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CRANIOFACIAL SKELETON (WE ROBERTS, SECTION EDITOR)

Part II: Temporomandibular Joint (TMJ)—Regeneration, Degeneration, and Adaptation

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Abstract

Purpose of Review Elucidate temporomandibular joint (TMJ) development and pathophysiology relative to regeneration, degeneration, and adaption.

Recent Findings The pharyngeal arch produces a highly conserved stomatognathic system that supports airway and masticatory function. An induced subperiosteal layer of fibrocartilage cushions TMJ functional and parafunctional loads. If the fibrocartilage disc is present, a fractured mandibular condyle (MC) regenerates near the eminence of the fossa via a blastema emanating from the medial periosteal surface of the ramus. TMJ degenerative joint disease (DJD) is a relatively painless osteoarthrosis, resulting in extensive sclerosis, disc destruction, and lytic lesions. Facial form and symmetry may be affected, but the residual bone is vital because distraction continues to lengthen the MC with anabolic bone modeling. Extensive TMJ adaptive, healing, and regenerative potential maintains optimal, life support functions over a lifetime.

Summary Unique aspects of TMJ development, function, and pathophysiology may be useful for innovative management of other joints.

Keywords Fibrocartilage · TMJ regeneration · Airway · Deciduous first molars · Condylar hyperplasia · Osteoarthrosis · TMD · Adaptation · Conserved traits · Propagation · Condylar distraction

Introduction

The mandible is a heavily loaded, highly mobile facial bone that articulates with the temporal bone at the base of the cranium. Temporomandibular joints (TMJs) are bilateral synovial articulations that facilitate a broad range of essential life-support functions and social interactions. Mastication, airway, communication, and facial form contribute to mating success. Part I of the current review addressed TMJ developmental physiology in preparation for the current assessment of regeneration, pathophysiology, and adaptation mechanisms.

This article is part of the Topical Collection on *Craniofacial Skeleton*

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Airway Development

The oropharyngeal complex (mandible, dentition, and pharynx) begins with pharyngeal arch development, involving head mesoderm, foregut endoderm, and neural crest cells. The mandible is formed from cranial neural crest cells via a hierarchy of gene regulation modules that govern formation, migration, and differentiation [1]. The mandible is a component of the pharyngeal arch which plays a lifelong role in development and maintenance of a patent airway. Hypoxia sensors [2] and respiratory reflexes [3] are important regulators of respiration that evolve simultaneously.

Airway defects are potentially lethal craniofacial anomalies. Chromosomal anomalies near the SOX9 gene [4] are associated with Pierre Robin syndrome (PRS), a mandibular developmental anomaly with a prevalence of 1 in 8500 live births.

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Relevant features of PRS are a severely underdeveloped mandible that is often associated with cleft palate. The latter occurs because the small mandible traps the tongue high in the developing nasal cavity, thereby blocking the elevation and fusion of the palatal shelves [5]. If the postnatal airway and feeding problems are successfully managed, either conservatively and/or with surgery [6], the mandible is capable of substantial growth and adaptation to help position the tongue anteriorly to maintain a patent airway. The development and successful management of this life-threatening anomaly demonstrates the importance of the mandible and TMJ for anterior posturing of the tongue.

Hemifacial Microsomia

Hemifacial microsomia (HM) is a relatively common (1 in 2000 live births) unilateral deficiency of the TMJ, ear, and associated structures. This anomaly falls into the oculoauriculovertebral spectrum (OAVS) [7]. Most cases of HM are genetically and phenotypically heterogeneous, so the precise etiology of the syndrome is unclear. Most studies suggest a unilateral vascular deficiency during development. There are recurring patterns of environmental factors, and family case reports of preauricular appendages, microtia, mandibular hypoplasia, and facial asymmetry. Chromosomal abnormalities and candidate gene studies suggest a multifactorial inheritance model [7]. Despite severe facial disfigurement and compromised oropharyngeal function, TMJ growth and adaptability render a surprisingly high health-related quality of life for affected children [8].

TMJ mobility has an important lifelong role in posturing of the mandible to support optimal masticatory and pharyngeal function. Maintaining a patent airway is challenging because of the increasing prevalence of negative environmental factors: atmospheric pollution [9], allergies [10], inflammatory disorders [11], and obesity [12].

Obstructive Sleep Apnea

Obstructive sleep apnea (OSA) is an increasingly serious health problem [13, 14], with a prevalence of 6.4% in women and 13.8% in men [15]. Continuous positive air pressure (CPAP) therapy is the gold standard for OSA treatment, but many patients are refractive to regular use of these unattractive devices. Other treatment options are medication [16], orthognathic surgical advancement of the jaws [14], hypoglossal nerve stimulation [17], or dental mandibular advancing devices (MADs) [18]. Therapeutic repositioning of the mandible requires TMJ adaptation. MADs are usually attached to teeth so they can cause substantial orthodontic problems, resulting in an uncomfortable and unattractive occlusion. Nocturnal wear of a MAD does not appreciably increase TMJ dysfunction, but OSA patients with preexisting crepitus or other disorders may fail to cooperate in regularly wearing the appliance [19].

Biomechanics

The critical teeth for the biomechanics of facial development are the deciduous first molars (Ds), which emerge to provide the initial posterior stops in occlusion at ~ 16 months of age [20]. Equal and opposite forces of posterior occlusion are evenly distributed between the cranium and mandible. Primary cartilaginous growth centers of the jaws [21, 22] revert to skeletal biomechanics to drive jaw growth and adaptation, via secondary growth mechanisms (condyle, fossa, facial sutures, and subperiosteal surfaces). The mandibular midline synchondrosis fuses to permit unilateral chewing [23], which is the primary mechanism of human mastication. The posterior palatal synchondrosis reverts to a midpalatal suture, which is a secondary growth site [24]. A critical event in facial and masticatory development is the emergence of the Ds, which increases the vertical dimension of occlusion (VDO) to help maintain a patent airway and establish full masticatory capability [20]. Although primarily associated with prenatal, primary growth mechanisms, IGF-2 continues to play an important role after birth [25]. Achieving posterior occlusal function (Ds eruption at ~ 16 months) occurs at about the same time the brain-pituitary-liver axis switches from IGF-2 to IGF-1. The latter is the principal endocrine factor for growth, development, and lifelong stimulus of the musculoskeletal system. There are no specific reports on relative IGF-2 and IGF-1 levels during the first 16 months after birth. It is hypothesized that the biomechanics of posterior occlusal function is the critical event in converting facial development from primary growth centers (IGF-1 control) to secondary growth sites (IGF-2 control).

Primary molar development is specified early in the development of the oropharyngeal complex [26], and it is a highly conserved genetic trait. Even when the Ds are deformed, they erupt and perform their critical mission of establishing the centric stops in posterior occlusion [27]. Full masticatory function promotes the development of the TMJ, particularly the articular eminence [28]. The latter achieves half of its adult dimension by 2 years of age [29], which is only 8 months after the Ds occlude. It is clear that the adult-like form of the TMJ is secondary to posterior masticatory function.

There are no published reports of congenitally missing first deciduous molars (Ds). Agenesis is common in the permanent dentition, probably because the affected teeth serve no critical life-support role, but congenital absence of deciduous (primary) teeth is rare [30]. A single child presented with multiple missing deciduous teeth (oligodontia) [31], but the Ds were present and effectively established the posterior

VDO. Collectively, these data indicate that the timely eruption of the Ds to expand occlusal function has important survival value for the human species. The authors are aware of only one patient with congenitally missing Ds. She was born with anhidrotic ectodermal dysplasia and raised in an affluent nation. At age 10, the patient presented with a complex acquired malocclusion. Correction of the severely decreased VDO and retrognathic mandible will probably require orthognathic surgery. The survival of a child, with such a severe functional disorder, would be unlikely in a primitive setting.

Regeneration and Adaptation

Adaptation to malocclusion, spontaneous and/or acquired, maintains adequate mastication, facial esthetics, and airway maintenance for achieving propagation. The TMJ adaptability of the stomatognathic apparatus is exposed to genetic and environmental challenges. Spontaneous mutations [32] affecting fundamental developmental processes may produce skeletal malocclusions that are lethal in a primitive setting [33, 34]. In affluent society, medical and caregiver support can provide adequate airway development and nutritional needs. Individuals with debilitating malocclusions survive, and their genes enter the pool, if reproduction is achieved [32].

Adult newts can regenerate the jaws and the dentition after an amputation distal to the TMJ. Blastema cells are derived primarily from muscle (mandible) and cartilage (maxilla) [35, 36, 37••, 38]. Both larval *A. maculatum* and adult newt regenerate alveolar cartilage and bone across defects created by excising a quarter to a half of the mandible [39]. Whether the TMJ itself can regenerate in these animals is not known. There are numerous reports of condylar regeneration in mammals. The fibrous articular layer is regenerated in marmosets (small primates) after making a full-thickness defect in the condylar head without damaging the articular disc [40]. The mandible has known regenerative potential [41, 42], including regeneration of the mandibular ramus and condyle after resection and stabilization of the remaining mandible with a titanium mesh [43–45]. The source of cells for repair of these defects is the periosteum [46]. Condylar regeneration is enhanced by the use of a functional appliance that propels the mandible anteriorly [47].

Hayashi et al. [48••] showed that the presence of the disc is necessary for condylar regeneration. The disc may be an essential environmental cofactor for periosteal activation. Rats in which the disc was excised in conjunction with a condylectomy failed to regenerate the condyle, even in the presence of a functional appliance. These data indicate the disc plays a direct role in inducing condylar regeneration [48••], but the relationship is not reciprocal because a damaged or missing disc fails to regenerate in the presence of the intact condyle. It is unknown if disc regeneration is possible in the regenerative environment associated with intracapsular fracture. Regenerated condyles have hinge and translation function (Fig. 1), suggesting that the original disc or disc-like connective tissue separates the condyle from the temporal bone.

Traumatized disc tissue can be induced to regenerate by implanting a collagen sponge into the defect [49]. The collagen sponge stimulates ingression of surrounding cells into the void and the missing tissue (fibrocartilage) is regenerated. Condylar fibrocartilage and adjacent bone can regenerate after fracture or excision from a healthy joint, but no regeneration occurs in the presence of DJD probably because the disc is destroyed. This lack of regeneration and adaptive potential has spurred research for restoring the TMJ with tissue-engineered implants involving osteoconductive and osteoinductive scaffolds alone, or with cells and growth factors [49, 50]. Complete biomimetic joints have been constructed for the distal mandible of selected patients [51, 52], but routine implantation of prosthetic devices is not imminent.

Condylar Fracture

The healing blastema for a fractured mandibular condyle emanates from the periosteum on the medial aspect of the ramus [53••]. This process is similar to the fetal origin of the condylar process [20]. Three-dimensional (3D) imaging with cone-beam computed tomography (CBCT) shows the sagittal and frontal views of a regenerated right condyle in a 48-year-old male, 18 months after it was fractured in a fall (Fig. 1). Note that the regenerated condyle typically occludes on the anterior eminence of the temporal fossa, probably because this is the position of the disc and fractured condylar head when it is displaced by the pull of the lateral pterygoid muscle (Fig. 1). Assuming condylar regeneration is a genetically controlled periosteal mechanism, upregulation of the gene(s) involved could be helpful for regeneration and repair of other joints.

Clinical management of TMJ condylar fractures is controversial [54]. Most clinicians prefer spontaneous healing for high condylar and intracapsular fractures, particularly in children and adolescents [53••]. A manipulative but closed management approach for intracapsular fractures is the use of an occlusal stent (orthotic) with elastic traction between the arches [55]. TMJ surgery may be complicated by avascular necrosis of the proximal segment [56], and the preauricular approach poses a risk for facial nerve injury [57]. However, good surgical results are reported for a modified open reduction and fixation procedure [58]. Lateral displacement of the fractured stump of the

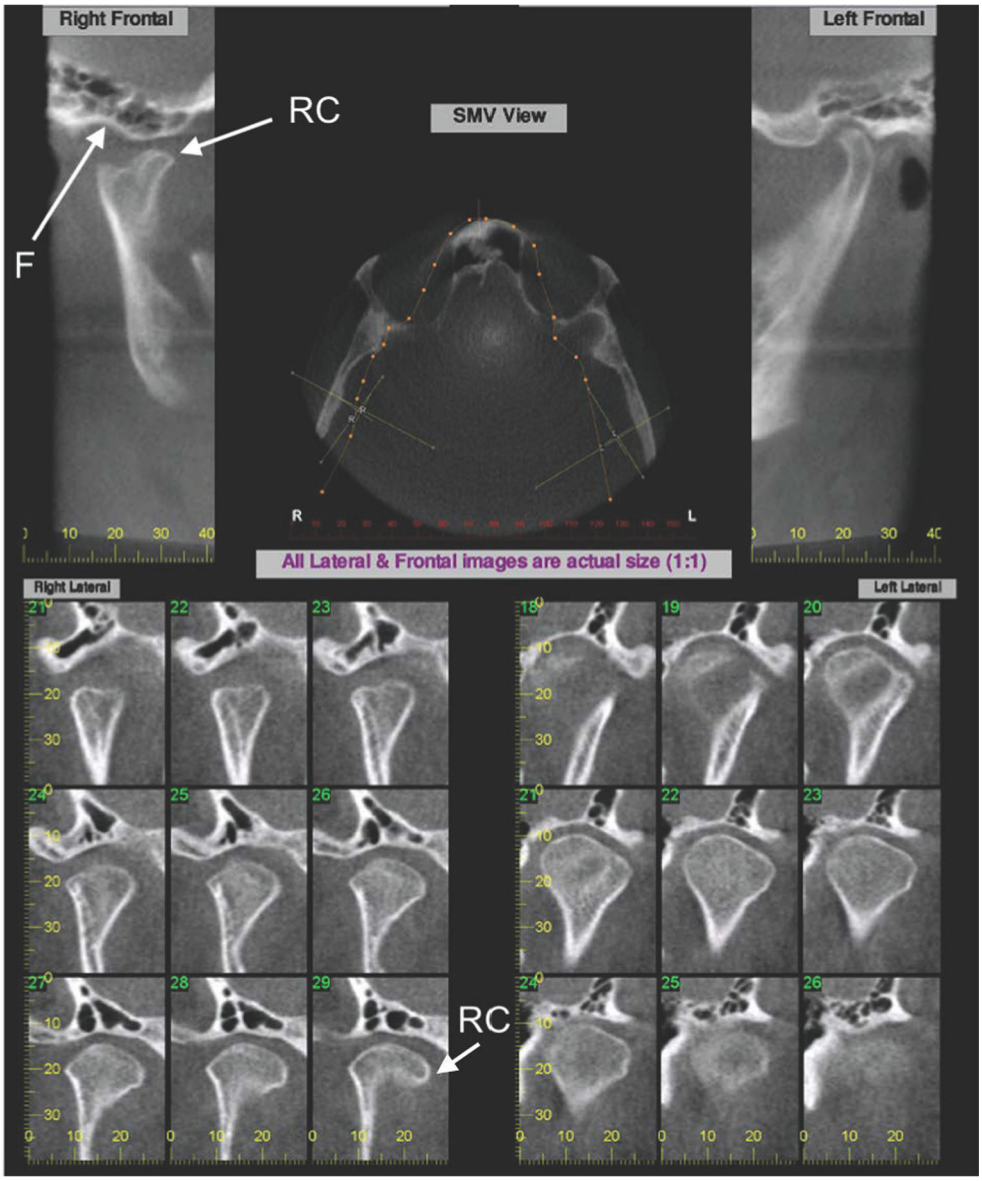

Fig. 1 A TMJ series from a CBCT scan shows the regenerated condyle (RC) in the sagittal (upper views) and frontal (lower views) planes relative to the temporal fossa (F) in a 48-year-old male who fell and fractured the right mandibular condyle 18 months previously. Note the blastema for the regenerating condyle (RC) grew anteriorly and medially from the internal surface of the ascending ramus

ramus is a strong predictor of ankylosis, so surgical intervention is indicated [59].

Mandibular Condylar Hyperplasia

Mandibular condylar hyperplasia (MCH), also deemed hypercondylia [60], primarily affects women (64%) and often results in facial asymmetry and TMJ dysfunction (Fig. 2) [62]. High condylectomy (HC) is performed when the condition is progressive. Although the literature shows large variations in etiology, diagnostic methods, and timing of the intervention, the overall conclusion is that HC is a suitable surgical method for correcting MCH [63]. The hyperplasia often "burns-out" (ceases to progress), so patients with modest asymmetry can be conservatively treated with orthodontics and restorative dentistry (Fig. 2) [61], but severe problems may require orthognathic surgery.

Temporomandibular Disorders

A comprehensive review of temporomandibular disorders (TMDs) is beyond the present scope because many clinical problems have strong psychogenic overtones [64], particularly relative to anxiety [65]. However, mechanical disorders are relevant since they are compensated by skeletal adaption and may elicit ear symptoms. In modern society, processed (soft) diets result in hypofunction of the stomatognathic system, and increasingly stressful lifestyles render an increasing fraction of the population susceptible to TMD [66]. Animal studies have confirmed that hypofunctional TMJs are more susceptible to

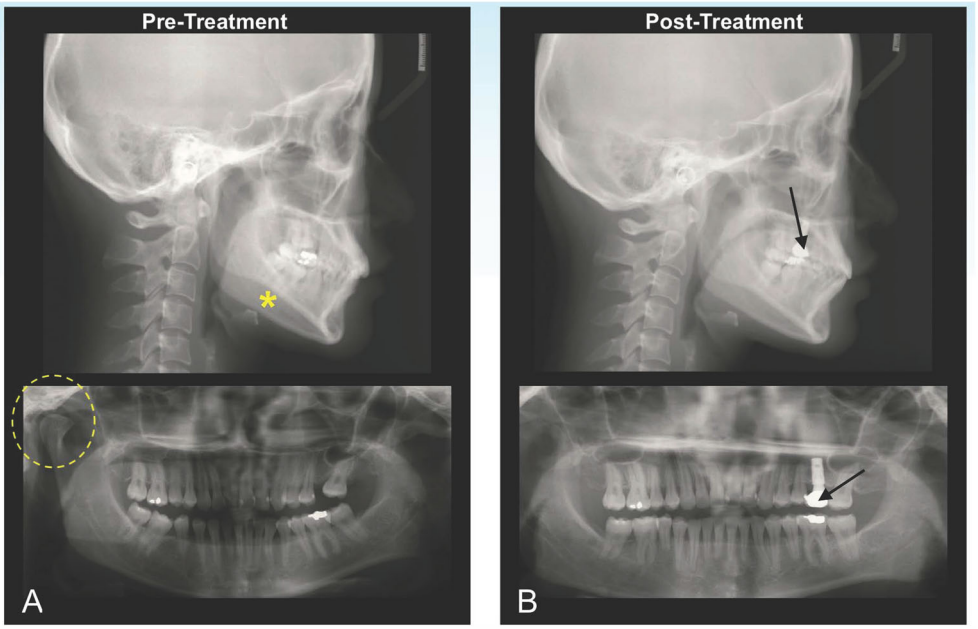

Fig. 2 **a** Pre-treatment of a condylar hyperplasia on the right side (dashed circle) produced an ~ 1.5-cm asymmetry between the right and left mandibular planes of the mandible (*) of a 30-year-old female. Reproduced with permission from [61]. **b** The hyperplasia was no longer progressive ("burned out") so the occlusal asymmetry was managed conservatively with orthodontics and an implant-supported prosthesis (arrow). Note that the asymmetry in the mandibular planes is reduced, but not resolved. Reproduced with permission from [61]

degeneration when loads on the jaws are increased [67]. Early extraction (< 8 years of age) of permanent lower first molars may produce acquired malocclusions with asymmetry and/or functional retrusion of the mandible that are associated with TMD [68].

TMJ development is distinct from other synovial joints. It is closely associated with ear development [20], so the signs and symptoms of TMD may include otology problems [66, 69]. Pruritus, otalgia, and aural fullness are the most common symptoms. Ear disorders are significantly correlated with female gender, TMD severity, and frequency of symptoms [69].

Advancement and posterior rotation of the mandible opens the pharyngeal airway, as well as providing intraoral access for dental and medical procedures. This is a well-known procedure in emergency medicine, anesthesia, and basic life support procedures. Placing an endotracheal tube for general anesthesia or extracting third molars may require very wide opening of the mouth and result in pressure on the teeth [70]. The TMJ is highly mobile and usually tolerates wide opening, but when the joint is unstable or anatomical limits are exceeded, the condyle may be displaced off the disc producing intracapsular damage. TMJ dysfunction after third molar extraction has a reported prevalence of 23% for all TMD patients in the age range of 12–20 years [71].

Management of TMJ Problems

Disc displacement (internal derangement) occurs when the condyle clicks off the disc during opening and/or closing of the mandible. If the disc is displaced anterior to the condyle, a closed lock may develop because the condyle cannot translate anteriorly. An acute closed lock can be reduced by pressing inferiorly in the retromolar area of the mandible, but only ~ 18% of patients completely recover normal function with no subsequent signs or symptoms of TMD. Reduction is least successful for advanced internal derangements with deformed discs [72]. Minimally invasive treatment for anterior disc displacement is successful in increasing mouth opening and reducing pain for most patients. However, TMJ pain and joint effusion are significantly related, inflammatory events that are often refractory to conservative treatment [73]. Fortunately, inflammatory TMJ degeneration is relatively rare.

Biomechanics may contribute to chronically clicking joints because they show fewer coincident stress-field paths and flatter stress-fields than controls during jaw opening and closing [74]. Clicks may be associated with unilateral crossbite, but orthodontic correction does not decrease the prevalence of clicks at 10-year follow-up [75]. Thus, disc damage incurred by a malocclusion may be irreversible with conservative measures. If a disc is damaged, manual manipulation is unlikely to effectively correct the derangement. On the other hand, relatively normal discs may reduce (click back into place), but others remain displaced (without reduction) during jaw movement. Occasional clicking with no pain or locking is usually well tolerated. Displaced discs that do not reduce may result in restricted opening (chronic closed lock) or an acquired overjet ("buck teeth") when the mandible is closed [76]. Disc displacement in female orthodontic patients is associated with altered skeletal morphology: decreased mandibular ramal and body length, in addition to more posterior positioning of the mandible, which is a Class II (retrognathic) malocclusion

[77]. Furthermore, TMD risk is associated with pneumonia, asthma, allergies, headache, general joint hypermobility, orofacial trauma, rheumatism, and orthodontic treatment [78].

Arthroscopic surgery for anterior displacement of the disc is successful at the initial 6-month recall [79], but poses increased risk of intra-articular adhesions in the long term [80]. Disc repositioning often results in malocclusion and may be unstable [81]. Partial or total prosthetic replacement of the TMJ and mandible may be the treatment of choice for patients with severe chronic pain [82], trauma, or cancer [83]. Previous TMJ prosthetic procedures were prone to complications such as functional overload [84] or persistent infections [85], but outcomes are expected to improve based on improvements in design and biomaterials [86]. Tissue engineering strategies to replace discs is emerging because TMD affects up to 25% of the population and there are often limited treatment options when the disc is badly damaged or destroyed [87].

TMJ Degeneration

Congenital disc degeneration is associated with human SHOX [88]. A developmental delay in condyle mineralization results in subsequent degeneration of condylar fibrocartilage, that is associated with the discoidin domain receptor 2 [89]. Loading a mineralized condyle results in development of an underlying fibrocartilage, but TMJ overloads contribute to degeneration [90], particularly with a history of hypofunction [67]. Within physiologic limits, the TMJ is capable of adapting to changes in functional loading [91], but habitual parafunction (bruxism and clenching) may result in TMD [66, 92, 93]. In a large university clinic sample ($n = 4204$), clenching and/or grinding was reported by 26.5% of TMD patients [94]. Frequent bone changes in 283 patients with degenerative joint disease (DJD) were condylar flattening (77.4%) and erosion (59.7%) [95]. Most DJD patients report little or no pain, but there is a positive correlation with erosion, and a negative correlation with osteophytes. A lower fractal dimension (decreased complexity) of the trabecular bone is noted in condyles of TMD patients with erosive and sclerotic changes [96]. Magnetic resonance imaging (MRI) evaluation reveals that disc displacement, joint effusion, and degenerative changes are relatively common in TMD patients [97]. The kinematics of TMJ motion in response to physiologic loading is best assessed with dynamic MRI [98].

Osteoarthrosis

Differentiating TMJ *osteoarthrosis* [99] from the *osteoarthritis* (OA) [100] of other joints is illustrated with a clinical case. Osteoarthritis in long bone joints is inflammatory and extremely painful [100], but chronic TMJ degeneration is rarely painful, probably because there is less inflammation [54, 94, 101]. The source and the site of the pain are coincident for OA, but TMJ pain is usually refereed from inflamed muscle fascia at a distant site [54, 102]. Trigger points in inflamed muscle are associated with painful spasms, which may be related to elevated muscle cytokines [103].

On the right side (Fig. 3), DJD produced severe sclerosis (hardening or induration) of trabecular bone extending from the superior surface of the temporal bone (brain interface) to the ascending ramus of the mandible. The pathologic mechanism for this form of trabecular sclerosis is anabolic bone modeling (formation) to strengthen overloaded trabeculae. In the absence of normal remodeling, bone thickness exceeds the diffusion limit for living bone, which is 100 μm from nearest blood supply [105]. The internal bone core dies and hypermineralizes, so the enlarged trabeculae appear more radiodense. Microdamage accumulates and if the overloaded trabeculae fracture, a bone resorptive response produces subcondylar radiolucent lytic lesions that are defined as radiologic "cysts" [106] or "pseudocysts" [95]. Little or no joint space is evidence of disc derangement or destruction (Fig. 3).

The radiographic picture on the left is relatively normal except for a single sclerotic lesion about 3 mm in diameter adjacent to a discontinuity in the subchondral cortex (Fig. 3). The diagnosis is severe chronic osteoarthrosis on the right, with a more incipient degenerative lesion on the left. Surgical intervention is rarely indicated [95], but there are no documented reports for conservative management of progressive DJD. In the absence of an evidence base, clinicians must rely on basic science principles [104, 105, 107]. Conservative treatment for the clenching, associated with the progressive degeneration, is achieved with a removable oral appliance (Hawley bite-plate) worn at night [104]. The bite-plate is constructed with premature occlusion of the lower incisors to prevent the molars from contacting. A periodontal ligament (PDL) polysynaptic reflex [108, 109] inhibits the forceful contraction of the powerful mandibular elevator muscles [110].

International arthroplasty guidelines vary but advanced joint disease (osteoarthritis of the hip or knee) is usually a strong indication for prosthetic replacement [100]. DJD of the mandibular condyle is rarely debilitating so it is managed conservatively, if treated at all (Fig. 3). If DJD produces facial asymmetry, the problem can be corrected with a sagittal split osteotomy [104]. The resistance to severe debilitation under adverse environmental conditions is a highly conserved trait of the TMJ. Compromised long bone function was certainly disabling in primitive society, but apparently not as life-threatening as a loss of mandibular function (airway and feeding).

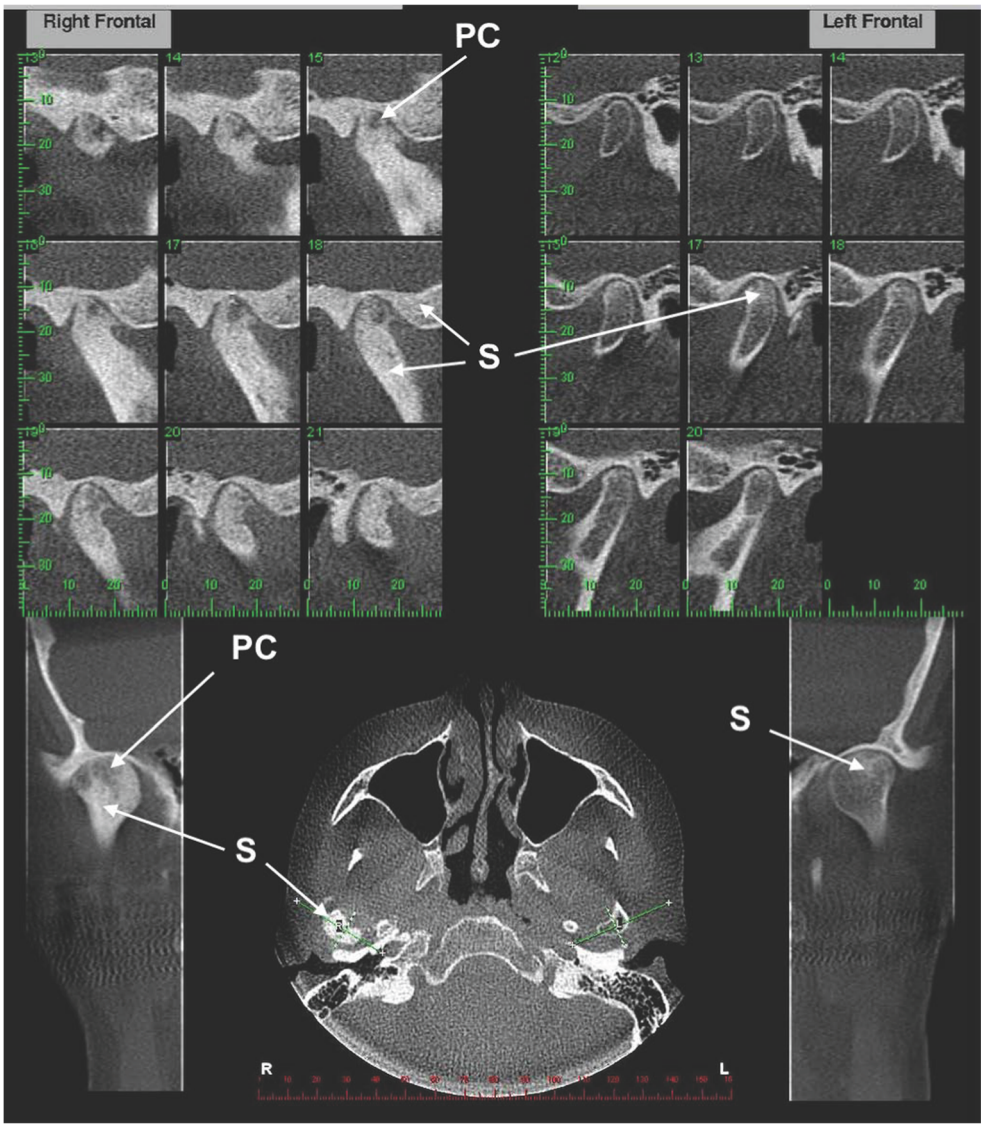

Fig. 3 A 64-year-old female presented with a 45-year history of bilateral closed lock and TMD. A TMJ series from a CBCT documents advanced DJD on the right side with sclerosis (S) with pseudocysts (internal radiolucent cavities) (PS). On the left side, there is an ~ 3 mm degenerative lesion with sclerosis (S) beneath a break in the subarticular cortex on the superior aspect of the condyle (see text for details). This illustration was originally published in [104]. *Reproduced with permission from Elsevier*

Distraction of a Degenerated TMJ

A 72-year-old female patient presented with unilateral symptoms of the left side: uncomfortable occlusion due to an excessive curve of Spee (curvature of the mandibular occlusal plane), decreased TMJ joint space (disc destruction), and condylar degeneration (Fig. 4a). Orthopedic correction was inferior distraction of the left condyle ~ 4 mm over 9 months via asymmetric adjustment of an acrylic occlusal orthotic. Removing posterior centric stops in occlusion on the right side produced a clockwise functional shift of the mandible in the frontal plane to create vertical occlusal space in the left posterior to correct the plane of occlusion (Fig. 4b). An implant-supported crown was used to restore the missing lower left first molar at the desired VDO, and the entire occlusion was aligned accordingly. Comparison of the original panoramic view (Fig. 4a) to the corresponding image after the condylar distraction (Fig. 4b) shows the orthopedic effects in the sagittal plane. CBCT evaluation (not shown) documented that the lytic lesions on the affected side were decreased in size or resolved following distraction. After a symmetric occlusion was restored (Fig. 4b), nocturnal clenching (parafunction) was controlled with a neurologic orthotic (Hawley bite-plate) [104]. Follow-up evaluation with CBCT 2 years later (not shown) documented a stable result, and 8 years later, there was no recurrence of TMD signs or symptoms.

Pathophysiology

The mandible and its temporal articulation are secondary skeletal structures formed after the initial nerve patterning for cartilage anlage and intramembranous ossification centers in other parts of the body [20]. This unique developmental pattern is

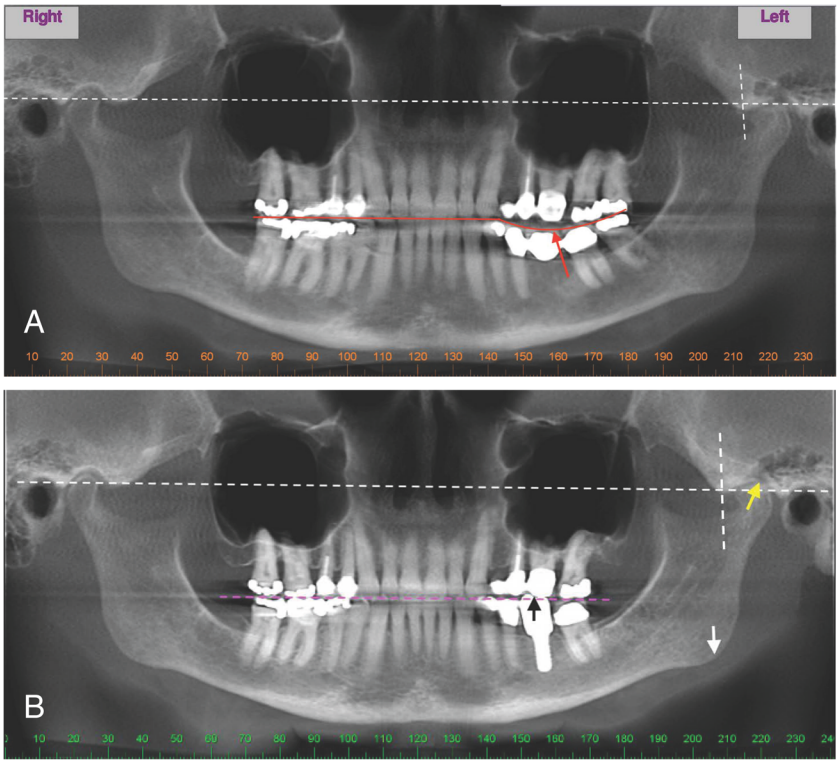

Fig. 4 **a** A 72-year-old female with a history of nocturnal clenching presented with a degenerated left condyle associated with an asymmetric occlusal plane (red line) in the left molar area (arrow). The lower left first molar was missing, and the three-unit fixed prosthesis that restored the edentulous space was constructed with a deep curve of Spee. Note that the superior margin of the degenerated left condyle is along a dashed line through superior margins of each internal auditory meatus (interporion line). The anterior margin of the left condyle is anteriorly positioned near a perpendicular dashed line through the articular eminence. *Reproduced with permission from Elsevier*. **b** The post-treatment panoramic radiograph shows the left condyle was distracted in the direction of the white arrow pointed inferiorly. With the vertical space provided by ~4 mm of condylar distraction, the asymmetric occlusal plane was corrected with an implant-supported prosthesis (black arrow pointed superiorly). Note the left condyle is positioned ~ 3 mm superior to the interporion line, and distally positioned in the temporal fossa (yellow arrow) about 5 mm distal to the perpendicular line through the eminence. See text for details. *Reproduced with permission from Elsevier*

associated with a less painful response to trauma, functional overload, and parafunction (Fig. 3). The difference in pathophysiology may relate to fewer pain receptors in TMJ-related tissues and/or a joint structure that is less prone to inflammation. The TMJ is the only joint formed with three separate condensations of mesenchyme [20], a developmental process that results in a disc and both articular surfaces cushioned with fibrocartilage. This unique developmental physiology results in a joint capable of resisting high loads and adapting to DJD with minimal painful inflammation. A degenerated TMJ can increase in length when it is mechanically distracted (Fig. 4). Despite the bizarre bone morphology associated with DJD (Fig. 3), the articular surface remains a vital modified periosteum that is capable of adaptation to maintain optimal function.

Predisposition to DJD may involve deficiencies in forming and maintaining fibrocartilage. Bone tissue of a degenerated TMJ remains vital because the condyle can be lengthened, but there is no evidence that fibrocartilage is regenerated. Correcting joint overloads decreases internal bone pathology (pseudocysts and sclerosis), but the irregular shape to the degenerated condyle remains (Fig. 4b).

Conclusion

The TMJ provides adequate, relatively pain-free mobility for the mandible to achieve its critical life support functions under a variety of adverse environmental conditions. Better understanding of the pathophysiology of the TMJ may provide insights for managing disorders in other joints.

Compliance with Ethical Standards

Conflict of Interest David Stocum declares no conflict of interest. W.E. Roberts is a section editor of the Craniofacial Section of Current Osteoporosis Reports, but this paper was reviewed by editor in chief David Burr.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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