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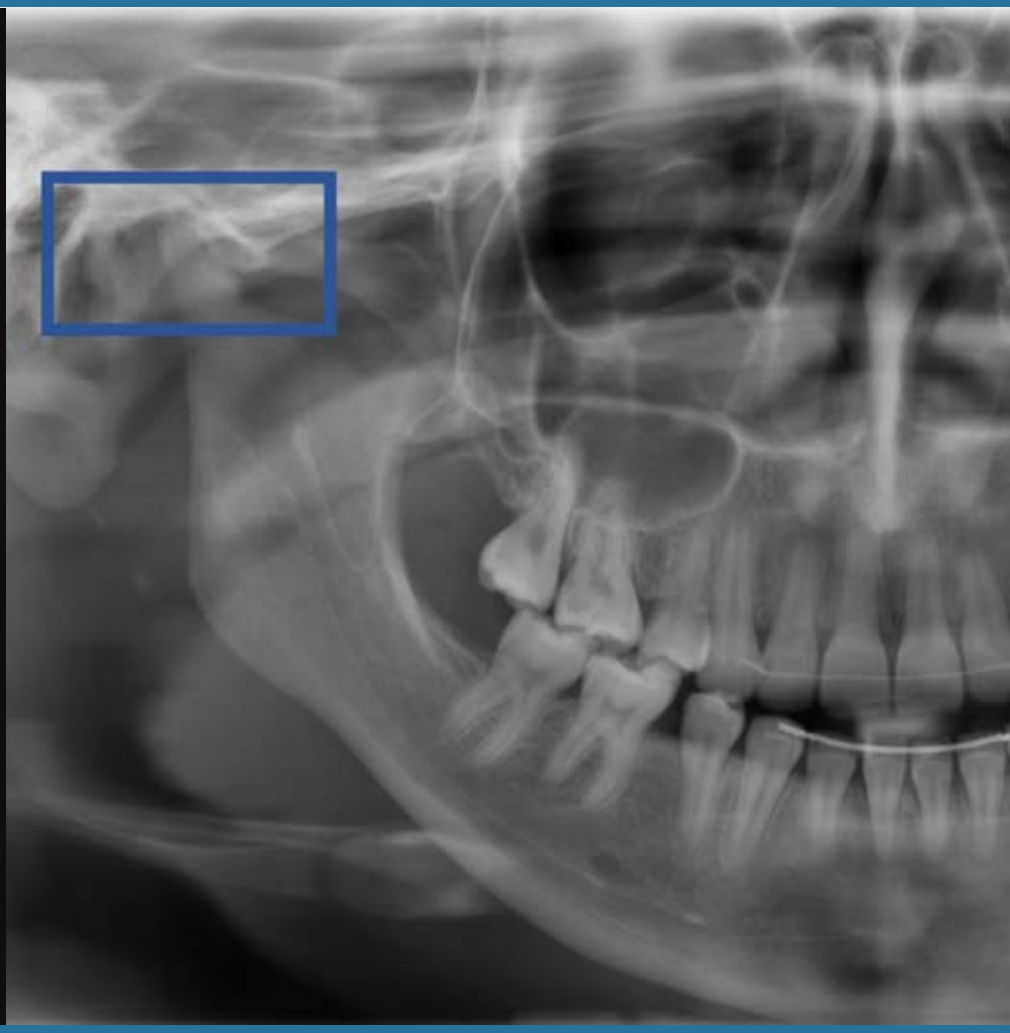

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PRACTICE

RESEARCH

Title picture: From the minireview of Michael Behr et al., here Figure 1: X-ray imaging of idiopathic condylar resorption. (Overall photo) Orthopantomogram of a patient with idiopathic condylar resorption. Blue frame: The X-ray on the right shows a section of the right mandibular condyle with anterior resorption of the condylar neck, p. 4–15; (Fig. 1: M. Behr)
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Idiopathic progressive condylar resorption

Question

What are the clinical signs and possible causes of idiopathic progressive condylar resorption?

Background

The term „idiopathic progressive condylar resorption“ has appeared in literature since the late 1990s [38]. It is a rare disease that involves the resorption of the mandibular condyle and it is accompanied by a progressive change of the bite position towards an Angle Class II [2, 3, 30, 46]. In the majority of cases, both condyles are affected [49]. The patient group consists mainly of young women subsequent to orthodontic or surgical treatment. The average age is 20.5 years [2].

Two forms of idiopathic condylar resorption can be distinguished:

- after the end of condylar growth in adulthood,
- during the growth phase in adolescents in which the growth rate of the condyle is restricted.

As seen on an X-ray, the mandibular condyle shows changes superiorly or anterior-superiorly (Fig. 1), but other condylar areas may also be affected. In the course of the disease, the shape of the condyle flattens; more pointed shapes are also found. On the other hand, the progressive destruction of the entire bone and an inflammatory infiltrate (articular effusion), which are typical of rheumatoid arthritis, as well as, typical repair processes with sclerosis, osteophytes (duckbill shape), or subcortical cysts, which are common in spondylitis and psoriatic arthritis are rarely found [49]. Wolford estimates the condylar height loss to be approximately 1.5 mm/year [46]. However, the resorption of the condyle at the level of the condylar neck seems to come to a standstill in the course of the disease. As the overall height of the ascending mandibular ramus is reduced by resorption, the occlusal surfaces of the distal molars occlude prematurely during jaw closure. This premature contact acts like a lever arm; it opens the bite anteriorly and increasingly shifts the mandible towards an Angle Class II, or distocclusion [49] (Fig. 2). The clinical symptoms described by the patients vary. In addition to pain in the immediate area of the affected joint, patients complain about discomfort in the masticatory muscles and joint sounds. Cracking sounds due to disc displacement are present in some cases, as are crepitation noises.

With respect to the differential diagnosis of idiopathic condylar resorption, it is distinguished from:

- bite openings due to parafunctions in the context of „bad habits“ such as thumb-sucking or malfunctions of the tongue,
- bite openings due to wearing splints with an anterior bite block (anterior bite splints),
- bite openings in the context of advanced periodontitis („flaring“),
- congenital syndromes with condylar hypoplasia, e.g. Goldenhar syndrome [9], Treacher-Collins syndrome [1], or acrofacial dysostosis [14],
- iatrogenic bite openings, e.g. temporary in the course of orthodontic therapy,
- juvenile arthritis – patients < 16 years of age with fever, skin rash, arthritis, and mostly negative rheumatoid factors [35],
- osteoarthritis and rheumatic diseases [42],
- osteoarthritis of the temporomandibular joints (no origin in rheumatic disease),
- rachitic open bite (vitamin D deficiency),
- tumor diseases.

The diagnosis is based on a diagnosis of exclusion (collaboration with rheumatologists, internists [endocrinologists], and other specialists), imaging, medical history, and collected clinical findings. MRIs or CT scans help to assess the extent of resorption in three dimensions. The estimation of the activity of the resorption process can be visualized using radionuclides (technetium methylene diphosphate, 99mTC-MCP) in single photon emission computed tomography (SPECT). This method requires a very strict indication in adolescent patients due to a radiation exposure of around 4–6 mSV [38].

Etiology

The etiology of idiopathic condylar resorption is to a large extent unclear [2, 49]. The spectrum of possible causes includes all possible disturbances in bone metabolism and it ranges from endocrine, hormonal or systemic diseases to mechanical stress factors which influence the tempo-

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romandibular joint. These various potential causes are discussed in more detail below.

Endocrine diseases

Endocrine diseases, nutritional deficiencies (anorexia nervosa), infectious diseases, cardiovascular diseases with vascular involvement, excessive physical stress as well as autoimmune reactions lead to various vascular damages and vasculitides. Such vascular changes also affect the blood supply to the condylar bone and can lead to inflammatory metabolic states in bone and cartilage.

The possible hormonal influences that are considered in relation to idiopathic condylar resorption are:

- corticosteroids,
- estrogens,
- parathyroid hormone (hyperparathyroidism),
- prolactin.

Hyperparathyroidism

Hyperparathyroidism, which is the excessive production and release of parathyroid hormone by the parathyroid glands, is considered to be a possible cause of idiopathic condylar resorption in literature [2]. However, it is unclear why this process would only affect the condyles of the temporomandibular joint in isolation and not other bone areas in the body. In the case of hyperparathyroidism, parathyroid hormone leads to an increased expression of the RANK ligand (RANKL, Receptor Activator of Nuclear Factor kappa B ligand) at the cell surface of osteoblasts and osteocytes or to its release as soluble RANKL. This RANK ligand binds to the RANK receptor of osteoclast precursor cells (preosteoclasts), which initiates their differentiation into mature, active osteoclasts that attach to the bone surface. So-called integrins in the cell membrane help to form a closed reaction space for the degradation of the inorganic and organic bone substance, the so-called Howship lacuna (Fig. 3). In this space, the pH is lowered by means of proton ATPases and the hydroxyapatite of the bone is dissolved [26]. The freed calcium and phosphate ions are released into the bloodstream. The release of antagonistic scavenger receptor, osteoprotegerin (OPG), by osteoblasts, among others, which binds to RANKL, can inhibit the binding of RANKL to the RANK receptor and thus regulate osteoclastogenesis (Fig. 3).

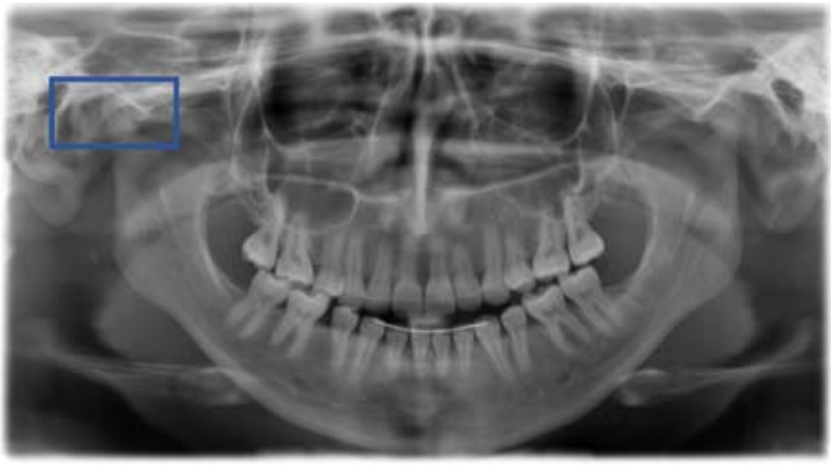


Figure 1 X-ray imaging of idiopathic condylar resorption. (Overall photo) Orthopantomogram of a patient with idiopathic condylar resorption. Blue frame: The X-ray on the right shows a section of the right mandibular condyle with anterior resorption of the condylar neck.

Corticosteroids

High corticosteroid levels demineralize bone. They stimulate the expression of RANKL (Receptor Activator of Nuclear Factor kappa B ligand) by osteoblasts (Fig. 3) and simultaneously inhibit osteoprotegerin release. As a result, the osteoblast population decreases and the number of osteoclasts increases. The effect of corticosteroids therefore occurs indirectly due to osteoblast inhibition [26].

Furthermore, corticosteroids regulate various metalloproteinases (collagenases), which cleave the collagen fibrils of the bone matrix and lead to a reduction of the bone matrix. The missing matrix can no longer be mineralized [11]. In osteoblasts, corticosteroids counteract the growth factor IGF-1 (insulin-like-growth-factor-I). IGF-1 stimulates the synthesis of type I collagen in the bone matrix as well as the mineralization of the matrix [11].

Apparently, corticosteroids which are applied directly into the joint can initially "repair" the cartilage microstructure to some extent. However, after 14 days of therapy with dexamethasone, for example, thinned collagen fibers with a very diffuse fiber structure are seen in animal experiments [7, 19]. These structural changes in collagen fibers are more extensive when dexamethasone is applied than in untreated osteoarthritis [19]. Possible damage to cartilage and chondrocytes depends on the dose and frequency of corticosteroid administration as well as on the type of corticosteroid [7].

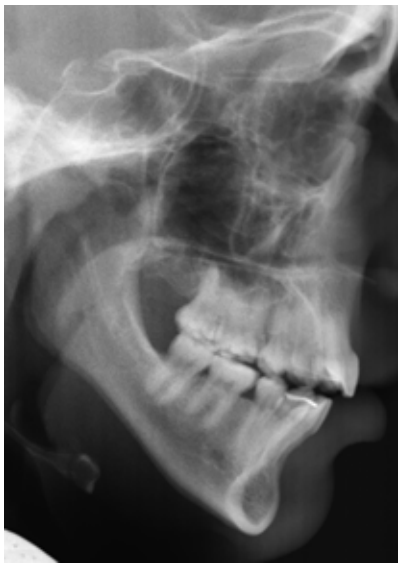


Figure 2 Lateral cephalometric X-ray of a patient with idiopathic condylar resorption. Although the molars are in occlusion, skeletal open bite is present in the anterior region.

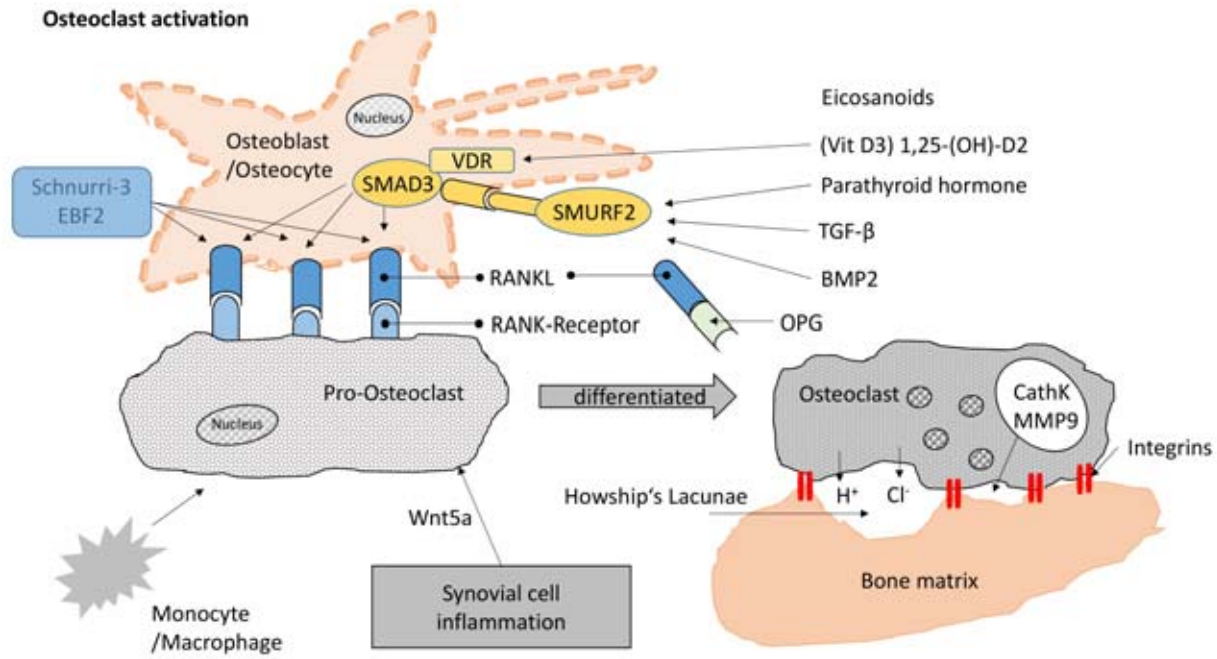


Figure 3 Schematic of osteoclast activation. RANKL = receptor activator of nuclear factor-κB-ligand; OPG = osteoprotegerin; CathK = cathepsin K; MMP 9 = matrix metallopeptidase 9; VDR = nuclear vitamin D receptor; BMP = bone morphogenetic protein; SMADs comprise a family of proteins that are signal transducers for TGF-ß signaling; Schnurri: key regulator of osteoblasts; SMURF = SMAD3 specific E3 ubiquitin protein ligase; EBF is a transcription factor of early B-cells of a protein coding gene

Prolactin

Prolactin is discussed as being another hormone that is responsible for the development of condylar resorption. [2]. The pituitary gland controls prolactin secretion. It is closely associated with estrogen metabolism. Prolactin stimulates the function of immune cells such as lymphocytes and macrophages, as well as, the release of cytokines, which in turn are involved in the breakdown of cartilage and bone.

Estrogens

Disturbances in estrogen metabolism can also play a role in the etiology of idiopathic condylar resorption. Based on studies performed on the great apes, it is known that female animals have more estrogen receptors in the region of the temporomandibular joint, whereas males have fewer [29]. This fits the observation made by Tsai [44]; in the synovial fluid of inflamed knee joints, estrogen and estrogen receptors are found more frequently than in healthy joints. Estrogens inhibit the synthesis of cartilage cells and, at the same time, promote the production of enzymes that degenerate the bone and cartilage matrix.

Systemic diseases

Included among the possible systemic diseases which can be associated with idiopathic condylar resorption are:

- psoriatic arthritis,
- rheumatism,
- spondylopathies,
- other autoimmune diseases.

Many of these diseases can be identified by specific markers in the blood. As illustrated above, the rheumatic disease forms mostly show other radiological manifestations; at present, it must therefore be assumed that idiopathic condylar resorption represents an independent clinical picture.

Mechanical causes

An important and possible chain of causation which can lead to the development of idiopathic condylar resorption are mechanical stress components such as:

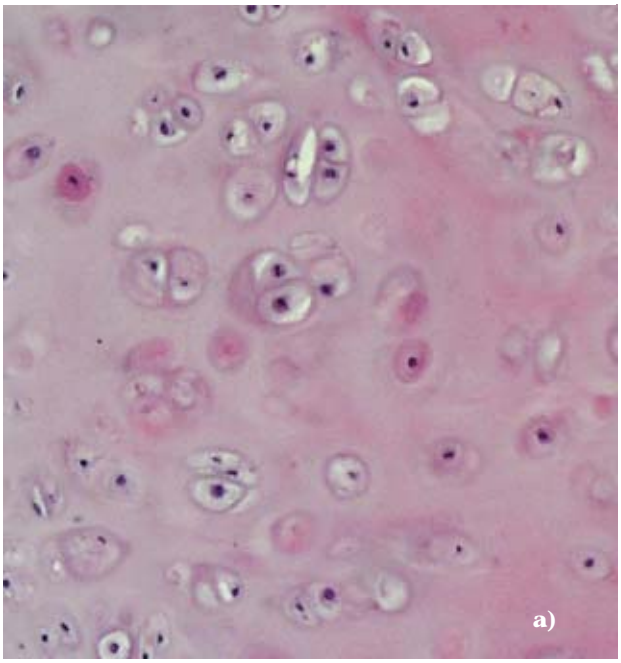
- a change in occlusion (orthodontics, prosthetics),
- parafunctions which damage the articular surfaces through mechanical load,
- traumas.

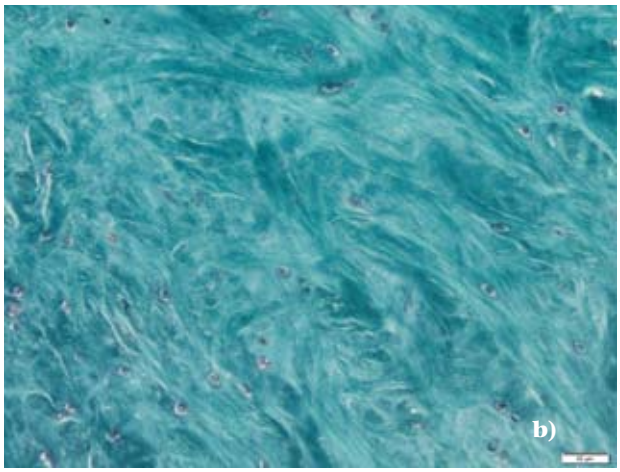
Mechanical stress is considered to be a possible cause of joint changes. The articular surfaces of physiologically functioning temporomandibular joints are largely load-free [6]. This is necessary in order to ensure that the ability of humans to speak through rapid, constantly changing movements of the lower jaw is retained. We know from animal experiments that a (normal) mechanical loading of the joint surfaces reduces or prevents the production and release of proteolytic enzymes in joints. Conversely, inactivity of a joint leads to the degradation of the articular cartilage [31]. Normal mechanical loading "protects" the joint surfaces.

Inflammatory processes in the temporomandibular joint can originate from 3 types of tissue: from cartilage, bone and cell structures of the synovial membrane.

Articular cartilage

The temporomandibular joint contains hyaline cartilage on the condy-





(Fig. 4a, b: from B. Miehe; Institute of Anatomy and Cell Biology, University of Greifswald)

Figure 4 Characteristics of cartilage cells of the temporomandibular joint: **a)** Cartilage cells with matrix. Hyaline cartilage in the cartilage layer of the mandibular fossa of the temporal bone. **b)** Fibrous cartilage of the articular disc. The collagen fibers are clearly visible.

lar process, in the mandibular fossa of the temporal bone and on the articular tubercle (Fig. 4a). The cartilage cells are scattered throughout the intercellular substance. The articular disc, on the other hand, consists of collagenous or fibrous cartilage (Fig. 4b). Unlike tissue structures, in which the cell walls lie close together, an extracellular matrix surrounds chondrocytes. In this way, each cell is separated from its neighboring cell. Both the cartilage capsule as well as the extracellular matrix are reinforced with fibrous structures. The magnitude of the load on the articulating surfaces determines the density and fiber reinforcement. A superficial fibrous layer with fibers arranged parallel to the articular surface, an intermediate layer having a fiber orientation in diverse spatial directions and a deep layer whose fibers run vertical to the articular surface and sprout into the mineralized subchondral zone of the bone can be distinguished [24]. Elastins, proteoglycans, hyaluronate and non-collagenous glycoproteins are the most important components of the extracellular matrix. Hyaluronic acid forms a scaffold to which proteoglycans with carbohydrate side chains attach to like plumage. Due to their hydrophilic character, the proteoglycans bind much water and, owing to their polyionic structure, sodium, potassium, calcium and magnesium as well. Under load, the hydrophilic (fluid-rich) structures distribute the forces in the cartilage evenly, much like a cushion. Collagen fibers are intercalated between the proteoglycans for stabilization. The so-called non-collagenous proteins of the extracellular matrix include fibronectins and laminins among others. Fibronectins have the function of connecting together collagen fibrils. Special heterodimeric transmembrane proteins such as the classical fibronectin receptor α5β1 bind fibrinonectin fragments (in the case of damages) and trigger growth factor gene expression via signal transduction in the chondrocyte. Laminins provide binding sites for collagen and glycosamines in the basement membrane region.

Cartilage cells develop differently depending on their future area of function. For instance, cartilage cells in the extremities progress through different developmental stages (Fig. 5). Initially, these cells form a cartilage matrix with type II collagen and proteoglycans. Afterwards, the cells mature into hypertrophic chondrocytes and express type X collagen. Finally, the hypertrophic chondrocytes differentiate. They secrete metalloproteinase-13 (MMP-13) so that the cartilage matrix is degraded and increasingly replaced by bone substance. In this way, piece by piece, the cartilage is transformed into bone. In contrast to the chondrocyte forms described above, the chondrocytes of articular cartilage exhibit a low turnover rate. They do not participate in the maturation process described above up to the point of ossification. Rather, they acquire a flat spindle-shaped form and store collagen types I, IX and X in the matrix (Fig. 5). Due to a high proportion of proteoglycans, the extracellular matrix is hydrated. Thereby, the cartilage cells function like a cushion under load and they facilitate frictionless sliding of the joint surfaces [48].

In the case of osteoarthritis, the special cells of articular cartilage undergo the stages of further maturation irregularly and, consequently, of later extracellular matrix degeneration. Processes similar to those of enchondral ossification take place afterwards (Fig. 5). This results in a loss of articular cartilage and the formation of osteophytes.

Cartilage responds to physiological mechanical load by increasing metabolism through mechanotransduction. Mechanosensitive ion channels and receptors such as integrins, among others, are involved in registering the changes. Under tensile

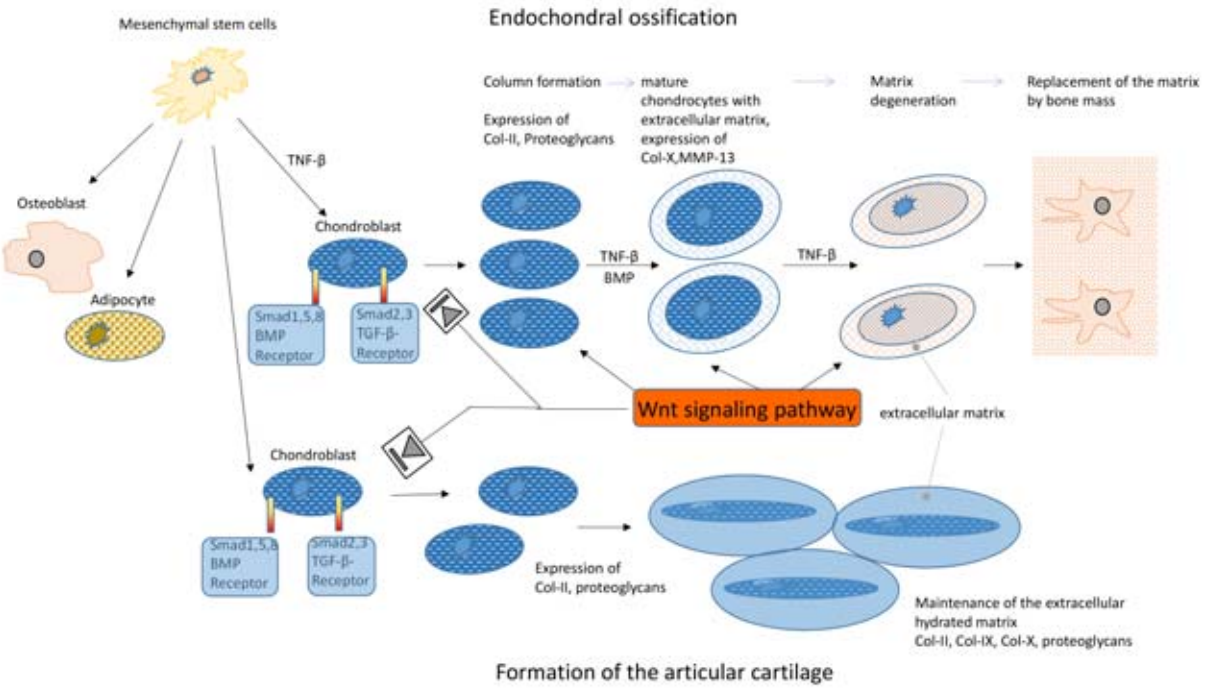


Figure 5 Differentiation of chondroblasts into chondrocytes. Development of chondrocytes during endochondral ossification and during specialization into articulating articular cartilage. Wnt (Wingless, Int-1), TNF-β (tumor necrosis factor β); Smad (intracellular proteins that relay extracellular TGF-β family signals from TGF receptors to the nucleus), MMP-13 (matrix metallopeptidase-13), BMP (bone morphogenetic protein), Col-II, IX, X (collagen type II, IX, X).

load, integrins and mechanosensitive ion channels promote the release of interleukin 4 and 10 (IL-4, IL-10) (Fig. 6). These interleukins increase the production of aggrecan (aggrecan binds H_2O and gives cartilage its „shock-absorbing function“) and simultaneously reduce the expression of matrix metalloproteinase-3 (MMP-3, collagen-dissolving) as well as the transcription of NF-κB (nuclear factor „kappa-light-chain-enhancer“ of activated B-cells). Furthermore, IL-4 and IL-10 inhibit the expression of interleukin 1 (IL-1) and tumor necrosis factor α (TNF-α) [31] (Fig. 6). A joint which performs physiological movements is thus „metabolically protected“. Diseased joints also benefit from this. In osteoarthritic joints, regular exercise increases the expression of IL-10 and promotes „healing“ of the joint surfaces [4,31].

Trauma and non-physiological stress on articular cartilage surfaces are manifested, among other things, by the appearance of fragments of, for example, collagen or fibronectin. With the help of phagocytic cells, the so-called A cells of the synovial membrane (s. below), the joint first attempts to eliminate these fragments. In the course of tissue damage, such fragments also bind to integrins and so-called Toll-like receptors (TLR) [23]. Integrins such as the classical fibronectin receptor α5β1 only bind to a single protein in the extracellular matrix. They cross-link proteins in the extracellular matrix on the extracellular side of the cell membrane with cytoskeletal proteins and actin filaments on the cytoplasmic side and mediate signals which are involved in regulating cell growth, cell differentiation or even apoptosis. The Toll-like Receptor (TLR) is part of the innate defense system and it recognizes structures that are found only on pathogens/foreign bodies, so-called PAMPs (Pathogen-Associated Molecular Patterns). The TLR controls the activation of the antigen-specific acquired immune system. In this way, NF-κB mediates the release of pro-inflammatory cytokines and chemokines such as IL-1, IL-6, IL-8, TNF-α, and ADAMTS (A Disintegrin and Metalloproteinase with Thrombospondin motifs) [21] (Fig. 7). This release leads to the breakdown of the extracellular matrix in the course of the disease. Released proteoglycans such as decorin, components of collagen fibers such as fibromodulin or other components of the extracellular matrix in turn activate further integrins and TLR, which then stimulate the release of IL-1 and TNF-α and trigger nitric oxide and prostaglandin E2. Another NF-κB activation pathway which results in the release of IL-6, IL-8, TNF-α, MMP-3, and ADAMTS also occurs via receptors for glycation end-products which are termed RAGE or AGE (Receptor for Advanced Glycation End-Products, Advanced Glycation End-Products) [25, 43]. However, this pathway is mainly associated with (natural) aging processes and is therefore not likely to be considered in young patients with idiopathic condylar resorption.

Cells of the synovial membrane

In addition to the chondrocytes of the articular cartilage and the subchondral bone cells, there are cells of the synovial membrane. In the case

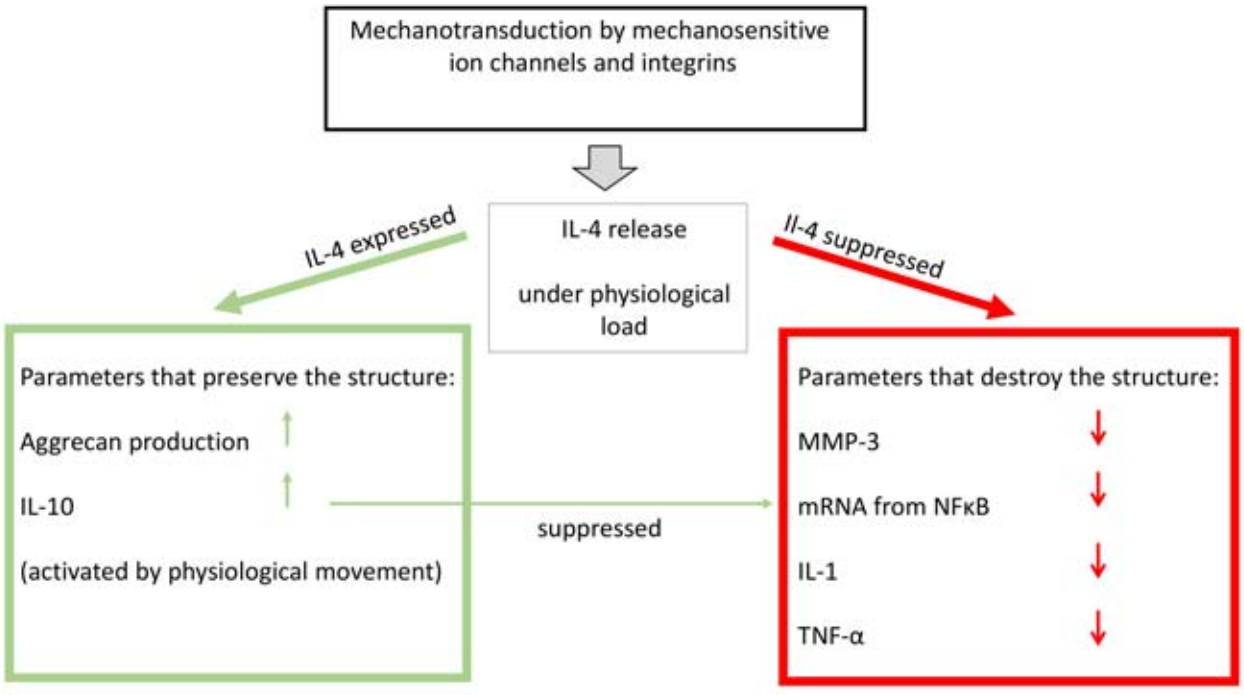


Figure 6 Processes that preserve and destroy the structure in articular cartilage under load application. IL-10 (interleukin-10), MMP-3 (matrix metallopeptidase-3), mRNA of NFκB (messenger ribonucleic acid of nuclear factor kappa B), TNF-α (tumor necrosis factor α).

of rheumatoid arthritis, it is considered with certainty that the origin of the autoimmune disease lies in the synovial membrane. The inflammatory changes then spread to the surrounding joint structures.

The inner joint space is bounded, on the one hand, by hyaline articular cartilage, which covers the mandibular condyle, the mandibular fossa as well as the articular tubercle and, on the other hand, by the synovial membrane of the joint capsule. The joint capsule consists of the fibrous membrane and the synovial membrane. The synovial membrane ends immediately at the cartilage edges. A synovial intimal layer, which is oriented toward the inner joint space, and a subintimal (subsynovial) layer can be distinguished [16]. The top layer consists mostly of 3 cell strata, which can form villi (Villi synoviales) and folds (Plicae synoviales). There exist 2 types of cells [18]: Type A is a round cell which is said to have phagocytic properties. This cell type is loosely embedded on the surface and it is found between larger fold and villus-forming B cells. The B cells are partially located in several cell layers behind one another and are in contact with blood vessels. The job of the B cells is to produce and, if necessary, modify the synovial fluid in the presence of pathological influences. The B cells are assigned the role of producing hyaluronic acids, proteins, as well as collagen and fibronectin [37, 40], whereas the A cells remove cellular debris and foreign molecules from the joint space. Some authors also describe a so-called intermediate type [17]. This is based on the idea that cell types A and B can merge together. At present, it is assumed that around three quarters of synovial cells are B cells [33].

Synovial fluid is a blood-plasma dialysate. It differs from blood serum mainly through its composition of proteins and hyaluronic acids. Hyaluronic acid is a dimeric molecule of D-glucuronic acid and N-acetyl-D-glucosamine. The functions of hyaluronic acid are, on the one hand, to ensure the sliding properties of the joint surfaces and to nourish the cartilage components; on the other hand, it is to regulate oxidative cell damage, to suppress the release of proteoglycans (inflammatory response) from cartilage as well as to participate in the chemotactic, proliferative, and phagocytic response of the joint to inflammatory changes [34]. The reported content of hyaluronic acid in synovial fluid is between 0.35 to 7.6 mg/ml [15]. On average, it is assumed to be 2–4 mg/ml [22].

In addition to hyaluronic acid, important components of the synovial fluid are the proteins albumin and γ-globulin. Both proteins are increasingly formed in the case of inflammatory joint diseases. Yet, only about 2 % of hyaluronic acid binds to proteins. Thus, its binding to proteins is not responsible for the rheological properties. Other proteins in synovial fluid are fibrinogen, immunoglobulin IgM, metalloproteinase inhibitor, α2-macroglobulin, and lubricin (glycoprotein). In addition to proteins, cytokines, collagens, enzymes, proteoglycans, fibronectin, uric acid, glucose, Na^+-, Cl^--ions, lipids, and cellular components such

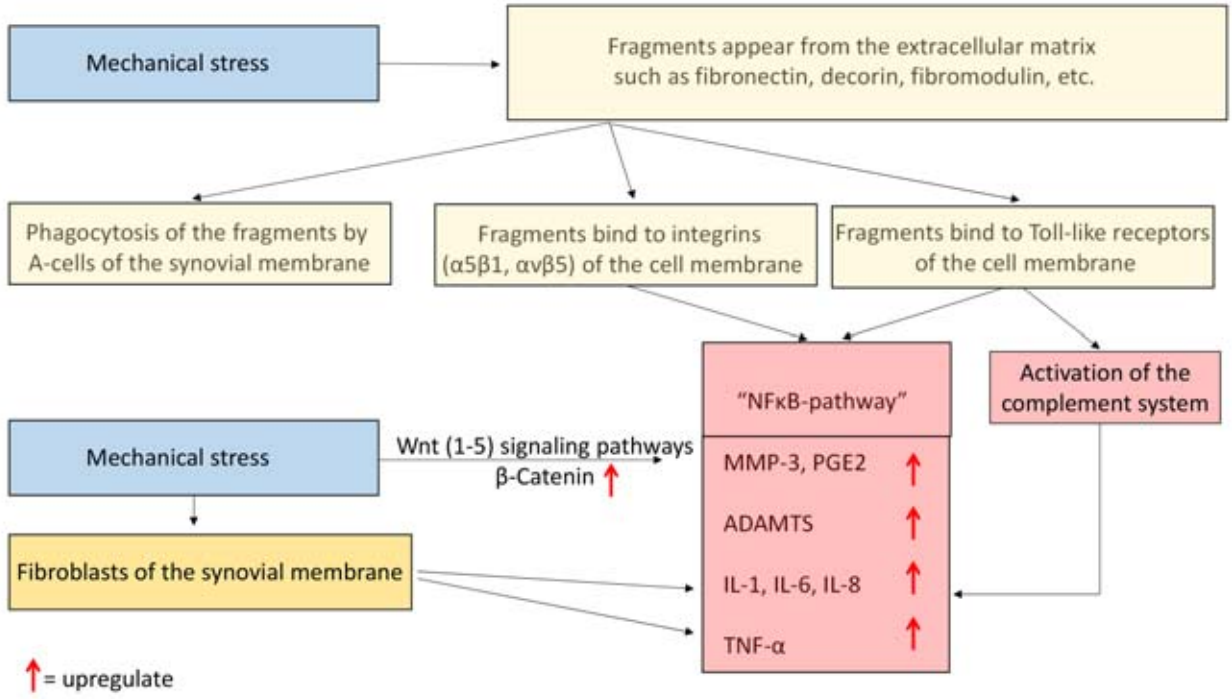


Figure 7 Processes and stages of cartilage matrix degradation under nonphysiological load. MMP-3 (matrix metallopeptidase-3), PGE2 (prostaglandin E2), ADAMTS (A disintegrin and metalloprotease with thrombospondin-1-like domains), IL-1, IL-6, IL-8 (interleukin-1, 6, 8), TNF-α (tumor necrosis factor α).

as lymphocytes, monocytes, phagocytes, and leukocytes can be detected [15].

In the case of mechanical stress, Schröder et al. [39] considered the fibroblasts of the synovial membrane, which constitute the main part of the cellular structures in the synovial membrane, as the cause of inflammatory changes. Synovial fibroblasts from healthy subjects were found to express increased levels of pro-inflammatory cytokines such as TNF-α, prostaglandin E2, and IL-6 under mechanical load. TNF-α blocks proteoglycan synthesis and it is one of the most important modulators of inflammation in chondrocytes, osteoblasts, and synovial fibroblasts. Interestingly, synovial fibroblasts obtained from osteoarthritic joints did not further upregulate TNF-α, prostaglandin E2, and IL-6 under mechanical load. This fact can be interpreted as follows: the upregulation of the abovementioned cytokines seems to play a special role only in the initial period of osteoarthritis, and in the course of the disease, other mediators maintain the inflammatory process [39]. In the case of inflammation, synovial cells also upregulate the release of Wnt5 and activate the so-called non-canonical Wnt signaling pathway (Fig. 3). The Wnt signaling pathway is discussed in more detail under the heading „bone“ (see below). This process ultimately activates osteoclasts and drives the destruction of the articular surfaces. The extent to which pathological processes in the synovial membrane are to be regarded as the starting point of idiopathic condylar resorption is currently unclear. The radiological findings – rarely joint effusion – tend to speak against it.

Bone

Disturbances in bone metabolism may be considered in the development of idiopathic condylar resorption, as bone is another structure of the joint. The bone of the mandibular condyle develops from mesenchyme through desmal ossification. Unlike other cranial bones, such as the temporal bone, there are no cartilaginous base structures in the mandibular bone. Articular cartilage also forms independently secondary to the mesenchyme (as does the articular disc) and attaches to the bony portion of the mandibular condyle [5, 28].

The so-called progenitor cells actively proliferate in bone tissue. These are spindle-shaped cells which are located in the area of the periosteum, endosteum and in the walls of the later Haversian canals. Under functional, as well as, under nonphysiological stresses of the bone, the progenitor cells differentiate into osteoblasts, which are in contact with osteocytes via their cytoplasmic processes. Osteoblasts possess numerous receptors for hormones, cytokines and other signaling substances.

Osteoblasts can develop in different ways. Some of them remain on the bone surface and they slow down their synthetic cell activities to become bone lining cells. The other

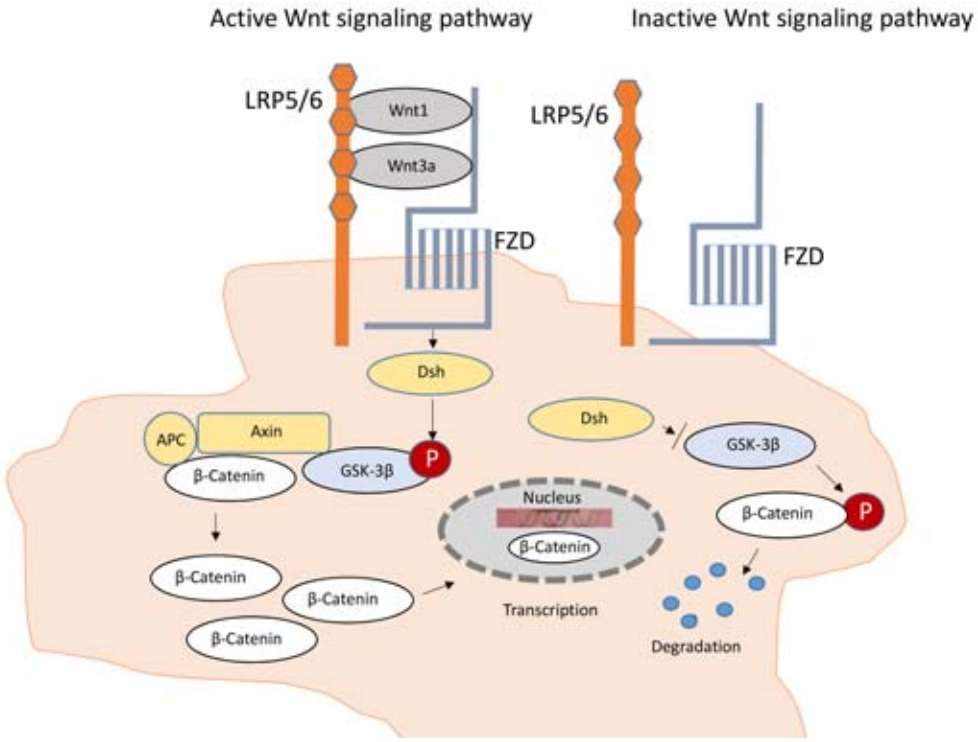


Figure 8 Active and inactive Wnt signaling pathways stimulating osteoblasts and osteocytes. Wnt1–5 (Wingless, Int-1), GSK-3β (Glycogen synthase-kinase 3), LRP4/5/6 (Low-density lipoprotein receptor-related protein 4/5/6), FDZ (Frizzled-receptor), CAMKII (Ca2+/Calmodulin-dependent protein kinase II), DKK1 (Dickkopf Gen 1), ROR1/2 (receptor tyrosine kinase-like orphan receptor), RhoA (Ras Homologue Family Member A), Rac (Rho family of GTPases), JNK (c-Jun N-terminal kinases), PKC (Proteinkinase C), Wnt/PCP (Wnt/planar cell polarity pathway), P (Phosphorylation).

part of the osteoblast population surrounds itself with osteoid. This basic organic substance of bone consists of type I collagen, proteoglycans, osteocalcin, osteonectin and osteopontin as well as various growth factors. The osteoid layer is increasingly mineralized. Ultimately, the cell cytoplasm is integrated into a bone lacuna. However, numerous projections emanate from there. These projections have contact with other osteocytes and with the bone canals. Via the projections, the „enclosed" osteocytes always exchange signals with the osteoblasts and with the surrounding bone lining cells [24].

In recent years, the molecular processes underlying bone homeostasis and bone diseases have been further elucidated. The various forms of the Wnt/β-catenin signaling pathway play a special role in head morphogenesis, in the homeostasis of bone metabolism and also in the case of pathological processes or mechanical load on the bone [10, 32] (Fig. 8). According to Wu et al. [48], any disturbance that affects the physiological function of the Wnt/β-catenin signaling pathway leads to dysregulations of osteoblast and chondrocyte function with changes in bone mass and degenerative joint disease.

At the level of mesenchymal cells, the Wnt signaling pathway inhibits the differentiation of these cells into chondrocytes or fat cells and promotes osteogenesis. The Wnt pathway further stimulates osteogenesis by expressing the Runx2 gene and by stimulating osteoblast differentiation. It prevents apoptosis of osteoblasts and suppresses osteoclast differentiation [20].

In the Wnt signaling pathway, we distinguish a canonical Wnt signaling pathway and a non-canonical pathway (Fig. 9). The non-canonical Wnt pathway is activated via Wnt5a and induces osteoclast differentiation. In particular, synovial cells release a lot of Wnt5a during inflammatory processes. Through this process, the gene ROR2 (receptor tyrosine kinase-like orphan receptor) is expressed, which converts osteoclast progenitor cells into active osteoclasts and activates the RANK receptors of the osteoclasts. In contrast, Wnt4 and Wnt16 block the expression of RANK.

When the canonical Wnt signaling pathway is stimulated, the concentration of β-catenin in the cell increases and this increases the transcription of genes in the nucleus [27]. In these cases, Wnt1 and Wnt3a first bind to the receptors „Frizzled" and LRP5/6 (low-density lipoprotein receptor-related protein) on the outside of the cell membrane. As a result, GSK-3β (glycogen synthase kinase 3) is phosphorylated and inactivated inside the cell. GSK-3β can no longer inactivate β-catenin in turn, so that a high availability of β-catenin in the cell increases transcription (Fig. 9). According to current knowledge, the receptor LRP5 is considered to have more tasks in the maintenance of bone mass in adults, while the receptor LRP6 is considered to have tasks mainly in embryonic bone devel-

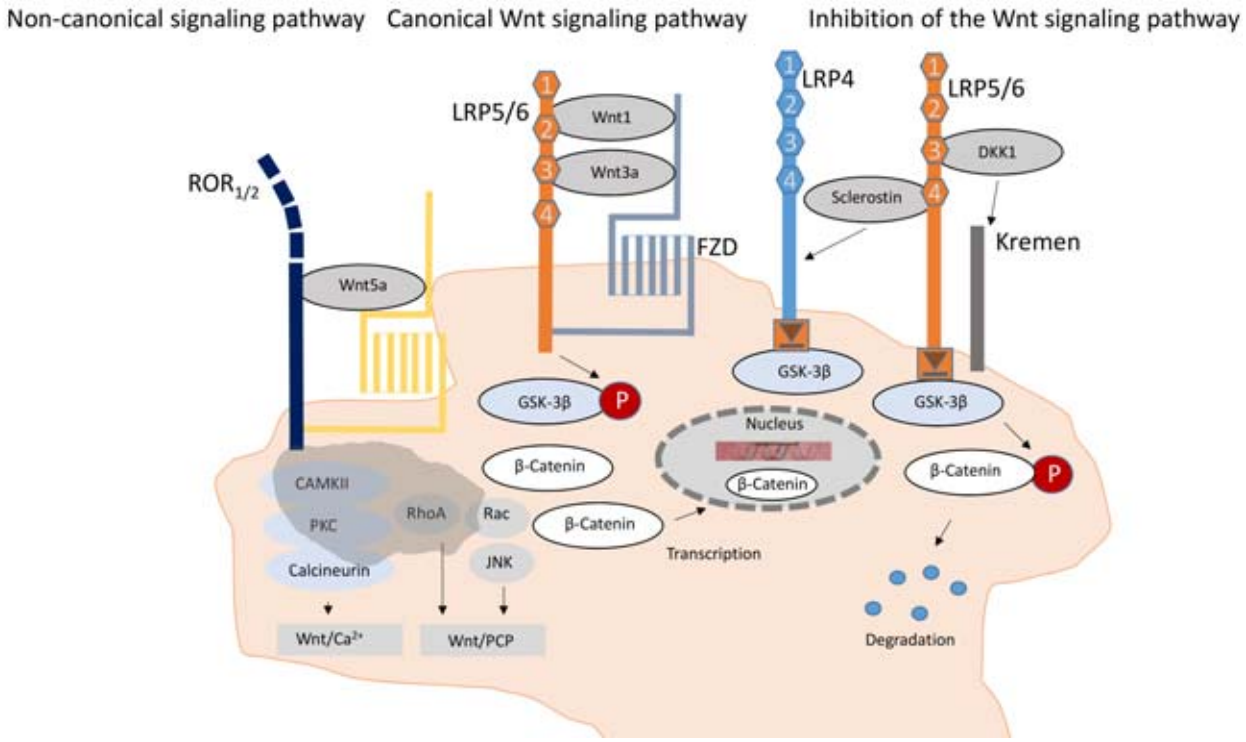


Figure 9 Currently known Wnt signaling pathways involved in the regulation of bone metabolism. Wnt1–5 (Wingless, Int-1), GSK-3β (Glycogen synthase-kinase 3), LRP4/5/6 (Low-density-lipoprotein-receptor-related protein 4/5/6), FDZ (Frizzled receptor), Dsh (Dishevelled), APC (Adenomatous-polyposis-coli-Protein).

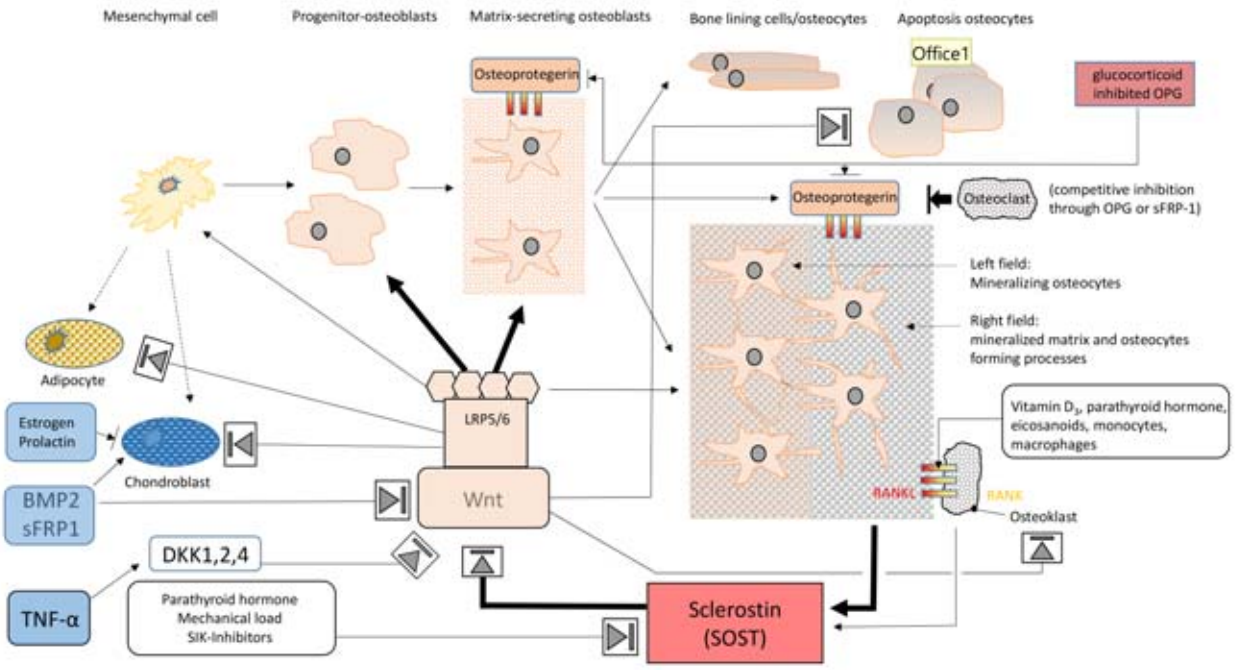
opment, with both receptors having overlapping functions [20]. Activation of the canonical Wnt pathway stimulates osteoblast maturation and bone mass formation (Fig. 10).

The canonical Wnt signaling pathway appears to play a central role in osteoblast differentiation. Inhibitors of the Wnt signaling pathway are genes of the so-called Dickkopf family such as DKK1 in combination with the gene Kremen. DKK1 binds to the BP1 and BP3 domains of the Wnt receptor LRP6 (lipoprotein-related protein 6) (Fig. 8). LRP6 and the Wnt receptor „Frizzled" are normally involved in signal transduction (ß-catenin) in the osteocyte. The Dickkopf protein and Kremen bind to LRP6 to block signal transduction for transcription in the cell. The Dickkopf gene family is considered essential in the development of the head and limbs in mammals [32]. In the case of patients with overexpression of DKK1, lytic bone lesions occur. DKK1 reduces the formation of bone mass and prevents the formation of osteophytes. In joints that are altered by inflammation, we find the expression of DKK1 especially in the synovial cells and in adjacent chondrocytes [13]. In rheumatoid arthritis, the serum concentration of 31.5 ± 2 pg/ml is twice as high as in healthy subjects, whereas the serum levels in patients with spondylitis are comparable to healthy subjects. DKK1 is regulated by TNF-α. In this way, the administration of TNF-α inhibitors lowers the serum level of DKK1 to a physiological level [13].

Unlike osteoblasts, osteoclasts are derived from the monocyte and macrophage cell lineages. The differentiation of an osteoclast precursor cell into an osteoclast is caused by the expression of RANKL and cytokines by osteoblasts and osteocytes. The binding of RANK ligand (RANKL) to the RANK receptor of osteoclast progenitor cells causes the osteoclast to mature and activate (Fig. 3). Osteoblasts and osteocytes counteract this process by secreting osteoprotegerin (OPG). The canonical Wnt signaling pathway enhances the expression of osteoprotegerin and counteracts osteoclast differentiation (Fig. 10) [27]. This increases bone mass. Excessive bone formation inhibits the gene SOST via sclerostin („bone formation inhibitor"). The normally high level of sclerostin release by osteocytes indicates that the Wnt/β-catenin system is usually switched to „off" (Fig. 10). The interplay of Wnt signaling and SOST regulate bone homeostasis. Mechanical load, IL-6, and parathyroid hormone suppress SOST expression in osteocytes and activate the Wnt signaling pathway. SIK (Salt-Inducible Kinase) inhibitors also hinder sclerostin release and the activation of RANKL so that osteoclast function and activation stops.

Statement: hypothesis on the etiology of idiopathic condylar resorption

Many of the regulatory pathways and mechanisms of bone metabolism that have been described above are only partially known and understood. Regarding the origin of idiopathic condylar resorption, defini-



(Fig. 1–3, 5–10: M. Behr)

Figure 10 Overview of regulatory mechanisms of bone metabolism under physiological and pathological conditions. Wnt1–5 (Wingless, Int-1), GSK-3β (Glycogen synthase-kinase 3), LRP4/5/6 (Low-density-lipoprotein-receptor-related protein 4/5/6), FDZ (Frizzled receptor), SOST (Sclerostin-gene),BMP2 (Bone morphogenetic protein 2), OPG (Osteoprotegerin), sFRP1 (Secreted Frizzled Related Protein 1), DKK1 (Dickkopf-Gen 1), RANKL (receptor activator of nuclear-factor-kappa B-ligand.

tively attributing it to a disturbance in a regulatory pathway is not possible at present. However, the following hypothesis should be formulated:

Imaging reveals that resorption mostly comprises of the anterior areas of the mandibular condyle. As already shown by Steinhardt [41], we normally find functional adaptations of bone and cartilage in the temporomandibular joint in the area of the articular tubercle as well as in the anterior area of the mandibular condyle; for example, in cases of an Angle Class II/2, bone and cartilage are reinforced. In deep bite, for example, before the mandibular body can be pushed forward, the condylar path along the tubercle is long until the dental arches are disengaged. As the disengagement of the anterior teeth progresses due to the rotational movement of the mandible, there is already a steady force vector in an anterior direction, which presses the condyle against the tubercle. In response to this functional stimulus, bone and cartilage physiologically respond through apositional growth. Figure 10 illustrates how mechanical load normally downregulates sclerostin and activates the Wnt signaling pathway so that bone mass can be formed. The „off“ position of the Wnt pathway is lifted by mechanical load and bone growth is activated. In idiopathic condylar resorption, the balance between the anabolic function of the Wnt pathway and catabolic function of the SOST/sclerostin gene in bone might be disturbed. The localized resorptions (in contrast to multifocal responses in systemic disease) suggest the presence of localized (over)load application and a simultaneous defect in the SOST gene/Wnt pathway regulatory loop. Instead of growing additional bone under load, the regulatory mechanisms that break down bone are activated locally under load. For example, the disturbance could be due to the SOST gene or the LRP5/6 as well as the receptor „Frizzled“.

Conversely, if the SOST gene and sclerostin expression are suppressed, changes like in osteoarthritis are seen in cartilage due to activation of the Wnt signaling pathway [12, 47]. Already Blechschmidt [8] and later Radlanski [36] pointed out that mechanical stimuli also lead to changes in the genome response (mechanical gene effects) and can obviously play a role in head morphogenesis and teratogenesis. In parallel to the disruption of the Wnt signaling pathway/SOST regulatory loop, fluctuations in estrogen and prolactin levels can contribute to damage of articulating surfaces. It is also known that especially chondrocytes can be very heterogeneous in shape, size and matrix [45], so that, in the case of idiopathic condylar resorption, less resilient chondrocyte types are perhaps present. Overall, the causes of idiopathic condylar resorption are likely to be a combination of local mechanical overload and disturbances in synovial membrane, cartilage, and/or bone metabolic regulatory loops.

Therapy

Due to the unknown etiology, no causal therapeutic concepts are avail-

able at present. Current therapy initially aims to achieve symptomatic pain relief by supporting the bite position with bite blocks. In acute pain conditions, additional naproxen with gastric protection (e.g. Vimovo 500/20 mg, 1–0–1, cave hypertension, pregnancy) have proven effective. An important therapeutic guideline is the above described fact that, through physiological joint movements, metabolic processes, which are protective for the joint structure, can be initiated or maintained (Fig. 6) [31]. Thus, the combination of adjusted splints (facebow, centric registration is a must!) to correct the bite position with intensive physiotherapeutic exercise is an important component for re-establishing the physiological movement patterns of the temporomandibular joint in order to promote the healing process. In this context, the instruction of daily exercises which are to be performed by patients themselves is essential for success [4].

Since resorption appears to stop over time (> 5 years), further interventions which aim to correct the bite position are first indicated once there is freedom from symptoms for a period of more than 6 months and no radiological changes in resorption are detectable. Surgical, orthodontic and prosthetic measures may then have to be performed a second time [3, 49].

Conflicts of interest

The authors declare that there is no conflict of interest as defined by the guidelines of the International Committee of Medical Journal Editors.

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(Photo: UKR)

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Jens C. Türp

The "problematic patient": what is the problem?

Illustrated by the example of temporomandibular disorders

Problem: When patients report pronounced physical complaints without sufficient somatic findings to substantiate them, practitioners sometimes refer to these patients as being "problem patients" or "difficult patients". When such an attribution is assigned, it usually denotes a difficult interpersonal relationship between practitioners and patients, which can be further exacerbated by deficits related to professional expertise, communication and dental fee schedules.

Discussion/Conclusion: On the basis of examples of persistent temporomandibular disorders/orofacial pain, it is recommended that professionally practicing dentists should live up to their responsibility and trust given to them by patients. For that purpose, dentists must be aware of their limits of competence and be cautious about overestimating their abilities. There are not only "difficult patients"; there are also "difficult dentists".

Keywords: health care provider's role; communication; dentist-patient relationship; pattern recognition; interpretation; clinical decision-making; craniomandibular disorders

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Halden picked up the phone ...
»Yes – – the countess shall then – –, how, – but that can be done without me, sister ...
Good, I'm coming ...«
And to us: »You must excuse me for a few minutes ... It's a somewhat difficult patient ... «

Max Schraut (pseudonym from Walther August Gottfried Kabel [1878–1935]): Harald Harst. Aus meinem Leben [From my life]. Volume 196 of the novel collection "Harald Harst": Doktor Haldens Patient [Doctor Halden's Patient]. Verlag Moderner Lektüre, Berlin 1925.

1. Introduction

According to Donner-Banzhoff [9], four fundamental, stratified medical functions can be distinguished in the relationship between the physician and patient:

- The physician as a healer: the patriarchal-acting expert with exclusive knowledge.
- The physician as a detective: the investigator for seemingly minor findings.
- The physician as a gatekeeper: the identifier of an indication for medically effective measures and justified claims in a solidarity-based health care system.
- The physician as a transparent, patient-oriented therapist: a partner in an equal relationship.

Considering the tasks involved and mutual expectations, it is understandable that not every patient encounter is free of problems. Hoefert [25] remarks: "The 'lucky' case for patients and physicians is always the one in which a certain (organic) cause for a disease is found and the corresponding therapy options are available." This prerequisite is not always a given, however. Particularly in the context of encounters where there is an obvious discrepancy between the disturbed subjective well-being of the patient and no conspicuous clinical and radiological findings, there is a high probability that the buzzword "problem patient" will be used by the dentist/physician. Due to its fuzzy definition and broad meaning, this term can cover a considerable number of people. Since an attribution with the label "problem patient" has a negative connotation, when possible, the term "difficult patient" [34, 40–41, 50] might be the better choice.

Dunkelberg et al. [10] state that patients who are experienced as being difficult by the practitioner are "evidently a problem of considerable extent". Physicians consider that 15 to 18 from 100 patients are "difficult" [27]. Comparable data is not available for dentistry; nevertheless, dental practices are familiar with such patients: based on the results of a nationwide survey in Austria (n = 145), Kreyer [36] reported that, in addition to occupational stress (constant time, scheduling, performance and quality pressure), confrontation with "problem patients" is a particular burden for dental practitioners, and that there is a genuine "fear on the part of dentists of their difficult patients". In any case, the dentists and dental staff involved "remember" [8] these patients for a long time usually. Table 1 summarizes frequent third-party descriptions for such persons.

Patients who are described as being "difficult" are extremely heterogeneous in terms of their complaints, behavior and background. For example, the management of children, anxiety patients and disabled people is often perceived as "difficult" by practitioners [36]. However, when speaking of "difficult patients" in the narrower sense, other persons are generally meant. Hoefert and Härter define "difficult" patient behavior "as a perceived deviation from the image of the 'desirable', or at least, 'normal' patient." As a rule, this involves dealing with insufficiently clarified, or inexplicable physical complaints and symptom evolution, coupled with patients' own behavior-related ("smart aleck" [36]) and other psychosocial peculiarities.

When evaluating this phenomenon, it should be clarified where the fundamental difficulty – or "the problem" – lies, and if, this is not something that needs to be searched for by the patients themselves.

2. The difficult patient?

The characterization of a patient as being "difficult" or "problematic" is an attribution that is assigned on the part of the practitioner [55]. Various authors [10, 40] indicate that such a perception is a relationship and communication problem, or in other

Adjective
pretentious
straining
smart-alecky
disappointed
sensitive
demanding
offended
stubborn
challenging
litigious
troublesome
narcissistic
annoying
nagging
refractory
blathering
recalcitrant
unfair
uncooperative
unsatisfied
reluctant to pay
time-consuming

Table 1 Some (translated) adjective descriptions from the German-language specialist literature (including [28]) for patients who are qualified as "difficult" by dentists/physicians.

Patient's side
Description of diverse, vague, unclear, partially variable somatic complaints
Excessive preoccupation with the (sometimes minor) symptoms
Lengthy medical history
High degree of concern
Increased psychosocial stress, presence of social pressure or conspicuous biographical events (e.g. separation conflicts, family members in need of care)
High prevalence of mental disorders or psychiatric diagnoses
Elaborate explanations
Strong, but unfavorable causal beliefs
Exact knowledge what is missing, what the cause is and how best to proceed, sometimes written down meticulously on a piece of paper, envelope or the like (*la maladie du petit papier* [5, 49])
Increased use of health care services (heavy user)
Attention-seeking, clinging, manipulative, demanding behavior towards the dentist/physician
Pre-informed (often misinformed) via the Internet
Unrealistic expectations regarding the health care providers and the therapy
Uncooperative behavior, lack of trust in the therapy (unwillingness to be treated), resistance to medical/dental recommendations
Frequent switching of dentists/physicians ("doctor shopping", "doctor hopping", "hospital hopping") [7]
Poor or no response to common therapeutic methods
Dissatisfaction
Ongoing procedures with other practitioners
Physician's side
Considerable time requirement (until shortage of time)
Difficult communication with the patient
Exclusive focus on somatic medical aspects
Dismissing, dominant communication behavior
Strong emphasis on visualization procedures
No consideration of psychosocial factors in diagnostics and therapy
Poor or unexplainable symptoms of the patient: despite great efforts, no causes for the complaints can be identified (discrepancy between subjective state of health and clinical/imaging findings)
In conflict with own professional standards

Table 2 Characteristics of a difficult physician-patient relationship (expanded after [10, 15, 27, 34, 40, 63]).

Overestimation of own knowledge and skills
Problems with decision-making when facing uncertainty
Disappointing therapeutic results (cave: iatrogenic damage due to overtreatment and incorrect therapy [39, 59, 61])
High strain, feeling of hopelessness, helplessness, disappointment, anger, frustration, aversion
Feeling of being taken advantage of by patients
Dissatisfaction, helplessness, disillusionment, self-doubt about your own competence
Patient-physician relationship
Strong differences between the "individual realities" (disease theories) of patient and physician/dentist [8, 26]
Absence of an explanatory model (disease theory) for the complaints from both sides [46]
Lack of a common basis for the initiation of meaningful diagnostic and therapeutic steps, expectation discrepancy regarding the ways and goals of therapy
Appearance of new problems at the end of the consultation
Patient as "expert killer" [36–37, 47]

Table 2 Continuation Characteristics of a difficult physician-patient relationship (expanded after [10, 15, 27, 34, 40, 63]).

words, a difficult *interaction* between the physician/dentist and patient. This assertion is supported by the realization that dealing with "problem patients" in dentistry is associated with a disturbed physician-patient relationship; it can take on the form of personal antipathy, emotional involvement and expressions of aggressiveness, for instance [36]. Kowarowsky [35] thus states: "The difficult patient does not exist. It takes two to tango." (Figure 1). Accordingly, Kreyer [38] suggests the following definition: "Problem patients, whose therapy can become a psychological, and sometimes, even physical burden for the dentist, are primarily those patients in whom it proves impossible to build a sustainable physician-patient relationship."

Langewitz [40] cautions that "the communication skills of physicians play a decisive role in the perception of a consultation as being difficult". Communication is not only a matter of concern between physicians/dentists and patients [11–12, 22, 53, 54, 67], but also between the treatment providers themselves [7]. Characteristics of the interpersonal relationship between patients and (dental) practitioners are summarized in Table 2.

3. Dealing with difficult patients

Especially those patients who are described as being "difficult" expect their practitioners to provide emotional support; for instance, this means responding to their complaints to a greater extent than is usually the case in patients with somatic problems [52]. The patients – many of whom have previously turned to other doctors without success – are primarily seeking for advice. The practitioners are thus faced with a special responsibility. This is the establishment and maintenance of a trust-based relationship, which is of critical importance. In order to achieve this, patients should be given sufficient time (not only during the first consultation) to talk about their complaints, concerns, expectations, and explanatory models of illness [14, 17]. This is rather unusual in a profession, in which (well-paid) doing dominates over (hardly-paid) listening, speaking and explaining [43]. Some strategies for dealing with patients who are usually perceived as being difficult are found in Table 3.

4. Interpretation

Clinically and radiologically, practitioners can reliably recognize only what they have learned before. Based on single or multiple previous experiences, the brain stores patterns which are used in comparable future situations (pattern recognition [18, 33]). Practitioners with many years of professional experience have developed this ability to a particular degree [13]. Consequently, they feel secure in their professional field. In spite of this, the acquired skills cannot be transferred to other fields in which one has only little expertise. Forgetting this principle can put patients at risk just as much as ignoring the progress in one's own field of expertise.

Pattern recognition (i.e. detection), as the first step in an interpretation, is followed by explanation and evaluation, and (if necessary) standardization [4]. Changes in the scientific evaluation of clinical findings, such as the question "a variation of normality or pathology?" (e.g. in the

Strategies
Confirmation of the credibility of the complaints: they are neither imagined nor deliberately pretended
Respecting and striving for openness, empathy, and appreciation towards the patient
Objectivity; avoidance of emotional reactions
Making personal expectations more realistic
Use of proven communication techniques: – patient, non-judgmental listening – creation of a clear time frame and structure for the consultation – directness; avoidance of misleading statements – using humor as a tool in conversations – targeted exploration of the patients' subjective concepts of the disease (disease theories), their beliefs regarding the causes and their preferences – involving patients in the decision-making process (shared decision-making)
Atmosphere- or situation-specific strategy when receiving vague feedback without justification [40], such as: – "Obviously, we're not going further past this point." – "I realize that I do not know how I can help you further on at this point."
Personalizing the relationship through self-revelation, e.g. "Thank you for telling me so clearly." [41]
Setting limits and organizing further help: – Addressing difficulties, confronting patients when their behavior is inappropriate – Referral of the patient – In hopeless cases: seeking advice from colleagues, recommending a change of dentist/physician
Avoidance of using trivial and random findings as an explanation for the complaints
Avoidance of unnecessary and redundant examinations
Waiving of non-indicated therapies
Up-to-date, trustworthy and reliable information [1]
Consideration of current therapeutic recommendations (guidelines etc.)

Table 3 Some strategies for dealing with patients who are perceived as being difficult (based on [7, 10, 40]).

case of an anterior disc displacement [58]) start at the level of explanation; this is then followed by, for example, clinical, ethical/moral or esthetic judgment of the observed phenomenon, for which, especially when it occurs frequently, action-oriented suggestions or recommendations (e.g. guidelines from specialist societies) are usually developed, if they do not already exist (standardization).

Valid interpretations of clinical phenomena must always be based on the current state of scientific knowledge. In this sense, regularly remaining up-to-date with developments in one's field of experise is indispensible. Failure to do so increases the likelihood that a clinical situation and the patients involved will be considered "difficult" or "problematic".

5. Are patients with temporomandibular disorders "difficult"?

Patients affected by temporomandibular disorders (TMDs) and/or orofacial pain (OFP) are at particular risk of being perceived as "difficult" as they are fundamentally different from those persons who are usually seen during routine dental practice [62]. By resorting to the traditional "craftsman's model" [23] in patients with OFP or impaired mandibular function, the practitioner's limits will quickly be reached. On the other hand, the introduction of a biopsychosocial view [11] within the framework of diagnosis and therapy [64] continues to pose considerable challenges [56]. Unfortunately, it can repeatedly be observed that dentists with little experience in the field of functional disorders tend to describe TMD and OFP patients as "psychosomatic", "psychologically disturbed" or "psychologically altered". Such an ad hoc assessment not only reveals a lack of professional expertise, but also violates the fundamental ethical and moral principles of the (dental) profession [cf. 17, 44]. The over-

Curriculum provider	Continuing education
Dental Academy Karlsruhe	Curriculum "Function and pain" [URL: https://www.za-karlsruhe.de/de/akademie/fortbildungsangebot/curriculum.html?curriculum=Funktion_und_Schmerz_2021.html]
Academy for Practice and Science (APW)	Curriculum "Bruxism" [URL: https://www.apw.de/iw/curricula/curriculum-bruxismus]
Academy for Practice and Science/ German Society of Craniomandibular Function and Disorders	Curriculum "Function, functional disorders, temporomandibular disorders, and pain" [URL: https://www.apw.de/curricula/curriculum-funktionsdiagnostik-und-therapie]
Academy for Practice and Science (APW)	Curriculum "Basic competency in psychosomatics" [URL: https://www.apw.de/curricula/curriculum-psychosomatische-grundkompetenz]
University of Greifswald	Master's program "Dental functional analysis and therapy" [URL: http://www2.medizin.uni-greifswald.de/dental/master/index.php?id=451]
Scientific society	**Working group**
German Pain Society	Interdisciplinary working group for orofacial pain [URL: https://www.schmerzgesellschaft.de/topnavi/die-gesellschaft/arbeitskreise/mund-und-gesichtsschmerzen]
German Society for Dental and Oral Medicine (DGZMK)	Working Group for Psychology and Psychosomatics [URL: https://www.akpp-online.de/]

Table 4 Training opportunities and working groups in the fields of functional disorders, orofacial pain, and psychosomatics in Germany. (Tab. 1–4: J.C. Türp)

whelming majority of patients are not "more difficult" than people who wish to be treated for the purpose of preserving, replacing or repositioning their teeth. When the patient is haphazardly put into the "psychological corner", the "difficulty" – or more appropriately: the problem – is on the part of the dentist. Practitioners must be conscious of their professional limits and express themselves with due caution regarding issues that are outside their area of acquired expertise.

A particular challenge is posed by patients with *persistent/chronic* OFP which goes beyond ordinary toothaches, especially when the pain cannot be detected and explained by structural lesions, as is almost regularly the case in dentistry. Non-specific complaints associated with a feeling of suffering and functional impairments occur relatively frequently in medicine (e.g. globe sensation; chronic fatigue syndrome) [57] and are collectively referred to as "functional body complaints" [51].

In the presence of pronounced pain syndromes (e.g. fibromyalgia syndrome; irritable bowel syndrome), one refers to "functional somatic pain syndromes" [21, 24]. Patients with chronic TMDs fall into this grouping [21]. When contacting these patients, the dentist is sometimes exposed to situations that are well known in medicine. For example, one may be encountering patients

- who appear at the initial consultation with (*fat folder)* [16, 32] that are filled with written documents (findings reports, results from imaging examinations, correspondence with reimbursers, etc.);
- who like to appear at their appointments with (usually small) pieces of paper [42] on which they have meticulously noted down new questions about their symptoms; these must be patiently worked through at first (*la maladie du petit papier*) [5, 49];
- whose (dental) medical documentation is disproportionately detailed – and the patient's medical/dental history record is correspondingly thick (*thick-file case*) [14].

Meetings in this very extreme form are the exception, even in university settings or special consultation facilities, where patients with functional disorders of the masticatory systems are exclusively attended to. Colleagues working in private practices should thus decide on how they would like to deal with such patients from early on. In cases where one's professional expertise is surpassed, early referral to appropriate centers, such as university dental facilities or specialized colleagues, is recommended. Yet, depending on the lo-

(Fig. 1: published in the satirical magazine "Punch, or The London Charivari" on October 20th, 1909, Pg. 277)

Figure 1 Difficult patient or difficult physician? Different expectations on the part of the patient and physician coupled with unfavorable communication.
(Based on a wood engraving by Henry Matthew Brock, published in the satirical magazine *Punch, or the London Charivari* on October 20th, 1909, Pg. 277: A doctor angry with his patient for trying quack medicine as well as his own prescription. URL: <A doctor angry with his patient for trying quack medicine as Wellcome V0011480.jpg> [last accessed on: January 4, 2021])

cation, it may not be easy to find a competent center for making a referral. This even applies to university dental clinics. The TMD field is not a domain in which an overwhelming number of dentists show interest. This is also reflected in the dental school curriculum. A survey conducted by Hugger et al. about 10 years ago [29] showed that only 2 of the 30 dental schools in Germany offered lectures on the subject of TMDs. To date, this situation has not changed significantly.

Given the fact that

- the field of TMDs/OFP thematically differs from other dental specialties in fundamental ways [56];
- this field is obviously not sufficiently integrated into dental education [29];
- new scientific evidence [66] grows annually in the form of high-quality articles [30] reporting, for example, on results from randomized controlled trials [65];
- the acquisition of profound expertise can only be achieved through
  - (a) continuing education and ongoing professional training;
  - (b) reading contemporary and relevant literature on the topic [60],
  - (c) regularly attending high-quality training events, and
  - (d) maintaining regular (daily) contact with affected patients over many years[1],

it can be concluded that a large proportion of TMD/OFP patients characterized as "difficult" are given this description only because there is often a lack of expertise on the part of the treating dentist [56]. Consequently, the patients are not appropriately diagnosed and managed. This point of view is confirmed by data from Kreyer [36]: according to the author, one of the main reasons for dentists' fear of patients who are deemed "difficult" lies in the dentists' perceived lack of their own professional competence. During face-to-face conversations, many colleagues openly admit their limited knowledge in the field of TMDs. It is simply not possible to have a sufficient level of knowledge regarding all types of complaints in the oral and maxillofacial area. However, dentists who are primarily handicraft- and surgically-oriented are not recommended destinations for these patients [50].

Moreover, it is difficult to perform a lege artis assessment of TMD/OFP patients due to billing-related restrictions, especially given the considerable amount of time that is sometimes required for taking a thorough patient history. Insufficient payment for the collection of this important data is a serious problem worldwide and this is disadvantageous for patients. A praiseworthy exception can be found in the tariff regulations of the Swiss Dental Association (SSO): it permits payment for the TMD-related patient history based on 5-minute intervals. This, however, is the only means for ensuring that the patient is given an adequate opportunity to speak. In both general medicine [19] and pain medicine [48], the medical history plays a key role in the evaluation of a clinical case. The combination of

- inadequate dental education and continuing education,
- anamnestically incomplete patient information and
- possible communication deficits

makes complex cases not only "difficult" and "problematic", but also inevitably leads to failure (even if the practitioner is not always aware of it)[2].

6. Discussion

For some dentists, the label "problem patient" may have the function of "relieving" them of a part of their responsibilities. However, with such a strategy, dentists rob themselves of one of the most valuable assets available to them in their dealings with patients: trust, which, as the Freiburg medical ethicist Giovanni Maio noted, is the "binding agent" in the relationship between the patient and

[1] The Canadian physician Sir William Osler (1849–1919) remarked: "To study the phenomena of disease without books is to sail an uncharted sea, while to study books without patients is not to go to sea at all." [2].
[2] Relevant postgraduate continuing education and training opportunities are summarized in Table 4.

the physician/dentist: "The need for trust comes into play [...] when the patient can no longer judge whether what the physician recommends is really good advice or not". [45].

For years, representative surveys in Germany have shown that, after firefighters (2019: 94 %), physicians (2019: 87 %) are the most highly regarded professional group in the population [6]. This indicates that being a dentist means to pursue a profession of trust [45]. This advance of trust must not be jeopardized by unprofessional, unscientific and/or unethical actions. By the simple use of terms like "problem patient", this can already be happening in clinical situations that are beyond the practitioner's usual patient cases.

Boland [3] advises: "Before we label a patient as a problem, we should analyze ourselves and our reactions to the patient and consider why we have this reaction." The characterization of people as "difficult patients" is an interpretation that, in some cases, is misleading and reveals professional and communicative deficits on the part of the dentist. There are also "difficult" (dental) practitioners [31].

7. Conclusion

Knowing the current status in his field of expertise, and at the same time the limits of his professional and communicative competence is a quality that distinguishes a dentist who acts professionally and practices "good dentistry" [20]. The overestimation of one's abilities is one of the greatest dangers for professional failure and a risk factor for creating "difficult" patients.

Conflicts of interest

The author declares that there is no conflict of interest as defined by the guidelines of the International Committee of Medical Journal Editors.

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(Photo: Basilisk, Basel)

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"Psycho"-Diagnoses as self-protection in professional insecurity

Introduction: Speculative etiological assumptions and blame are widespread in the case of medically unexplained symptoms, and particularly in the case of pain.

Materials and Methods: In the article, findings are presented that demonstrate a high inter- and intra-individual variance of pain and thus the "subjectivity" of pain experiences.

Discussion/Conclusion: The meaning of the treatment context is discussed as well as possible errors and "traps" in the treatment. A critical reflection of one's own diagnostic and therapeutic routines is encouraged.

Keywords: patient-doctor relationship; placebo; nocebo; ICD-11; stigmatization; chronic pain

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"No luminary can be sure that he knows everything – in medicine this has become well known." [27].

The luminary was in ancient times the leader of the choir in Greek tragedies. As an expert in a particular field, he is in modern times "setting the tone". In medical field this expression names an outstanding expert, mostly head of a clinic, who is often the "last resort" in particularly difficult cases [27]. As his opponent, a "special" type of patients, called the "luminary killer", was introduced to the scientific literature by Beck on pain [4]. The concept "luminary killer syndrome" should explain why treatments fail in some patients, why the relationship between them and the doctor is characterized by distrust instead of trust, and by hostility instead of empathy. The patients are characterized by the following 3 features:

- a diffuse pain symptomatology with a variety of examinations and invasive procedures
- the lack of a clear somatic diagnosis and thus a meaningful therapy,
- a pathological doctor-patient relationship.

"This disease refers to indeterminate and functional pain conditions in the abdomen, neck and back. The impossibility of attributing it to a clinically defined somatic disease leads to a multitude of diagnostic efforts that become more aggressive with increasing failure" [4].

This "aggressiveness" is also implied in the term itself. Beck chose it "to attract attention so that patients with this condition can be better understood" [4].

This psychodynamic concept was developed on the basis of a group of 20 patients (2 men, 18 women), who were all seen for an interview and 10 of whom were in psychotherapy. Further details and data are missing. Beck asserted that the cause of this suffering is narcissistic personality traits in all patients, which show up as fragile self-esteem combined with being easily offended and highly sensitive. He goes on to say, that all of them lacked basic trust and were unable to develop reliable human relationships. This is connected with another characteristic: the persistence of an external ideal object. Finally, it is suspected, that the patients concerned idealize human relationships, and disappointment is thus predetermined [4].

Dissemination of the diagnosis "luminary killer": the stigmatization of patients with complex diseases

The terms "luminary" and "luminary killer", which have fallen somewhat out of fashion, were chosen for this article because they indicate an existing problem in a pointed but prototypical way in the treatment of patients with unclear symptoms. Originally conceived for patients with pain, the "luminary killer" has found its way into other areas of medical literature. The term has been and is used for craniomandibular dysfunctions [25], burning mouth syndrome [2], diarrhoea [26], in dermatology [22], ear, nose and throat medicine [12] and in fertility medicine [42]. These are all patients with special problems in diagnosis and therapy. It is difficult to distinguish between simulation, "doctor-shopping", aggravation of symptoms like pain and demonstrative illness behaviours [47].

All of these terms are concerned with attributing causes and, implicitly, blame: these are mental disorders of patients, often with manipulative tendencies. In this context of mistrust, explanations such as deception, lies or at least intentional motives are used to explain the clinician's own therapeutic failure.

According to Beck it is "a real psychosomatic suffering" [4]. Differential diagnostic problems exist with regard to other functional, psychosomatic, psychogenic and somatoform disorders. These are also assumed to be caused by a "real" mental illness. What these diagnoses have in common is that the cause lies primarily with the patient. The aim is to identify "difficult" patients as early as possible and to refer them to the most suitable treatment for them: psychotherapy. Patients usually do not accept this alternative to somatic treatment or only with considerable reservations.

Treatment-resistant facial pain has been a pioneering factor in the development of concepts for "psychologically induced pain". George Engel's influential work, entitled "primary atypical facial neuralgia", deals exclusively with unclear facial pain. The case presentations and conclusions concern 19 female patients and one male patient. The subtitle is "A hysterical conversion symptom" [13]. Typologies, terms and diagnoses for patients with "medically unexplained" pain of different localizations have their roots in the 19th century concept of hysteria.

Technical terms and diagnoses develop in the scientific context and consensus of the time. They reflect the respective state of knowledge of professional but also social constructions of illness and health. They should not be evaluative, but in many cases, they are, which often only becomes clear in retrospect: "insanity", "moronism", "idiocy" were official diagnoses in scientific classifications for many years. Also "hysteria" – and the different variants of psychogenic disorders (conversion, psychogenic pain, somatoform disorders) derived from it – were and are conceptual snapshots of scientific ideas. From today's perspective, they are associated with negative evaluations and are now less and less accepted socially and scientifically [31]. For this reason, the term "somatoform" has been largely ignored in America [28]. In everyday clinical practice, there are terms based on this that can be the basis for insult claims. In informal collegial discussions, derogatory slang expressions are common. For one patient with pain after a dental implant, the cryptic diagnosis "HGM" was found on the dental referral form to a university clinic. When asked, it turned out to be an acronym for "Has Gone Mad". In fact, it was a case of malpractice that was only recognized by the advanced diagnostics after the referral. Obviously, pain is a burden for the practitioner as well, in some cases with professionalism and empathy being lost. A contribution by Goldman is appropriately titled "Patients with chronic pain must cope with chronic lack of understanding on the part of the practitioner" [19].

Possible causes of interaction problems

There are many problem areas in dentistry that offer considerable po-

tential for conflict. For the treatment of patients with prosthesis intolerance [17, 53], mouth and tongue pain [15], occlusal dysesthesia [23], bruxism [37] and craniomandibular dysfunctions [25], competencies far beyond dentistry are required. Professionalism in diagnostics and therapy requires – in addition to the necessary technical expertise – the competence to shape relationships, create trust and involve patients in decisions. Friendliness, compassion and interest are necessary prerequisites for this. Unfavourable are prejudices, negative evaluations and resulting derogatory behaviour towards patients, verbal and non-verbal. The equality of people on the basis of their origin, skin colour, ideological orientation, physical or mental limitations is regulated by constitutions and laws in most countries. Whether these "prohibitions of discrimination" also reflect everyday life and especially attitudes is a central research question in the social sciences that is not easy to answer empirically.

For example, no prejudices were found in an open survey of over 25,000 health care workers on attitudes toward people with disabilities. The test subjects then additionally processed the Implicit Association Test, which records "automated" evaluations that are not subject to conscious control. Even this "professional" group showed clear reservations and latent discrimination towards disability and disabled people [45].

In another study, physicians anonymously commented on the question: How high do they estimate the proportion of simulators among their patients with back pain? [24]. Half of the surgeons suspect less than one malingerer in 10 patients. Every 10th surgeon, however, assumes that half of his patients are simulators. A trusting relationship in treatment is unlikely in this initial situation, creating an iatrogenic interaction problem in this case, not reflecting a mental disorder on the patient's side. Appreciative communication with patients is not only an important factor for patient satisfaction. Patients experience less pain after an empathic and respectful conversation than patients who experience degradation [44].

However, a "pathological relationship", distrust and devaluation are not limited to the "doctor-patient" interaction. Numerous studies show the widespread stigmatization experienced by people with chronic pain [9]. Stigmatization is the devaluing and discrediting reaction to people who possess a certain characteristic that deviates from social norms. In this case, people who do not conform to the usual biomedical norm, and where the pain clearly has physical causes. Stigmatization affects far more than the patients' experience in the health care system. People with chronic pain are also viewed sceptically and negatively in the family, at work and in social contacts if "clear findings" are lacking and complaints do not – as "normally" expected – disappear over time.

Are "luminaries" the better pain diagnosticians and practitioners?

High expectations are placed on diagnostics and therapy performed by luminaries, they should be outstanding. Is many years of experience and ascribed high competence an advantage?

Pain is not directly measurable, but requires communication. People can take different paths to achieve this: verbal messages, gestures and facial expressions, aids such as visual analogue or numerical rating scales. How well doctors and patients agree in their assessment was checked in an emergency room [29]. For this purpose, the pain data of 200 patients (data from 0 = no pain to 10 = strongest imaginable pain) and external assessments by the treating physicians (also 0–10) were compared. In an ideal world, there should be no differences between the intensity data for ideal patients and ideal practitioners. The influence of "experience" and "gender" on discrepancies between self and external assessment was examined, i.e. the difference between patient data and expert rating. The doctors consistently estimated the pain intensity of their patients as lower than did the patients themselves. The difference was particularly large among the "real luminaries", i.e. the experienced practitioners: Compared to newcomers, the "expert rating" is the furthest removed from the patients' experience. Surprising effects were also seen for the "gender" factor: Female doctors rated the pain of both men and women higher, i.e. closer to the patients rating than their male colleagues. "Experienced" male doctors were particularly far off the mark: they underestimated the pain intensity of female patients most significantly.

Who heals is right – or is in the clinical evidence pitfall

The reputation of luminaries is based primarily on their treatment competence. "He who heals is right" is an occasional justification for "eminence based" treatment successes. Surgical doctors in particular quickly gain a reputation as "luminaries". Specialization in one field, many years of professional socialization in hierarchically structured fields of work, and increasingly higher expectations of success have side effects. As one shoulder surgeon remarked: "Hardly any surgeon realizes how much their own perception can be deceptive. When you operate day after day and see that many patients feel better afterwards, you quickly think that this is because of you. That is why it is so important to conduct good studies. This is the only way to find out whether surgery really helps or not. Unfortunately, it is then often very difficult to transfer the study results into daily treatment practice. The doctors, but also the patients simply believe that the treatment, whose ineffectiveness has just been scientifically proven, is still effective" [21].

In the meantime, studies have been conducted on arthroscopic shoulder and knee surgery that have been shown to have no effect beyond that of a "sham operation" [3, 34, 41].

One possible explanation for the preservation of invasive procedures despite their dubious effectiveness is the "clinical evidence trap" in which the surgeon and patient get caught in "real life": While in studies medical interventions like drugs or procedures are tested "blinded" against placebo, both patient and surgeon go

into treatment in everyday clinical life with "open eyes" and high expectations. For patients, it takes place in an impressive setting, which in itself means a considerable placebo effect [5]. This is a favourable prerequisite for treatment success, although often due to nonspecific effects and less to the "elimination" of a suspected pathology.

In addition to the (at least initially) high expectations, the practitioners' conviction of the effectiveness of their method plays an important role. A very complex study entitled "Socially transmitted placebo effect" showed for the first time "that the expectations of providers regarding the efficacy of a treatment can significantly influence the treatment results of patients" [8]. What was new about this study was that the practitioners themselves were initially "patients" of a very convincing placebo treatment with the following "legend": the effect of an analgesic ointment was to be tested against a "placebo". The practitioners experienced this effect themselves first. They were given heat stimuli. Under placebo conditions the temperature was 47 °. In order to simulate the effect of a real "drug" (in fact also a placebo), the temperature was lowered to 43 ° during its application. This clearly perceptible difference convinced the practitioners of the effect of the ointment. They then carried out this test themselves with patients under simulated "strict" conditions. Indications of differences between the two substances, the effectiveness or comments were prohibited by instructions. The "patients" were also exposed to pain stimuli under "real drug" or placebo conditions. However, the temperature in this case was always 47 ° and was therefore identical for both ointments applications. Although the "patients" had no experience of the effect themselves, a clear placebo effect was also observed in them. In this case, it was achieved exclusively through the non-verbally conveyed expectations of the practitioners.

Under "real" conditions and additional verbal communication, the "socially transmitted placebo-effect" might be considerably stronger. By unconsciously shaping the patients' expectations of success, the "success" of their own treatment method becomes repeatedly confirmed and will be continued

The increasing aggressiveness of therapies described by Beck, as well as repeated unsuccessful and increasingly drastic interventions, are also called cascades [33]. The "Failed Back Surgery" of orthopaedics for back pain corresponds to the excessive tooth extraction ("the pain is often in the last tooth not yet extracted"). The common motto is "if in doubt, cut it out". Treatment options discussed for chronic facial pain were also aggressive. For example, in Engel's classic study, electroshock and lobotomy are considered, although there is no evidence for these, as he himself notes [13]. If a clear somatic diagnosis is missing and yet invasive diagnostic and therapeutic procedures are repeatedly performed, is a pathological doctor-patient relationship not normal? However, expressions such as "something must happen now" come from desperate patients and practitioners alike. This is associated with the assumption that "if nothing else works, you can still operate" – a generally wrong assumption and the beginning of many disastrous cascades. In a recent review of burning mouth pain, it is "the unexpected failure of interventional therapies that ultimately leads to a correct diagnosis" [11].

Deciding when to perform interventional procedures, is particularly relevant for surgical disciplines: The British neurosurgeon Henry Marsh, himself a recognized outstanding expert in the field of brain surgery, states: "Neurosurgery is certainly not about steady hands. It is about what the doctor has in his head; it is about judgment. It takes 3 months to learn how an operation works. Three years to learn when to perform it. And 30 years to learn when not to do it" [30].

Mental disorders are no explanation for chronic pain

"Psychosomatic" as a residual category in the absence of somatic explanations is associated with high risks of over-, under- or inappropriate treatment. The "classic" psychosomatic disorders of the past are gastric ulcer/duodenal ulcer, bronchial asthma, rheumatoid arthritis, neurodermatitis, essential hypertension, hyperthyroidism as well as the inflammatory bowel diseases ulcerative colitis and Crohn's disease. They are now regarded as physical diseases with psychological factors of influence. The assumed causation of "psyche" was wrong. For example, stomach ulcers are usually caused by the bacillus Helicobacter pylori and less by emotional stress. For Crohn's disease, which in the German-speaking countries also was considered to belong to the "holy cows of psychosomatics" [1], the causes are now discussed on the basis of prospective long-term studies. They are actually related to childhood experiences: the amount of antibiotics that had to be taken during that time [36].

A central question concerns the specificity and causal relevance of mental disorders for the explanation of chronic pain. The traditional classifications have been developed through studies with patients in specialized institutions. They refer to patients who could not be treated satisfactorily in the usual care process, who were "left over" after several selection processes. The "publication bias" based on these studies gave the impression that patients with chronic pain are a homogeneous group with a high prevalence of mental disorders and great potential for conflict and problems in treatment.

Epidemiological studies, in which the frequency of mental disorders in people with chronic pain was recorded, clearly put these assumptions into perspective. In a worldwide study involving more than 85,000 people, a higher probability of anxiety and affective disorders was indeed found in people with chronic pain compared to the pain-free population. However, the frequency of diagnoses was mostly below 10 % [10]. No statements on causality can be deduced from this. Prospective studies show that these are bidirectional relationships: "A persistent pain disorder at the start of the disease predicted the occurrence of a mental disorder to the same extent as a mental disorder at the start of the disease predicted the later occurrence

of persistent pain" [20]. Pain can trigger and aggravate depression – and vice versa. It therefore makes sense to integrate both areas (with necessary differentiations and focal points) into treatments. Premature and sweeping psychological labels are used to exclude patients with pain problems. Even with unclear somatic findings in other medical fields, both this "functionalization" of "psychodiagnoses" and their reliability as etiological explanations are increasingly being questioned [38]. In any case, even with standardized interview procedures, the diagnosis of somatoform disorders has proven to be less reliable and poorly repeatable than diagnoses of anxiety disorders or affective disorders [51].

Personality disorders also do not seem to have a specific meaning for the aetiology of pain, as a study with the meaningful title "Patterns of normal personality structure among chronic pain patients" shows [46].

The "pain personality" [14], the migraine type [52], the pain as a variant of depression [7], and "atypical" neuralgia [18] are further examples of historically interesting but retrospectively speculative ideas that have done little to advance our understanding of pain and chronic pain. However, the stigmatization associated with them has contributed greatly to the lack of understanding of those affected [9, 50]. The reliability of diagnostic procedures in medicine and psychology has increased in many areas. The use of standardized interviews and questionnaires in psychological diagnostics has improved the quality of studies and led to the elimination of familiar but speculative "diagnoses". The increasing sensitivity to language, for its possible significance as a placebo and nocebo, has increased. Paternalistic communication, a common feature of "the luminary", is now obsolete, not least for legal reasons [39]. Especially in the case of complex health problems, equal communication is a prerequisite for successful treatment.

Subjectivity of pain as a result of contextual conditions

Our traditional concept of pain is based on an almost essential connection between somatic damage and pain – and it is misleading. "There is no direct connection between somatic pathology and the intensity of pain" [43]. Nociception is not pain. Only after and through processing in different areas of our brain do nociceptive signals become danger signals and thus pain [32]. Pain is subjective. Even under simple experimental conditions with standardized pain stimuli, the intensity of pain experienced is highly variable between individuals: More than 300 subjects were exposed to multiple short heat stimuli of 48 °. The intensity should be assessed on a scale from 0 (= no pain) to 100 (= strongest imaginable pain). The values of the test persons ranged – with an average value of 71.8 – almost over the entire spectrum of possible values [16]. If a standardized experimental condition already leads to a confusing variety of experience on the part of the persons concerned, a further increase in variance is inevitable in complex situations with social, biological and psychological influences. These additional factors lead to further increased variance within individuals across settings: people experience pain differently depending on the personal significance of situations, not in a standardized way. For example, pain caused by overstraining in sports is easily dismissed while the same person can hardly stand the pain of dental treatment.

Chronic pain is usually etiologically and therapeutically complex. Categorical classifications into "healthy" and "sick" are initially a useful decision algorithm for acute somatic problems. In chronic pain, these concepts seldom take effect and lead to considerable problems, as Patrick Wall, a physician and one of the most renowned pain researchers outlined: "The full power of the classical medical profession which is pathologically based has concluded that there is 'nothing wrong' in pathological terms with the great majority of chronic pain patients. Since this conclusion is unquestioned and since the only generally accepted alternative is that there must be a design fault in human mental processing" [48]. Pain and especially chronic pain are now understood as a biopsychosocial phenomenon. The decisive difference to diseases with causally clear pathogenic factors is the inter- and intraindividual variation of risk factors: the significance of somatic, psychological and social influences can rarely be considered in isolation. Statistically (and in reality), the focus is not on dichotomous but on dimensional relationships and models. The individual components (e.g. risk factors) only partially contribute to clarify variance. In addition, these are usually no pathological changes, but variations around the norm, which only leads to pain problems or chronification in combination and interaction with each other.

Bruxism in this sense is no longer understood as a disorder requiring treatment "in otherwise healthy people" [37]. Back pain is also "statistically normal" and can only to a small extent be attributed to structural pathology. Most patients have a completely healthy spine for their age, and anxiety disorders are also found in only a few. Nevertheless, the combination of (muscularly induced) back pain, the widespread fear of serious causes ("slipped disc") and social stress ("If I can't work, how will I manage?") can lead to diagnostic decisions, in which widespread secondary findings are interpreted as central, are operated on and become the actual problem. This concerns most medical specialties with interventional procedures. Alf Nachemson, a recognized expert in spinal column surgery, states: "Back pain is not only about the spine, it is also about the brain" [49].

Advances in genetics, brain research, and epidemiology have contributed to a growing understanding of the complexity of pain and its chronification and have laid the scientific foundation for the biopsychosocial model that is now widely accepted. This model was published in 1977 by Engel, who thereby fundamentally revised his concept of the "pain personality" published almost 20 years earlier. The view of pain requires a broader perspective: instead of an individual psychopathology, somatic and social aspects and their interactions are integrated. This made oversimplified characterizations of patients obsolete. For these reasons, too,

teams are better suited for diagnosis and treatment. Interdisciplinarity and multimodal procedures characterize current treatment options. This initial situation is hardly compatible with the role of "luminary".

When treating chronic pain, whether of the face, head, back or shoulders, to name a few sites, medical expertise, diagnostic and therapeutic competence is an important but usually not sufficient basic requirement. The active involvement of patients is increasingly seen as an essential factor for treatment success. For craniomandibular dysfunctions, the "splint" is only one treatment component. Self-observation and the application of muscle relaxation procedures are the patients' task, which is met all the more reliably the more trusting the relationship with the dentist is, and the more plausible and acceptable the instructions and information provided are. This development – involving patients as partners in therapy – is now found in almost all forms of chronic pain. Instead of devaluating and excluding patients with dubious diagnoses that are unacceptable to them, medical, psychological, physiotherapeutic and social influencing factors are treatment goals in interdisciplinary multimodal programs.

As a consequence of these developments, chronic pain is a separate diagnosis in the ICD-11 and no longer included in the chapter for mental disorders. The criterion "lack of somatic cause" is considered too unscientific and has no significance for the diagnosis. Somatic, psychological and social factors are self-evident components of this "new" concept. The differentiation into primary and secondary chronic pain is also new. This takes into account the different somatic initial conditions and treatment options. The diagnosis is largely descriptive, and speculative assumptions about aetiology are avoided. Psychogenic pain or somatoform pain disorder as a mental disorder categorically distinguishable from the norm is no longer included in the ICD-11. Instead of a categorical one, a dimensional concept is consistently implemented. It is a continuum of various factors that contribute to varying degrees and not categories that can be clearly separated diagnostically: "normal" versus "disturbed" or "healthy" versus "sick".

This change to dimensional rather than categorial concepts was also realized for personality disorders: with the exception of the borderline personality disorder, all other types of disorders have been grouped under the generic term "personality disorder". Depending on the degree of different personality traits, specific profiles result. The lack of stability of personality disorders over the life course is one of the reasons for this revision [6, 40]. And here, too, we find a classificatory continuum with varying degrees of severity: From norm variants to pronounced stress instead of healthy versus sick, normal versus disturbed.

Concluding remarks

Tensions and conflicts between patients and therapists are relationship problems. To understand the reasons, it is necessary to look at both sides. Patients who do not fit into the system, because of their unclear complaints, personality characteristics or incompatibility with particular therapists, are still labelled as disturbed [39]. In fact, they disrupt clinicians' familiar routines and cognitive schemata. This danger is particularly prevalent in dentistry. Time pressure is high and a clear orientation towards a treatment algorithm that focuses on the (very successful) treatment of acute pain becomes a dead end for patients with unclear symptoms. This dynamic of interaction problems between doctor and patient has been summarized by the spinal surgeon Nachemson: "it is becoming clear … that ill-conceived diagnostic behaviour on the physician's part can lead to abnormal illness behaviour in patients, and this, in turn, may lead to abnormal treatment behaviour" [35].

Conflicts of Interest

The author declares that there is no conflict of interest within the meaning of the guidelines of the International Committee of Medical Journal Editors.

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Is the concept of somatoform prosthesis intolerance still up to date?

Introduction: Until recently "somatoform prosthesis intolerance" covered a wide range of patients with diffuse symptoms.

Material and Methods: Meanwhile, new dental conditions have been established so that it is possible to differentiate among Burning Mouth Syndrome (BMS), atypical odontalgia (persisting [idiopathic] dental alveolar pain), occlusal dysesthesia, and somatoform prosthesis intolerance. These clinical pictures can be categorized under diagnosis of "somatic symptom disorders", which was newly established in 2015. It is marked by a duration of symptoms of more than 6 months, intense preoccupation with those symptoms, and a significantly reduced capability to cope with everyday life. The formerly used diagnosis "somatoform prosthesis intolerance" can likewise be understood as a subcategory of specific dental somatic symptom disorder.

Conclusion: Based on available clinical experience it can be assumed that this diagnosis will be particularly applicable for patients that are equipped with objectively well-fitting fixed and/or removable dentures but experience difficulties with them and therefore attract attention with somatic stress symptoms. A structured approach is necessary for initial and basic treatment. This is described by the S3-guideline "functional disorders".

Keywords: somatoform prosthesis intolerance; burning mouth syndrome; occlusal dysesthesia; atypical odontalgia; somatic stress disorder; functional disorders

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Review

In 1921, Moral and Ahnemann [33] described the course of disease of a 50-year old patient, who complained about tongue pain, in a paper on borderline cases: "Her depiction appears unclear and blurred ... if pain showed up on the right side of the tongue once, it appeared on the other side at the next examination [...] suddenly also here [...], so that the pain can also be lead from one nerve region to another [...]". The authors found no clinical abnormalities for the mentioned complaints. They described the prostheses as well-crafted and occluded, the elimination test was negative, meaning that the patient was complaining about the same amount of discomfort while not wearing prostheses. The authors highlight the uselessness and specifically the damage caused by countless treatment attempts, which usually lead to chronification. They believe that the desire to help tormented patients leads to therapeutic errors and mishaps. The authors are giving a lot of attention to a goal-oriented, possibly interdisciplinary somatic diagnosis of exclusion. They do not consider it the dentist's job to – according to them – treat hysteria, but rather to perform necessary dental treatment. The difficulties of making the diagnosis in the manifold and multifaceted clinical picture indicate, that for routine dental measures, usually "... superficial recording of the anamnesis is sufficient", and with that the borderline cases depicted by them are mostly unrecognizable. The clinical picture of "hysteria", which according to Moral and Ahnemann is based "... on a disorder of a normal relationship between processes of our conscience and our physicality", for which they determine a basic condition "... that hysteria is an illness of the soul and that a treatment should be used; ...", which was described in 1859 by the French physician Briquet [5] in his work "Traité clinique et thérapeutique de l´hystérie". He also shows a descriptive approach to analyze the disease similarly to Moral and Ahnemann [33]. He lists a variety of physical and mental symptoms, which appear in "hysteric" sick patients in the form of a protruding leading symptoms or in combination with multiple complaints, possibly alternating with different emphasis. In the meantime, the work of Briquet has been picked up by many authors. Essentially, the attempt was made to systematize his observations assisted by Guze [17–19]. With the introduction of the DSM-III in 1980 [3], the Briquet-Syndrome was first incorporated as a framework of the prototype of somatization disorder in its own category in a clinically binding classification system. Müller-Fahlbusch and Marxkors [30] shaped the term "psychogenic prostheses intolerance", which had already been used by Peterhans in 1948 [36]. Based on an interdisciplinary research project conducted in 1976 [39], Müller-Fahlbusch and Marxkors understood this as "complaints that do not fit the picture of the respective findings.

Diagnostic criteria of a psychogenic prosthesis intolerance according to Müller-Fahlbusch	
1.	Discrepancy between description of symptoms and anatomical limits
2.	Discrepancy between chronology of symptoms and complaints and the known development known to us by clinical experience
3.	Ex non juvantibus (a normally helpful treatment does not lead to success)
4.	Unusual co-participation of the patient in the course of the disease
5.	Coincidence of biographic-situational results and beginning of the complaints

Table 1 Diagnostic criteria of a psychogenic prosthesis intolerance according to Müller-Fahlbusch [34]

Criterion A	**Somatic symptom(s)**	
	are distressing result in disruption of daily life	
Criterion B	**Psychological Characteristics regarding physical symptoms**	
	Exaggerated and persisting thoughts on the seriousness of the present symptoms	cognitive dimension
	Persisting and pronounced high level of anxiety regarding health of symptoms	emotional dimension
	Excessive effort in time and energy that is expensed for the symptoms	behavioral dimension
Kriterium C	**Burden of symptoms for longer than 6 months**	

Table 2 Somatic Symptom Disorder: For a diagnosis according to DSM-5 (Diagnostic and Statistical Manual of Mental Disorders) criteria A, B (at least 1 of 3 psychological dimensions) and C must be met [11, 25].

List of symptoms	
• Stomach pain or indigestion	
• Back pain	
• Aching arms, legs or joints	
• Headache	
• Chest pain or shortness of breath	
• Dizzyness	
• Fatigue or lack of energy	
• Insomnia	
Severity of somatic burden	
0 – 3	None to minimal
4 – 7	Low
8 – 11	Moderate
12 – 15	High
16 – 32	Very high

Table 3 Somatic symptoms scale to determine the somatic symptom burden (SSS 8) [28]. A sum score of 0 = "none" to 4 = "very high" is formed to answer the "how high was the burden caused by the mentioned symptoms during the past week".

The complaints are more general, less tangible and do not allow for direct conclusions about the prosthetic work" [31, 34]. While Marxkors understood this term in the prosthetic context, Müller-Fahlbusch extended this viewpoint with psychiatric aspects. In an interdisciplinary study he diagnosed 57 % of patients with psychogenic prosthesis intolerance with phasic and chronic depression, 21 % of patients with abnormal personality disorder and 19 % of patients with an abnormal experience response. He classified about 3 % of patients in the category of schizophrenics. Only in the course of further cooperation psychosomatic diagnostics developed, but further down the line, a viewpoint in psychosomatic diagnostics which was especially expressed in the catalogue compiled by Müller-Fahlbusch [34] of 5 diagnostic criteria to recognize psychosomatic conspicuous patients (Tab. 1).

Müller-Fahlbusch attaches special importance to the time of treatment of a possibly necessary somatic therapy and depicts recommendations of how to deal with these patients. Just like Haneke [20], he recommends the regulation of psychiatric drugs, usually antidepressants. Balters [4] advises psychological care of the ill that is supposed to help turn the loss of their teeth into something positive. Marxkors [31] warns against overpowering when dealing with difficult patients and to not expand treatments against the wishes of the dentist, just because the patient wishes or demands it. Other authors [8, 48] recommend to consider solid constrictions in "patients with psychosis". All authors are in agreement regarding a crucial and necessary interdisciplinary cooperation.

In 2008, the term "psychogenic prosthesis intolerance" was replaced by the term "somatoform prothesis intolerance" [13]. With this, the necessary adjustment of the nomenclature in general medicine occurred [12]. Besides the Burning-Mouth-Syndrome, the somatoform pain disorder and body dysmorphic disorder (as a special form), the somatoform prosthesis intolerance presents a relevant subdivision of somatoform disorder: "The characteristic of somatoform disorder is the repeated presentation of physical symptoms in combination with persistent demands after examinations, despite repeatedly negative results and reassurances by doctors, that the symptoms are not based on any physicality. If there is some organ pathology present, it does not explain the nature and extent of the symptoms and the pain and the internal investment of the patient". With the classification of the illness pattern of "somatoform prosthesis intolerance", the first criterion according to Müller-Fahlbusch takes on an extended dimension. While Müller-Fahlbusch relates the discrepancy to anatomical structures – "medical psychology and psychosomatics does not work without anatomy" [34] –, demanded with the inclusion of somatic findings, that the complaints within context are evaluated of possible, also pathological findings, regarding their nature, expansion and intensity in order to detect an available discrepancy to the mentioned complaints.

Characteristics of a "somatoform prosthesis intolerance"

Fundamentally, the question is raised whether or not the term "somatoform prosthesis intolerance" summarizes this group of patients accurately enough today, or if further classifications exist by now that allow a more precise distinction and with that more targeted treatment options. There is hardly any data on who is typically affected by these symptoms. Studies [32, 39] could show, that women aging between 60 and 70 sought out a specialized consultation 5-times more frequently. The main symp-

Symptoms	Burning-Mouth-Syndrome	Persisting idiopathic facial pain	Atypical odontalgia	Occlusal dysesthesia
Diagnosis criteria	– daily burning/burning pain or feeling of dysesthesia – > 3 months – at least > 2 hours per day	– pain nearly all day – at most low impairment of night's sleep	– daytime pain, brief to persisting – unimpaired night's sleep – > 4 months – spontaneous beginn, that can (often) be delayed following the trauma of a peripheral trigeminal nerv (experiencing pain during such course of action increases the risk)	– awareness only during waking state
	– women, – > 50 years old – reduced quality of life – increasing intensity during the course of the day	– fluctuating intensity – at most just beginning anatomically limiting expansion – "peculiar" disease causation modell	– diffuse expansion tendency – variable intensity – pain amplification through peripheral stimuli – allodynia/hyperalgesia – uncertain pain elimination – ex non iuvantibus	– "occlusally fixed" model of illness – intensive occupation with the disorder – Chronification tendency with many unsuccessful changes in occlusion – dismissive evaluation of previous practitioners – glorification of the current practitioner – psychosocial burdens/impairment at the beginning and/or over the course – further physical ailments usually without objectifiable cause and plausible course of therapy
	– no local/general medical and psychological causes	– no relevant pathological results	– missing adequate pathological results	– no relevant malocclusion
Screening/ documentation forms (if available)		Pain diary regarding modulation factors, possibilities of relief and accompanying symptoms		– Chronification: GCS [43, 45]. – anxiety, depression: HADS [21]; PHQ-4 [27]; DASS [20,34]. – emotional stress: SRRS [1, 22]; – somatization: BL-R / BL-R' [46] – localisation of pain: full body drawing [42] of all existing pain regions.

Table 4 Typical characteristics, screening options, differential diagnoses and supportive information in diseases appearing diffuse in the orofacial

toms listed by affected patients are pain, burning of oral mucosa and adaptation disorders (mostly related to prostheses and often specifically related to the "difficulty to bite down)". These symptoms can appear localized or radiate further into the oral cavity and are solely associated with the oral cavity based on the patients' understanding of the clinical picture. Usually the symptoms last longer than 6 months. The patient's path in search of relief is characterized by countless diagnostic procedures and therapy attempts ("doc-

Symptoms	Burning-Mouth-Syndrome	Persisting idiopathic facial pain	Atypical odontalgia	Occlusal dysesthesia
Differential diagnoses (screening, if available)	secundary burning of the oral mucosa	neuropathological pain (possibly triggered by surgery in the specified pain region)	dental causes	objectifiable malocclusion craniomandibular dysfunction/bruxism CMD screening https://www.dgfdt.de/documents/266840/3732097/CMD-Screening+DGFDT/cc704187-a983–4eed-893c-614ae3969bd1 bruxism screening https://www.dgfdt.de/documents/266840/3732097/Bruxismus+Screening+02_20/8039b42a-9640–47e9-bd9f-0717a3c4d423 functional status https://www.dgfdt.de/documents/266840/406693/Erfassungsformular+Funktionsstatus+2012/1d692d7a-bf94–4509–9035-a091a82d58f7?version=1.0)
Supporting information (if available, retrievable from DGZMK: https://www.zahnmedizinische-patienteninformationen.de/patienteninformationen)	https://www.zahnmedizinische-patienteninformationen.de/documents/10157/903264/Zungen-und_Schleimhautbrennen/	https://www.zahnmedizinische-patienteninformationen.de/documents/10157/1129556/268572_1567299_Chronischer+Kiefer-+und+Gesichtsschmerz.pdf		https://www.zahnmedizinische-patienteninformationen.de/documents/10165/1430990/PI+Bruxismus-final.pdf https://www.zahnmedizinische-patienteninformationen.de/documents/10157/1129556/268572_1567355_Kiefergelenkschmerz.pdf/

Table 4 Continuation Typical characteristics, screening options, differential diagnoses and supportive information in diseases appearing diffuse in the orofacial

tor hopping, doctor shopping"). It is not uncommon to observe that patients affected, as well as people close to them subject their entire lives to these complaints and show a severely reduced quality of life. These main symptoms are accompanied by complaints about dry mouth or altered sense of taste. The courses of complaints are individually different and vary regarding their intensity. These characteristics regarding course and duration, understanding of the disease or dealing with the complaints are key criteria of the newly added diagnosis of "somatic stress disorder", which can therefore be seen as a superordinate category.

Somatic Symptom Disorder (SSD)

SSD refers to a new classification in the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) [7, 25, 26, 41]. This should diagnostically record about 30 % of patients in basic care, that are severely affected by existing physical symptoms and restricted in their daily lives. In order to make this diagnosis, it is irrelevant if the characteristics listed in table 2 were triggered by a somatic and/or mental reason. In general medicine, the severity of the burden is determined by respective points on an 8-symptom scale (SSS 8) [16, 28] (Tab. 3). Typical dental symptoms have not been recorded in the SSS 8. In order to assess a potentially generally existing problem, this symptom scale can be inquired within the context of general anamnesis in a regular dentist appointment. This offers the chance to recognize tendencies and risks of expansion into the jaw and face region with possibly necessary dental measures. Depend-

Initial basic care		Extended basic care	
In the mild form		When visiting the doctor repeatedly and "doctor-hopping-tendency"	
Recognition	Comprehensive anamnesis – main symptoms – accompanying symptoms – impairments – patient behavior – initial recording of findings – demand preliminary findings – avoid redundancies – further examinations with reserved and strict indication	simultaneous diagnosis	To emphasize the equality of physical and psychosocial influencing factors – Attention: do not act on "pressure by the patient" "slow down" – Goal-oriented, clear setting – No technical supplementary examinations to calm down the patient – explain regular findings – demonstrate prognosis/risks of a one-sided somatic approach
Reassurance	Evaluation of findings and risks – Announce "expectable" normal findings – No downplaying model of disease in order to expand psychosocial viewpoint	Accomplishment	– biopsychosocial explanation model originating from subjective disease theory e.g. using individual amplifiers, modulation factors as well as possibilities of relief – clarify expectations and correct if necessary – emphasize autonomy, develop "active" coping strategies – regular appointments independent of discomfort
Advice	– "take the patient seriously" – emphasize credibility – work out positive resources	Possibly initiate interdisciplinary cooperation	– Transparent findings assessment – Motivate patients to take up psychosocial therapy options – dental findings to avoid the polypragmatic approach "monitoring"
Supporting information	https://www.awmf.org/uploads/tx_szleitlinien/051–001p3_S3_Funktionelle_Koerperbeschwerden_2020–01.pdf		
Therapeutic "support"	wait and see: Patient monitoring and supervision while avoiding measures that are not strictly indicated		

Table 5 Diagnostics and therapeutic approach [following 37]
(Tab. 1–5: A. Wolowski)

ing on the severity of the disorder, it has to be decided if an explanation of risk by the dentist is sufficient or if an interdisciplinary approach has to be pursued. It can be helpful for such a decision to differentiate between relevant dental clinical pictures. The necessity of this differentiation also results from the fact that an interdisciplinary setting with treating "non-dentists" requires explicit details on dental context.

Dental diseases with symptoms seeming diffuse

It can be differentiated between dental symptoms within the group of somatic stress disorders by using a complaint-related classification. The leading symptoms are burning of the oral mucosa, pain and occlusal "malfunctions".

Burning of the oral mucosa: Scala et al. [40] differentiate the secondary burning of oral mucosa that can be diagnosed following an underlying dental, general and mental disorder of idiopathic burning of the oral mucosa, which is classified as BMS [47]. According to the currently valid definitions [24], the diagnosis BMS is based on a diagnosis of exclusion. The different current definitions regarding BMS differ mainly in the specification of total duration and the daily course. Because BMS has not been defined uniformly in literature, it cannot be differentiated regarding mental factors if this is the cause for a secondary burning of the oral mucosa or if mental factors arise following an (idiopathic) BMS. Different levels of anxiety are listed and in 20 % of BMS patients, the phobia of cancer can be observed. Depression and somatization disorder are named as further diagnoses [2, 6, 14, 27, 29, 37] (Tab. 4).

Pain in the sense of persistent idiopathic facial pain (PIFP)/ atypical odontalgia (persistent [idiopathic] dental pain): PIFP refers to the pain, that does not meet the criteria of a facial neuralgia and is not associated with signs of an organic lesion. "The pain is present, mostly continuous, one-sided and difficult to locate. Sensitivity symptoms or other deficiencies are not present. Further examinations including X-Ray diagnostics of the face and jaw are without pathological

findings. Either trauma, or an operation of face, jaw and teeth can cause the pain. However, there can be no current pathological local findings" [10], because that would categorize it as a diagnosis of exclusion [15].

A localized form of the PIFP is described as atypical odontalgia, in which a pathomechanism of a neuropathological persistent pain comparable to phantom pain is taken on [15, 44]. Based on missing pathological findings, this is also a diagnosis of exclusion. Endodontic procedures are described as risk and trigger factors or as an experienced painful dental treatment before a tooth extraction (Tab. 4).

Occlusal dysesthesia: The symptoms of occlusal dysesthesia (OD) describes the phenomenon, that patients complain about pain originating from their occlusion, which is clinically not objectifiable. Most patients affected are burdened mentally and show characteristics of depression and/or anxiety. They are often solely focused on a somatic/occlusal cause of their pain and every therapy attempt according to the rules with mostly rotating practitioners almost always leads to intensification of the complaints. The median age described for these symptoms in literature is 52 years (plus/minus 11 years), which also goes along with clinical experience in specialized consultation. Etiological factors discussed are psychopathological causes, neuroplasticity, phantom phenomenons and changes of proprioceptive stimuli and transmission [9] (Tab. 4).

Structured approach

Given the mostly complex and diffuse ailments and also the psychological strain of the affected, it is the most important goal to identify influencing factors early and inform comprehensively, so that affected people are actively included in the diagnostic and possibly therapeutic process. The guideline on "functional disorders", published in 2019 [37], which specifically included the clinical picture of somatic stress disorder on the spectrum of the summarized clinical pictures, presets a structured approach. These are based on the severity of the course of the disease and are classified into the always necessary basic care and the extended care during longer hospital stays as well as multimodal therapy. It should be emphasized in this context that further dental treatment support should be maintained and no either-/or-principle should be initiated with the referral to other specialist disciplines. This issue is written out to supposedly lead to a better mutual understanding of co-practitioner and patient. The basic principle here is maximum transparency. This requires a sustainable and with that resilient doctor-patient-relationship, which is supported by a structured approach (Tab. 5).

Which symptoms remain of the "somatoform prosthesis intolerance"?

In conclusion, the question is raised if the diagnosis "somatoform prosthesis intolerance" is justified today. This can be understood as a subgroup of dental-specific disease in the sense of a somatic stress disorder in patients whose leading symptom is burning of the oral mucosa, pain and/or occlusal difficulties to general physically severely burdening symptoms. Based on the available clinical experience one can assume that this diagnosis applies especially to patients that are fitted with (fixed and/or removable) prostheses and experience difficulties with them and show signs of somatic stress. The diagnosis "somatoform prosthesis intolerance" should not be a diagnosis of exclusion, but rather it is more important to detect indications of somatic and psychosocial influences, if a differential exclusion of the specific clinical pictures follows. It is to be expected that the diagnosis "somatoform prosthesis intolerance" can overlap with the described dental diseases. A valid and identical approach according to psychosomatic basic care for all differential diagnoses is helpful and crucial for the practitioner.

Conflicts of interest

The author declares that there is no conflict of interest as defined by the guidelines of the International Committee of Medical Journal Editors.

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(Photo: A. Wolowski)

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A subtle trap – occlusal dysesthesia

Introduction: Patients complaining of uncomfortable and unpleasant tooth contacts are encountered in the dental practice time and time again, as well as in the fields of physiotherapy, pain therapy, and psychotherapy. These tooth contacts are neither clinically identifiable as premature contacts nor associated with other disorders (e.g., of the periodontal tissues, dental pulp, masticatory muscles, or temporomandibular joint). It is not uncommon for patients to experience this perceived occlusal discomfort as a constant impairment of their oral or physical well-being. This is often accompanied by psychosocial problems. The cases discussed in this article often concern patients suffering from occlusal dysesthesia (OD), although a differential diagnosis must always be carried out to distinguish OD from occlusal disease.

Methods: This article presents clinical features of occlusal dysesthesia that are relevant to everyday practice. These features are explained based on the current guideline "Occlusal Dysesthesia – Diagnostics and Management" published by the Association of the Scientific Medical Societies in Germany (AWMF) and by means of case examples. Psychopathological factors, neuroplasticity, phantom phenomena, and changes to the transmission of proprioceptive stimuli and perception have been discussed as etiological factors of OD; however, the exact connections have not yet been extensively researched or fully understood. Invasive occlusal therapy is not advisable. The use of dental splints is also a controversial topic of discussion in the literature. Patient counselling and education about the nature of OD ("information therapy") that aims to explain and defocus is a recommended measure. Other therapeutic alternatives include cognitive behavioral therapy, specialist medical treatment of possible comorbid psychological factors, pharmacotherapy, and the prescription of physical activity.

Conclusion: Despite professional therapy, treatment of affected patients is often unsuccessful.

Keywords: occlusion; lost bite; false bite; occlusal discomfort; occlusal disease; occlusal dysesthesia

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Introduction

"This might sound funny, but I've lost my bite!"

Patients complaining of uncomfortable and unpleasant tooth contacts are encountered in the dental practice time and time again, as well as in the fields of physiotherapy, pain therapy, and psychotherapy. These patients often experience their occlusal discomfort as a perpetual constraint on their oral or even whole-body well-being. According to latest knowledge, occlusion is considered a low risk factor for the development of painful musculoskeletal disorders inside and outside the masticatory organ, and in this context should be understood only as a cofactor and not as a sufficient condition on its own [8, 17, 28]. Nonetheless, the widespread view remains that humans can only tolerate their occlusion if it fulfills certain conceptual rules.

Based on these classical views in dentistry with regard to the "optimum bite", the consulting dentist will often undertake invasive procedures in the cases described above. Unfortunately, however, such an approach usually leads to unsuccessful therapy attempts, conflicts, and a complete loss of trust between dentist and patient. If the costs of treatment are high, it is not unusual for therapeutic efforts to be followed by legal proceedings. As the title of this article suggests, these cases often involve patients who are suffering from occlusal dysesthesia (OD).

Without claiming to be exhaustive, this article presents and discusses clinical features of occlusal dysesthesia that are relevant to everyday dental practice. This discussion is based on the guideline of the Association of the Scientific Medical Societies in Germany (AWMF), "Occlusal Dysesthesia – Diagnosis and Management" [1, 11] and the authors' experiences as practitioners, as well as those of experts appointed in legal disputes. The article also includes several patient quotations that the authors consider typical of the clinical picture of OD. Here we would like to express our gratitude to the authors of the guideline, whose explanations have provided a valuable basis for making decisions when treating patients suffering from OD and a helpful aid for dental experts.

Treatment methods

Diagnostics

"It all started back in 1988 when I received an inlay on tooth 14. The contact with the opposite tooth was much too strong. All of a sudden, I was unable to move my left leg back while dancing – from then on, nothing was right anymore. […] With every dental treatment I received, things just got even worse! I've brought you all the models made over the years, in case you would like to see them. […] Please help me! I'm at my wits' end."

In general, patients do not consciously perceive the contact between antagonist teeth in the upper and lower jaws [23]. The substantial difference in perception experienced by patients who "suffer" from OD in the truest sense of the word is clearly to be found in the AWMF guideline's definition of the condition. This defines OD as "a condition in which tooth contacts that are neither clinically identifiable as premature contacts nor associated with other conditions (e.g., of the periodontium, dental pulp, masticatory muscles, or temporomandibular joints) are continuously (for more than 6 months) perceived as uncomfortable or unpleasant. The clinical findings do not bear a clear relationship to the nature and severity of the symptoms reported. The patients suffer from severe psychological and psychosocial strain." [1].

Psychological factors, neuroplasticity, phantom phenomena, and changes in the transmission and perception of proprioceptive stimuli have been discussed as etiological factors of OD, although the exact links have not been researched in much detail [9, 19, 21].

"It was all rather inconvenient at the time. I was on business abroad (in Spain) to set up a branch there. Of course, that's when my tooth chose to break, and I had to go to the dentist there. […] Something had not been right with the crown from the beginning. It felt as if I had just this one tooth in my mouth. The dentist always said that everything would be fine, yet he reground the crown countless times. At this stage, my jaw and neck had already begun to hurt."

The onset of OD is often connected to dental treatment, and commonly happens in conjunction with a stage of life that the patient has

Diagnostically important and frequently encountered signs of OD in the context of the specific anamnesis are [16, 19, 24, 25, 27]:
Complaints exist for longer than 6 months (frequently a long-standing medical history with numerous changes of practitioners and negative emotions towards the previous practitioners)
There is a focus on the conscious perception of the occlusion
The trigger was a dental treatment (regardless of the intensity)
The complaints have a relevant influence on living and experience
Non-specific complaints are attributed to the occlusion
Frequently, extremely detailed descriptions of the occlusal disturbances using specialized terminology
Despite clarification, there is a vehement insistence on the person's own pathophysiological beliefs
Repeated changes to the occlusion remain unsuccessful

Table 1 Diagnostic evidence that can indicate the presence of OD (modified after [1]).

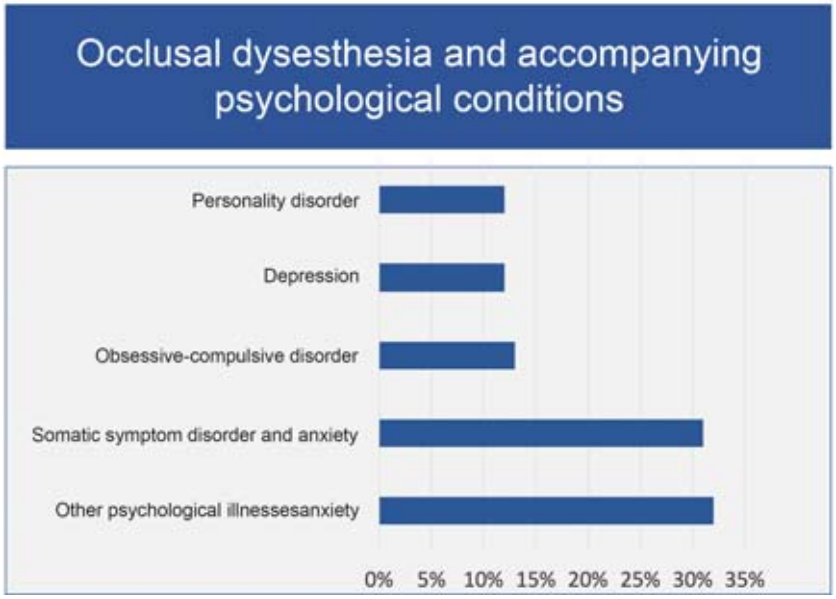

Figure 1 As a rule, occlusal dysesthesia is accompanied by additional psychological stresses, of which, an illustration in percentage frequency is shown for a selection of them (modified after [1])

found stressful [5, 26]. The type and complexity of the dental intervention does not appear to have an effect [23]. OD occurs in isolation or in combination with temporomandibular joint (TMJ) disorders [12]. Occlusal interventions aimed at eliminating non-specific symptoms have been described as iatrogenically contributing to the development of OD [24]. In most cases, it is middle-aged women who visit the dentist with symptoms of OD [9, 25] (women are affected approximately five times more often than men). Current data indicate that the average age of onset for the condition is 45 [9, 14]. Only adults appear to be affected [1] (Table 1).

"I just want to bite the way I used to. I want my old life back!"

Questionnaires for evaluating possible cofactors of occlusal dysesthesia
Localization of pain • Full body mapping of all areas of pain
Chronification • Graded Chronic Pain Scale (GCPS))
Anxiety and depression • Personal Health Questionnaire 4 (PHQ-4) • Hospital Anxiety and Depression Scale (HADS) • Depression-Anxiety-Stress Scale (DASS)
Emotional Stress • Social Readjustment Rating Scale (SRRS) • Depression-Anxiety-Stress Scale (DASS)
Somatization • Symptoms list (B-LR and B-LR' symptoms lists) • Somatic Symptom Scale (SSS-8) • Personal Health Questionnaire 15 (PHQ-15)

Table 2 Questionnaires to evaluate possible cofactors of occlusal dysesthesia (modified after [1])
(Tab. 1 and. 2, Fig. 1 and. 2: Adoption of the contents of the tables and figures from [1])

Over time, patients with OD generally become fixated on their occlusion [15, 23, 24]. It is evident that the described symptoms play a central role in the lives of those affected, and that the patient's environment is tightly interconnected with his or her situation. Pseudo-scientific posts on the internet confirm that those affected ascribe a clearly exaggerated pathophysiological potential to their occlusal disorders, usually involving extensive effects on the general health of the entire body. This situation often also causes patients to become extremely anxious. OD fulfills the criteria of a "somatic stress disorder" (DSM-5 300.82). It is often accompanied by other psychological problems [9, 22, 25] (Fig. 1).

"No dentist listens to me properly – they all immediately want to pigeonhole me as a loony!"

If the affected patient's medical history provides corresponding indications of OD, the extent of his or her symptoms can be recorded by means of suitable and frequently used questionnaires (Table 2). If such findings are obtained, the results must be discussed with the persons concerned. However, a delineation of mental or psychiatric symptoms does not fall within the area of competence of the dentist and must be carried out by an appropriate specialist.

"Surely you can also see that the shape of my crowns is not correct. As a result, my lower jaw has lost its stability and is always slipping to the left."

Somatic findings are characterized by a discrepancy between the patient's subjective occlusal sensations and the occlusal findings. Patients with OD usually describe their complaints in very vivid and precise terms, and generally go far beyond the degree of explanation used by untroubled patients to describe occlusal interventions.

Occlusal disease compared with occlusal dysesthesia

It is important to differentiate OD from occlusal disease (Fig. 2). The main difference is that occlusal disease can have dentogenic, myogenic, or arthrogenic causes. This means that the discomfort mentioned by

the patient can be clearly and convincingly objectively identified by means of standard dental diagnostics. In this case, subjective sensations and objective findings coincide.

Thus, a patient merely stating that his or her bite is not or is no longer correct should not necessarily lead to the diagnosis of OD. Additional diagnostic information should be obtained first.

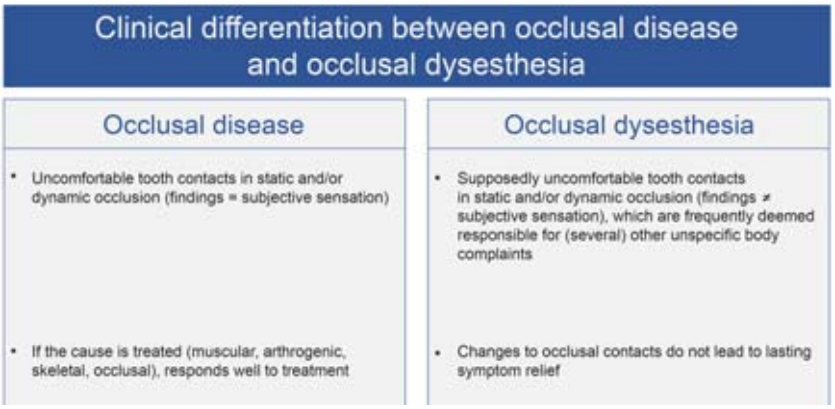

Figure 2 Clinical differentiation between occlusal disease and occlusal dysesthesia (modified after [1])

Management

"I've heard that you are a very good dentist. My previous dentists didn't examine me as thoroughly as you have. I'm sure you'll be able to sort me out."

Because the symptoms of OD are an expression of a functional condition, it should be emphasized at this point that they cannot be effectively treated by means of dental interventions, but instead require further specialist medical care. It is therefore more appropriate to speak of management than of treatment. Even if the presumed solution often seems obvious to those affected, and they vehemently demand the implementation of occlusal therapy in accordance with how they expected to be treated, it is advisable to repeatedly offer non-invasive measures and therapy alternatives from outside the field of dentistry.

"Your predecessor almost succeeded. But when almost everything was fine, in the end, he didn't want to grind down the point where I told him to any further."

It should always be noted that interventions to treat a patient's occlusion will not bring lasting success if the patient has OD. After apparent initial success, the occlusal "corrections" will often be ineffective or even lead to a worsening of symptoms [14, 25]. In most cases, this creates a lasting strain on the dentist-patient relationship. If invasive interventions are performed simply at the request of the patient, despite the fact that the described sensations cannot be objectively substantiated by means of established dental procedures, then the dentist is simply straying away from the rules that underlie the practice of their profession. In the case of any possible subsequent dispute, no plausible justification exists for such actions. Based on the current guideline [1], the question of a differential diagnosis between OD and occlusal disease is likely to be raised in any future legal disputes.

Because data regarding the management of patients with OD is very limited, the following explanations are based solely on an expert-based consensus derived from the guideline. When a patient has OD, the primary goal of any therapeutic efforts is to improve the patient's oral-health-related quality of life by means of extensive patient education and defocusing [3, 21]. This is only possible if mutual trust exists between doctor and patient; this means that the dentist takes the patient seriously and that the patient is convinced of the practitioner's competence. The general recommendation is to avoid confrontational discussions with the patient and, in the context of information therapy, to repeatedly offer them alternative ways out of how they usually interpret their physical perceptions. This is certainly a sensible and helpful approach; given billing arrangements, however – at least for dentists residing in Germany – it is difficult to achieve. Owing to the above mentioned cofactors of OD, the importance of a psychological or psychiatric therapeutic approach again becomes clear at this point. An essential feature of information therapy is to make it clear to patients that, compared with healthy people, their perception of their occlusal contacts is heightened [13]. Many patients tend to constantly "check" their occlusion in the form of static and dynamic biting behaviors. This can increase the patient's fixation with their occlusion and also constitutes a risk factor for TMJ disorders [4, 10, 20], because biting behaviors performed with little force and for a prolonged duration can trigger pain within the jaw muscles [7]. Therefore, in the case of myofascial pain, patients should be given instruction that aims to prevent them from consciously checking their occlusion.

Invasive occlusal therapy is not recommended. The use of oral splints is a topic of critical discussion in the literature and, if splints are used at all, they are recommended as a short-term therapy to reduce irritation and thereby possibly achieve defocusing [6, 9, 25].

The therapeutic considerations just mentioned will now be elucidated by means of the example of an affected patient, who for many years originally wanted a comprehensive (unindicated) prosthetic restoration of all teeth in the upper and lower jaws. As the result of talking therapy that aimed to achieve defocusing, the patient learned to accept her clinical picture of OD. Because the patient's perception of her occlusion remained heightened, she has since adjusted her mandibular occlusal splint – made for her by one of the authors – by adding targeted occlusal contacts in the form of a few small cellulose "underlayings". The patient did this by way of self-therapy, without consulting a

dentist. According to the patient, this allowed her to return to a normal everyday life that was no longer dominated by her occlusion. This example is not intended to establish this type of splint therapy as the preferred form for patients with OD, but serves as evidence that even partial successes can often signify the start of a patient's return to normal life.

The recommended therapy approach favored by many authors is cognitive behavioral therapy that aims to change a patient's perception of his or her occlusal contacts [2, 6, 9, 13, 18, 21, 26]. As already mentioned above, any comorbid psychological factors should be treated by the appropriate specialist. In Germany, dentists do not have the power to refer; as a result, cognitive behavioral therapy cannot be initiated by the dentist, but is instead usually arranged by the family physician. In spite of this, it is important for the dentist to provide the patient with an explanation of the findings made.

With regard to possible pharmacotherapy, everyday clinical practice suggests that many patients reject this form of therapy. This is due to the lack of a specific medication for OD, and the fact that patients do not wish to use the antidepressant or neuroleptic drugs often used in this context.

Similar to the treatment of chronic pain, the recommendation of physical activity can be a promising therapeutic option for treating OD. Depending on the physical constitution of the patient, possible suggestions include forest walks, dancing, yoga, or endurance sports. Group physical activities can also be used as a way to re-engage socially.

Outcome/Conclusion

"I thought you were an expert, but apparently you don't know what you're doing either!"

The symptoms of OD are indicative of functional disease. For this reason, OD cannot be treated effectively by means of dental interventions, but instead requires further specialized medical care. If OD is suspected, a differential diagnosis should be performed to distinguish it from occlusal disease. In addition, it is advisable to use validated questionnaires (Table 2) to screen for non-specific risk factors, so as to better capture the disease profile. From clinical experience, the authors have found that a large proportion of patients determinedly evade the therapeutic efforts presented in this article, and continue to search for a supposed specialist who can – as the patient sees it – comprehensively solve their problem by means of invasive therapy methods.

Conflicts of interest

The authors declare that there is no conflict of interest as defined by the guidelines of the International Committee of Medical Journal Editors.

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