances. However, with advancements in the science and understanding of aligners this has become somewhat of an antiquated notion. Both systems have been shown to work well, and it really is an "apples to oranges" comparison with an emphasis on the skill and experience of the clinician. Chapter 7 deals honestly with an aspect of aligner orthodontics that is seldom discussed. The environmental data provided are startling. Avoiding plastic waste with strategies to manufacture aligners only as needed, selection of packaging and auxiliaries and aspects to consider as responsible clinicians.

In summary, this book provides fundamental information for clinicians to better diagnose and treat orthodontic patients with aligners. Despite its comprehensive nature, easy-to-follow clinical forms and charts are provided to allow readers to more readily apply this wealth of information toward patient care. This book is a real asset for those embarking on the journey of learning about aligners.

James Mah, DDS, MSc, DMSc
Interim Dean
University of Nevada, Las Vegas

Foreword of the first edition

In 1993, Toni Morrison received the Nobel Prize for literature. Of what relevance is this fact to this textbook? Well, Mrs Morrison once said: "If there's a book you really want to read but it hasn't been written yet, then you must write it." This was, in all likelihood, the motive behind Julia Haubrich and Werner Schupp's work on this textbook given that, in my opinion, it is the only one of its kind.

The authors are experienced orthodontists who work in the same private practice and who began using the Invisalign System shortly after its introduction into Europe. Very soon after this, they became devotees of this novel treatment modality, in which they have gained extensive experience since then.

This textbook opens with a chapter on diagnostics. It is for the reader to decide whether to examine patients to the extent and depth that is described here. One should at least be aware of the complex, interrelated physiology of the human being who exists at the end of every tooth, and be prepared to make individualized and appropriate referrals to other disciplines, which are specialized in muscle or joint problems.

Guest author John Morton has contributed a short chapter on the biomechanics of aligners. This is followed by a broad presentation of all kind of malocclusions, the accompanying symptoms, the rationale behind the selected treatment approaches, and the various outcomes achieved. Each patient is documented with high quality intra- and extraoral photos and radiographs. Every reader stands to benefit from this chapter, irrespective of his or her level of experience with the Invisalign System. Impressive as the treatment results are, the authors, in their admirable self-criticism, still point out minor flaws.

The last chapter of the textbook deals with the advantages (and some disadvantages) of the Invisalign System. Its content may help patients and clinicians alike in deciding whether this system is the optimal choice for a particular situation.

This author does not wish to take any more time from the reader, who has ahead of them a challenging, but worthwhile read. Study the text, read sections of it selectively, skim through , or return to it over and over again in the best interests of your patients.

Prof R-R Miethke
2016

Abbreviations

ADDWR – *anterior disc displacement with reduction*
ARAS – *ascending reticular activating system*
BROM – *back range of motion*
CBCT – *cone-beam computed tomography*
CGRP – *calcitonin gene-related peptide*
CNS – *central nervous system*
CO – *centric occlusion = maximum intercuspation*
COPA – *condylar occlusal positioning appliance*
CPG – *central pattern generator*
CR – *centric relation; physiological centric relation = physiological TMJ relation*
CRT – *cotton roll test*
EPSP – *excitatory postsynaptic potentials*
FR – *formatio reticularis*
FR – *Fränkel appliance (Funktionsregler in German)*
GABA – *gamma-aminobutyric acid*
HIP – *habitual intercuspation position*
ICC – *interclass correlations*
IED – *incisal edge distance*
IPR – *interproximal reduction*
MRI – *magnetic resonance imaging*
MRT – *magnetic resonance tomography*
MSS – *musculoskeletal system*
NS neuron – *nociceptive neuron*
OISD – *orthodontic implant site development*
OPG – *orthopantomogram*
OSAS – *obstructive sleep apnea syndrome*
PAPA – *preparatory anticipatory postural adjustment*
PDL – *periodontal ligament*
RANKL – *receptor activator of NF-kappaB ligand*
RCP – *retruded contact position*
RCT – *randomized controlled trial*
RJM – *rhythmic jaw movements*
RPE – *rapid palatal expansion*
SHIP – *Study of Health in Pomerania*
TAD – *temporary anchorage device*
TCB – *therapeutic construction bite*
TMD – *temporomandibular dysfunction*
TMJ – *temporomandibular joint*
TMS – *temporomandibular system*
TNF – *tumor necrosis factor*
TPP – *torque pressure points*
VTS – *virtual treatment simulation*
WDR – *wide dynamic range neuron*

1

PHYSIOLOGY AND FUNCTIONAL NEUROANATOMY OF THE TEMPOROMANDIBULAR SYSTEM AND MUSCULOSKELETAL SYSTEM

The nervous system, with its peripheral and central portions, is a complex and coherent system which can be divided into individual blocks, communication though inseparable. The central nervous system (CNS) requires information from the periphery to plan the motor activities and functions and be able to control these. Considering the motor cortex in the precentral gyrus and somatotopic representation of the contralateral muscles (homunculus) as well as the sensory portion, a significant proportion of the temporomandibular system (TMS) drops directly into the eye area (Fig 1-1). Many actions are done unconsciously. Most of the information from the TMS is processed in the brain stem, the trunk encephali, consisting of the midbrain (mesencephalon), bridge (pons), and prolonged spinal cord (medulla oblongata). The basal ganglia are an integral part of the motor system, which become highly active already directly after birth.[1, 2]

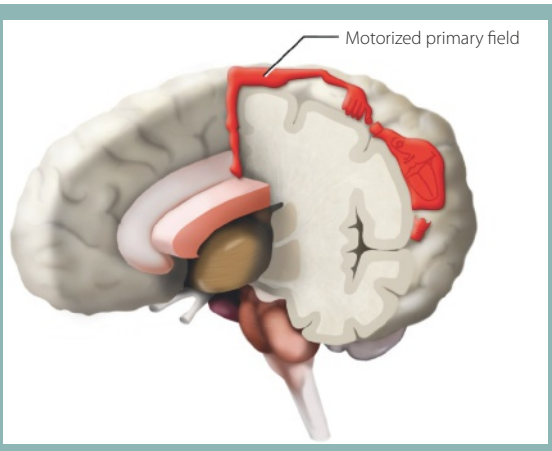


Fig 1-1 Motorized primary field of the gyrus precentralis. Graphic: Lovric & Bohr from Boisserée, Schupp, Kraniomandibuläres and Musculoskeletal System, courtesy Quintessence Publishing.[2]

The function of the basal ganglia

The subcortical basal ganglia are involved in many processes such as perception, learning, memory, attention, motor function and select central inputs and outputs, both of a cognitive and emotional nature, to maintain stability with change, namely allostasis.[3–6] For our human survival, its selection of movements and establishment of a corresponding sequence of movements to our current context of events is indispensable.[7] The basal ganglia are directly involved in this process. The basal ganglia return on existing patterns of movement and save new movement patterns by repeating the new patterns. The basal ganglia select, sort, and integrate congenital learned movement patterns associated with cognitive and emotional mental information.[7]

Directed motility controls the flow of directed voluntary movements. The interaction of sensorimotor regions in the nervous system, in compliance with the basal ganglia in voluntary movements, is shown in Figure 1-2.[8] The center for purposeful movement is the subcortical motivation area. Further processing takes place simultaneously and is rational in the frontal cortex and emotional in the limbic cortex. There is thus no muscular activity without the influence of the limbic system. Regarding the associative cortex, the advance takes place in the basal ganglia, the nucleus tegmental pedunculopontine informs among others the cerebellum, but also the cortex, basal ganglia, and thalamus according to an "integrated interface." The nucleus tegmental pedunculopontine is connected to motor nuclei in the brain stem and spinal cord.[9, 10] From the thalamus the information enter into the brain stem and from there via the dorsolateral pathways of the spinal cord to the muscle that performs the idea. An immediate feedback follows as somatosensory information into the cerebellum, the associative, and in the limbic cortex.

The basal ganglia receive their input from the sensory organs, from the muscles, tendons, and other deep somatic tissues, as well as from connective tissues. This information of the cortex is communicated to the basal ganglia via the thalamus. Cortical areas of the orbitofrontal cortex, the anterior cingulate cortex, the lateral prefrontal cortex, and areas of the motor cortex control striatal core areas, which lie in the dorsolateral and ventromedial caudate nucleus, in the putamen and nucleus accumbens, and which are connected to the globus pallidus as a starting region of the basal ganglia area (Fig 1-3). The globus pallidus communicates with the ventral and medial thalamic nuclei, which mediate a rear projection to cortical output structures.[11] The basal

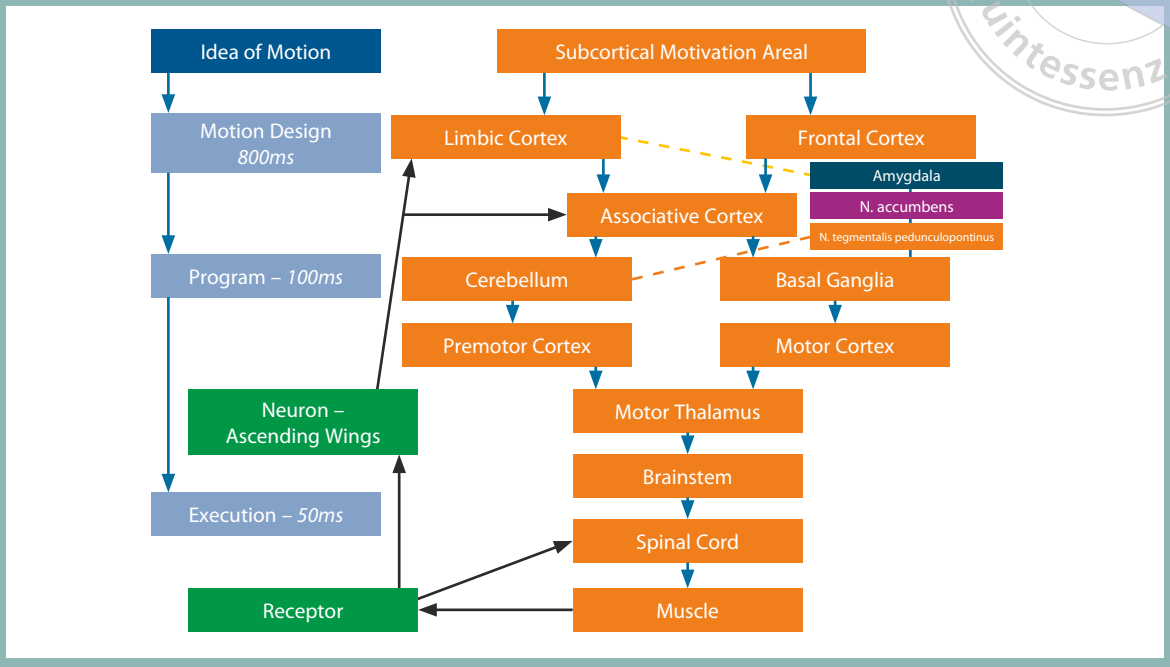


Fig 1-2 Planning process of directed motility in consideration of the basal ganglia, modified by Rettig.[8] Immediate feedback from the muscle follows as somatosensory information into the cerebellum and into associative and limbic cortec (black arrows).

ganglia decide after receiving this information whether a motor, an emotional, motivational, or cognitive instruction takes place or not. The selection is made according to the criteria of maintaining allostasis. Beyond the direct interconnection in the basal ganglia, the activity is increased in the thalamus, and inhibited via indirect and hyperdirect interconnection in the basal ganglia. The resulting net income determines accordingly whether the stimulation of a motor, emotional–motivational, or cognitive region in the cortex takes place or not.

Without the involvement of the basal ganglia, the motor cortex areas cannot trigger or control any voluntary movement. The basal ganglia prepare any directed motility and control it. This is where the implementation of the cortical motion planning into specific exercise programs happens. These exercise programs control the development of power, direction, and speed of a movement sequence. A further function of the basal ganglia is the emotional and motivational influence of action preparation and action selection on limbic shares. Under the influence of the limbic system, the process of selection of action, inhibiting unwanted and finally the activation of desired action sequences is carried out here.[12–14]

The basal ganglia are positioned exclusively subcortically. The cortex radiate fibers into the basal ganglia from widely distributed areas. From here, they go through the thalamus back to the cortex, resulting in a bowing movement – which is why they are called "loops." Cortical areas, the basal ganglia, and the thalamus are thus connected in the form of loops together. Complex conscious planning, unconscious and involuntary motor experience, and emotional aspects draw circles on parallel tracks between cortex, basal ganglia, thalamus, and cortex. This parallel processed information produces an activation, which is the basis of voluntary movement. According to their different functions, the fibers are divided into five separate tracks, which are grouped into two systems[12]:

- *The motoric dorsal lap*
- *The limbic ventral lap*

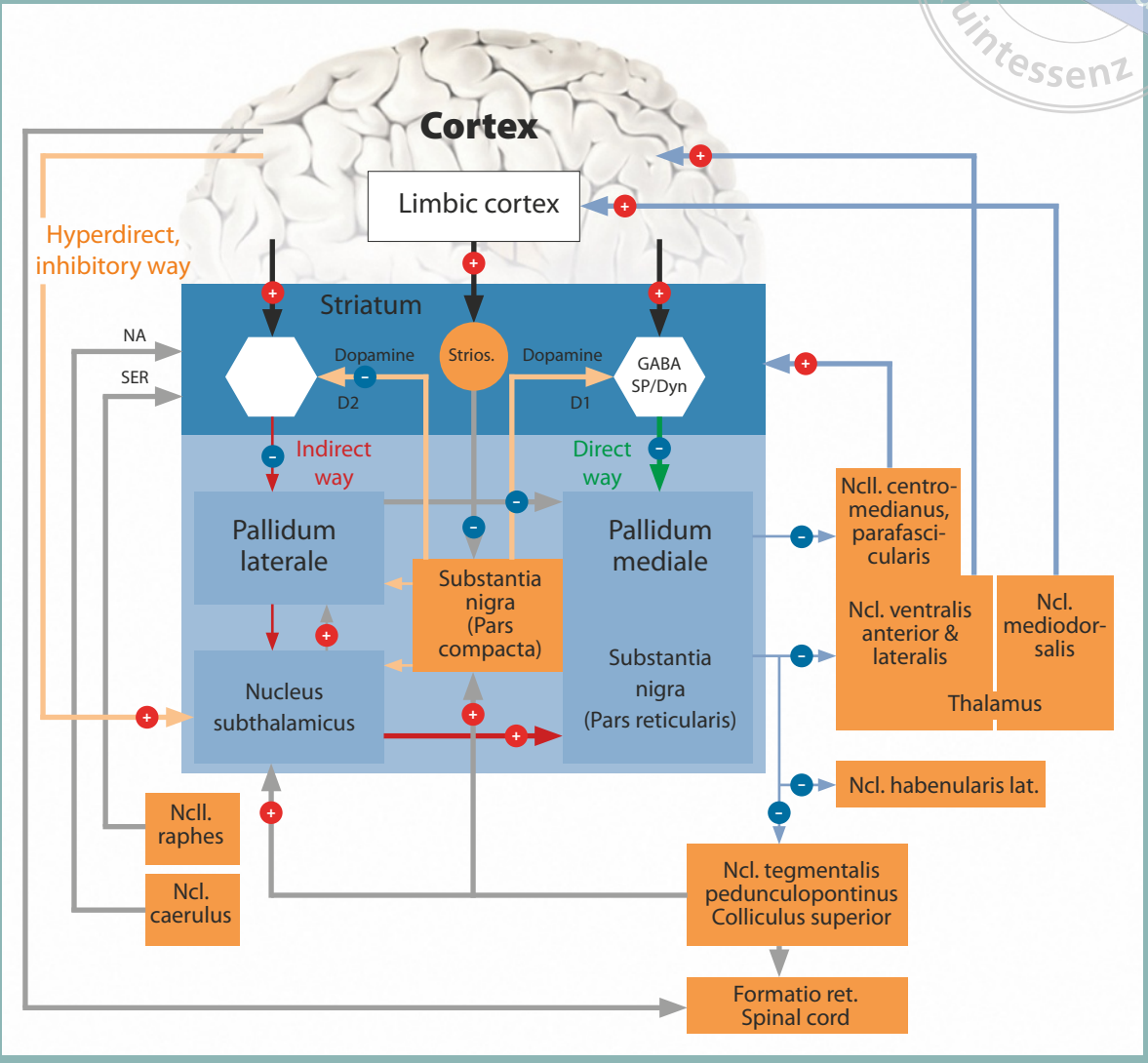


Fig 1-3 Basal ganglia interconnection in detail, Indirect way: inhibiting; Direct way: exciting; Hyperdirect way: inhibiting. mod. Asan[15] with courtesy of Damir del Monte (www.damirdelmonte.de) (cortex, limbic cortex, cortical areas, basal ganglia, and thalamus are connected underneath in three loops: 1. Direct, exciting way; 2. Indirect, inhibitory way, 3. Hyperdirect, inhibitory way, formatio reticularis = spinal cord).

The basal ganglia consist of (mod. according to Asan[12,15]):

- *Striatum*
  - Striatum dorsale
  - Nucleus caudatus (main part)
  - Putamen (main part)
- *Striatum ventrale*
  - Nucleus caudatus (ventral)
  - Nucleus accumbens
  - Putamen (ventral)
  - Pallidum
  - Pallidum laterale (dorsal, ventral)
  - Pallidum mediale (dorsal, ventral)
  - Associated nuclei
  - Nucleus subthalamicus
  - Substantia nigra

In the basal ganglia interconnection, three routes can be distinguished:

- *direct, exciting way*
- *the indirect, inhibitory way*
- *the hyperdirect, inhibitory way*

The direct, exciting path projects by GABAergic neurons (inhibiting) go from the striatum to the globus pallidus medial, as well as into the substantia nigra pars reticularis. From there, the forwarding occurs via GABAergic neurons into the thalamic nuclei. The concatenation of two inhibitory neurons leads to a disinhibition, which is an excitation of thalamic nuclei.

The indirect, inhibiting path of the striatum projects to the external, lateral pallidum segment, which inhibits the subthalamic nucleus with GABAergic neurons. The subthalamic nucleus acts via neurotransmitter glutamate excitation to the pallidum media, which thereby inhibits movement impulses in the thalamic nuclei.[8, 12]

The hyperdirect, inhibiting pathway acts via the striatum, pallidum laterale, and nucleus subthalamicus to the medial pallidum.

The nucleus accumbens (Fig 1-2) includes afferents to the limbic system, making an integrative interface between the basal ganglia and limbic system. It plays an important role in pleasurable and motivational states and therefore is considered an antagonist of the amygdala, which processes mainly negative key stimulus.[12]

The striatum and the subthalamic nucleus, as two separate structures, give input into the basal ganglia. Both play a role in motor control, but also in cognitive function.[16] The cognitive loop runs from the prefrontal cortex to the nucleus caudatus and from there parallel to the motor loop over the substantia nigra, globus pallidus, and anteroventral thalamic nuclei back to the prefrontal cortex.[17] The efferent connection of the basal ganglia occurs via globus pallidus and substantia nigra with structures, which initiate movements as well as physiological and cognitive processes. Typically, an inhibitory effect takes place. The basal ganglia thus inhibit spontaneous motoric, physiological, and cognitive activity of the organism. According to Schwarting,[18] the function of the basal ganglia is to select between the diverse afferents that are of the utmost importance for the organism. As a result, after Schwarting,[18] the corresponding processes are disinhibited, while irrelevant or incompatible processes are further inhibited. Due to the direct interconnection, the activity of the thalamus is excited; via the indirect and hyperdirect interconnection it is inhibited. The net result determines whether the activity of a cortex region is inhibited or uninhibited via the thalamus. This means that it is determined here whether a motor, a physiological, or even a cognitive process is initiated or not.[18] The basal ganglia decide, in the spirit of allostasis, what process is important for the organism.

Since the communication of the motor, the occulomotoric, the cognitive loop, and the limbic loops among themselves is not fully understood yet, no definite statement can be made about how and where the inhibition of the functions is carried out among themselves. The motor loop receives the starting point of the motion planning from the ventral, emotional loop. Since the CNS is always energy-saving, probably at a high and constant proprioceptive and nociceptive input, allostatically will inhibit those functions that are not directly necessary for the survival of the organism, in which case more cognitive processes are inhibited. This means inferentially that with high and constant nociceptive and proprioceptive stimulation from the temporomandibular system, motivational activities are inhibited and motoric activity, important for allostasis functions, would be disinhibited.

The importance of the basal ganglia for the function of the temporomandibular system

The motor activity of the temporomandibular system is determined by the basal ganglia and the simultaneous movements coordinated. The occlusion has direct influence on the mandibular movement and muscle tone. Both the receptors of the periodontal ligament (which has free nerve endings with ramification to the layer of cementoblasts as mechanoreceptors) and nociceptors (such as Ruffini corpuscles as mechanoreceptors especially in the apex area with a resolution up to 10 µ,[19] as well as from the temporomandibular joints), constantly carry information among the trigeminal nerve into the CNS. In a modified, unphysiological temporomandibular

joint (TMJ) position and occlusal trauma, the resulting information provides a reflex response directly to the muscles of mastication for the adaptation, which is controlled by the basal ganglia.[7] If the altered TMJ position or occlusal trauma last for a longer time, a slowed and decreased coordinated movement of the lower jaw and a higher muscle tone are the result. Even unconscious movements can occur. From a malocclusion develops a motor response that leads to an altered relation of the mandible to the maxilla. The thus performed stimulation of the proprioceptors of the temporomandibular joints leads compensatorily to a muscular adjustment. This explains the effect of occlusal splints, which solve faulty occlusal contacts and lead to a normalization of the condyle position.[20]

Errors in the occlusal relationship induce sensory stimulation of the CNS via the trigeminal nerve.[2] This results in a change in the dopaminergic neurons. Experimentally created occlusal interferences in rats led to a neurochemical change in the dopamine and norepinephrine activity in the striatum, frontal cortex, and the hypothalamus. Interestingly, in cases of tooth overcapping, thus increasing the mandibular incisor height in rats with composite for a day, a significant increase of DOPA (3,4-dihydroxyphenylalanine) accumulation could be found; in parallel, there was a significant increase in dopamine – levels in the hypothalamus and an increase of dopamine and noradrenaline-levels in the frontal cortex. After 14 days, the elevated levels fell back to control values, except in the left striatum. If, on the other hand, incisors had been shortened in the upper jaw and the lower jaw, as well as if an incisor was reduced only in the lower jaw, no change of DOPA, dopamine, or norepinephrine levels was found.[21] This is consistent with our clinical experience that a faulty anterior contact, along with the absence of posterior support and displacement of the temporomandibular joints are the most common causes of temporomandibular dysfunction (TMD),[22] possibly also affecting the musculoskeletal system.[2] Areso concluded from this that prolonged occlusal interferences lead to a central change of catecholaminergic neurotransmitter with anterior contacts, and thus leads to motor changes.[21]

Nakamura found in a study on rats that the basal ganglia control the muscles of mastication. By microinjection of picrotoxin (PTX), a gamma-aminobutyric acid (GABA) antagonist in the nucleus caudatus and putamen, a rhythmic jaw movements (RJM) was triggered.[23] An injection of GABA in the substantia nigra lead to sustainably increase of the orofacial muscle activity.[24] Other studies confirm the hypothesis that the basal ganglia control the chewing muscles.[25–32] Masuda also confirmed that the movements are controlled by the basal ganglia, here in the globus pallidus and putamen.[33]

Brain neuroimaging

Brain neuroimaging has been used to investigate the brain signature of chronic orofacial pain, including trigeminal neuropathic pain and pain related to temporomandibular joint disorders (TMD).[6] Ernst et al[34] investigated the effects on pain (with a VAS scale from 0 to 100), movement kinematics, and cerebral representation by a 3-month mandibular splint therapy. In their study, the patient's pain ratings decreased about 60%, while kinematic and electromyographic characteristics over therapy were not significantly altered. During therapy, they observed a decrease of functional magnetic resonance imaging (fMRI) activation magnitude in the primary somatosensory cortex (S1) and secondary somatosensory cortex (S2), and insular cortex during occlusion. Left hemispheric anterior insula and the cerebellar fMRI activation decrease were associated with decrease in pain over time.[34] Youssef et al studied the differential brain activity in subjects with painful trigeminal neuropathy and painful TMD. With brain neuroimaging they found that acute pain is associated with coactivation of numerous brain regions, including the thalamus, insala, and the cingulate cortices. A similar set of brain structures is not activated in all chronic pain conditions, particularly chronic neuropathic pain, which is associated almost exclusively with decreased thalamic activity. Neuropathic pain was associated with cerebral blood flow decreases in a number of regions, including the thalamus and primary somatosensory and cerebellar cortices. In contrast, painful TMD was

associated with significant cerebral blood flow in areas in regions commonly associated with higher-order cognitive and emotional functions, such as the anterior cingulate and dorsolateral prefrontal cortices and the precuneus, also in motor-related regions as well as within the spinal trigeminal nucleus.[35]

Patients with trigeminal neuropathic pain and TMD patients showed consistent functional and structural changes in the thalamus and the primary somatosensory cortex, indicating the thalamocortical pathway as the major site of plasticity. The trigeminal neuropathic patients showed more alterations at the thalamocortical pathway, and the two disorders showed distinct patterns of thalamic and insular connectivity. Functional and structural changes were frequently reported in the prefrontal cortex and the reward processing in chronic orofacial pain.[6] Based on previous evidence, Lin et al hypothesized that both trigeminal neuropathic patients and TMD patients would show a common pattern of functional and structural changes within the pain-related network. Trigeminal neuropathic pain patients would show more changes in the thalamocortical pathway compared to TMD patients, which is predominantly associated with the abnormality within the peripheral musculoskeletal system. The prefrontal cortex, the limbic system, and the circuitry of reward processing changes in relation to chronic orofacial pain.[6] In patients with neuropathic pain, the thalamus showed hypoactivity during resting but hyperactivity during allodynia.[6]

The dorsolateral and the ventrolateral prefrontal cortex play a key role in modulating pain, particularly with cognitive re-appraisal. The consistent engagement of the prefrontal cortex in trigeminal neuropathic patients and TMD pain patients highlights the role of psychological factors in chronic orofacial pain.[6]

Nebel et al showed that TMD modifies cortical response to tactile simulations.[36] TMD patients suffer from persistent facial pain and exhibit abnormal sensitivity to facile stimulation with brain neuroimaging. They recorded cortical responses evoked by low-frequency vibration of the index finger in patients with TMD and healthy controls. The primary auditory cortex was activated in patients with TMD. TMD patients also showed greater activation bilaterally in the anterior cingulate cortex and contralaterally in the amygdala. The study presents evidence that central processing of unconscious tactile simulations is abnormal in TMD patients.[36]

Lickteig et al examined the changes in cortical activation during occlusal splint therapy with a functional MRI. They found that the cerebral activation during occlusion was decreased after therapy in bilateral sensorimotor regions and in the left posterior insula, right superior temporal cortex, and bilateral occipital lobe.[37]

The segmental dysfunction

The segmental dysfunction is a reversible, hypomobile musculo-articular dysfunction. The segmental dysfunction has a limited or missing joint play. The joint receives the passive reserve function.[38] From an osteopathic medical point of view, also the tooth in the periodontal ligament (PDL) has to be seen as a joint. Treatment for the segmental dysfunction – and accordingly the temporomandibular joints – should be started as soon as possible to avoid structural pathologies. Motor control dysfunction already exists before the pain develops. Start the treatment of nociafferences as soon as possible and as efficiently as possible to avoid activation of the sympathetic nervous system and pain chronicity.[38] According to Beyer, even very brief stimulations of motor neurons or interneurons can lead to a long-lasting change of the motor unit, which in itself may evoke changes in muscle tension, change in body posture, impaired movement, and pain. As he points out, the origin of the painful stimulus and the region where the pain occurs do not necessarily coincide. If one part of the segment (sclerotome, myotome, dermatome, viscerotome) is disturbed over a longer period, the dysfunction disperses first in the segment itself, then over the segments along to cranial and caudal, as well as over the muscular, fascial, and articulation chains, giving rise even to disturbances of stereotypes. This spread will often occur in a very short period of time; sometimes it may only take two days. In manual medicine we can almost always observe such functional associated symptoms. A "primary lesion" can no longer

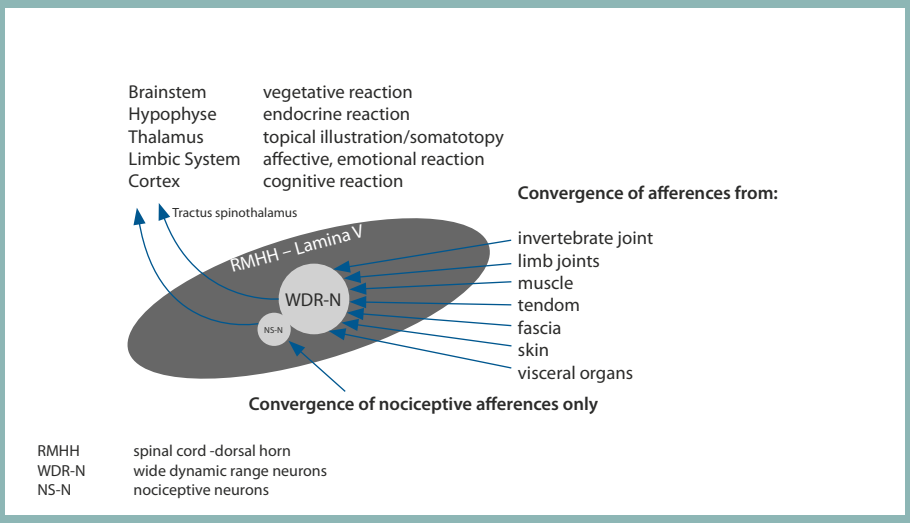


Fig 1-4 Convergence of nociafferences from different structures in the wide dynamic range neurons and their signal transduction.

be identified as the disturbances sustain each other reciprocally. Treatment in the region of the cranial or upper cervical nerves can reduce muscle tension in the periphery and thus correct asymmetries.[39]

Muscle tension and muscle spasm can be triggered through nociception from a neighbored joint or the PDL. The origin of pain can be the joint, TMJ inclusive, but the source of pain is a neighboring muscle.[40] Even if the joint is the cause of the referred pain, it can itself be painless. Due to the convergence, various overlays of different sources of pain are possible, the so-called principle of summarized stimuli.[40]

The convergence of nociafferences come from the:

- *Intervertebral joint*
- *Limb joints, TMJ*
- *Muscle*
- *Tendon*
- *Fascia*
- *Skin*
- *Visceral organs.*

These nociafferences converge in the wide dynamic range neurons (WDR neurons) in the lamina V of the dorsal horn in the spinal cord. They reach the CNS within the tractus spinothalamicus (Fig 1-4). This convergence in the WDR complex leads to a missing discriminability of pain (Fig 1-4) (see also "The trigeminus and its complex relationships"). The WDR complex does not only obtain information from the nociceptors, but also of proprioceptors and mechanoreceptors.

Wu and Hirsch showed obvious differences in the prevalence of TMD between adolescents of different ethnic origins (Asians and Europeans). These differences cannot be attributed to cultural differences alone, which implies the involvement of genetic factors in the etiology of TMD.[41]

We describe here the structural side of a temporomandibular and musculoskeletal dysfunction without denying the influence of the psyche. Hans Georg Gadamer, a German philosopher (1900–2002) has described the influence of the psyche, summing it up in one sentence: "The strongest analgesic agent and one of the most important life engines is the joy of succeeding."[42]

The influence of occlusion and the muscles onto the load in the temporomandibular joint and the periodontal ligament

Unlike all other joints in the musculoskeletal system, the temporomandibular joints are not guided neuromuscularly alone in its final end position.[43–45] The occlusion will determine the final end position. In Figure 1-5a, the

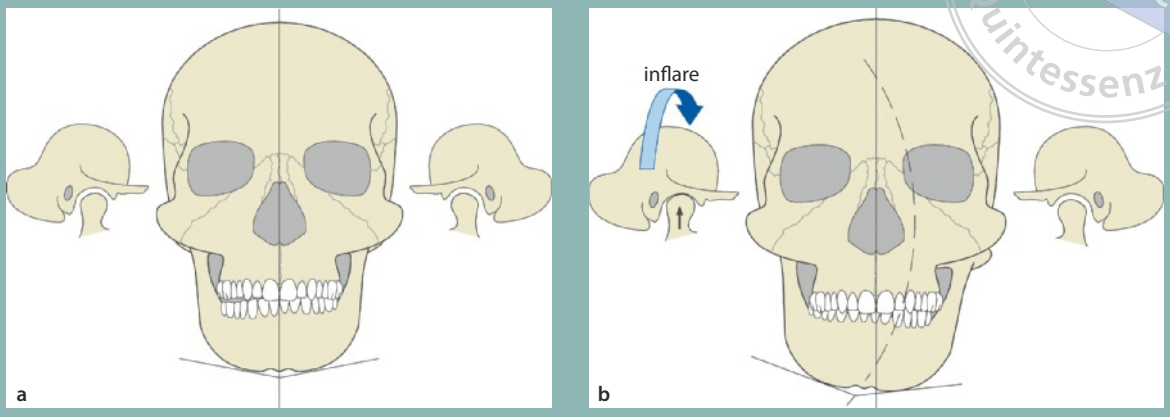


Fig 1-5 (a) In a physiological bilateral condyle position, occlusal contacts exist in a centric position only on the left side. In this unilateral missing support, there is no occlusal contact on the right side. (b) From the centric position shown in (a), the mandible slides during biting to the right, the teeth occlude now in habitual intercuspation bilaterally. In this case, the right TMJ displaces posteriorly and cranially with the result of compression in the bilaminar zone. From the displacement of the condyle results an internal rotation of the temporal bone. The face appears convex to the left with a shortened right side of the face.

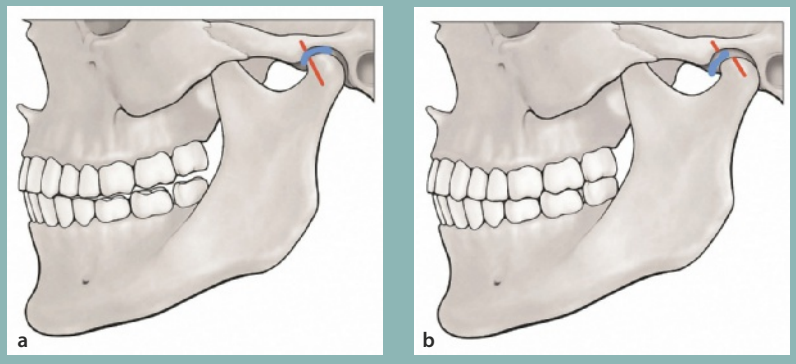


Fig 1-6 TMJ disorders in lateral aspect. (a) Lack of posterior support with contact limited to the incisor area in physiologic condyle position, giving a posterior open bite. (b) Habitual contact on the molars then moves the condyles into a backward position, displacing the disk to anterior and positioning the joint on the bilaminar zone. Graphic: Lovric & Bohr from Boisserée, Schupp, Kraniomandibuläres and Musculoskeletal System, courtesy Quintessence Publishing.[2]

temporomandibular joints are shown in a physiological and symmetrical position. Here is an occlusal contact in a centric position only on the left side and there is a non-occlusion on the right side.

If the patient bites firmly in this situation, the lower jaw swings to the right, the teeth occlude now bilaterally in habitual intercuspation. In this case, the right TMJ displaces posteriorly and cranially with the result of compression in the bilaminar zone.

From the displacement of the condyles results an internal rotation of the temporal bone.[2, 46] The facial scoliosis is convex to the left, which results in a shortened right side of the face (Fig 1-5b).

The occlusion exerts a direct influence on the TMJ position and the corresponding force vector in the joint[47] (Fig 1-6).

A joint is the connection between two bones. A joint combines joint mobility with simultaneous restriction of unwanted mobility as the subluxation. Bones are not optimized for friction forces. Therefore, the bony structure is covered in the joint with cartilage. The articular cartilage is nourished by diffusion from the synovial fluid. Repetitive joint overloading results in osteochondritis dissecans and ultimately osteoarthritis.[48] A mechanical overload in the temporomandibular joint results in an expression of green fluorescent proteins (GFPs), which

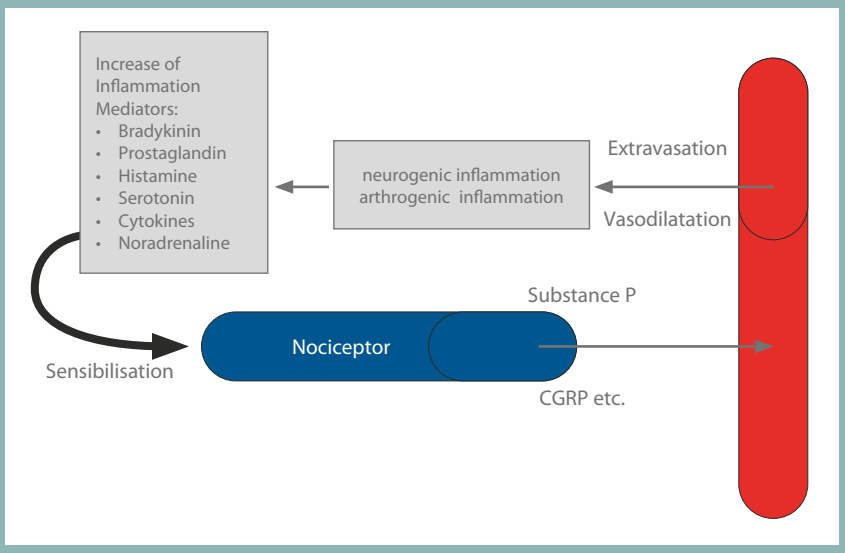


Fig 1-7 In an arthrogenic inflammation which is autoimmune (inflammatory rheumatism) or degenerative by improper loading, the nociceptors release inflammatory mediators. This leads to vasodilation and extravasation of vessels; a neurogenic and arthrogenic inflammation occurs. This leads to a further release of inflammatory mediators, leading to a sensitization of nociceptors, thus an increase in the sensitivity of nociceptors. A vicious circle is thus created (mod. according to Zieglgänsberger in Böhni et al[51]).

shows the effect of the mechanical stress and the related cell response in the articular cartilage. A first mechanical stress in the jaw joint leads according to this animal study first of all to a thickening of the cartilage, an adaptive remodeling.[49]

The damage of a joint occurs in addition to metabolic, traumatic, or tumor causes by the mismatch between capacity and load. Incorrect load occurs due to disturbed statics. A uniform compressive load on the other hand protects against arthritis. The cartilage has to keep its proper structure, including the synovial membrane. With progressive axial malposition, the constantly increased load will increasingly cause loss of hyaline cartilage and bony substance. The hyaline cartilage does not regenerate, and early treatment intervention is necessary. The static-dynamic regulation is increasingly decompensated; the formerly painless procedure becomes increasingly painful. Arthritis arises once inflammatory mediators are released from the detritus.[50]

Due to the constant mechanical agitation in the joint, but also from the PDL and from all other deep somatic tissues, the nociceptors release inflammatory mediators (Fig 1-7). These include, among others:

- *Bradykinins*
  - inflammatory mediator, cause of pain, vasodilatation, increase of permeability
- *Calcitonin gene-related peptide (CGRP)*
  - Is a strong vasodilator, which is released among others during migraine attacks from neurons of the trigeminal nerve. It is the crucial mediator in a migraine attack by its vasodilation action. It also triggers the release of IL-1ß and IL-6, resulting in the local sterile inflammation. Here nociceptive neurons are stimulated and thus the typical headache in migraine is triggered.[51, 52]
- *Prostaglandins*
  - Prostaglandins bind to a variety of cell surface receptors. The different signal induction paths are responsible for multiple effects. PGE2 binds to four different receptors that activate, inhibit, or even cause antagonistic reactions depending on subtype, which leads to both muscle contraction and muscle relaxation. PGE2, PGE1, and PGI2 are vasodilatory and indirectly promote nociception through sensitization of peripheral nociceptors. They stimulate the transmission and processing of pain stimuli in the spinal cord and brain and are thus involved in the formation of hyperalgesia and allodynia. Acetylsalicyl acid (eg, aspirin) has an analgesic, anti-inflammatory, and antipyretic effect by inhibiting cyclooxidase and thus the formation of prostaglandins.

- *Interleukins and tumor necrosis factor*
  - IL-1ß, IL-6, and tumor necrosis factor (TNF) are inflammatory mediators that are responsible for the systemic effects of inflammation. They are primarily responsible for rubor, calor, and tumor. IL regulates mainly communication between macrophages B and T cells. TNF-α activates monocytes and neutrophils. Tumor necrosis factor is a cytokine of the immune system and is involved in inflammation, always in rheumatoid arthritis.
- *Substance P (SP)*
  - Substance P (SP) is mediator of inflammation and thus involved in arthrogenic and neurogenic inflammation, as well as pain trigger, vasodilator, and increasing permeability.

Free nerve endings can be found in the temporomandibular joint in the bilaminar zone and in the lateral and anterior part of the joint capsule. Studies show the involvement of inflammatory mediators on the occurrence of pain in the TMS, as well as the PDL. These free nerve endings show CGRP and SP. CGRP and SP are involved in blood flow and pain in the TMJ.[53] With a TMJ dysfunction, there is a higher concentration of SP in the synovial fluid.[54] The clinical symptoms of internal derangement of the TMJ are thought to be associated with the degree of synovitis. It was concluded that the expression of SP seems to be closely related to histopathological changes of the human TMJ with internal derangement.[55] The score of cells that stained for SP in joints with internal derangement was significantly higher than that in controls.[56] Synovial fluid TNF-α levels and pain levels (VAS scores) were found to be increased in patients with internal derangement as the stage of the disease progresses.[57] CGRP was significantly increased in the arthritic trigeminal ganglia in the temporomandibular joint of rats. SP, CGRP, and neuropeptide Y in the arthritic temporomandibular joint were significantly increased as compared to the controls.[58]

In all groups, the levels of SP, CGRP, and neuropeptide Y-like immunoreactivity were higher in the trigeminal ganglia than in the TMJs. The results clearly demonstrate a close interaction between increased neuropeptide release from sensory and sympathetic neurons after induction of arthritis in the rat.[59] The immunoreactivity of two inflammatory mediators, CGRP and SP, was measured in the trigeminal ganglia and brainstem to characterize an adjuvant-induced inflammation within the rat temporomandibular joint at various acute (6, 24, and 48 h) and intermediate (10 day) time intervals. Concentrations of adjuvant-related neuropeptides were compared to those in both contralateral vehicle-related tissues and non-injected controls. SP in the ganglion on the injected side was significantly increased for all four time periods. In brainstem subnucleus caudalis, CGRP was significantly increased for all four time periods. SP immunoreactivity in the subnucleus caudalis was significantly increased for the initial three time periods, but by day 10 had been reduced to that of the control. These data show that the pattern of changes in neuropeptides following the induction of inflammation is different between SP and CGRP. Moreover, the pattern of change varies between the brainstem and the trigeminal ganglion. This suggests that the two neuropeptides may have different roles in the inflammatory process, and that this process may be modulated by different mechanisms at the brainstem and ganglion.[60] The aim of the following study was to test the hypothesis that TMJ inflammation alters the excitability of trigeminal root ganglion neurons innervating the facial skin by using behavioral, electrophysiological, molecular, and immunohistochemical approaches. The number of SP-immunoreactive neurons in the inflamed rats was significantly increased compared with that in the naive rats. These results suggest that TMJ inflammation can alter the excitability of medium- and large-diameter trigeminal root ganglion neurons innervating the facial skin and that an increase in SP/NK1 receptors in their soma may contribute to the mechanism underlying the trigeminal inflammatory allodynia in the TMJ disorder.[61] Fifteen male Sprague-Dawley rats were randomly assigned into occlusal interference group (n = 12) and control group (n = 3). In the occlusal interference group, 0.4 mm thick crowns were bonded to the rats' first maxillary molar. SP expression was the most obviously increased at 5 days in both sides and gradually decreased to the level of control. Experimental occlusal interfer-

ence-induced masticatory muscle pain was associated with peripheral sensitization of nociceptive neurons rather than muscle damage and inflammation.[62] Bradykinin in synovial fluid might be useful as an index of the degree of synovitis in the TMJ.[63]

The neuropeptides SP, CGRP, and neuropeptide Y have all been found at high levels in the synovial fluid of arthritic TMJs in association with spontaneous pain, while serotonin (5-HT) has been found in association with hyperalgesia/allodynia of the TMJ. Interleukin-1 beta (IL-1 beta) and tumor necrosis factor alpha have been found in arthritic TMJs, in association with hyperalgesia/allodynia of the TMJ as well as spontaneous pain, but not in healthy TMJs. Interleukin-1 beta has also been related to radiographic signs of joint destruction. Prostaglandin E2 (PGE2) and leukotriene B4 are both present in the arthritic TMJ and PGE2 has been shown to be associated with hyperalgesia/allodynia of the TMJ. Very little is known about pain and inflammatory mediators in muscles. However, we know that serotonin (5-HT) and PGE2 are involved in the development of pain and hyperalgesia/allodynia of the masseter muscle in patients with fibromyalgia, whereas local myalgia (myofascial pain) seems to be modulated by other, as yet unknown mediators.[64]

The inflammatory mediators lead to neurogenic or arthrogenic inflammation. The neurogenic inflammation results in a sensitization to the persisting mechanical stimulus. The nociceptive stimulation by bradykinin increases the mechanical sensitivity of the free nerve endings of nociceptors in such a way that they react painfully to non-painful stimuli. Hyperalgesia through to allodynia can be the consequence. From the increased sensitization, misinformation results in terms of allegedly high mechanical load. This means that even if the load of "the bite" remains consistently high, it is experienced by the neurological system as a higher load. This results in increased nociceptive stimulation. Motor stereotypes change. The change of motor stereotypes leads to muscular activity increase.[65] Nociceptive afferents from deep somatic tissues, which will not exceed the threshold of action potential, and therefore not perceived as painful, can sensitize spinal cells. It follows the activation of dormant synapses and the reduction of the threshold. In the spinal cord, pronounced changes occur in the interconnection in the spinal dorsal horn; a functional reorganization of the spinal cord takes place.[40] Through an ongoing nociceptive influx of impulse, inhibitory interneurons decay, which are normally always active and inhibit nociceptive neurons. Cell death is caused due to the fact that nociceptive afferents release glutamate and SP in excess as a result of permanent strong excitement. This leads to a strong excitation of interneurons with opening of Ca^{2+} channels, leading to cell damage; hence, the excitement toxicity develops. Due to the sinking of the pain-inhibiting interneurons, the nociceptive neurons of the spinal cord are permanently uninhibited and overactive. The anti-nociceptive system is in malfunction. In this way, pain can also occur without the action of external stimuli.[65]

Since no specific nociafference of a muscle, a joint, or the PDL of a tooth exists, this results in a lack of central discriminability of localization. This leads to a central cognitive disorder. There is no specific pattern of pain for a particular source of pain. Nociceptive activity from a neighboring area (joint, muscle, PDL, etc.) can produce an activation of muscle motor neurons in the sense of a spasm. Due to the convergence, overlays of different sources of pain are possible, which is referred to as principle of summarized stimuli.[40]

Muscle tension and muscle spasms can be triggered by nociafference from an adjacent joint. The effects of inflammatory irritant application to the rat temporomandibular joint on jaw and neck muscle activity is shown in a study. The study results showed that an injection of mustard oil in the TMJ results in a sustained and reversible activation of jaw muscles, which may be related to the reported clinical occurrence of increased muscle activity associated with trauma to the TMJ.[66]

Non-sensitized receptors in muscle tissue and in other deep somatic tissues respond only to strong stimuli. The muscle or joint capsule are less sensitive to pain in a non-sensitized state. Nociceptive afferents from all deep somatic tissues such as joints, intestines, or neuropathic altered nerves can produce motor system activation in the affected segment. This results in a motor reflex response with sometimes extreme muscular tension.

These muscular responses to nociceptive stimuli make the clinical diagnosis of malfunctions and the locating of the primary nocigenerator so difficult.[52, 65, 67]

Especially in the temporomandibular joint with anterior disc displacement, antagonistic joint surfaces are incongruent. This results in minimal joint contact surfaces at high repetitive stress especially with compressed joint space. But even with a centered disk position the load results in compression of the joint surfaces in the lower and the upper jaw joint space to repetitive high stress vectors. With simultaneous retral displacement of the condyle, compression in the bilaminar zone and repetitive stimulation of the mechanoreceptors and nociceptors also occur The temporomandibular joints are "stress-loaded," meaning that force of the bite in occlusion is transmitted through the temporomandibular joints into the cranium.[68, 69] In animal experiments (pig) it has been proven that when chewing, a considerable load (stress loading/strain) occurs during joint compression. This study assumes that a protrusive occlusal splint reduces the stress generated by the muscle strength in the TMJ. The study also showed that the stress in the TMJ leads to a distortion in the temporal bone ("bone squamosal"). In mastication it occurs in both joint partners, TMJ and os temporale, a distortion with different aspects.[47] The condyle–fossa distance varies during the chewing cycle. The condyle–fossa distance is smaller at the final closing movement than in mouth opening. The condyle–fossa distance is less on the mediotrusion (balancing side) than on the laterotrusion side (working side). Likewise, the condyle–fossa distance is less during crushing hard food as when chewing soft food. Results of this dynamic stereometric study show that both temporomandibular joints are stress-loaded during the chewing cycles. On the mediotrusion side, the force vector is higher than on the laterotrusion side.[70] In another animal test (monkeys), a force transmission from the TMJ into the cranium, particularly here in the zygomatic bone (zygomatic arch) and the temporal bone could be shown.[71] In a Class I and a Class II relationship, the maximum force vector is on the mediotrusion side; in a Class III relationship rather more on the laterotrusion side. The maximum distortion of the mandible occurs in menton.[70]

Composition of the mastication muscles and their pain process in muscles

The anatomy of the jaw muscles is complex. The motor units within the *M. masseter*, *M. temporalis,* and the *M. pterygoideus medialis*, the elevators, are arranged in a highly complex manner within each muscle. The muscle fibers on the whole of the *M. masseter* do not run from the zygomatic arch to the ramus but rather there are small compartments of short fibers divided by aponeurotic sheaths and arranged in a so-called pennate manner. Therefore, when motor units on one side of a compartment contract, force can be generated at an angle, the pennation angle to the long axis of the muscle, with a force vector at an angle to the force vector that would be generated if muscle fibers passed directly from the zygomatic arch to the ramus without pennation. Patterns of pennation vary between muscles, which are thus classified as unipennate, bipennate, and multipennate. Examples of all three can be found in the jaw muscle. The *M. masseter* is an example for a multipennate pattern. These complexities of muscle-fiber architecture, together with selective activation of certain motor units within one muscle, provide a wide range of directions with which forces can be applied to the jaw and thereby contribute to the enormous range and sophistication of jaw movement that are possible. When generating a particular movement of the jaw, the sensorimotor central nerval system that drives voluntary movements is not organized in terms of specific muscle to activate. Rather, it sends a command signal to activate those motor units, in whatever muscles are available, that are biomechanically best suited to generate the force vector required for that particular jaw movement.[72, 73]

The origin of muscle tension and pain in the muscle can be in the muscle itself, as in an adjacent joint. In a TMD, participation of the chewing muscles is common and requires targeted treatment[1] and exploration for the cause.

"Pain of the muscles of mastication is the sole complaint in about three out of four CMD patients, while the rest have pain either restricted to the TMJs alone or involving both the TMJs and the masticatory muscles."[74]

5

TREATMENT OF DIFFERENT MALOCCLUSIONS WITH DIFFERENT ALIGNER SYSTEMS

This chapter will provide examples of different malocclusions and their treatment with aligner orthodontics, step by step. The topics will show different malocclusions, but with a focus on one main aspect of the malocclusion when determining treatment for this particular problem. No further details will be given for treatment of other issues in the patient. The aligner systems shown are "outsourced aligner systems" (Invisalign, Air Nivol, Angelalign) and "in-office aligner systems" (OnyxCeph Software).

Topic 1.1

In-office aligner treatment

Dr. Fayez Elkholy, Dr. Julia Funke

Introduction

Aligner treatment has been established as an almost invisible orthodontic treatment option over the past two decades. It offers certain advantages to both practitioner and patient. Since the introduction of the concept of moving teeth with appliances based on setup models and the introduction of different aligner materials, several aligner concepts were introduced. The first concept was based on CAD/CAM planning and fabrication of the aligners in an external laboratory and, at that time, was solely provided by Invisalign (Align Technology, Santa Clara, CA, USA). After the introduction of new digital printers and the advances of the aligner setup software, more companies emerged in the orthodontic field; eg, CA Digital (Scheu Dental, Iserlohn, Germany), Accusmile (Forestadent, Pforzheim, Germany), and eCligner (Seoul, South Korea). The second concept was mainly used for in-office aligner fabrication on plaster models with the teeth displaced manually for each setup step. This concept was limited only to a certain range of movement due to the inaccuracy of the manual tooth movement on the plaster models. This limitation was, however, eliminated by the introduction of more affordable digital planning and printing solutions, allowing not only for more accurate but even more complex but controlled tooth movements. The latter dramatically increased the interest for in-office aligner fabrication. In this chapter we will introduce a step-by-step overview of the current digital workflow for in-house aligner fabrication.

Different aligner approaches

Generally, two different aligner approaches are applied in clinical practice. The main differences between these approaches lie in the amount of tooth displacement and number of aligners fabricated for each setup step. The first approach comprises dividing the tooth movements into small setup increments of up to 0.2 mm and circa 2° for linear and rotational movements, respectively, with the fabrication of one aligner with uniform aligner thickness on each setup step. The second approach is based on larger setup increments of up to 0.5–0.75 mm. On each setup, however, aligners of different thicknesses of 0.4–0.8 mm, depending on the system used, are thermoformed. This sequential aligner thickness provides a gradual force increase for each setup step. A general advantage of this approach is mainly in the larger setup increments, reducing the number of models.

The originally recommended aligner sequence for this approach was 0.5 mm, 0.625 mm, and 0.75 mm. However, evidence showed that this aligner sequence would induce high force application on the teeth, leading to overloading of the periodontal ligament as well as a non-uniform force increase within the setup steps. Therefore, a new aligner sequence with a thinner initial "leveling" aligner with a thickness of 0.4 mm and omitting the 0.625 mm foils was established. In this manner, the newly recommended gradational aligner sequence was 0.4 mm, 0.5 mm, and finally the 0.75 mm aligner.

Digital workflow for the in-office aligner fabrication

The process of conventional analog aligner fabrication is well known to almost every dental practitioner, beginning with impression-taking, separating and moving the teeth on the plaster setup models, and finally thermoforming the aligners on these models. The general steps of digital in-office aligner fabrication are not much different. In truth,, however, an in-depth understanding of the

digital aligner workflow, as well as the presence of suitable software and hardware is crucial for a successful aligner treatment (Fig 5-1-1). The digital workflow usually begins with the creation of a 3D model of the patient's dentition, usually followed by importing and preparing the 3D models, tooth separation, and creation of the digital setup. The separated model can then be used in the actual aligner software and, if required, placing the different modifications (eg, attachments or pressure points). Lastly, comes the staging process, ie, creating the single setup steps and exporting each step as a separate 3D model which will be used for thermoforming the aligners.

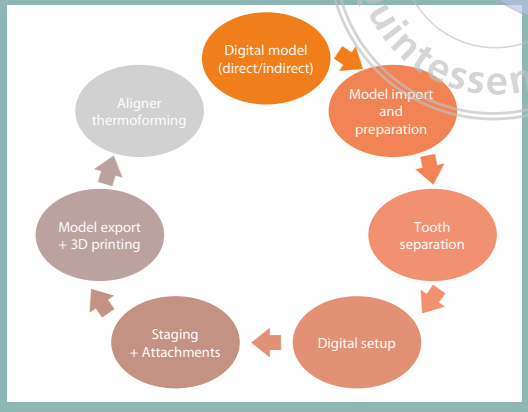


Fig 5-1-1 Overview of workflow for the digital in-office aligner fabrication.

Meanwhile, different in-house aligner planning programs are available in the orthodontic field. A few of these programs are presented in Table 5-1-1. In the following sections, however, we will only compare a few of these options, including Archform (St. Gallen, Switzerland) and OnyxCeph3 (Image Instruments, Chemnitz, Germany).

Table 5-1-1 In-house aligner planning programs available in the orthodontic field

Material	Name	Manufacturer	Film thickness [mm]	E-Modul [MPa]	Spacer foil included	Use
PET-G Polyethylene-terephthalate Glycol	Duran	Scheu Dental, Iserlohn, Germany	0.4 / 0.5 / 0.625 / 0.75	2200	no	Tooth movement
	Clear-Aligner	Scheu Dental, Iserlohn, Germany	0.4 (extra-soft) / 0.5 (soft) / 0.625 (medium) / 0.75 (hard)	2200	no	Tooth movement
	Biolon	Dreve, Unna, Germany	0.5 / 0.625 / 0.75	2020	no	Tooth movement
	Erkodur	Erkodent, Pfalzgrafenweiler, Germany	0.5 / 0.6 / 0.8	2200	yes	Tooth movement
	Track A	Forestadent, Pforzheim, Germany	0.5 / 0.63 / 0.8	N.A.	yes	Tooth movement
PP Polypropylene	COPY-PLAST	Scheu Dental, Iserlohn, Germany	1.0	450	no	Attach-ment tem-plate
Multilayer Multilayer (Cycloaliphatic Polyester / Polyether Poly-urethan) PU Polyurethane	Smart Track	Align Technology, Santa Clara, CA, USA	0.76	N.A.	no	Tooth movement
	Zendura FLX	Bay materials, Fremont, CA, USA	0.38 / 0.625 / 0.76 / 1.02	1256	no	Tooth movement
	Zendura A	Bay materials, Fremont, CA, USA	0.38 / 0.625 / 0.76 / 1.02	1938		

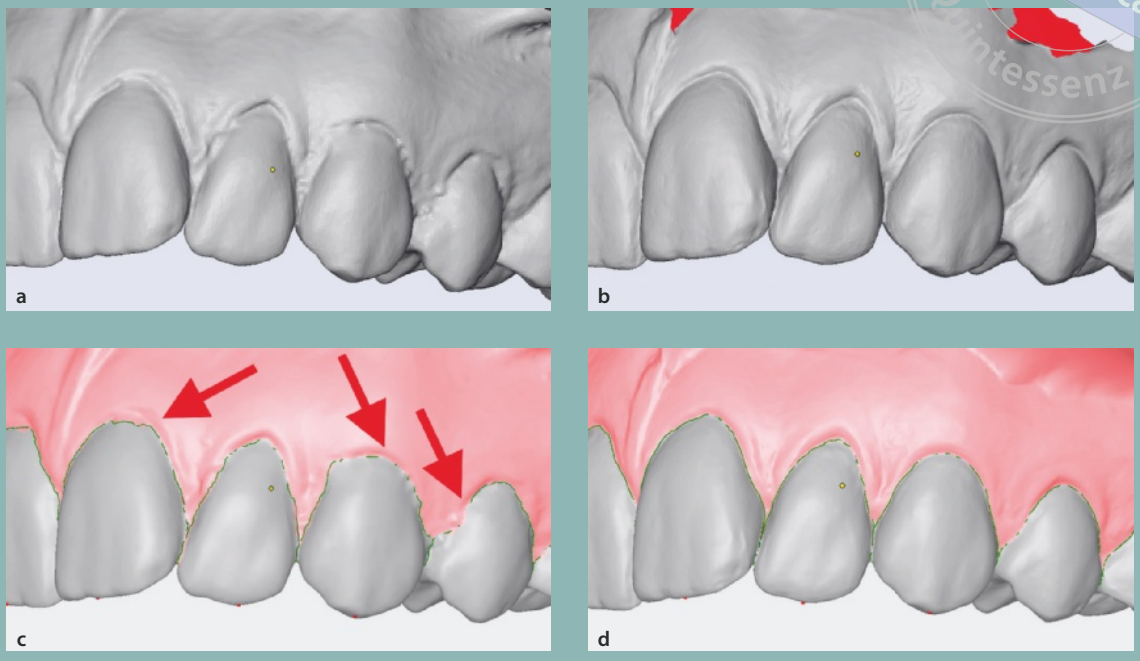


Fig 5-1-2 (a–d) Comparison of 3D models acquired indirectly by means of (a) a plaster model scan and (b) an intraoral scan as well as the corresponding segmented models from (a) the model scan and (b) intraoral scan, respectively. As illustrated by the red arrows (c), the plaster model scan contained more artifacts hindering a sharp definition of the exact crown edges (d).

Digital model acquisition

The first step in digital aligner planning is the acquisition of a virtual 3D model of the patient's teeth. This step should be performed thoroughly to facilitate the further model manipulation steps. Currently, there are two main options to digitize the patient's dentition, either indirectly by digitizing the patient's plaster models or directly by intraoral scanning. The first method is accompanied by higher laboratory costs. Moreover, the quality of the 3D models created is usually inferior to those created by intraoral scanning due to the expansion of the plaster used, as well as the presence of artifacts or air bubbles, especially in the gingival margin region (Fig 5-1-2). Intraoral scanning, on the other hand, usually produces well-defined crown contours as well as a distinct gingival margin which is of an utmost importance for shape-driven appliances, eg, aligners. This aspect is also important to allow for an exact tooth segmentation in the designated software (Fig 5-1-2c, d).

However, in some aligner systems, the aligners extend beyond the dental areas, covering some soft tissue regions to speed up the post-thermoforming finishing of the aligners in the laboratory. For these systems, other regions should be thoroughly captured by the practitioner to ensure the comfortable fit of the aligners and to avoid pressure areas on the soft tissues. These include the oral and vestibular gingival and vestibular areas, sublingual region, labial, buccal, and lingual frenula, as well as the maxillary and mandibular buccal vestibule (Fig 5-1-3). Furthermore, just like any normal orthodontic diagnostic and treatment planning process, the occlusal condition of the patient should be accurately registered to avoid a post-therapeutic occlusal instability. Most scanners provide a live examination of the occlusal contacts after performing the occlusal scan (Fig 5-1-4).

In addition to the quality and orientation of the scanned models, the export capabilities of the 3D model or intraoral scanner is also an important factor enabling the practitioner to further utilize the model in the further 3D analysis, manipulation, and segmentation in the designated software.[1]

Fig 5-1-3 Screenshots from the Trios intraoral scanner interface showing (a) an upper and (b) lower jaw scan. The green marked areas illustrate the important areas to be considered while performing an intraoral scan for aligner fabrication.

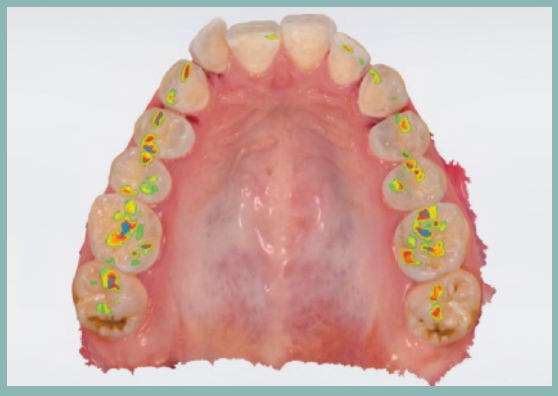
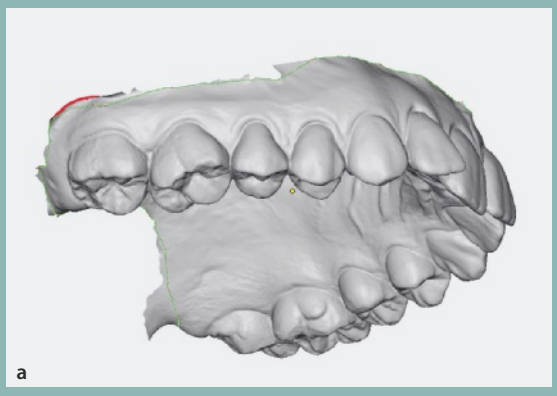
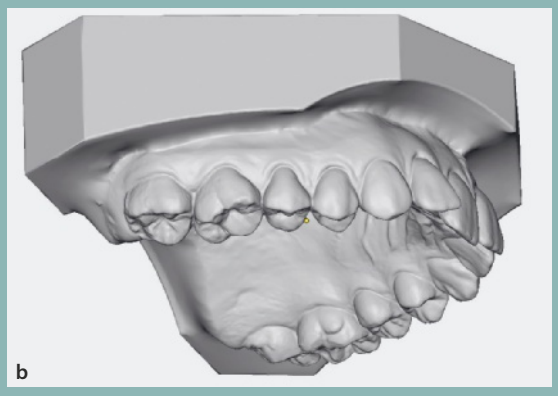


Fig 5-1-4 (right) Screenshot of the intraoral scanner interface (Trios 3, 3Shape) showing the visualized occlusal contacts after the digital bite registration and model aligning.

Fig 5-1-5 (below) An imported intraoral scan (a) before trimming with the green line marking the borders of the model, as well as (b) after trimming and adding a suitable model base in OnyCeph3.





Model import and preparation

After acquisition of the intraoral or model scan, the exported 3D models should be properly prepared for further processing. The preparation steps usually include trimming the redundant areas from the 3D Scan, filling small holes, and smoothing the edges of the 3D scan. This feature should be preferably integrated in the aligner planning software, otherwise, an external program should be used, requiring additional export and import steps. However, it is not yet incorporated in all programs. OnyxCeph3, for example, provides this feature in its standard import and preparation module (Fig 5-1-5). Furthermore, the models should be oriented according to the occlusal, tuberosity, and midpalatal raphae plane (Figs 5-1-6 and

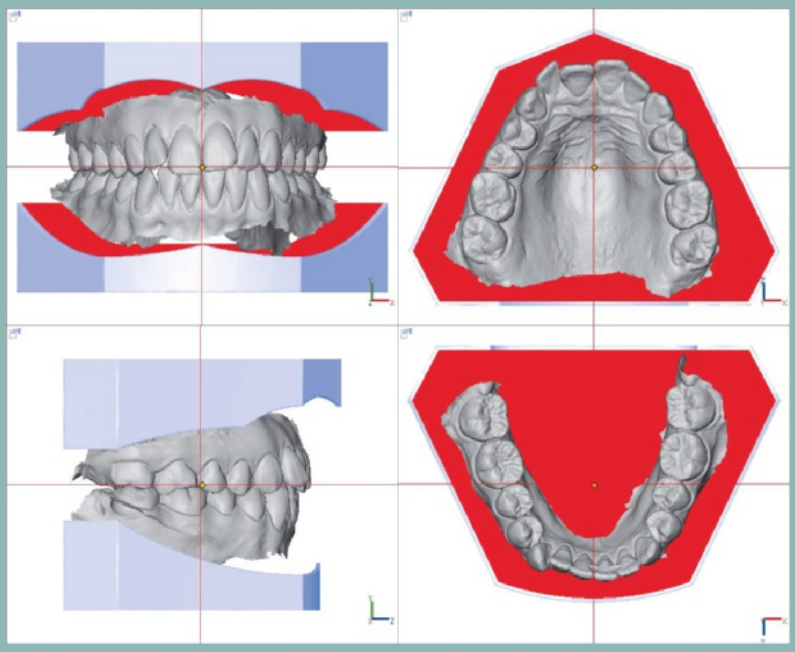


Fig 5-1-6 Model preparation module of OnyxCeph3 showing the different planes used for model orientation including the occlusal, tuberosity, and midpalatal raphae planes.

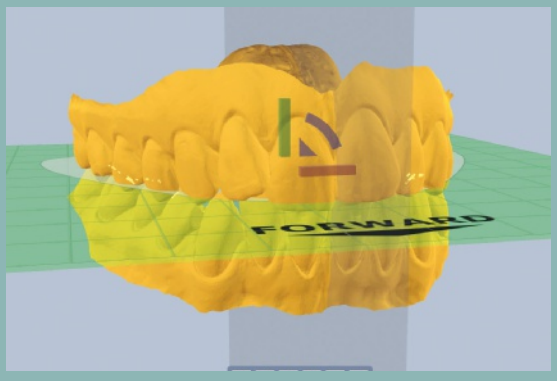


Fig 5-1-7 Model preparation module of Archform, showing the different planes used for model orientation with the option to tilt/translate the model in relation to these planes.

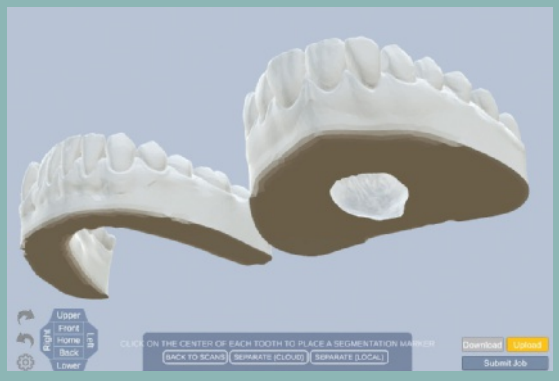


Fig 5-1-8 Capped upper and lower models during model preparation in the Archform software.

5-1-7). This orientation is sometimes used by the aligner planning programs as reference planes for the different tooth movements. Other programs, however, only provide the ability to use single tooth coordinate systems for a single tooth. The final steps of the model import process are usually by final fine trimming as well as adding and fusing an appropriate model base to the scanned and oriented virtual models (Fig 5-1-5).

Some programs do not include this feature; they do, however, allow capping of the upper and lower virtual models after manual adjustment of the model orientation and closure of the capped areas with a flat surface, creating a closed horseshoe model of both jaws (Fig 5-1-8).

Model segmentation and tooth separation

The next step is the segmentation of the virtual models. During this process the software divides the single crowns from the virtual model. Some programs require a manual definition of the crown margins, while others

Topic 1.2

Flow Chart of the In-Office Aligner Technique

1. Scan

- Maxillary and mandibular arch, maxillary with palate
- Occlusion
  - *TMD no → scan in habitual intercuspidation*
  - *TMD yes → scan in therapeutic condyle position*

2. Match and Prepare

- Matching scan into OnyxCeph3 software
- Preparation and segmentation of virtual models (Fig 5-1-2-1)

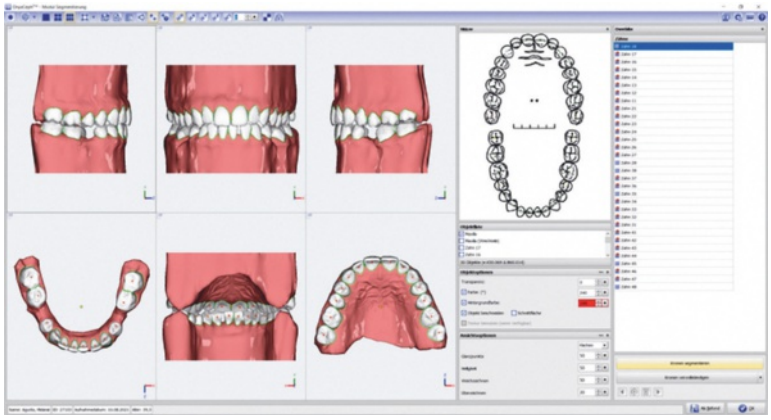


Fig 5-1-2-1

3. Basic Thoughts in Orthodontic Treatment Planning

- What is the main goal of the treatment?
- Which teeth should not be moved? (Fig 5-1-2-2)
  - *No movement of these teeth at this stage because of anchorage*
  - *No movement of these teeth is necessary*
  - *TMD – treatment with COPA – onlay in phase 1, do not move the teeth with COPA onlay and the antagonists*

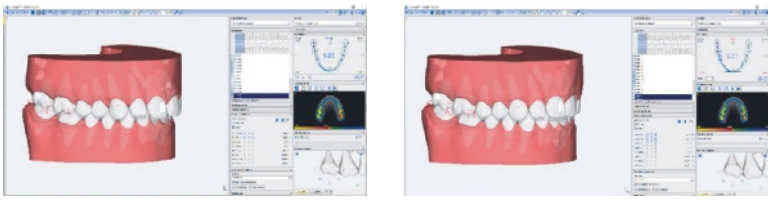


Fig 5-1-2-2

3. Basic Thoughts in Orthodontic Treatment Planning

- Transversal movement
  - *If necessary, start VTS with transversal movement*
  - *Transversal correction tooth by tooth*
  - *Transversal correction entire arch (Fig 5-1-2-3)*
  - *Not necessary*
- Sagittal movement
  - *Necessary, see cl. II/III*
  - *Not necessary*
- Vertical movement
  - *Necessary, see deep bite, open bite*
  - *Not necessary*

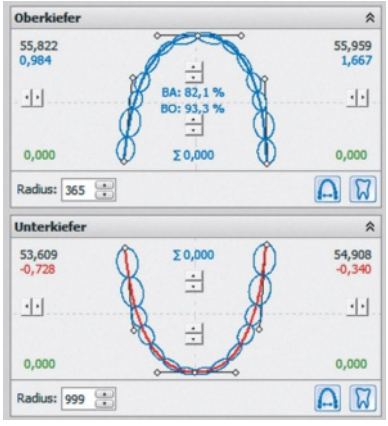


Fig 5-1-2-3

4. Treatment Planning Related to the Anomaly in Virtual Treatment Simulation (VTS) in OnyxCeph3 – V.T.O.3D

1 Spacing

- Space closure
  - *From anterior, from posterior or in combination*
- Space allocation for symmetry and possible later restorative dentistry
- Avoid tipping especially when closing space from posterior -> slow staging and angulation with translation
- Avoid space closing by means of mesialization of molars of more than 3 mm → auxiliaries needed

2 Crowding

- Extraction
  - *Planned before VTS*
  - *Planned within the VTS*
- Anterior proclination if sufficient buccal alveolar bone and gingiva existing
- Expansion
  - *In combination with buccal corridors*
  - *If compatible with periodontal situation and gingiva type*
- Distalization maxillary/mandibular

Topic 1.3

Attachments and bonding procedure

Orthodontic treatments with aligners require the use of attachments, which are composite resin bulks attached to tooth surfaces. Attachments have widened the clinical indication of aligner orthodontics over the years, allowing treatment of difficult and complex cases.[1, 2]

Cai et al examined the optimization of configuration of attachment in tooth translation with transparent tooth correction by appropriate moment-to-force ratios with biomechanical analysis. They concluded that the attachments can be designed and placed appropriately to improve tooth movement.[3]

On the other hand, the absence of these supplementary elements could lead to undesirable inclinations of teeth during translation movements, as described by Mantovani et al[4] or Gomez.[5]

In their study examining the effect of gingival margin design on retention of thermoformed orthodontic aligners, Cowley et al concluded that aligners with a 2 mm straight gingival margin design had the highest retentive forces when compared to aligners of the same material and attachment type. Due to the longer aligner margin in in-office aligners, attachments can be used optimally as the retention is higher than with scalloped aligner margins.[6]

The material of the composite used for the attachments has been examined in several studies, showing that different composite materials could be used for the attachments.[7–9]

An experimental *in vitro* study by Vincenzo D'Antò et al examined the influence of dental composite viscosity in attachment reproduction. The results of this study demonstrated that the use of different composites with different viscosities does not influence the shape and volume of attachments reproduced with a template on extracted teeth. Furthermore, the orthodontic composite showed more overflow respect when compared with the flowable one. Though this study showed more overflow with hard composite, the authors believe that the use of hard composite and particularly less abrasion during more complex treatment allows better anchorage.[10]

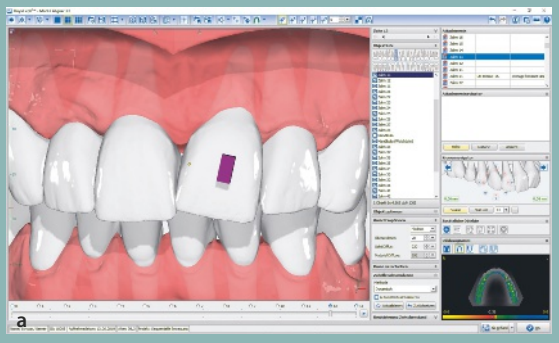
Still, transferring the attachment from a virtual situation with a template onto the teeth seems to be a challenge. The precision of the attachment after the bonding procedure with an attachment template shows different aspects depending on the template material used. A study by Weckmann et al was performed to test the influence of attachment bonding protocol on precision of the attachment in aligner treatment with a thermoplastic foil of 0.6 mm thickness (Erkodur). The largest deviation was found using high viscous composite without a perforation in the attachment reservoir with a median difference of 0.41 mm. The most accurate result delivered the two-phase procedure with high viscous composite with a median difference of 0.13 mm. Regarding the excessive composite area bonded around the attachment, the smallest amount was found for the protocol with high

Table 5-1-3-1 Description of different attachment types we recommend for different movements; depending on the movement, size and shape may vary

Type	Movement	Size/length	Size/depth
Rectangular vertical (Fig 5-1-3-1)	- Angulation - Derotation - Bodily movement	2, 3, or 4 mm	1–1.5 mm
Rectangular vertical tipped (Fig 5-1-3-2)	Derotation	2, 3, or 4 mm	1–2 mm
Rectangular horizontal (Fig 5-1-3-3)	Vertical movement: - Anchorage (eg, on neighboring teeth of desired intrusion) - Extrusion	2 or 3 mm	1–1.5 mm
Beveled (Fig 5-1-3-4)	- Extrusion	2 or 3 mm	1–2 mm
Semilunar (Fig 5-1-3-5)	- Extrusion	2 mm	1–1.5 mm
Torque element (negative attachment, Fig 5-1-3-6)	- Torque	2 mm	–0.7 mm

viscous composite for both types of attachments. The median difference for the ellipsoid attachment was 7.40 mm^2 and 6.20 mm^2 for the rectangular attachment. The largest amount of excess composite was detected in the protocol with low viscous composite without a perforation in the attachment reservoir. The amount of excess for the ellipsoid attachment was 33.50 mm^2, and for the rectangular attachment 19.85 mm^2.[11]

Bruno et al studied the bonding procedure with an Invisalign attachment template vs a Spark attachment template. The Spark group showed, in general, a lower frequency in debonding in comparison with the Invisalign group, as 87.5% of patients in the Spark group did not show any bonding failure versus 27.5% in those of the Invisalign group. At template removal, the Spark template showed less attachment debonding compared with the Invisalign template. The Spark template can be considered more effective in attachments transferring to the tooth surface than the Invisalign one. The hypothesis of the present study can be extended to try to explain the results that have emerged, in that the Spark template appears to be less rigid than that of Invisalign, and that,



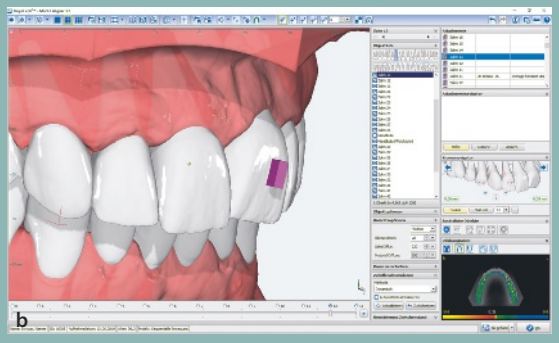
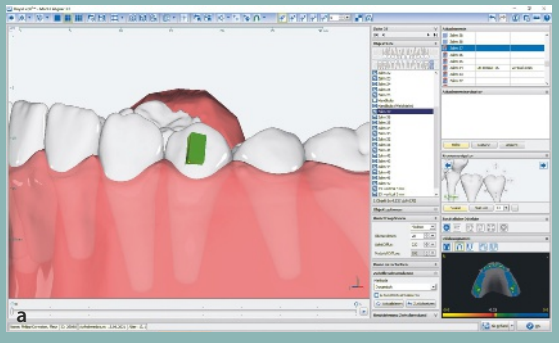
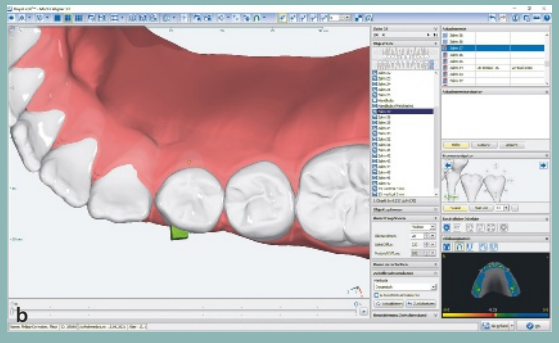


Fig 5-1-3-1 (a, b) Rectangular vertical attachment.





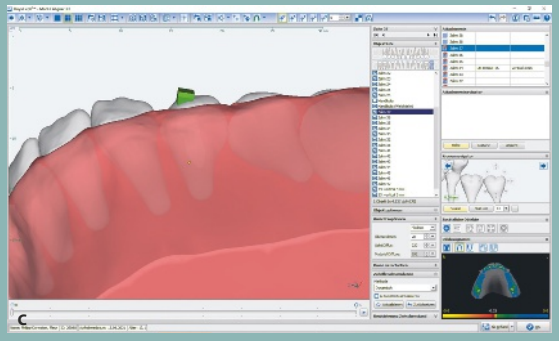
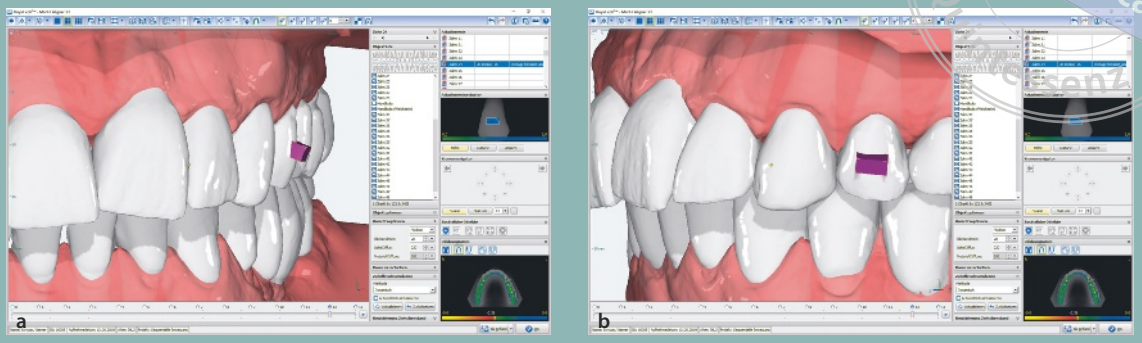


Fig 5-1-3-2 (a–c) Rectangular vertical tipped attachment.



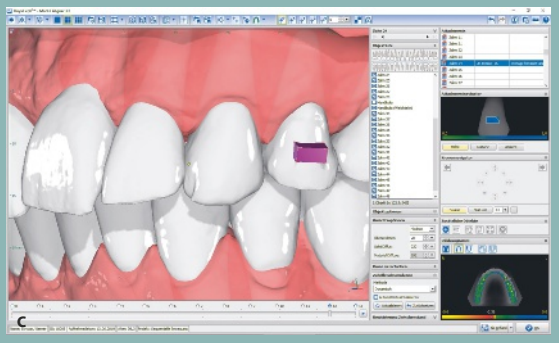


Fig 5-1-3-3 (a–c) Rectangular horizontal attachment.

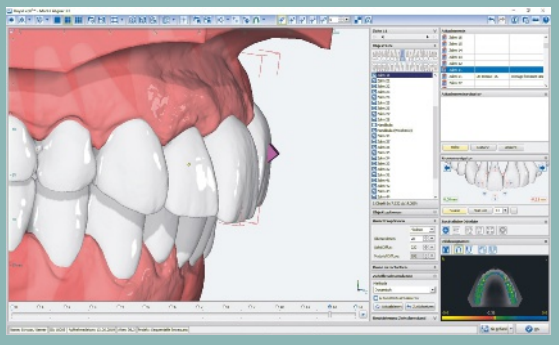


Fig 5-1-3-4 Beveled attachment.

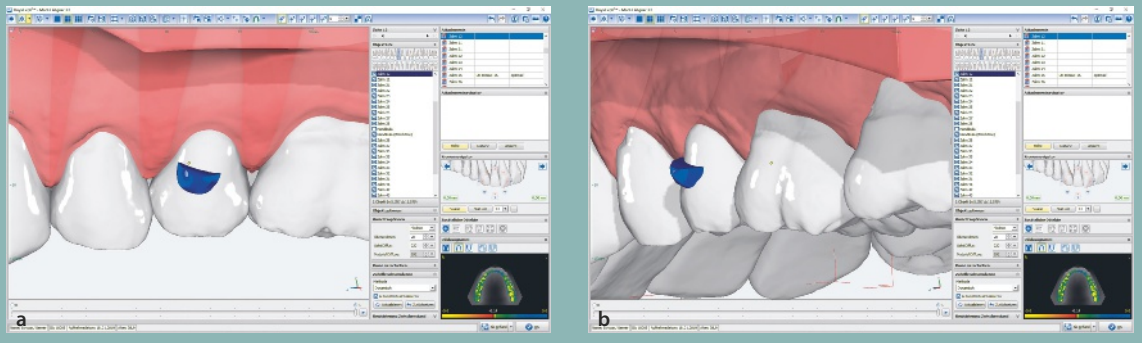
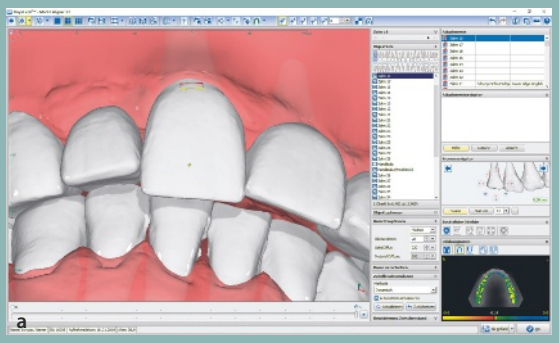


Fig 5-1-3-5 (a, b) Semilunar attachment.



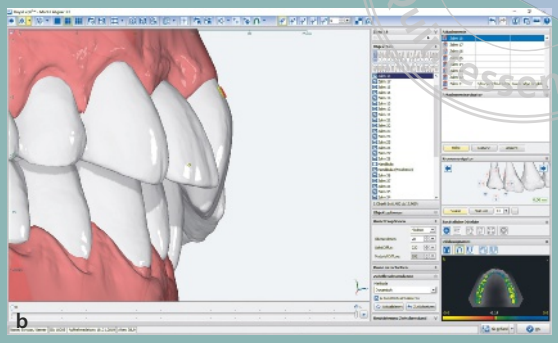
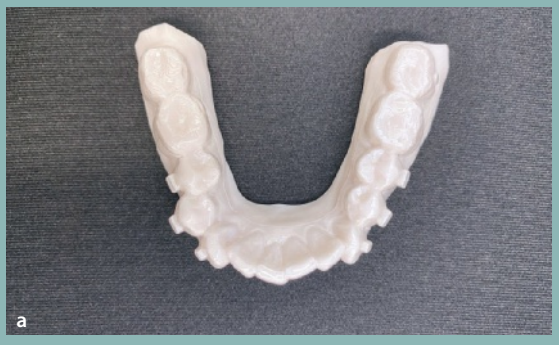


Fig 5-1-3-6 (a, b) Torque element.



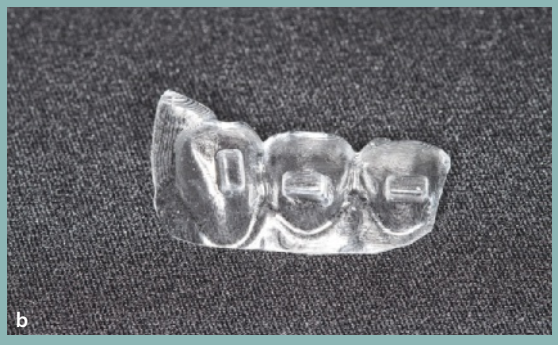


Fig 5-1-3-7 (a) Attachment template on the model made of 0.4 mm Duran (Scheu Dental) with a vertical rectangular attachment on teeth 33, 43, and horizontal rectangular attachments on teeth 34, 35, 44, 45 for transfer. (b) Template prepared for bonding of attachments 33, 34, 35.

therefore, this might allow a less traumatic template removal from the dental arch and from the just-bonded attachments.[12]

As a further hypothesis, we refer to the fact that the Invisalign attachment template has grooves due to the scan lines compared with the line-free Spark attachment template. These lines of the Invisalign attachment template are reflected in the bonded Invisalign attachment. Plastic often remains in the undercuts built by these grooves when the Invisalign attachment template is removed.

To avoid the inaccuracy of the bonding process, the in-office aligner treatment offers the option of bonding the attachment directly and before scanning (see, eg, Chapter 5, Topic 4). The scanner recognizes the attachment exactly like enamel, so that the volume of the attachment is just as precise as the volume of the enamel. The aligner fitting with respect to the attachment is the same as the enamel. The setting of direct bonded attachment requires experience and knowledge of biomechanics in aligner orthodontics for the correct position and shape.

Alternatively, we design the attachment in the OnyxCeph software and determine the size, angulation, and position of the horizontally or vertically attached rectangular attachment (Figs 5-1-3-1 to 5-1-3-3) according to any other possible shape of attachments (Figs 5-1-3-4 to 5-1-3-6). For the attachment template, we use 0.4 mm Duran (Scheu Dental), a crystal-clear foil without grooves (Fig 5-1-3-7) allowing perfect transferal of the virtually planned attachments. A maximum of three attachments bonding simultaneously is easiest. The attachment template should be adapted onto the tooth surface with an instrument such as, eg, a Heidemann spatula on all sides

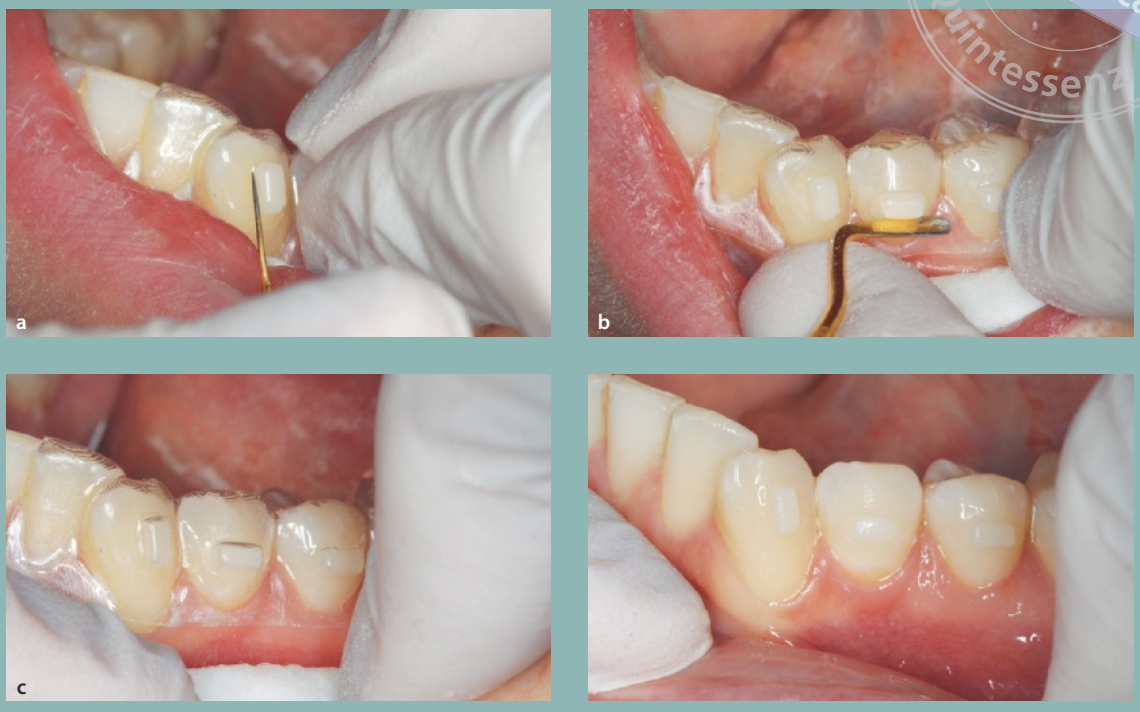


Fig 5-1-3-8 Adaption of the attachment template onto the tooth surface with an instrument such as a Heidemann spatula (a, b); attachment template in situ with transferred attachments (c).

Fig 5-1-3-9 Transferred attachments on teeth 33, 34, and 35.

of the attachment and finally held at the lower edge toward the gingiva with the spatula to obtain optimal transferal (Fig 5-1-3-8). In our experience, a high viscous composite (sealer: OptiBond, Kerr, FL, composite: Enamel plus HFO (GDF)) allows best transferal and esthetics (Fig 5-1-3-9).

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Topic 11

Class II non-extraction treatment in adults

Lin and Melsen showed that in the case of a moderate buccal Class II tendency, 55% of the molars were in a Class I relationship when evaluated from the lingual side. In Class II relation molar rotation occurred in 74% of the cases. The rotation is around the mesiolingual cusp. This fact shows the need for derotation of maxillary first molars in Class II treatments.[1] Figure 5-11-1 shows a mild Class II molar relationship on the right side at the start of treatment planning. With a rotation of tooth 16 around the distopalatal root to distal, it was possible to achieve a Class I molar relationship without further distalization (Fig 5-11-2). Figure 5-11-3 shows the amount of movement in the superimposition tool. This needs to be taken in consideration when treating Class II patients as it may help to reduce treatment time and improve the occlusal outcome.

Class II non-extraction treatment can be performed well with aligner orthodontics. This included the combined treatment of a skeletal Class II in the growing patient as well as Class II treatments with distalization or even a combination with surgery in an adult.[2-5]

When we want to perform distalization in the maxilla to transfer a Class II relationship into a Class I relationship, the biomechanics need to be considered and understood in detail. Figure 5-11-4 shows the force effects occurring when teeth are moved. Because exertion of a force by one body on another generates an equal and opposite reaction, anchorage is needed whenever force

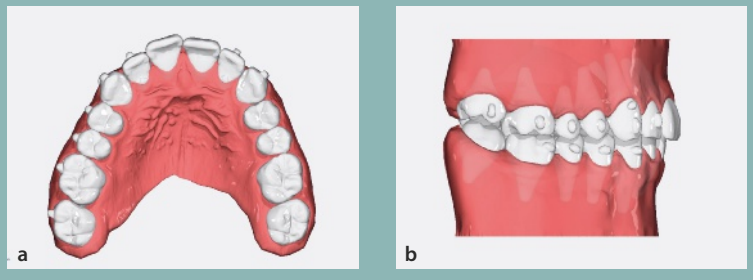

Fig 5-11-1 (a, b) Initial situation transferred into the OnyxCeph software with minor Class II division on the right side, tooth 16 shows a mesiobuccal rotation.

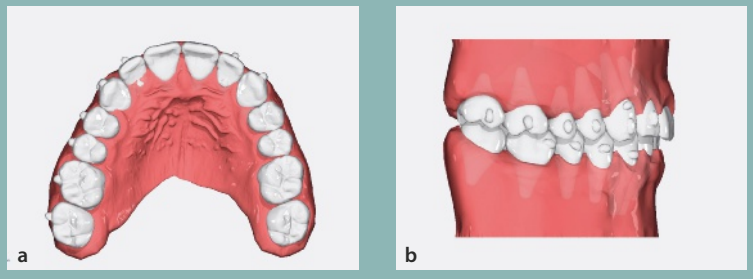

Fig 5-11-2 (a, b) Planned final outcome in the OnyxCeph software: after rotation of tooth 16 with the mesiobuccal cusp to distal, the rotation has led to a Class I molar relationship.

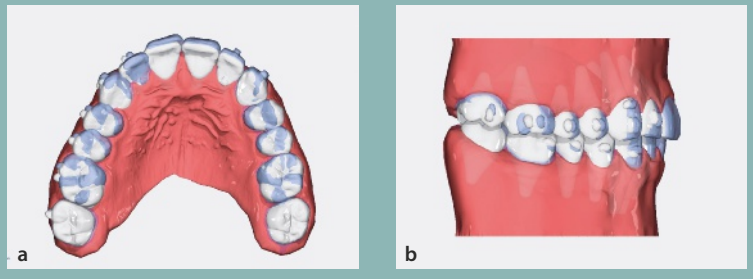

Fig 5-11-3 (a, b) Superimposition of the initial situation (purple color) and the planned virtual outcome (white color), demonstrating the amount of rotation of tooth 16 around the distopalatal root.

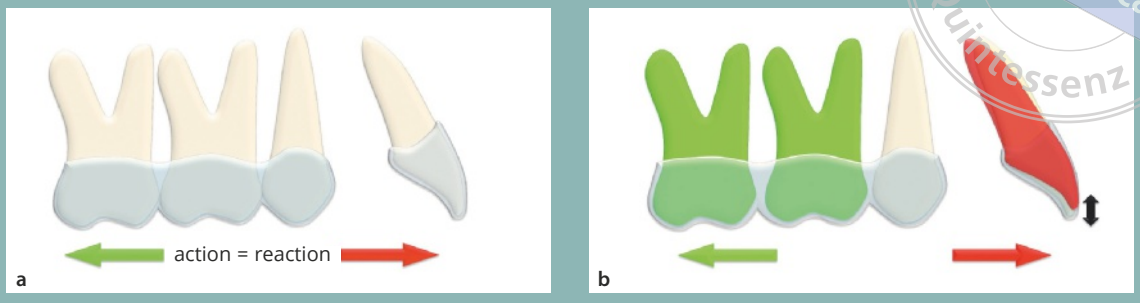

Fig 5-11-4 Tooth movements under force. (a) A force exerted by one body on another generates an equal and opposite reaction (Newton´s third law). (b) Loss of anchorage can lead to anterior proclination and relative intrusion of a tooth.

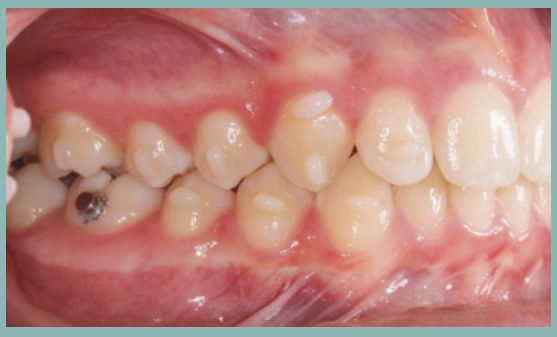

Fig 5-11-5 Composite hook (HF enamel) on a canine and button on a first molar for insertion of Class II elastics during aligner treatment. For better anchorage of the aligner during distilization, vertical attachments are also added on tooth 13, 14, 15, and 16.

Fig 5-11-6 (a) A precision cut (elastic hook) in an aligner for the elastic (b). A button cut-out can also be made in the opposite arch (c) (Align Technology). Alternatively, cuts can be performed chairside with instruments such as the "the gun" (Dr. Schwarze, Hammacher Dental).

is used to move teeth. Particularly when distalization is required with the Invisalign system, anchorage needs to be provided with Class II elastics to avoid undesired mesial counterforces.

Loss of anchorage may manifest with anterior proclination, resulting in a relative intrusion of the maxillary incisors; this will hamper the fitting of an aligner in this region. Attachments on the maxillary incisors at the start of the distalization of maxillary canines may be helpful for major anchorage.

In almost all patients with Class II relationship and planned distalization in the maxillary arch, we use Class II elastics from a clear hook on the maxillary canine to a bonded button on the mandibular first molar (Fig 5-11-5). To avoid rotation of the canine under the force of the elastics, an attachment is placed on the canine.

An alternative approach is to place a precision cut (elastic hook) directly in the aligner for the elastic wear. A button cut-out for bonded hooks or buttons can be requested in the opposite arch (Fig 5-11-6).

Based on our experience, it is best to use the following procedure to treat Class II relationships:

1. Start exclusively with the single distalization of the second molar.

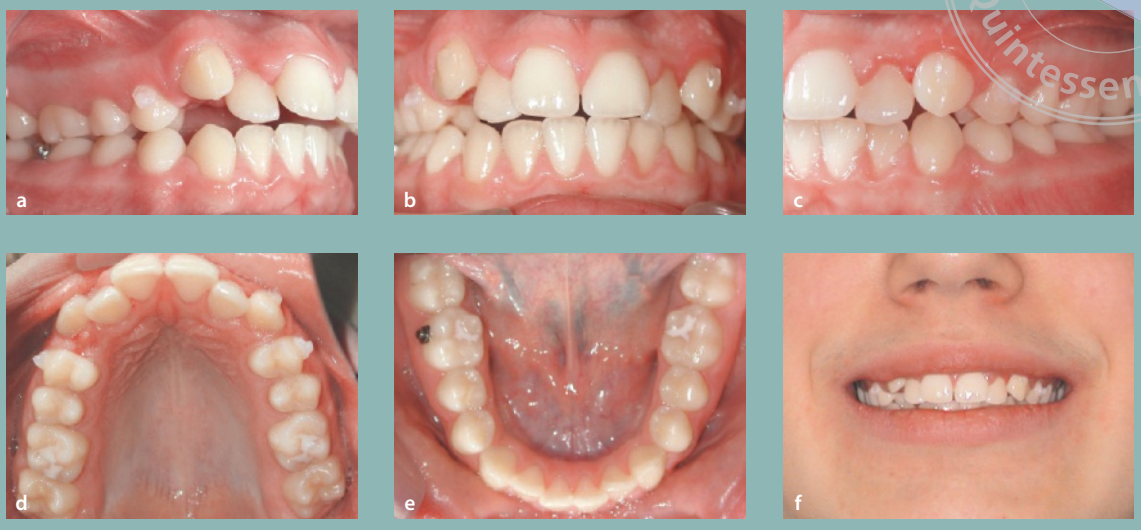

Fig 5-11-7 (a–f) Initial presentation with buttons and hooks placed (button on tooth 36 came off prior to picture taking and needed to be rebonded).

2. When the second molar is distalized 50%, start distal movement of the first molar.
3. Start premolar distalization when the second molar has reached its final position.
4. Start canine distalization when the first molar has reached its final position. Attachments on 12, 11, 21, and 22 can help to obtain major anchorage when the canine starts distalization.

The patient is asked to wear the elastic not only at night during the movement of the second molar, but also to wear it for an additional 3 hours during the day with the start of movement of the first molars. The patient is also advised to wear the elastics during the night for at least 3 months after active treatment is completed.

We consider the force of the Class II elastics for anchorage to be optimal at a maximum of 100 g for Class II and a maximum of 80 g for Class III.

Patient 1: Class II with highly erupted maxillary canines

This patient had a Class II relationship, crowding, and rotations in both arches, and highly erupted maxillary canines (Fig 5-11-7). He was one of the first patients we treated with distalization in the maxillary arch and the Invisalign treatment.

Diagnosis:

- *Class II relationship*
- *Crowding and rotations in the maxillary and mandibular arches*
- *Frontal open bite*
- *Highly erupted tooth 13 more than 23.*

Therapy:

- *Invisalign*
- *Myofunctional therapy*
- *Distalization in the maxillary arch with Class II elastics*
- *Alignment of the maxilla and mandible*
- *Extrusion of the highly erupted teeth 13 and 23.*

Treatment

At the start of treatment, vertical rectangular attachments were placed on maxillary canines for extrusion, as well as hooks on teeth 14 and 24 and buttons on teeth 36 and 46 for Class II elastic wear. ClinCheck software was used for

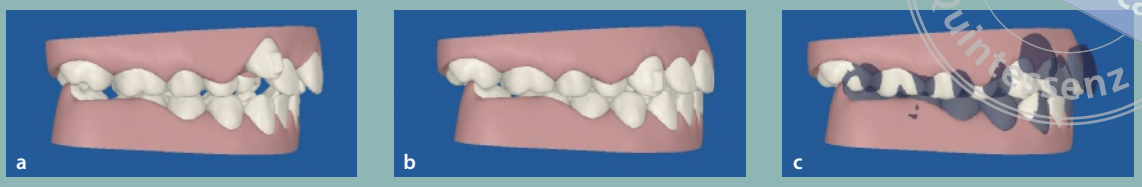

Fig 5-11-8 ClinCheck software results. (a) Initial situation on the right side. (b) Planned final situation. (c) Superimposition to show planned movements (blue, initial tooth position; white, final tooth position).

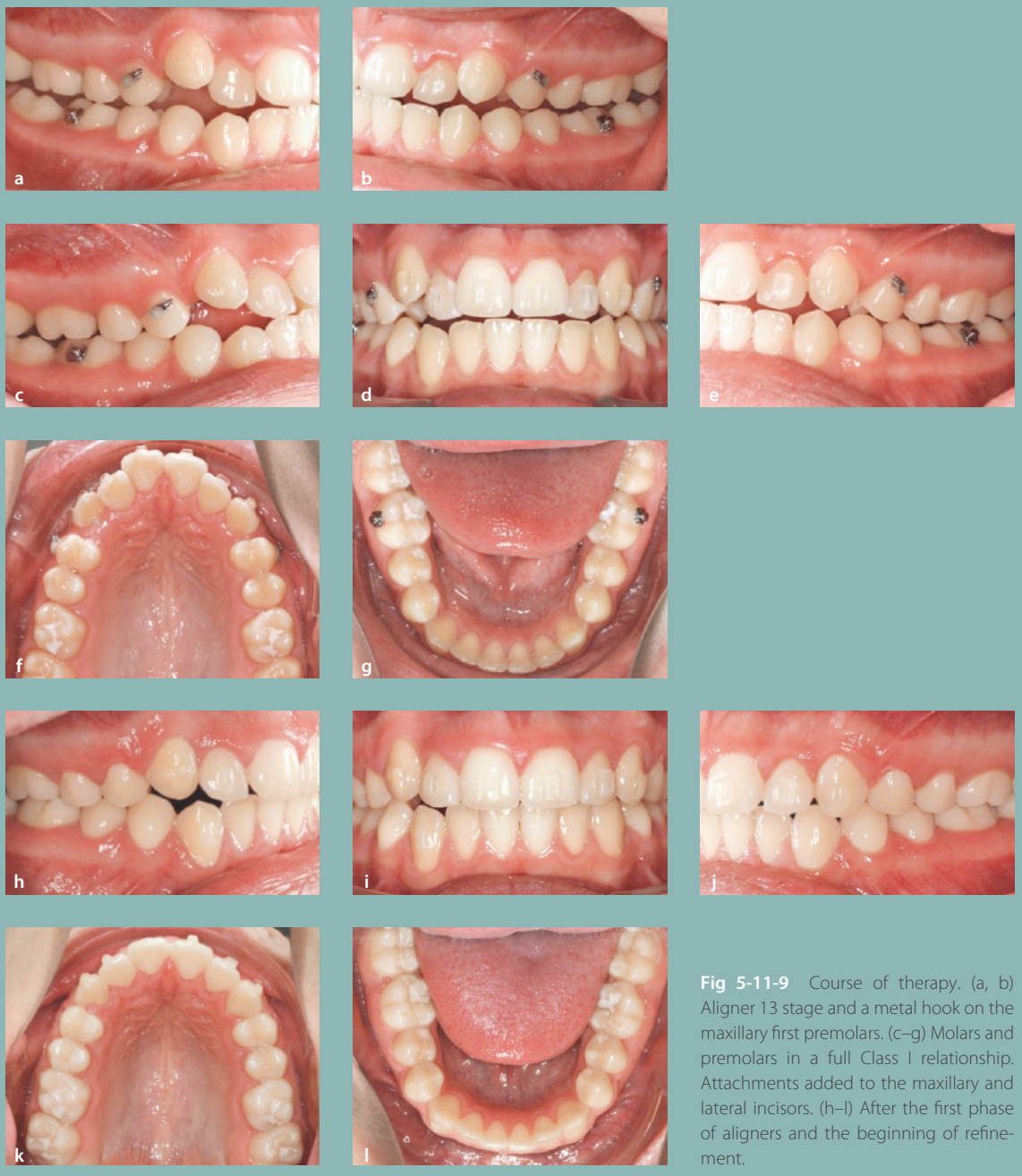

Fig 5-11-9 Course of therapy. (a, b) Aligner 13 stage and a metal hook on the maxillary first premolars. (c–g) Molars and premolars in a full Class I relationship. Attachments added to the maxillary and lateral incisors. (h–l) After the first phase of aligners and the beginning of refinement.

Fig 5-11-10 (a–e) Final result in Class I relationship and functional overjet and overbite and harmonically aligned arches.

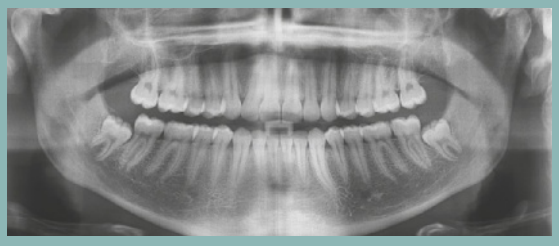

Fig 5-11-11 Final orthopantomography with good root inclination of maxillary posteriors after distalization. No root resorptions or pathologies are present, teeth 38, 48 were advised for extraction.

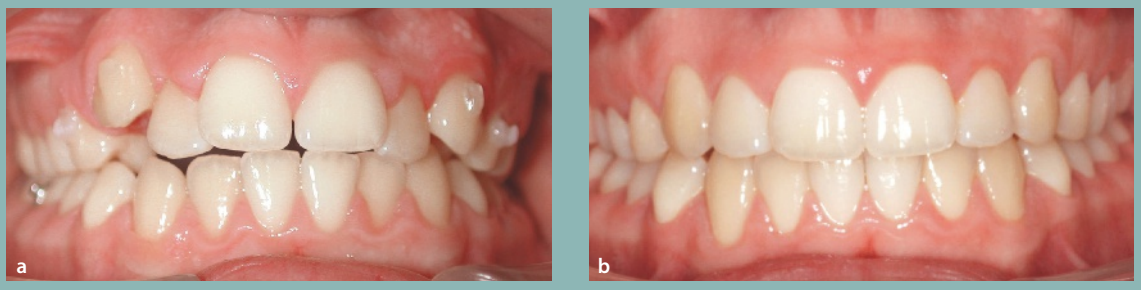

Fig 5-11-12 The initial (a) and final (b) frontal views.

planning, with 51 aligners in the first phase (Fig 5-11-8). Because of the large number of aligners, the patient was advised to change these every 10 instead of 14 days, which we used in the early years of aligner treatment. Figure 5-11-9 show the course of therapy. The transparent plastic hook on the maxillary first premolars was replaced during the treatment with a metal hook. Once the molars and premolars were in a full Class I relationship, attachments were added to the maxillary and lateral incisors for major anchorage during the upcoming distalization. After the first aligner phase, refinement occurred as tooth 13 still needed some extrusive movement to end up in sufficient canine guidance and there was still some rotation of the mandibular anterior teeth to correct.

The patient showed no decalcification, white spots, or root resorptions at the end of orthodontic treatment (Fig 5-11-10). The final orthopantomogram showed no pathologic findings (Fig 5-11-11). Extraction was advised for teeth 38 and 48. Comparison of initial and final views confirmed the success of treatment, with extruded teeth

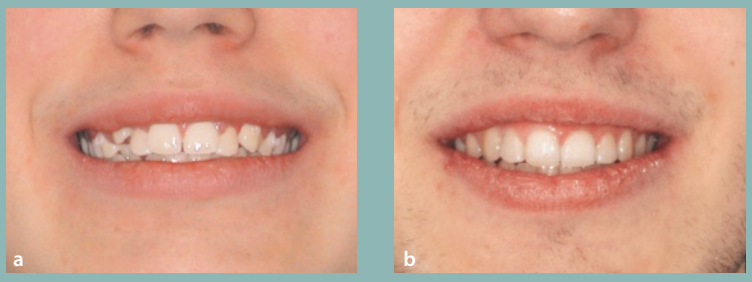

Fig 5-11-13 The initial (a) and final (b) frontal views showing the improved smile esthetics.

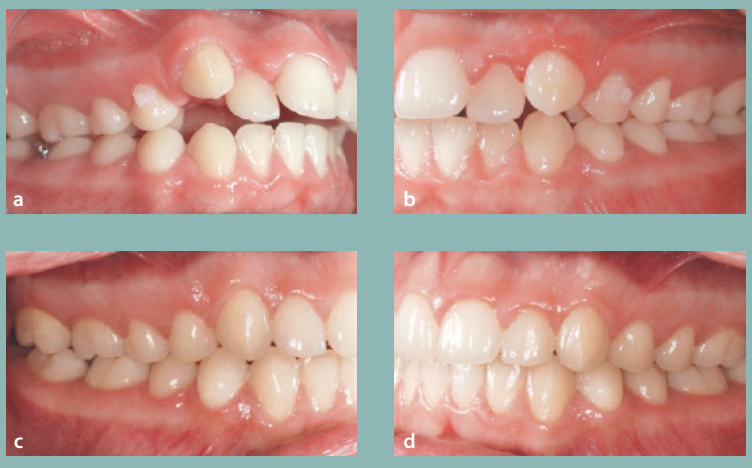

Fig 5-11-14 The initial (a, b) and final (c, d) occlusion.

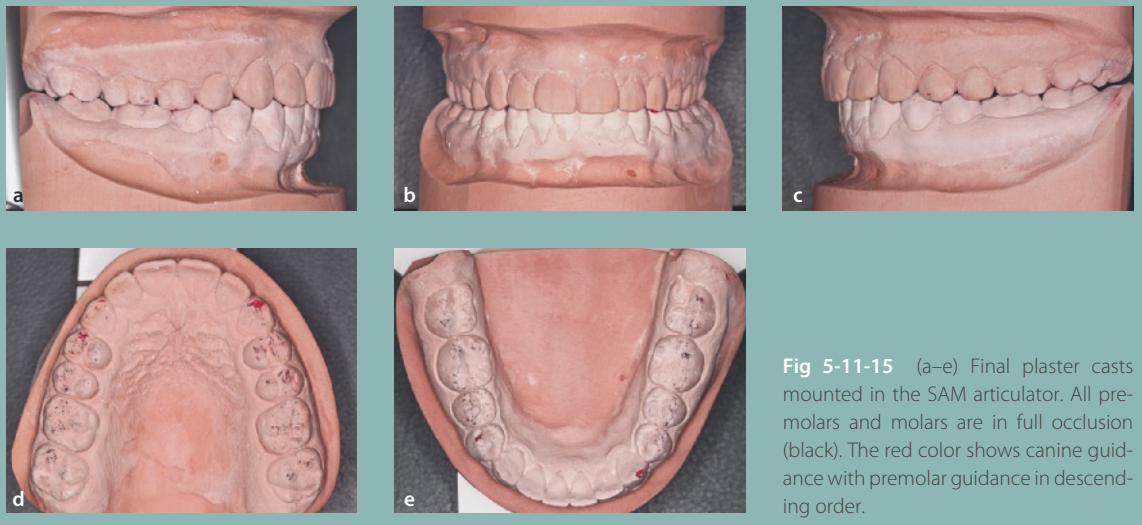

Fig 5-11-15 (a–e) Final plaster casts mounted in the SAM articulator. All premolars and molars are in full occlusion (black). The red color shows canine guidance with premolar guidance in descending order.

13 and 23, distalization, and a physiologic anterior relationship. The smile esthetics had been improved, with the maxillary dentition following harmonically the lower lip curve (Figs 5-11-12 to 5-11-14).

The final plaster casts mounted in the SAM articulator show the premolars and molars in full occlusion, with canine guidance and premolar guidance in descending order (Fig 5-11-15).

This book presents useful tips and strategies on how to integrate aligner orthodontics (AO) successfully into clinical practice, whether outsourced or with in-office aligner treatment. The authors review the diagnostic protocols and the biomechanics of aligners before presenting AO protocols. With the support of accompanying case documentation, discussion of each malocclusion includes information on the associated symptoms, the rationale behind the selected treatment approaches, and the various outcomes achieved. The separation into sections on each malocclusion helps patients and clinicians in deciding whether this system can provide optimal treatment outcomes for a particular clinical situation. This is a practical manual for any clinician interested in the treatment modality of AO.

Dr. Werner Schupp

Dr. Schupp graduated in dentistry in 1985 from the University of Münster and continued his studies here as a postgraduate student of orthodontics under the direction of Prof. Dr. U Ehmer. Collaboration in the orthodontic private practice of Dr. DE Toll followed and since 1990, he has been in private practice as an orthodontics specialist in Cologne, Germany. In his office, over 6000 patients have been treated with aligner orthodontics. He is certified in Manual Medicine and Osteopathy for Orthodontics. Dr. Schupp is a founding member and past president of the German Board of Orthodontics and Orofacial Orthopedics, and a board member of the German Society of Aligner Orthodontics (DGAO). He is also founder and editor-in-chief of the *Journal of Aligner Orthodontics (JAO)* and an advisory board member for the periodical *Manuelle Medizin*. He has published various articles concerning orthodontics, function, and pain therapy. He has written the books *Funktionslehre in der Kieferorthopädie* and *Kraniomandibuläres und Muskuloskelletales System*, as well as the first edition of *Aligner Orthodontics*, published in 2016 (Quintessence Publishing) and translated in several languages. Dr. Schupp is also visiting professor at the Capital University, Beijing, China.

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