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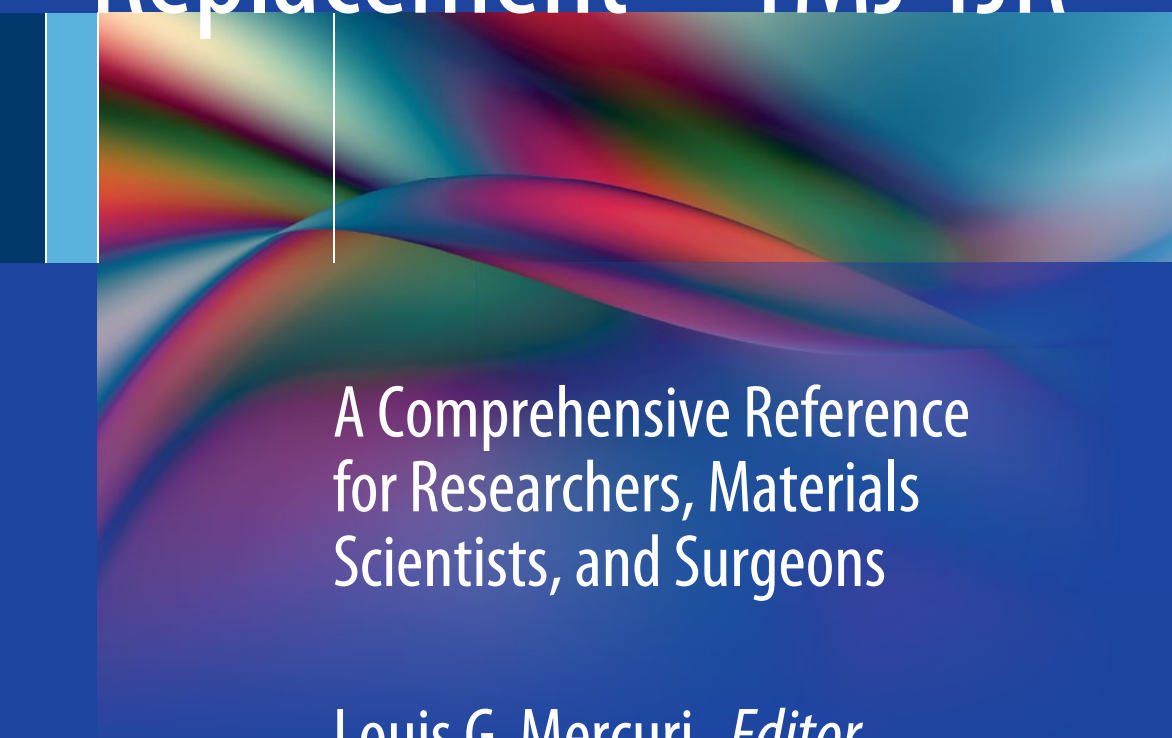
# Temporomandibular Joint Total Joint Replacement – TMJ TJR



A Comprehensive Reference for Researchers, Materials Scientists, and Surgeons

Louis G. Mercuri *Editor*

*Foreword by*
Joshua J. Jacobs, M.D.

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# Temporomandibular Joint Total Joint Replacement – TMJ TJR

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*To my mentors Gustav Kruger, Daniel Laskin, S. Elmer Bear, and notably Larry Peterson who cultivated my interest as a student in oral and maxillofacial surgery; all the students and residents who consistently pushed me to look for answers; and to my research colleagues who keep me asking "why."*
*Most importantly, to my wife Joan, whose love, encouragement, and understanding have been a constant source of strength and determination in my career.*

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# Foreword

Joint replacement has been one of the great success stories of modern medicine. Lower extremity joint replacement, in particular, has revolutionized the treatment of end-stage diseases involving the hip and knee, and total hip and total knee arthroplasty are among the most commonly performed and successful procedures. In the USA, alone over one million hip and knee replacements are performed on an annual basis. For these large lower extremity joint replacements, survivorships in excess of 90 % at 10 years are typical and will likely be surpassed with improvements in surgical technique, implant materials, and implant design.

While hip and knee arthroplasty are considered to be very reliable and effective procedures, this is not the case for other joints such as the ankle, elbow, and wrist where the anatomical and biomechanical milieu may be more complicated. This is also the case for the temporomandibular joint (TMJ). Although temporomandibular joint disorders are not nearly as common as osteoarthritis of the hip and knee, there is a large patient population that is affected, often leading to considerable disability. In the appropriate patient population, TMJ arthroplasty can be a very effective treatment, and like other joint arthroplasties, restoration of function, maintenance of fixation, and minimization of implant and periprosthetic bone and soft tissue degradation are key in determining the ultimate success of this intervention.

In this volume, the authors have provided a valuable addition to the extant literature by summarizing the state of the art and science in TMJ arthroplasty. There are many scientific advances summarized in this book that are relevant to understanding of the performance of TMJ arthroplasty and also provide a pathway to improve the ultimate outcomes of this intervention. This book is recommended to biomaterials scientists either in training or in practice who are working in the area of TMJ arthroplasty as well as to clinicians either in training or in practice who care for patients with TMJ disorders. Kudos go to the authors for their scholarly contributions to this important topic.

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# Preface

The practice of reconstructive orthopedic surgery would be unthinkable and impossible without the availability of alloplastic joint replacement devices. In the 1960s, posed with the problem that resection arthroplasty was an uncertain procedure with recurrent deformity and limited motion as common complications, Sir John Charnley (Fig. 1) developed a successful low-friction total alloplastic joint replacement device. Since that time, with the evolution of surgical techniques, implant materials, and designs, excellent long-term function and quality-of-life improvement results have been reported along with device survival rates exceeding 90 % after 10 years.

Temporomandibular joint (TMJ) reconstruction presents unique problems because of the integral and complex roles the TMJ plays in establishing and maintaining proper form and function within the stomatognathic system. The TMJ not only acts as a secondary growth center for the mandible in prepuberty but also is essential to the functions of mastication, speech, airway support, and deglutition in both child and adulthood.

Alloplastic materials have been employed for decades in the management of primary and secondary TMJ pathology. Prior to the early to mid-1980s, the primary reasons for TMJ reconstruction were the management of developmental maxillofacial deformities, ankylosis, severe inflammatory joint disease, or TMJ replacement after ablative tumor surgery or trauma. Most of these early reports of the use of alloplastic material were single cases with no long-term follow-up; hence, complications were often unreported.

Thereafter, along with these form and function challenges, there arose a group of patients who presented requiring TMJ reconstruction having previously undergone multiple failed TMJ surgical procedures. Many of these patients' TMJs were anatomically distorted and functionless secondary to the failure of interpositional materials such as Proplast—Teflon (Vitek, Houston, TX) and/or silicone rubber (Dow-Corning-Wright, Arlington, TX). Early in the 1990s, it was discovered that failure of these materials had caused wear-related foreign body giant cell reactions resulting in significant end-stage TMJ anatomical architectural changes necessitating total joint replacement (TJR).

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As the number of these unfortunate patients grew (an estimated 26,000 Proplast-Teflon containing TMJ devices had been implanted in the USA between its introduction in the early 1980s and 1992), interested reconstructive surgeons began developing goals to reach a physiologically reasonable, biologically rational, and technically achievable TMJ TJR outcome taking into consideration not only TMJ form and function but also these patients' neurological and psychological needs. Utilizing time-tested orthopedic technologic and materials science advances, custom and stock TMJ TJR devices were developed, approved, and manufactured to manage these and future end-stage TMJ disease cases. Furthermore, modern TMJ TJR surgeons also realized that due to the complex nature of joint anatomical and related masticatory muscle functional relationships, it was unreasonable to expect that a reconstructed TMJ could be returned to "normal" premorbid function. There will always be some functional disability involved with any reconstructed TMJ.

In the multiple-operated, anatomically distorted patients, chronic neuropathic centrally mediated pain will be a major component of their disability. Therefore, it is important for both surgeon and patient to understand that the primary goal of any type of TMJ reconstruction is the restoration of objective mandibular form and function. Any subjective pain relief gained must only be considered as of secondary benefit.

Based on evidence from the orthopedic, biomedical engineering, materials science, and oral and maxillofacial surgery literature, and the expertise of the contributing authors, this book discusses the role TMJ TJR can play as a salvage device in the management of patients with severe, debilitating end-stage TMJ anatomical disorders.

The biomechanics and biomaterials chapters present the basics of TMJ biomechanics and the rationale for the biomaterials used in the development and manufacture of modern TMJ TJR devices. A chronological historical review provides readers with information on the successes and failures associated with TMJ alloplastic devices so that, in the future, the successes can be built upon, and the failures avoided.

**Fig. 1** Professor Sir John Charnley, FRS. 1911–1982 (Wroblewski BM. Professor Sir John Carnley (1911–1982). Rheumatology. 2002. 41" 824–5

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In the following chapters, the clinical indications and contraindications, surgical techniques, and outcomes for custom and stock TMJ TJR devices are presented, together with the diagnosis, avoidance, and management protocols for common TMJ TJR device complications and failure.

In the tribocorrosion chapter, the role of this latest advance in materials science analysis for the study of functional material wear and the peri-articular tissue responses will be discussed. In the following chapter, the complex, controversial, and vexing issue of alloplastic TJR material hypersensitivity will be considered in detail.

Finally, the potential for the development and use of bioengineered tissue in the design and production of viable TMJ TJR replacement devices will be presented and considered.

This text is designed to be the first comprehensive reference of its kind not only for reconstructive surgeons and materials scientists but also for all TMJ researchers as they seek to improve the management of end-stage TMJ disease for patients.

Chicago, IL, USA Louis G. Mercuri, DDS, MS

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# Part I
# Biomechanics and Biomaterials

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# Chapter 1
# TMJ Biomechanics

**Hannah J. Lundberg**

## 1.1 Anatomy

The TMJ is a bilateral joint where movement of one side is dependent on the other. Movement direction and magnitude are governed by the shape of the contacting surfaces, ligaments, and muscles. Upper and lower joint compartments are separated by a fibrocartilaginous articular disk. The disk articulates with the mandibular condyle in the lower compartment and against the articular eminence in the upper compartment (Fig. 1.1). After total joint replacement (TJR), the TMJ becomes a single compartment joint with one intended articulation between the mandibular condyle and glenoid fossa components.

### *1.1.1 Contact Surfaces*

In the natural, non-implanted TMJ, the articular disk is concave on both surfaces. This allows the bony components of the joint to remain congruent during a wide range of mandibular movements. In TMJ TJR, the articulating surfaces are replaced, and the congruency depends on the design of the implant.

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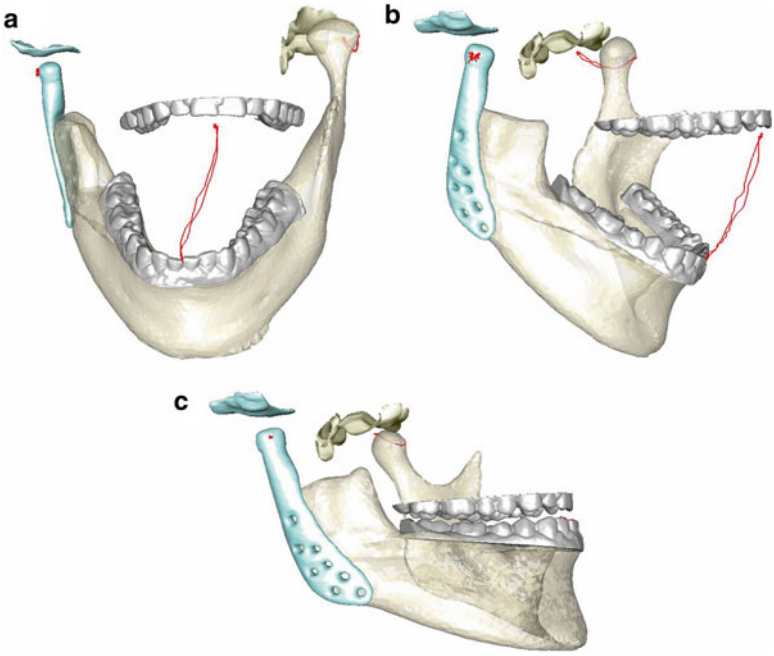


**Fig. 1.1** Bilateral temporomandibular joint (TMJ) showing a total joint replacement (TJR) on the right side and the natural joint on the left side. Trajectories (*red lines*) of the interincisal point of the mandible and the right and left lateral condylar poles computed using dynamic stereometry. Mandible, teeth, and metal TJR components are also visible. (**a**) Frontal view of one opening and closing cycle, (**b**) oblique view of one opening and closing cycle, and (**c**) oblique view of protrusion of the mandible (Reprinted with permission from Leiggener et al. [1])

### *1.1.2 Ligaments*

Ligaments passively constrain the motion of the TMJ. The temporomandibular ligament is composed of oblique and horizontal parts. The oblique part attaches to the neck of the condyle and the articular eminence to limit the mandible from moving inferiorly and posteriorly and limit mandibular rotation during mouth opening. The horizontal part attaches to the lateral condylar pole, the posterior disk, and the articular eminence to resist posterior condylar movement [2]. The stylomandibular ligament extends from the temporal bone styloid process to the posterior ramus of the mandible coursing between the masseter and medial pterygoid muscles. It functions to limit mandibular protrusion [2]. The sphenomandibular ligament passes from the spine of the sphenoid bone to the mandibular ramus and may also attach to the disk medially [3]. The function of the ligament is not agreed upon. It may suspend the mandible, limit anterior translation, or have no function depending on the source [2]. The intra-articular disk ligaments attach the medial and lateral disk to the condylar poles. The anterior and posterior disk ligaments attach the disk to the temporal bone and condyle and function

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to hold the disk in position between the condyle and the articular eminence during mouth opening and closing. They function to restrict motion to rotational movement in lower joint compartment and to anterior-posterior translation in the upper joint compartment with little medial-lateral movement.

### *1.1.3 Muscles*

Muscles influencing TMJ motion include the masseter, temporalis, medial pterygoid, lateral pterygoid, suprahyoid (digastric, geniohyoid, mylohyoid, and stylohyoid), and infrahyoid (sternohyoid, omohyoid, sternothyroid, thyrohyoid). As a group, the masseter, temporalis, and medial pterygoid are the major muscles that elevate the mandible and close the mouth. The lateral pterygoid and digastric are the primary muscles that depress the mandible and open the mouth.

The masseter is a rectangular muscle which can be divided into superficial and deep components. The superficial originates from the anterior zygomatic arch, while the deep component originates from the medial zygomatic arch. Both components have a common insertion on the mandibular ramus. The main function of the masseter is elevation of the mandible during mastication, the masseter bringing the teeth into occlusion during chewing [4].

The temporalis is a fan-shaped muscle originating in the temporal fossa. The muscle lies medial to the zygomatic arch and inserts on the coronoid process of the mandible in the infratemporal fossa. The temporalis elevates the mandible during mastication to bring the teeth into occlusion. Because the muscle is fan shaped, muscle fibers originating in the anterior temporal fossa tend to move the mandible anteriorly, while muscle fibers originating in the posterior temporal fossa tend to move it posteriorly. Activation of these muscle fibers helps stabilize the joint in the glenoid fossa [5]. If coronoidectomy, removal of the coronoid process, is required during TMJ TJR, the influence of the temporalis on the mandible is lost.

The medial pterygoid is a rectangular muscle that can be divided into superficial and deep components. It originates at the pterygoid plates of the posterior maxilla and inserts on the medial ramus and angle of the mandible. The medial pterygoid elevates the mandible. It can also help move the mandible laterally when activated with the opposite side lateral pterygoid muscle. When working bilaterally with the masseter and temporalis, the medial pterygoid causes closing of the jaw. When working unilaterally, it causes mandibular deviation toward the contralateral side [5].

The lateral pterygoid is divided into a superior and inferior head. The origin of the superior head is the infratemporal surface on the greater wing of the sphenoid. The origin of the inferior head is the lateral pterygoid plate. The lateral pterygoid inserts anteriorly on the pterygoid fovea at the neck of the mandibular condyle and the TMJ capsule. The lateral pterygoid protrudes the mandible, pulls the articular disk forward, and contributes to mandibular lateral movement when active with the contralateral medial pterygoid. The inferior head functions during opening and

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protrusion by exerting an anterior, lateral, and inferior force on the mandibular condyle. The superior head contributes to jaw closing by stabilizing the disk on the condyle during closing [5]. When TMJ TJR is performed, the lateral pterygoid is removed with the mandibular condyle.

The suprahyoid muscles consist of the digastric, mylohyoid, geniohyoid, and stylohyoid muscles. The digastric muscle consists of an anterior and posterior belly. The anterior belly originates from the digastric fossa of the mandible, and the posterior belly originates from the temporal bone mastoid notch. Both bellies meet at an insertion at the hyoid bone. The digastric muscle depresses and retracts the mandible and elevates the hyoid bone. Activation of the digastric muscle aids in forced jaw opening by stabilizing the hyoid bone. Posterior bellies are active in swallowing and coughing [5]. The mylohyoid muscle stabilizes and elevates the tongue and the floor of the mouth. The geniohyoid muscle lies beneath the mylohyoid muscle and elevates the hyoid. The stylohyoid muscle elevates the hyoid and base of the tongue [5]. In the absence of the lateral pterygoid after TMJ TJR, the suprahyoid muscles are recruited to assist in mandibular opening.

The infrahyoid (sternohyoid, omohyoid, sternothyroid, and thyrohyoid) muscles are also called strap muscles. The sternohyoid muscle depresses the hyoid and functions in speech and mastication. The omohyoid muscle is lateral to the sternohyoid muscle and also depresses the hyoid. The sternothyroid and thyrohyoid muscles are deep to the sternohyoid. Together the sternothyroid depresses the larynx, and the thyrohyoid depresses the hyoid and elevates the larynx.

## 1.2 TMJ Kinematics

In the native TMJ, the upper and lower joint spaces above and below the disk are responsible for different types of movement. Rotation occurs at the lower joint, a hinge joint, between the disk and the mandibular condyle [2]. Translation occurs at the upper joint, between the disk and the articular fossa. The upper joint allows translational motion because of loose attachments between the disk and the temporal bone [2]. In TMJ TJR, there is no disk resulting in a single joint space. Rotation and translation can occur, although translation is greatly reduced [6] leaving almost pure rotation [7, 8]. Reasons for the reduced translation include the removal of the attachment of the lateral pterygoid muscle, the TMJ TJR device articular surface geometry [8–12], and tissue and muscle fibrosis especially in multiply operated patients [9, 13–16]. TMJ TJR patients can, however, regain some of translation by recruiting the suprahyoids, masseter, and medial pterygoid muscles [16].

Mandibular motions include depression (mouth opening), elevation (mouth closing), protrusion (chin anterior jutting), retrusion (posterior sliding of the teeth), and lateral deviation (sliding the teeth laterally on either side). Main functions are chewing, talking, and swallowing which are achieved by the action of muscles and constrained by ligaments and the TMJ contacting surfaces. Two different biomechanical

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environments are present: first, the case where there is resistance to movement, for example, chewing, biting, or clenching, and second, the case where there is no resistance to movement or empty-mouth movements. Empty-mouth movements occur without contact between the teeth or contact with food between the teeth [17].

Several methods have been employed to measure native TMJ and TMJ TJR kinematics including optoelectronic, electromagnetic, dynamic stereometry, and ultrasound systems. In the optoelectronic method, radiopaque passo-reflective markers are placed on the face or teeth. Marker motion is recorded with multiple cameras, and mathematical operations determine mandibular and TMJ motions. Electromagnetic tracking methods use a magnetic source to track the movements of electromagnetic sensors attached to the face, teeth, or dental appliances. Another method is called dynamic stereometry [18, 19], where imaging data (e.g., magnetic resonance or computed tomography) is synched with dynamic jaw tracking such as that performed with optoelectronic and electromagnetic methods. Finally, a method using ultrasound has been used to measure motions of the native TMJ and TMJ TJR cases [20]. This method uses a mandibular frame and a face bow. The mandibular frame has four ultrasound emitters, and the face bow has eight ultrasound receivers. Kinematics of the mandible can be determined using time lapse analysis of sequentially emitted ultrasound pulses. All the methods for measuring TMJ kinematics are subject to limitations that the markers on the teeth and face may interfere with normal movement. Table 1.1 summarizes the translations that the implanted and non-implanted TMJ undergoes during the movements described below.

### *1.2.1 Mouth Opening and Closing*

During opening, or depression of the mandible, electromyography (EMG) studies have found that the digastric and inferior head of the lateral pterygoid muscles are active [17, 21]. Gravity also depresses the mandible. During closing, or elevation of the mandible, the temporalis, masseter, and medial pterygoid are active. The superior head of the lateral pterygoid acts eccentrically during closing to keep the disk forward while the mandibular condyle rotates backward.

During maximal mouth opening, the linear distance traveled by the interincisal point of the mandible reaches about 38–50 mm in subjects with normal jaw function [12]. The normal mandible can rotate 29–35° [22, 23]. Rotation accounts for 11–25 mm of mouth opening, and translation accounts for the remaining mouth opening. The mandible moves anteriorly and inferiorly. During mouth closing, the mandible moves posteriorly and superiorly, and the TMJ undergoes the reverse translation and rotation.

To determine the amount of maximum mouth opening attributed to rotation versus translation of the mandible, Ferrario et al. obtained three-dimensional motions from normal subjects using an optoelectronic system [24]. The majority of movement, ~77 %, was mandibular rotation. The percentage of motion attributed to rotation increased as mouth opening progressed and then decreased during closing. Motion was different for men and women, of which some was attributed to

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**Table 1.1** Interincisal and condylar translations during opening, protrusion, and lateral deviation motions for normal subjects and subjects with TMJ TJRs

| | | | Opening | | | | Protrusion | | | Lateral deviation | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| *Normal subjects* | | | Incisor linear distance | Incisor deviation | Condylar linear distance | | Incisor linear distance | Condylar linear distance | | Incisor linear distance | | Condylar linear distance | |
| Celebi et al. [12], Voiner et al. [8], Yoon et al. [16], Komistek et al. [30], Naeije et al. [25], Travers et al. [7], and Ferrario et al. [24] | | | 38–51.3 | 2.2–2.7 | 11.9–19 | | 8–12 | 8.7–9 | | 7–12 | | Ipsilateral: 2.7–3.1 | Contralateral: 7.1–7.9 |
| *TMJ TJR subjects* | | | | | | | | | | | | | |
| Study | Subjects | TJR | | | Natural TMJ | TMJ TJR | | Natural TMJ | TMJ TJR | Toward Natural TMJ | Toward TMJ TJR | Natural TMJ | TMJ TJR |
| Linsen et al. [28] | 8 Hypomobility | TMJ Concepts, custom and stock Biomet | 25.67 | | 14.05 | | 1.94 | | | 1 | | | |
| | 9 Instability | | 33.39 | | 17.49 | | 3.1 | | | 1.1 | | | |
| Leiggener et al. [1] | 1 Unilateral | TMJ Concepts | 43.3 | 8.8 | 24.8 | 5.4 | 11.3 | 13.4 | 1.4 | | | | |
| Voiner et al. [8] | 13 Bilateral | Stock Biomet | 24.9 | | | | 2.5 | | | 3 | | 3.1 | 2.9 |
| | 5 Unilateral | Stock Biomet | 28.7 | | | | 5.6 | | | 3.8 | 6.7 | | |
| Yoon et al. [16] | 13 Unilateral | Christensen fossa only | 35 | 5.8 | 12.2 | 7.1 | 5.1 | 6.9 | 3.4 | 5.1 | 9.4 | op[a], 3.4; nonop, 2.1 | op, 3.1; nonop, 6.4 |
| Baltali et al. [13] | 14 | Christensen fossa only | 35.52 | | 13.31 | 11.37 | | | | | | | |
| Komistek et al. [30] | 5 | Christensen fossa only | | | 7.1 | | | | | | | | |
| | 5 | Christensen TJR | | | 1 | | | | | | | | |

All measurements are reported in mm

[a]"Op" indicates the operated TMJ, "nonop" indicates the non-operated TMJ

mandibular size. Both mandibular size and degree of mandibular rotation were correlated to the distance reached at maximum opening. Some subjects also had asymmetrical opening and closing motion profiles. The authors noted that previous studies have conflicting findings about the percentage of mandibular motion attributed to rotation versus displacement throughout opening and closing.

Many studies have been performed to relate the amount of mouth opening to movement of the TMJ. Mouth opening results in a combination of joint rotation and translation at the TMJ. Rotation occurs in the lower joint compartment between the condyle on the disk. Translation occurs in the upper joint compartment between the disk and the articular eminence. A reference point on the mandibular condyle must be chosen to transform movement of the interincisal point to movement of the condyle within the TMJ.

Naeije et al. studied kinematic and anthropometric factors that contributed to maximum mouth opening and condylar movement for normal subjects using an optoelectronic system [25]. The kinematic condylar center was used as the reference point to relate mandibular movement to condylar movement. Maximum mouth opening reached an average of 51 mm and passed through 35° of rotation. This corresponded to 19 mm of condylar translation: 4 mm inferiorly and 18 mm anteriorly. The biggest determinants of maximum mouth opening were the angle of rotation and mandibular length. Angle of rotation was positively related to forward translation and negatively related to downward translation. Condylar translation also decreased with increasing age.

Travers et al. compared interincisal to condylar movements during mouth opening for normal subjects also using an optoelectronic system [7]. They reported movements relative to a terminal hinge axis or the axis the mandible would rotate about given pure rotation rather than the kinematic condylar center. Incisal straight line distance traveled was 46 mm, while condylar straight line distance traveled was 12 mm. Incisal straight line distance traveled was correlated with mandibular rotation, but not with condylar straight line distance traveled. There was a high amount of variability in condylar distance traveled measurements, and the authors concluded that it may not be feasible to use condylar distance traveled as a clinical indicator of TMJ function.

### *1.2.2 Mandibular Protrusion and Retrusion*

The masseter, medial pterygoid, and lateral pterygoid muscles act together bilaterally to produce protrusion or anterior movement of the chin. The posterior fibers of the temporalis, the digastric, and suprahyoid muscles produce retrusion, or posterior movement of the chin, when acting bilaterally.

The normal mandible is able to protrude 8–12 mm [12], enough to allow the upper and lower teeth to align in the superior-inferior direction. Protrusion involves only anterior and inferior translation of the upper TMJ compartment against the articular eminence. Conversely, retrusion involves only posterior and superior translation of the upper TMJ compartment against the articular eminence. Protrusion is restricted by the posterior discal attachments. Retrusion is restricted by the temporomandibular ligament and retrodiscal tissue.

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### 1.2.3 Lateral Deviation

The lateral and medial pterygoid muscles deviate the mandible to the opposite side. The temporalis muscle can deviate the mandible to the same side depending on which muscle fibers activate. Together, the temporalis and lateral pterygoid muscles act as a force couple: the mandible rotates about the condyle on the side where both muscles are active resulting in lateral deviation of the mandible. Lateral deviation occurs during chewing because the temporalis muscle also elevates the mandible.

The normal mandible is able to laterally deviate between 7 and 10 mm [12] or the full width of one central incisor in each direction. One condyle rotates around a superior-inferior axis, and the other condyle translates anteriorly. When biting on one side, lateral deviation occurs by rotation of one condyle an anterior-posterior axis and depression of the other condyle. This results in frontal plane mandibular motion. At the TMJ, rotational movement on one side occurs concurrently with gliding on the other side. Both forms of lateral deviation occur together for chewing and grinding.

### 1.2.4 Kinematics of TMJ TJRs

Many studies have investigated the TMJ kinematics of normal subjects or subjects with temporomandibular disorders, but fewer have investigated the kinematics of TMJ TJRs. TMJs with TJRs have different kinematics because of the geometry of the bearing surfaces of the device components and the loss of bony and soft tissue components that govern normal movement.

Maximum interincisal opening increases postoperatively for TMJ TJR patients with functionally restrictive end-stage TMJ disease. Mercuri et al. found that TMJ TJR patients could obtain 24.9 mm of opening before surgery. After TMJ TJR with a patient-fitted joint replacement, maximum interincisal opening increased 36 % after 3 and 10 years and 74 % after 14 years [26]. In a similar study of 56 patients with a median 21-year follow-up, maximum interincisal opening increased from 25.8 mm to 36.2 mm after TJR [27].

Wolford LM et al. compared the amount of interincisal opening between patients with stock (TMJ Inc., Golden, CO) prostheses and patients with patient-fitted (TMJ Concepts, Ventura, CA) prostheses [10]. The two prostheses have different articular surface geometries and material composition. The stock prosthesis is a metal-on-metal design, while the patient-fitted is an ultrahigh weight molecular polyethylene-on-metal design. For the stock device subjects, interincisal opening increased from 23.4 mm preoperatively to 30.1 mm postoperatively. For the patient-fitted subjects, interincisal opening increased from 27.4 mm preoperatively to 37.3 mm postoperatively. In another study by the same group, maximum interincisal opening increased from 27.5 mm preoperatively to 32.6 mm 5 years postoperatively for subjects with patient-fitted prostheses [11].

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Patients with a unilateral TMJ TJR have asymmetrical motion during opening, including lateral deviation toward the non-implanted side [1, 9]. Leiggener et al. used dynamic stereometry to measure the kinematics of the mandible and TMJs during opening for one patient with a unilateral TMJ TJR (Fig. 1.1). The study found that although the patient could obtain maximum opening, there was a strong lateral deviation of the mandible toward the TMJ TJR side potentially resulting in increased loading on the contralateral joint.

Linsen et al. used ultrasound-based jaw tracking to measure the mandibular motion of 17 TMJ TJR patients before and at least 1 year after TMJ TJR surgery [20]. Eight patients were preoperatively categorized as having condylar hypomobility or decreased opening from intra-articular ankylosis. The remaining seven patients were preoperatively categorized as having condylar instability or loss of condylar guidance within the mandibular fossa. Patients had a unilateral or bilateral patient-fitted components (TMJ Concepts, Ventura, CA), custom TMJ TJR devices (Biomet Microfixation, Jacksonville, FL), or stock TMJ TJR implants (Biomet Microfixation, Jacksonville, FL). Interincisal motion and condylar point motion were described as total linear distance traveled or the curvilinear distance traveled during maximum mouth opening, protrusion, and lateral deviation. In the hypomobility group, interincisal opening increased from 12 mm preoperatively to 26 mm 1 year postoperatively, and mandibular rotation increased from 9 to 19°. Condylar point movement during opening increased from 2 to 14 mm. Protrusion increased from 1.2 to 1.9 mm. Lateral deviation was approximately 1 mm and did not change after surgery. For the instability group, preoperative interincisal opening of 33 mm did not change 1 year postoperatively. Condylar motion during opening increased from 13 to 17 mm, although the change was not significantly different. Both protrusion (preoperative 6 mm, postoperative 1 mm) and lateral deviation (preoperative 7 mm, postoperative 3 mm) decreased after surgery. Increased translation was seen in the hypomobility group despite the loss of the lateral pterygoid. The authors attributed this to compensations from other muscles, gravity, increased motion of the healthy joint side which indirectly increased motion of the TJR side, or "pseudo-translation."

Voiner et al. compared maximum opening, protrusion, and lateral deviation motions for subjects with and without TMJ TJRs using electromagnetic jaw tracking [8]. TMJ TJR subjects had bilateral or unilateral stock Biomet prostheses and were tested at least 6 months after surgery. Maximum interincisal linear distance traveled during opening was 50, 25, and 29 mm for controls, bilateral TJR, and unilateral TJR, respectively. Protrusion was 6.7, 2.5, and 5.6 mm. Right or contralateral excursion was 8.9, 3.1, and 3.8 mm. Left or ipsilateral excursion was 8.1, 2.9, and 6.7 mm. Distance traveled was significantly different during opening, protrusion, contralateral excursion, and ipsilateral excursion for the control than the bilateral TJR subjects. Distance traveled was only significantly different for contralateral excursion between the control and unilateral TMJ TJR subjects. The authors state that the Biomet stock prosthesis has a large circumferential lip on the fossa component to prevent dislocation of the condylar component. Because the fossa is thick, the center of rotation is

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also moved inferiorly which can result to pseudo-translation of the implanted joint as reported by Linsen et al. and Van Loon et al. [28, 29] (Table 1.1).

## 1.3 TMJ Forces and Muscle Forces

Because there are no available techniques to measure forces in the native or TMJ TJR in humans, animal, in vitro, and mathematical models are necessary to estimate in vivo joint and muscle forces.

### *1.3.1 Animal Models*

TMJ forces have been measured in macaques [31, 32] and baboons [33]. Mandibular condylar neck strains have been measured in macaques [4, 34] and miniature pigs [35]. Brehnan et al. measured the joint loads during chewing for one macaque using piezoelectric foil [31]. TMJ loads reached a maximum of 13 N for molar chewing and 18 N for incisor biting. The magnitude of joint loading did not change while chewing soft versus hard foods, although the force waveforms were more consistent while chewing hard foods. In a follow-up study with a refined experimental technique, Boyd et al. measured the joint loads during chewing in two macaques [32]. Loads on the TMJ varied widely depending on the activity: 60–173 N during open-mouth aggressive behaviors, 7–153 N during molar chewing, and 7–10 N during drinking. TMJ loads on the working side were about two times greater than non-working side loads. Contrary to their previous study, chewing hard food produced larger TMJ loads than chewing soft food.

Hohl et al. measured TMJ loads in a baboon during simulated biting by replacing the mandibular condylar neck with an instrumented prosthesis [33]. The prosthesis allowed the TMJ and occlusion to remain intact. Bite force was simultaneously measured. The trigeminal nerve was stimulated bilaterally with currents ranging from 1 to 6 mA to contract the masseter, temporalis, medial pterygoid, and lateral pterygoid muscles simultaneously. With increased stimulation current, both the bite and joint forces increased. For stimulation currents greater than 5.5 mA, joint force decreased even though bite force continued to increase. This was attributed to the lateral pterygoid bending the condyles anteriorly. Bite forces of 2–32 N corresponded to TMJ loads ranging from 4 to 33 N. For all simulations, TMJ loads were 0.56–2.25 times the bite force.

The above animal studies provided valuable information about TMJ loading. First, the TMJ is loaded during a variety of activities. Second, the working side TMJ load appears to be greater than nonworking side TMJ load during chewing and biting. Third, as bite force increases, TMJ load increases. There are limitations, however, to the application of the animal data to the human TMJ. First, the TMJ biomechanics of the macaque and baboon are not the same

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as humans; therefore, the measured joint loads may not be the same in humans. For example, the length of the condyle in the macaque is greater than in humans, a variable that affects TMJ load [25]. Second, bite forces were only measured for the baboon model and were much smaller than reported human bite forces. This could be because the measurements were taken during stimulated muscle action instead of natural chewing/biting conditions, because humans are larger than baboons and have masticatory muscles that are capable of generating larger bite forces or as a result of the surgery and invasive measurement techniques.

### *1.3.2 In Vitro Models*

Hatcher et al. performed an in vitro test on a dry skull with synthetic muscles and disk and various transducers to measure condylar force, muscle force, and bite force [36]. The deep masseter, superficial masseter, medial pterygoid, anterior temporal, and posterior temporal muscles were modeled with Kevlar strands. The distribution of applied muscle forces were based on the relative physiological cross-sectional area of each muscle. For unilateral biting, the load at the balancing side TMJ was higher than the load at the working side TMJ. Occlusal forces were higher than TMJ loads on both the balancing and working sides.

Celebi et al. developed a TMJ motion simulator which can be used with either a cadaveric or surrogate skull [12]. The motion simulator was used to compare motion before and after unilateral TMJ TJR. Cables were inserted in the center of muscle attachments to simulate the jaw elevator muscles, the lateral pterygoid (superior and inferior heads combined), anterior digastric, geniohyoid, and mylohyoid muscles. Cables for the suprahyoid muscles ran through a surrogate hyoid to replicate the correct lines of action. Cables for the combined action of the elevator muscles were attached to the anterior mandible. Muscle forces were applied to produce motions. The muscle forces were bounded by the maximum muscle forces predicted by a mathematical model [37]. A three-dimensional laser scanner and fluoroscopy that were used to ensure motions were achieved. To produce maximum opening, a 119 N lateral terygoid force, a 50 N geniohyoid and digastric force, and a 23 N mylohyoid force were required. Smaller forces were required for maximum lateral deviation: 75 N for the lateral pterygoid, 5 N for the geniohyoid and digastric, and 1 N for the mylohyoid. Maximum protrusion required the highest lateral pterygoid force, 150 N, but smaller geniohyoid and digastric (14 N) and mylohyoid (1 N) forces. Maximum interincisal opening was approximately the same before and after unilateral TJR surgery. Lateral deviation was eliminated on the non-implanted side after TJR, and protrusion was greatly reduced. When the lateral pterygoid muscle was reattached to the mandible through a predrilled hole in the implant, lateral deviation on the non-implanted side improved (to approximately 7 mm) but did not reach preoperative values (approximately 10 mm).

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Protrusion did not increase after lateral pterygoid reattachment, but there was more vertical jaw movement during protrusion.

### *1.3.3 Mathematical Modeling*

Mathematical models are needed to predict TMJ and muscle forces during daily activities. Inputs to mathematical models include external forces (weight of the mandible, bite forces, or chin cup forces) and internal forces (from muscles, joint reaction forces) or motions. The location of action of external and internal forces on the system is also necessary inputs. Assumptions that are usually made include that muscles follow straight lines of action spanning between the muscle origin and insertion points. For muscles that attach to bone over large areas and have fibers running in more than one direction, the muscle is often split into functional parts. Updates to this include using pulleys to produce a curve in the mylohyoids [38] or the use of contact spheres to produce locations where muscles can wrap around bone, for example, with the temporalis, masseter, and medial and lateral pterygoid muscles [39]. One study also modeled a three-dimensional masseter muscle in a finite element analysis (FEA) model that contained a structural representation of the muscle fibers [40]. Muscle lines of action can be measured from cadavers or with medical imaging techniques. Muscle lines of action are important variables because they define the moment arms that muscles have about the TMJ. Occlusal forces can be measured using force transducers in between the teeth. Normal masticatory loads typically are assumed to be in the range of 250–450 N [41, 42]. Clenching and maximum isometric muscle contractions can result in much higher bite forces. One study measured maximum bite forces during isometric contractions of 597 N for women and 847 N for men [43].

Numerical models can be categorized as static or dynamic. Static models use principles of static equilibrium where the mandible is analyzed at a given position around which joint, muscle, and occlusal forces applied to the mandible must balance so that no acceleration is produced. Static models are useful for determining associations among parameters such as joint, muscle, and occlusal forces and patterns of muscle activation. Many static models of the TMJ have been developed in two or three dimensions. Two-dimensional models can only predict the resultant of right and left TMJ and muscle forces. Applications of static models include tooth clenching or biting.

Dynamic models use equations of motion where muscle forces on the mandible cause motion which is constrained by joints, contact between bodies, and passive structures such as ligaments. Models can use forward or inverse dynamics methods. For forward dynamics, muscle forces are applied to the mandible which results in motion and reaction forces (joint and occlusal). For inverse dynamics, motion and any external forces (e.g., bite forces) are applied to the mandible, and the internal (muscle) forces necessary to produce the motion are determined. Applications of dynamic models include mouth opening/closing and chewing.

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In both static and dynamic analyses, an indeterminate problem results because of the large number of unknown muscle forces and the multiple activation patterns that could be used to produce the motion or static equilibrium. Optimization methods have been used to solve this problem but require optimization criteria (e.g., minimization of joint force). The next sections summarize different types of mathematical models and their major findings.

#### 1.3.3.1 Static Models

Early static biomechanical models were two-dimensional and did not separate the jaw into right and left TMJs. Accordingly, the models were symmetric, and muscle, occlusal, and joint loads were equal on each side. Barbenel et al. investigated biting at different tooth contact points, under different angles between the occlusal force and plane, and using two different objective functions for optimization to solve the equations of static equilibrium [44]. The static, two-dimensional model had four muscle components modeled as straight lines: masseter, temporalis, medial pterygoid, and lateral pterygoid whose locations were measured from cadavers. Two objective functions were evaluated: (1) minimize joint force and (2) minimize total muscle force. The TMJ load could be less than half the occlusal load or greater than 2.5 times the occlusal load depending on the location of tooth contact and the angle that the occlusal load made with the occlusal plane. Highest TMJ loads – given an equal occlusal load – occurred with tooth contact at the incisors. This finding has been replicated by many studies. With both objective functions, TMJ loads were less sensitive to the angle that the occlusal load made with the occlusal plane than the location of tooth contact. The objective function that minimized joint force resulted in higher TMJ loads when the occlusal load was directed more anteriorly. The objective function that minimized total muscle force resulted in the opposite relationship where TMJ loads were higher when the angle of the occlusal load was directed posteriorly. In addition, only the masseter was active, a finding that is invalid based on electromyography (EMG) data [45]. Therefore, the authors suggest that using minimization of total muscle force as an objective function is not physiological. When the minimization of joint force objective function was used, only the lateral pterygoid and temporalis were active [46], again a finding not consistent with EMG data.

In another study, Barbenel et al. investigated the effect of the direction of the TMJ load and the magnitude of lateral pterygoid activation during molar biting and used EMG data to further constrain their model [47]. Surface EMG was used to measure the muscle activity for the masseter, temporalis, and medial pterygoid during molar biting where the occlusal force was perpendicular to the occlusal plane. A model assumption was that muscle force is linearly proportional to the measured EMG potential. Lateral pterygoid activation was parametrically varied. Minimum TMJ load was 2.7 times occlusal load, and TMJ load could reach over four times the occlusal load. Minimum TMJ load occurred at the lowest lateral pterygoid force and for TMJ loads oriented 8° from vertical in the posterior direction.

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Hekneby et al. also used a static model to investigate clenching at the first premolar and second molar [48]. Forces included in the model were a resultant muscle force for the muscles of mastication, an occlusal force, and a TMJ reaction force. The occlusal force was set to 29.4 N. All forces were perpendicular to the occlusal plane. Occlusal plane and moment arms were defined from measurements of 25 mandibles from young male cadavers. In agreement with Barbenel et al., TMJ loads were greater with tooth contact at the first premolar than the second molar. Maximum TMJ load was 107 N, and maximum resultant muscle load was 136 N.

Similar to Barbenel et al., Pruim et al. predicted TMJ loads during biting at the first premolar and first and second molars from muscle forces determined from EMG activation [49]. Muscles investigated included the combined action of the masseter and medial pterygoid, anterior temporalis, posterior temporalis, openers, and lateral pterygoid. The lateral pterygoid was assumed to only act parallel to the occlusal plane. Muscle activity and bite forces were measured for seven male subjects. The model used two-dimensional static equilibrium equations to determine muscle tension, TMJ load, and lateral pterygoid force from the EMG potentials and bite forces. The authors did not find a pattern between muscle tension and bite moment, although higher bite moments lead to higher antagonist force moments of the opener muscles. Bite position had a smaller impact on the relationship. High TMJ loads were present (mean 1297 SD 503 N for total force right and left). TMJ loads were higher for the first molar and premolar than the second molar, consistent with other studies.

Three-dimensional models allow the model forces to differ on the right and left sides and allow investigation of unilateral activities. An early attempt was that of Hatcher et al. who developed a three-dimensional model of biting and performed in vitro testing to validate their model predictions [36]. Six muscles were modeled on each side: deep masseter, superficial masseter, medial pterygoid, anterior temporal, posterior temporal muscles, and lateral pterygoid. An assumption that the TMJ load on the right and left joint were equal in the medial-lateral direction was necessary. Muscle forces were directly input to the model as either proportional to cross-sectional area alone or proportional to cross-sectional area multiplied by an EMG potential representing muscle activation level. The mechanical model consisted of a dry skull with synthetic muscles and disk and transducers to measure condylar force, muscle force, and bite force (see 1.3.2. In Vitro *Models* section above). The mechanical model did not include the lateral pterygoid muscle. The agreement between the mathematical and mechanical model was good; even though sometimes the magnitude of differences in the forces could be large, the same trends were followed by the mathematical and in vitro models. Sensitivity studies were performed to assess potential sources of error between the mathematical and in vitro models. The model results were most sensitive to the variation in muscle parameters associated with the anterior temporalis and deep masseter. A 20 % change in the force of the muscles and 6.5 mm change in the location of their attachment points could result in a change of 15–20 % in the occlusal and TMJ loads. When equal muscle loading was present on each side, turning the lateral pterygoid on or off changed the direction but not the magnitude of TMJ load.

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Faulker et al. performed a follow-up study of unilateral biting using the validated mathematical model developed by Hatcher et al. [50]. Tooth contact was varied between the first, second, and third molar on the left side. The direction of occlusal force was parametrically varied by 10° anterior or posterior from perpendicular to the occlusal plane. In agreement with previous studies, muscle and TMJ loads decreased as tooth contact location moved posteriorly for equal occlusal forces. The working side TMJ carried half as much load as the balancing side regardless of tooth contact location (313 N balancing side TMJ load compared to 188 N working side load for a 500 N occlusal force). TMJ loads were approximately half the magnitude of occlusal forces, and occlusal forces were approximately half the magnitude of muscle forces. TMJ loads were highest for occlusal forces directed anteriorly from perpendicular to the occlusal plane. Changing the direction of the occlusal force resulted in wide variation in the direction of the working side TMJ load but not the balancing side TMJ load which was always directed approximately perpendicular to the occlusal plane.

Osborn et al. describe a three-dimensional model that included 13 muscles on each side: anterior superficial masseter, anterior deep masseter, posterior deep masseter, posterior superficial masseter, medial pterygoid anterior, medial pterygoid posterior, large vertical temporalis, temporalis oblique anterior, temporalis oblique posterior, lateral pterygoid upper, lateral pterygoid inferior, lateral pterygoid superior (upper and inferior are for lower head and superior is for upper head), and anterior digastric [51]. They simulated bilateral (symmetric) biting with an occlusal force located at the central incisor or first molar that was perpendicular to the occlusal plane. TMJ load was constrained to perpendicular to the surface of the eminence. Two different optimization objective functions were investigated: minimize muscle force and minimize joint load. For minimizing joint load, the model predicted zero joint load until the occlusal force was at least 127 N at the first molar or 39 N at the central incisor. Muscle forces could be asymmetrical, and the oblique temporalis and pterygoid lateral inferior muscles dominated the solution. Because this muscle activity is not consistent with EMG studies of muscle activity during biting, the authors concluded that minimizing joint load was not a physiologic optimization objective function. For minimizing sum of muscle force, very different muscle activity occurred with increasing occlusal forces. With an occlusal force up to 196 N on the first molar, the anterior superficial masseter and temporalis oblique anterior were active. When the anterior superficial masseter reached maximum activation, the large vertical temporalis and medial pterygoid anterior become active and the temporalis oblique anterior deactivated. Next when the medial pterygoid anterior activated maximally, the medial pterygoid posterior also activated. Finally, after the medial pterygoid posterior maximally activated, the posterior superficial masseter became active to increase occlusal force. A similar pattern of muscle activation was seen for tooth contact at the central incisor.

Another three-dimensional model was used to determine the maximum unilateral and bilateral bite forces and resulting TMJ loads that could be generated for different locations of tooth contact and mandible positions [52]. The optimization objective function was to minimize the activation of the most activated muscle.

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The model was created from measurements of one male cadaver aged 65 years. Nine muscle components on each side were included in the model: deep masseter, superficial masseter, medial pterygoid, superior lateral pterygoid, inferior lateral pterygoid, superficial anterior temporalis, superficial posterior temporalis, deep temporalis, and anterior digastric. The direction and location of contact for the TMJ reaction force was defined as the location of minimum distance between the condyle and eminence and in the direction perpendicular to both surfaces. Tooth contact was simulated at the first and second incisors, canine, first and second premolars, and first molar. The direction of the occlusal force was parametrically varied. Three different mandible positions were investigated for biting: (1) edge-to-edge contact position (anterior top and bottom teeth lined up edge-to-edge), intercuspal position (natural occlusion), and (2) open position (10° of open rotation from the edge-to-edge contact position). Maximum occlusal forces ranged from 585 to 967 N depending on tooth and bite type. This corresponded to TMJ loads that ranged from 43 N for tooth contact at the second molar in the edge-to-edge contact position to 513 N for tooth contact at the first incisor in the open position. The latter maximum TMJ load occurred for an anteriorly directed occlusal force. The maximum TMJ load for a posteriorly directed occlusal force was 456 N for a 585 N occlusal force. Consistent with other studies, incisor occlusal force caused higher TMJ loads than molar occlusal forces. Occlusal forces directed laterally loaded the working side TMJ more than the balancing side TMJ; occlusal forces directed medially loaded the working side TMJ less than the balancing side TMJ. Occlusal forces were smallest when the mandible was in the open position. Maximum occlusal forces were produced when most muscles except for the lateral pterygoid were maximally activated. In the open mandibular position, joint forces could be greater than the occlusal force if tooth contact was located at the first incisor. Unlike EMG studies of muscle activity, balancing side muscle activity was not necessarily greater than working side muscle activity. The digastric muscles also contributed during maximal biting even though they are a jaw opener. Very different muscle recruitment patterns, for example, no activity in the anterior temporalis, could still lead to almost maximal biting forces.

May et al. reported bite, muscle, and TMJ loads predicted by a three-dimensional model during bilateral clenching [42]. Like Barbenel [47], muscle force was calculated from muscle cross-sectional area and EMG potential. Two optimization objective functions were investigated: (1) minimize the sum of squared muscle activations (equivalent to minimizing muscle stress) and (2) minimize the sum of squared muscle forces (restricts the force of large individual muscles). Model assumptions included that the anterior-posterior component of the TMJ load must act in the posterior direction, the medial-lateral TMJ load component was equal on the right and left side, and the superior-inferior component of the TMJ load acted inferiorly or into the mandibular condyle. EMG activity and occlusal forces were measured for 25 subjects during maximal clenching. EMG activity was measured for the masseter and temporalis. A force sensor measured the bilateral occlusal forces at the first molars. Model predictions were compared for solutions where the temporalis and masseter muscle forces were predicted from EMG activity or left as model

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unknowns. The temporalis forces were not different whether forces were calculated from EMG activity measured in subjects and muscle cross-sectional area or forces were predicted by the model without EMG data. Conversely, masseter forces predicted by the model were not the same as those calculated from EMG activity measured in subjects and muscle cross-sectional area. The two objective functions and methods for predicting muscle forces did not result in different TMJ load predictions. Average predicted TMJ loads were 260 N, 172 N, and 152 N for men, women, and women with temporomandibular disorders.

In a series of studies, Schindler et al. used a three-dimensional mathematical model to investigate the muscle and TMJ loads generated during bilateral clenching [53]. Ten male subjects performed feedback-controlled clenching via a three-dimensional bite force transducer. Muscle activity was measured using surface EMG for the masseter, anterior temporalis, posterior temporalis, and anterior digastric muscles. Muscle activity was measured using fine wire EMG for the medial and lateral pterygoid. Feedback was provided to the subjects to produce occlusal forces of a certain magnitude and direction, measured at the midpoint between the first molars. Maximum voluntary contractions of the jaw muscles under various movements were also generated in order to normalize the EMG measured muscle activity. Musculoskeletal models were created for each subject using magnetic resonance tomography. Area of muscle attachments was also measured from the images. Model assumptions included that the condyles were only under compression; therefore, the vertical component of the TMJ load was directed in the inferior direction, and the medial-lateral TMJ load was equal to zero on one side. Model results using muscle forces calculated directly from EMG potential and cross-sectional area were compared to results predicted by three different optimization objective functions: (1) minimization of joint force magnitudes, (2) minimization of overall muscle force, and (3) minimization of elastic energy of the contractile properties of the muscle tissue (based on the pennation angle and length of the muscle fibers).

Varying the bite angle resulted in large variation in TMJ loads and muscle forces. The lateral pterygoid and posterior temporalis muscles tended to have increased muscle forces with occlusal force angles directed more vertically, while the masseter and anterior temporalis had the opposite behavior. Lateral occlusal force directions resulted in highest muscle forces in the anterior and posterior temporalis on the side of the direction of the occlusal force and in the medial pterygoid, lateral pterygoid, and masseter on the opposite side of the directed occlusal force. Maximum TMJ loads of about 150 N were seen for medial and anteromedial directed occlusal forces, and minimum TMJ loads of about 50 N were seen for purely vertically directed occlusal forces. The optimization objective function which minimized the contractile elastic energy produced the closest results to that when using muscle forces directly from EMG data and cross-sectional muscle area. The conclusions of the study were that the medial pterygoid was the most heavily loaded muscle in all the biting activities and TMJ loads were much higher with more horizontally directed occlusal forces.

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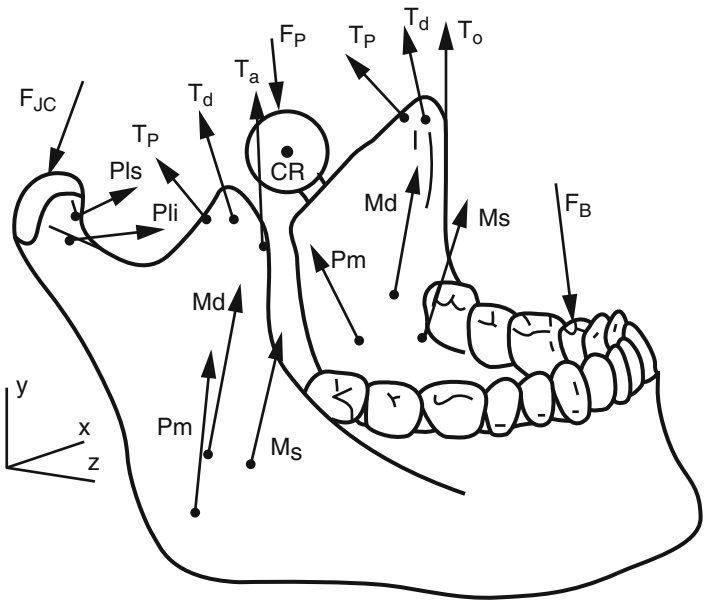


**Fig. 1.2** Model developed by Van Loon et al. to investigate the forces acting on the mandible with a unilateral TMJ TJR. Muscle forces in the model include superficial masseter (Ms), deep masseter (Md), medial pterygoid (Pm), right inferior head of the lateral pterygoid (Pli), right superior head of the lateral pterygoid (Pls), anterior temporalis (Ta), posterior temporalis (Tp), and deep temporalis (Td). $F_B$ indicates the occlusal force, $F_{JC}$ indicates the TMJ load on the non-implanted side, and $F_P$ indicates the TMJ TJR load (Reprinted with permission from Van Loon et al. [55])

In a subsequent study, Rues et al. reported unilateral and bilateral submaximal biting at different tooth contact locations [54]. Occlusal forces were varied from 50 to 400 N for bilateral canine biting, bilateral premolar biting, bilateral molar biting, and unilateral molar biting. Similar to other studies of maximal biting, the TMJ loads during submaximal biting decreased as the location of tooth contact moved anteriorly. For example, a bilateral 200 N occlusal force resulted in a 125 N TMJ load for molar tooth contact, a 155 N TMJ load for premolar tooth contact, and a 190 N TMJ load for canine tooth contact. In addition, working side TMJ loads were smaller than balancing side TMJ loads for unilateral molar biting.

Van Loon et al. published the only mathematical model which includes a TMJ TJR [55]. The TJR is unilateral with a natural TMJ on the opposite side (Fig. 1.2). The mathematical model uses the same data and assumptions as that reported by Koolstra et al. [52] but does not have a lateral pterygoid on the side of the TJR. TJR geometry consists of a perfect sphere for the head of condyle. The model was used to investigate the maximum loads experienced by the TMJ TJR and the effect of the location of the center of rotation of the TJR on the developed maximum loads. The center of rotation of the TJR condyle was varied from the same superior-inferior location as the center of the condyle on the natural TMJ side to 15 mm inferior to the

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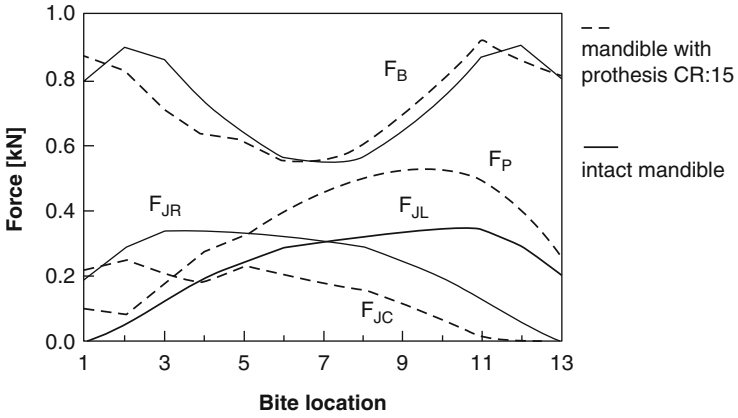


**Fig. 1.3** Maximum occlusal forces ($F_B$), non-implanted TMJ loads ($F_{JL}$ and $F_{JR}$), and unilateral TMJ TJR loads ($F_P$ and $F_{JC}$). For non-implanted TMJ loads, $F_{JL}$ and $F_{JR}$ indicate the left and right sides, respectively. For unilateral TMJ TJR loads, $F_P$ indicates the right (prosthesis) TMJ, and $F_{JC}$ indicates the left (non-implanted) TMJ. The *x*-axis indicates bite location where 1 is the second molar on the right (prosthesis) TMJ and 13 is the second molar on the left (non-implanted) TMJ. Results shown are for a TJR center of rotation 15 mm below the center of rotation of the non-implanted condyle (Reprinted with permission from Van Loon et al. [55])

natural TMJ condylar center. The direction of the TMJ TJR load was parametrically varied. The same locations of tooth contact as Koolstra et al. were investigated.

When the TMJ TJR was present, although the magnitude of maximum occlusal force was relatively unchanged, the maximum occlusal forces generated were not symmetric for tooth contact on the TMJ TJR versus natural TMJ side (Fig. 1.3). When a TMJ TJR was simulated, the joint loads in the prosthesis were higher than the non-implanted case, and the joint loads through the natural TMJ side were lower than the non-implanted case. TMJ TJR loads were highest for biting on the contralateral side (the TMJ TJR was the balancing side). TMJ TJR loads for biting on the same side (the TMJ TJR was the working side) were lower than working side TMJ loads with two natural joints. The absence of the lateral pterygoid muscle was considered responsible for increased loads on the TMJ TJR side.

#### 1.3.3.2 Dynamic Multibody

Multibody dynamic modeling techniques have been used to investigate chewing and opening and closing activities. Hannam et al. investigated chewing using a three-dimensional model of mandible and hyoid dynamics [56]. The model was developed from computed tomography imaging of one adult male. Muscles in the model included the anterior, middle, and posterior temporalis, deep and superficial masseter, medial pterygoid, superior and inferior lateral pterygoid, anterior digastric, sternohyoid, posterior digastric, stylohyoid, mylohyoid, geniohyoid.

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Soft tissues were modeled as springs including thyrohyoid, cricothyroid, and cricotracheal membranes. A compressible food bolus was simulated. The modeled chewing kinematics were 0.7 s per cycle in duration where first the incisor point moves left and then 17–20 mm of gape right to open the jaw. Next jaw closing occurred by first moving the mandible 5 mm right with the chewing side returning to the rest position before contralateral side. Hyoid movement was toward the midline, anterior, and superior up to 3–5 mm by the end of jaw opening. The jaw moved medially when the food bolus made contact with the teeth at the end of closing. Muscle activity was asymmetrical for the lateral pterygoid and mylohyoid muscles to allow lateral deviation of the mandible. Muscle activation was less for the openers than the closers when crushing the food bolus. Muscle activity was also asymmetrical in closing. The mandibular condyle of the working TMJ returned to the starting position before the contralateral condyle. The working side lateral pterygoid slowed posterior condylar point movement, while the contralateral lateral pterygoid prolonged the return of the contralateral condyle. The authors concluded that the timing of lateral pterygoid muscle activity was critical for lateral movement of the mandible and food bolus compression.

De Zee et al. also investigated chewing using an inverse dynamics model [57]. The model was created using computed tomography images from one male cadaver. The optimization objective function for inverse dynamics was to minimize muscle effort. Motion and occlusal force were input to the model, and muscle and TMJ loads were output. To validate the model, comparisons were made to jaw tracking data with simultaneous EMG measurements and bite force (clenching) measurements. The data was collected from one male subject performing cyclic protrusion, chewing without force, incisal clenching, and unilateral clenching at the right and left first premolar. EMG envelopes matched well for measured and predicted muscle activity. Forces were greater on the balancing side (336 N) for unilateral clench (occlusal force 441 N) than the working side (234 N), consistent with results of static biting.

Another investigation of chewing was performed by Sellers and Crompton using a forward dynamics model [58]. The maximum TMJ loads generated from muscle forces were predicted for various tooth contact locations. Four muscles were modeled on each side: temporalis, medial pterygoid, masseter, and lateral pterygoid. The muscle activation levels were parametrically varied between “on” and “off” and the food bolus location varied between each tooth on one side from incisors to molars. Damped springs were used to model constraints on the motion of the TMJ and food bolus. Vertically oriented TMJ loads reached a maximum of approximately 560 N on the balancing side TMJ. The TMJ load was higher on the balancing than working side. Balancing side TMJ load changed relatively little for different tooth contact locations. The sensitivity of the model to the location of muscle attachments, TMJ load location, and TMJ and food bolus spring stiffness was evaluated. Bite forces reached 1079 N in the superior-inferior direction corresponding to a 311 N working side TMJ load. The temporalis muscles had the biggest contribution to the occlusal force in the vertical direction, while both the temporalis and masseter had the largest contribution to the TMJ load in the vertical direction. The lateral occlusal forces

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were influenced the most by the balancing side temporalis muscle. The lateral TMJ loads were influenced the most by the balancing side temporalis and working side masseter. Occlusal forces in the anterior direction were most influenced by both masseters and the working side temporalis muscles. TMJ loads in the anterior direction were most influenced by both masseters, the working side temporalis, and the balancing side medial pterygoid muscles. The origin of the temporalis and masseter muscles and the stiffness of the TMJ spring constraints had a large effect on anterior-posterior occlusal forces. TMJ loads were relatively insensitive to changes in model parameters. Stiffness of the constraints on the food bolus had almost no effect on the generated occlusal or TMJ loads.

Multibody dynamic models have also been used to investigate jaw opening and closing. Koolstra et al. describe simulated unloaded jaw opening under various muscle activations [37]. Twelve muscles were simulated on each side: superficial, deep anterior and posterior masseter, anterior and posterior temporalis, medial pterygoid, superior and inferior lateral pterygoid, digastric, geniohyoid, and anterior and posterior mylohyoid. The resistance of a food bolus on muscle and TMJ loads was also investigated during jaw closing. TMJ loads reached 85, 45, and 15 N for 100, 50, and 10 % activation of the opener muscles, respectively. Load was present on the TMJs because the muscles responsible for opening the jaw had to overcome the increasing passive tension of the jaw closing muscles as opening progressed. For jaw closing muscle activations of 10 % or more, the lateral pterygoid had to be activated to 100 % to prevent jaw dislocation. The TMJ loads were 90 and 10 N for 10 and 1 % activation of the jaw muscle closers, respectively. For a 50 N incisal resistance load from food, TMJ loads increased to 145 N bilaterally. For an 80 N unilateral second molar resistance load from food, the TMJ load was 145 N on the balancing side and 110 N on the working side.

Another study from the same group compared the predicted TMJ loads during unloaded maximal jaw opening and closing [59]. Muscle activation profiles were applied according to previously reported EMG data [45] and reached a maximum of 50 % for opener muscles and 4 % for closer muscles. Opening had higher TMJ load than closing (43 vs. 10 N). The kinematics of the jaw predicted by the model were not symmetric for opening and closing; the jaw moved 0.45 mm more anteriorly during opening than closing. TMJ loads were always greater, and the jaw always moved farther anteriorly than closing during jaw opening even for variations of 25–75 % in maximum muscle activation levels of the opener muscles and 2–8 % in maximum muscle activation levels of the closer muscles.

Peck et al. studied *loaded* wide jaw opening using a dynamic model [60]. The force needed to push or pull the mouth open was measured with a transducer in five subjects. The force was then applied to the dynamic model and wide opening replicated. The effect of articular eminence shape was also investigated. Eight muscles were modeled: anterior temporalis, middle temporalis, posterior temporalis, superficial masseter, deep masseter, medial pterygoid, lateral pterygoid, and anterior digastric. A 5 N load could pull the jaw open to 50 mm in the tested subjects. To replicate this with the mathematical model, the jaw closer muscles needed some activity (0.18 %) to maintain the correct resting jaw position. Opening was attained

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using by activations of the opener muscles of approximately 25–30 %. The model predicted maximum TMJ loads of 28 N during wide opening and a steep articular eminence contact surface. The anterior digastric and lateral pterygoid muscle forces reached a maximum of 11.6 and 16.8 N, respectively. Similar to that found by Koolstra et al. [37], the closer muscles contributed passively to TMJ loads during unloaded jaw opening. The middle temporalis had the highest maximum force resulting from passive tension (10.1 N).

#### 1.3.3.3 TMJ TJR Finite Element Modeling

Finite element analysis (FEA) is used across a broad array of industries and research to investigate internal loadings. In biological engineering applications which investigate joint mechanics, FEA is widely used to model bone and other biologic tissues, the interfaces between bone and implants, and total joint replacements. FEA is useful to investigate situations not easily experimentally investigated and for performing parametric studies where single variables are changed in each analysis. FEA models require input data including kinematics and kinetics and require assumptions of the boundary conditions. Like the mathematical models described above, FEA models must also undergo rigorous verification and validation in order to ensure their accuracy and predictive ability. FEA for the investigation of TMJ TJR has been performed to investigate the interface between the TJR, screws, and bone of the mandibular component [61–70], to investigate TJR geometry [66, 71, 72], and to investigate different loading conditions [73–75].

## 1.4 Summary

Although many sophisticated models have been created to investigate TMJ biomechanics, there is a need for mathematical models of the mandible implanted with bilateral or unilateral TMJ TJRs to reflect the unique model assumptions necessary for TMJ TJRs. Besides the obvious geometrical differences, the muscle forces may be drastically different due to the surgery and previous conditions. A coronoidectomy is often performed during TJR surgery; therefore, temporalis function is compromised [9]. This affects the ability to produce vertical force between the teeth. TMJ TJR patients also often have decreased muscle tone in the masseter and medial pterygoid [9].

Because mathematical models are necessary for prediction of muscle forces and joint loads, one of the biggest challenges to determining TMJ TJR biomechanics is the lack of data for validating mathematical models. Models must be validated to ensure that they are applicable to in vivo conditions. Validation is usually performed with direct in vivo measurements or data from in vitro experiments of which little information is available. Without experimental and in vivo data for model validation, it is difficult to determine what simplifications are acceptable and ensure that accurate predictions of TMJ TJR behavior can be made.

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# Chapter 2
# TMJ TJR Biomaterials

**Robert E. Baier and Anne E. Meyer**

## 2.1 Introduction

All engineering constructs require appropriate materials to meet safety and effectiveness standards. It is an unfortunate observation that in the decades of the 1970s and 1980s, synthetic materials were utilized in the management of temporomandibular joint (TMJ) pathology that proved to be inappropriate and—sometimes—even dangerous. This was a failure that remains an embarrassment to the biomaterials community and to the bioengineering designers who selected those materials and are now redoubling their current efforts to do better.

The biomaterials available for implants have been surveyed as well as their sterilization and preparation for implantation [1]. Material wear under functional loading as demonstrated in Fig. 2.1 from a retrieved, failed TMJ ramus component with a polymethylmethacrylate (PMMA) condyle bearing surface was and is still a serious warning that assumptions about the TMJ being an "unloaded" joint were flawed [2]. Some many billions of bacterial-sized particles were distributed into the tissues surrounding the TMJ, generating inflammatory conditions that caused patient-reported pain and suffering, plus loss of jaw function.

Metal-on-metal articulations, using cobalt-chromium-molybdenum alloys, demonstrated metallic wear particles that although less obvious were more troublesome [3, 4]. Figure 2.2 illustrates a failed metal-on-metal (MoM) TMJ total joint replacement (TJR) explant from an era when its manufacturer judged "…this suggests that the smaller amount of particulate metal debris generated by cemented cobalt–chromium alloy prostheses may be due to better wear resistance…" [3].

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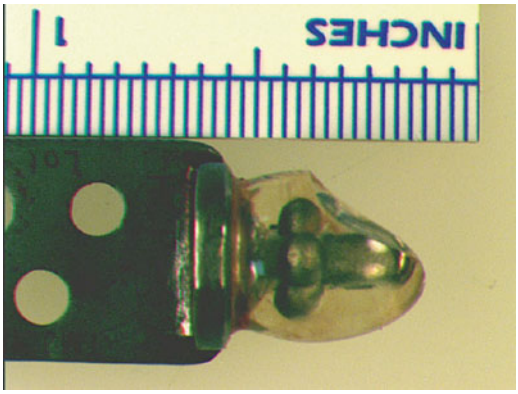


**Fig. 2.1** Condyle of TMJ implant, removed from patient after significant material lost from polymeric component

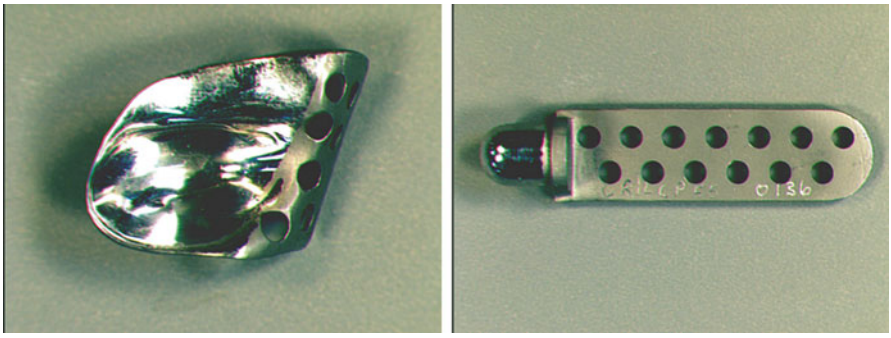

**Fig. 2.2** Components of a metal-on-metal TMJ implant after removal from a patient. Close inspection of the articulating components demonstrated degradation and wear [*Note*: Photos are of different magnifications]

Another problem is that if fixation screws of different material compositions are utilized, the local tissue reaction to the dissimilar metal electrochemical gradient can lead to osteolysis, loosening, and ultimately device failure. Less obvious is the MoM wear-producing where coefficients of friction (CoF) >0.6 are reached, whereas natural joints display a much lower CoF (<0.1).

Regulatory concerns and legal liabilities are so great that introduction of new biomaterials for prosthetic uses is a difficult and expensive commercial endeavor [5]. So bioengineers are forced to modify both the engineering designs and surface properties of established devices to meet the continuing patient needs [6]. Likely improvements will come from the use of clinically proven structural ceramics and a return to a low-friction bioengineered TMJ articulation that mimics nature (See Chap. 12 by Feinberg).

Anticipating improved regulatory oversight, any new implantable devices should not be introduced by simple replacement. Potential new biomaterials and implants

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must forecast and accept TMJ loading requirements (see Chap. 1 by Lundberg) and the probable consequences of future wear particle generation (see Chap. 10 by Mathew).

Following the trend of coating of dental and orthopedic implants with layers of calcium hydroxyapatite (HA), manufacturers introduced Proplast (PTFE)-HA (Vitek, Houston, TX) coating onto TMJ implants. When wear resulted in the release of PTFE-HA particles into the adjacent tissues, it triggered differentiation of monocytes to osteoclasts that in some cases perforated skull base structures [7].

This and a simultaneous problem arising from the use of silicone rubber caused the FDA to force manufacturers to remove these products from the medical/dental marketplace. Biomaterial suppliers followed suit, and this limitation of material supply has not been adequately resolved by subsequent "hold harmless" federal legislation [8], which many corporate attorneys considered inadequate.

## 2.2 Materials Utilized in the Manufacture of TMJ TJR Fossae and Ramus/Condyle Components

Currently, the FDA-approved materials for use in the manufacturing of alloplastic temporomandibular joint replacement (TMJ TJR) devices are cobalt–chromium alloys (Co–Cr–Mo), commercially pure titanium (cpTi), alloyed titanium (Ti6Al4V), and ultrahigh molecular weight polyethylene (UHMWPE). There are no materials that are automatically "biocompatible," since this is a single word that requires both *safety* and *effectiveness* for the intended function. The first challenge from a material standpoint came in 1960 from Sir John Charnley who developed an alloplastic total hip joint device with metal femoral stem and perfluorocarbon (Teflon™) acetabular cup. This combination resulted in excessive particulate wear and subsequent failure.

This joint replacement system later utilized a metal-backed UHMWPE acetabular cup articulating with a stainless steel femoral head component that was cemented in place with PMMA [9]. Modifications of this device utilizing titanium, titanium alloy, and cobalt–chromium–molybdenum alloys have now become standard for low-friction total joint arthroplasty in orthopedic surgery [10] (Tables 2.1 and 2.2).

**Table 2.1** Titanium and titanium alloy

| |
|---|
| Commercially pure titanium (>99 %) spontaneously acquires a protective oxide having free-radical-scavenging properties thought to be critical to osseointegration (close approximation to the bone, making a biomechanically sound bone/implant unit) |
| Titanium6Aluminum4Vanadium alloy has much lower free-radical activity, does not osseointegrate well, but is closer in stiffness to the bone and more easily machined |
| Neither Ti nor TiAl6V4, through the predominantly titanium dioxide (TiO2) surface layers, provides low friction with tissues or other materials |

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**Table 2.2** Cobalt–chromium–molybdenum alloys

| |
|---|
| The alloy's surface oxides, as with titanium and its alloys, provide excellent corrosion and pitting resistance. When polished, these alloys usually acquire permanent molecular-level coatings of abrasive-carrying fatty acids that convert the original "lost wax" castings to a low-surface-energy and hydrophobic character that minimizes subsequent bonding potential |
| In the alloys' clean states, bone adhesive will attach well. In their coated states, they are used for the blood clot-resisting struts of heart valves. This is an example of where the one word "biocompatible" is *not* adequately descriptive, since the "bio" needs differ substantially from place to place. It is important to specify both their safety *and* effectiveness for the intended use |
| Much higher modulus than bone, occasionally lending to stress shielding and bone failure |

## 2.3 Titanium (ASTM F-64) and Ti6AlV4 Alloy

Unalloyed titanium was chosen for endosteal implants and bone plate fixation devices because the element was always covered with a thin ($\ll$10 μm), free-radical-reactive but corrosion-resistant oxide ($TiO_2$). This metal-oxide layer provides a favorable surface for osseointegration of device components with the host bone. This quality, along with its strength and machinability, coupled with the extensive literature demonstrating its suitability when used in appropriate clinical applications, makes titanium the metal of choice for the manufacture of the major structural components of alloplastic total joint devices and dental implants [11].

The alloy of titanium (Ti), aluminum (Al), and vanadium (V) (Ti6Al4V) combines relatively high mechanical strength, ductility, and resistance to pitting and crevicular corrosion. This alloy also forms the aforementioned protective surface oxide in air and fluids, but that oxide is modified by the alloying components and does not demonstrate the same beneficial reactivity with free radicals generated during the inflammatory processes associated with implantation.

Alloyed or not, titanium is not optimal for bearing surfaces for total joint components. Laboratory data from joint simulators have shown titanium and its alloys to be more subject to contact surface wear compared to cobalt chromium alloys or smooth ceramics when articulated against polyethylene [11]. Under load, unalloyed and alloyed titanium is susceptible to abrasion, fretting, and galling if exposed to frictional sliding motions, resulting in the formation of debris which can lead to third body wear, foreign body reactions, host bone osteolysis, and failure of the devices [12].

Attempting to overcome low indentation hardness and wear resistance of titanium, nitrogen ion implantation and chemical nitriding have been used to prepare titanium as an articulating surface, but limitations have been noted when exposed to third body abrasion and wear phenomena [12] (see Chap. 10 by Mathew).

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## 2.4 Cobalt–Chromium–Molybdenum Alloys (ASTM F-75)

The cobalt-based alloys were chosen for early orthopedic device components because these materials could be cast easily into component shapes, the material could be polished to a smooth surface, the final product was relatively hard and strong, and they were biocompatible and appeared wear and fretting resistant in early testing [11].

Cobalt (Co), chromium (Cr), and molybdenum (Mo) are the primary elements in a cobalt alloy system. The refining process results in approximately 1 % nickel (12) in the cobalt constitution, most of which is retained in the final alloy, but it is not always present in the final prostheses. Chromium adds strength and chemical inertness through the formation of a chromium oxide passivation layer. The molybdenum provides resistance to corrosion, especially pitting and crevicular corrosion, and adds strength to the alloy [12].

These properties led to the use of cast cobalt alloys in MoM total joint systems that clearly showed fretting and wear, as well as porosities in thin section castings. These findings, along with the material's high modulus of elasticity and low fatigue strength, led to device component fractures, pain, loosening, and subsequent failures [11, 12].

MoM cobalt–chrome alloy total hip replacement systems are in a second cycle of being removed from the market. These MoM devices appear to function well only where the articulation is at least semi-constrained. Even perfect congruency, precision, and accuracy between the articulating components cannot assure that a MoM articulating system can function with limited wear, fatigue fracture, and failure. MoM geometry has been all but abandoned by the orthopedic community, even though dimensional tolerances less than 0.001 in. (25 μm) with an interface of 200–300 μm had been achieved [12].

The TMJ is not a constrained joint, but has rotational, translational, and lateral movements due to the multi-vector force influence of the masticatory muscles on the mandible. These basic anatomical functional characteristics make it an unsuitable joint for MoM TMJ TJR devices.

Cobalt–chromium–molybdenum alloy, with its ability to be cast, strength, polishability, and biocompatibility as well as its excellent wear characteristics as a bearing surface against a UHMWPE fossa, presently makes it the standard for the condylar component in orthopedic and TMJ TJR systems.

Research into the use of ceramics such as aluminum and zirconium oxides as the mobile-bearing surface in total joint systems was prompted by the decrease in wear

**Table 2.3** Ultrahigh molecular weight polyethylene (UHMWPE)

| |
|---|
| Extremely low moisture absorption and low coefficient of friction, but still subject to frictional wear and particle production, especially after sterilization-induced oxidation |
| Can apparently be improved in wear resistance by additional radiation treatment beyond that required for sterilization. Possible simultaneous surface energy increases can predispose to tissue invasion and attachment |
| High impact strength, but particle production remains a problem for generating aseptic device loosening via inflammatory processes |

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exhibited by these materials when compared to cobalt–chrome against UHMWPE. To date, there are no total TMJ reconstruction systems utilizing ceramic condylar heads, but proposals to employ the new generation of structural ceramics are emerging [13].

## 2.5 Ultrahigh Molecular Weight Polyethylene

After prompting by international groups of orthopedic surgeons, Charnley introduced high molecular weight polyethylene (UHMWPE) as a bearing surface in total hip replacement in 1962 as a replacement for polytetrafluoroethylene (PTFE [Teflon™]) which was found to have poor wear properties under load. Failed PTFE caused the formation of large volumes of intra-articular wear debris, massive foreign body giant cell granulomas, and catastrophic device failures before it was abandoned [14]. This scenario was unfortunately reproduced with the use of Proplast-Teflon (Vitek, Houston, TX) in the TMJ two decades later.

UHMWPE is a linear unbranched polyethylene chain with a molecular weight of more than one million. Most medical grade UHMWPE used today has a molecular weight of three to six million [14], but extra-high irradiation shows promise of increasing that value and decreasing wear susceptibility [15].

UHMWPE is characterized as ductile, with a low coefficient of friction (<0.3) and high tensile strength making it an ideal material to form the stable articulating component for a total joint replacement system. UHMWPE rarely fails catastrophically because of a single high stress or strain exceeding yield or break strains. Rather, it may fail because of wear or fatigue damage under repeated loading.

UHMWPE is considered to have excellent wear and fatigue resistance for a polymeric material, although osteolytic response to its wear debris in orthopedic joints continues to drive efforts to reduce wear further [14]. There are still a number of efforts being made to improve the clinical performance of polyethylene by making it stronger, more scratch resistant with more resistance to fatigue and chemical degradation. The most promising approach appears to be strengthening UHMWPE by cross-linking the polymer chains with covalent bonds to convert the linear chains into an interconnected three-dimensional network. Cross-linking can be achieved by physical or chemical means. A potential drawback to cross-linking would be loss of ductility and fatigue life. Further research is ongoing to determine the long-term potential for this technology [15].

## 2.6 Polyethylene Wear Particulation and Third Body Wear Phenomena

Polyethylene debris is the most common wear particle isolated from the tissues following total hip revision surgery. Polyethylene wear has been directly linked to aseptic loosening of cemented acetabular components via a process of macrophage-mediated foreign body reaction to these particles.

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The TMJ, especially in the multiply operated patient, is variably exposed to functional loads, but none as great as to which the hip is exposed. TMJ TJR involves the elimination of both the functional influence of the lateral pterygoid and temporalis muscles on mandibular function. This decreases the bite force by approximately 50 %; therefore, the subsequent load delivered to the polyethylene is theoretically reduced. Patient-fitted TMJ TJR devices are designed and manufactured to comply with the host bone anatomy. The bearing surface geometry of these patient-fitted TMJ TJR devices is designed so that there is no period when the condylar component is articulating in an aberrant pattern that might exacerbate polymer wear debris. However, clinically this cannot be guaranteed. Finally, over the many years of use of total TMJ devices utilizing a UHMWPE fossa, none has been documented to require removal due to polymeric particulation. Mercuri has reported in 2 publications the histology of intra-articular fibrous tissues removed at 1, 2, and 5 years from patients functioning with a cobalt–chromium–molybdenum/UHMWPE articulation with no evidence of polymer particulation failure [16, 17].

Third body wear is a phenomenon which occurs when polymer, cement, or metal debris particles become trapped between the bearing surfaces of a joint replacement device creating excessive abrasion and wear at the bearing surfaces. This leads to inflammation, macrophage-mediated foreign body reaction, osteolysis, loosening of the components, and ultimately device failure.

However, there still are patients with PMMA condyles articulating with cobalt–chrome alloy fossa liners where the worn PMMA condyle exposes the metal trunnion to the metal fossa (Fig. 2.1). The particulation resulting from the worn PMMA condyle can lead to the aforementioned third body wear phenomenon, foreign body reaction, and failure of the device. The MoM joints and use of nitrided titanium condyles against UHMWPE discussed above have similar potentials for wear and particulation device failure (Fig. 2.2).

In summary, UHMWPE today remains the fixed component bearing surface of choice in TMJ TJR, as it has been for over 30 years in orthopedic TJR. No other polymer has performed as well in this demanding application. Despite the success of this material, there remain opportunities for improvement in wear and particulation issues.

It is clear that TMJ TJR fabrication utilizing computer-aided design and computer-aided manufacturing (CAD/CAM) processes developed from protocol patient imaging has made great strides in improving these devices. Utilization of such prostheses to reconstruct patients with major TMJ and mandibular defects has been reported to have good long-term outcomes [17–19]. The use of commercially pure titanium (cpTi) mesh as the backing for the fossa component provides the significant advantage of promoting osseous ingrowth stability to that component.

## 2.7 Surface Quality of Implanted Devices

There are important surface qualities based on extensive analyses of various prosthetic devices that must be addressed [10]. First, the surface cleanliness and surface free energy qualities of such biomaterials have been noted to directly affect their

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acceptance in the biologic environment. “Many available studies of biomedical implants have focused on the long-term behavior of the materials without initially characterizing the devices prior to placement. This is a crucial point…” [20]. The elemental and chemical states at the surface of any device vary a great deal from standard bulk values. This is particularly true of the above-noted alloy materials which are known to exhibit surface enrichment of certain elements although constituting only minor proportions in the bulk alloy.

Reported modifications have identified changes from machining, polishing, and sterilization, consisting of oxide growth, environmental contamination, and the blooming of unintentionally added impurities (in some instances, lead in dental implant-grade titanium) [20]. What is desired, if the tissue reaction to the material is favorable, is integration of the implant material into the host tissues. Commercially pure titanium has been shown to exhibit such a favorable reaction (osseointegration), while the titanium alloy has been reported to be not as favorably reactive [21, 22]. Osseointegration depends on the appropriate titanium oxide’s integration with living Haversian bone while in contact with load-bearing titanium [23, 24]

Electron spectroscopy for chemical analysis (ESCA or XPS), Auger electron spectroscopy (AES or SAM), and surface energy evaluations (by critical surface tension and contact angle measurements) have been the dominant methods for the analysis of surface qualities [20, 25]. These tools specifically supply data on the outermost 1–10 nm of an implant material, and AES supplies both lateral specificity and depth profiling. This synergistic information is crucial to proper material selection. Of special concern is that various sterilization methods drastically affect the surface chemistry of the metals and alloys, sometimes even leading to inadvertent carbon overcoats and reactive debris [26, 27].

## 2.8 Future Prospects

Early evaluations of CAD/CAM-generated fossa and condyle models (Fig. 2.3) for one patient-specific device constructed from lithium disilicate (LS2) dental ceramic have shown excellent bone bonding prospects with no troubling screw fixation and with a large safety factor as evaluated by finite element analysis (FEA) methods [13]. Following observations in orthopedics, the ceramic-on-ceramic hardness minimizes wear and particle generation but can create annoying “squeaking” sound during function. A squeak-suppressing ceramic-bound dry monolayer lubricant, based on the concept of a favorably low critical surface tension (CST) [28], is being tested, but it risks being worn away faster in the TMJ than in nonabrasive blood [29] which has shown clinically thrombo-resistant performance for over 40 years [30].

Near-term, it is likely that the CAD/CAM-fabricated custom fitting TMJ TJR devices will continue to dominate the field. Wear-resistant UHMWPE materials will substitute advantageously for the lower molecular weight and less cross-linked polymers that have heretofore been available.

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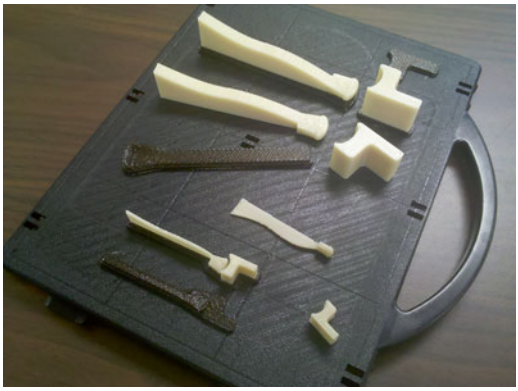

**Fig. 2.3** CAD/CAM-produced prototype models of fossa and condyle components. The *light-colored* model components shown in this photo are an ABS polymer. Lithium disilicate dental ceramic is proposed for CAD/CAM production of clinical components

For the longer term, favorable imaging, computational, prototyping, and adhesive bonding results bode well for a possible material conversion to well-performing ceramics for an improved bioengineered TMJ TJR construct. Because of the low-friction requirements in the TMJ, these may have to accommodate polyurethane-bound inserts of hyaluronic acid-supplemented tough tissue (like glutaraldehyde-preserved pericardium [31]) that will produce very small but metabolizable wear debris particles.

As an intermediate "fix," such tissues could be considered to be bonded to the bearing surfaces of existing TMJ TJR devices to take advantage of the significantly reduced coefficients of friction possible in tissue-on-tissue articulations [32]. Recent literature supports these concepts: "…boundary lubrication can be crucial for the disc. Therefore, the morphological integrity, surface roughness, and efficacy of joint lubricants appear to be critical in minimizing disc friction. In addition, the importance of boundary lubricants in mitigating disc friction may lend support to the debated practice of intra-articular lubricant injections" [31, 33].

All these forecasts are based on the conclusion that existing FDA-approved biomaterials will not be supplemented in the near future, for both regulatory and litigious reasons, while their modifications for alternative uses will be acceptable.

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# Chapter 3
# History of TMJ TJR

**Louis G. Mercuri**

> *"Medicine like all knowledge has a past as well as a present and a future and … in that past is the soil out of which improvement must grow."*
>
> *Alfred Stillé 1813–1900 [1]*

Revisiting the past is an important first step in understanding the use of alloplastic materials in temporomandibular joint (TMJ) reconstruction. The literature cited is intended to demonstrate first, how in the past many surgeons recognized the need for alloplastic TMJ reconstruction devices for the management of particularly difficult clinical situations; and secondly, how the materials in those devices mirrored their introduction into industry, science, and medicine. Further, the history of the resulting material failures in some cases that raised concerns about the use of TMJ devices will be discussed.

## 3.1 Historical Perspective

The hieroglyphics dating back to 5000 BC mention the problem of the ankylosis of joints and the management of jaw dislocation [2]. The first written account of joint surgery was by a French barber-surgeon of the Renaissance, Ambrose Pare, who in 1536 performed the first joint excision on a patient with a destructive infection of the elbow [3]. Between 1536 and 1840, surgical excision was the only treatment reported for severe joint disease [4, 5]. In 1778, John Hunter was among the first to explore the surgical management of ankylosis of human joints [6]. Barton, in 1826, proposed the concept of pseudo-articulation in the treatment of ankylosis of the extremities [7].

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In 1840, a New York surgeon John Murray Carnochan was credited with the idea of interposing material between the surfaces of a diseased joint. He reported an attempt to mobilize a patient's ankylosed TMJ by placing a small block of wood between the raw bony surfaces of the residual mandible after creating a gap at the neck of the condyle [8]. In 1891, Gluck reported total joint arthroplasties using ivory prosthetic TMJ and hip joints which he stabilized with cement made of colophony, pumice, and gypsum [9].

During the intervening period between these reports and the 1980s, the use of alloplastic materials in the TMJ was primarily for the management of ankylosis, reconstruction of mandibular function and form after ablative tumor surgery, trauma, and degenerative disease.

As any new alloplastic material was introduced, surgeons confronted with these difficult clinical conditions attempted incorporating that material into a TMJ device to manage these dysfunctions [10–14]. It should be noted that in most instances, early reports were single cases, and often the follow-up was typically less than a year with the only criteria for success being that the patient could open their mouth, if they were reported at all.

## 3.2 Silicone Elastomers

Silicone elastomers are elastic materials that contain linear silicone polymers that are cross-linked in a 3-dimensional network. Silicone rubber, one form of inorganic synthetic silicone elastomer, is made from a cross-linked, silicon-based polymer strengthened with a filler that acts as a reinforcing agent to impart certain mechanical, chemical, or physical properties. In general all silicones (polydimethyl siloxanes) are noted for their high thermal stability, biocompatibility, hydrophobic nature, and electrical and release properties [15, 16].

Because of these properties, and its ease of manufacturing and shaping, silicone rubber is used in a wide variety of products including automobiles; products for cooking, baking, and storage of food; clothes such as underwear, sportswear, and footwear; electronics; medical devices and implants; and in home repair and hardware products such as silicone sealants [16].

Silastic,® a combination of the words "Silicone" and "Plastic," was patented by Dow Corning (Midland, MI, USA) in 1948 and refers to silicone elastomers, silicone tubing, and some cross-linked polydimethyl siloxane materials that they produce [17, 18].

After reports of experimental evidence that silicone rubber was biologically inert when used as a joint replacement material in 1966 [18], in 1968, it was introduced to the medical community as an interpositional material in the reconstruction of arthritic or destroyed joints in the hand [19, 20]. Braley remarked that a new era in the use of non-autogenous implants was developing. He further commented on the astonishing lack of reaction evoked by medical grade silicones, and how this material was opening many surgical doors to the reconstructive surgeon, but suggested

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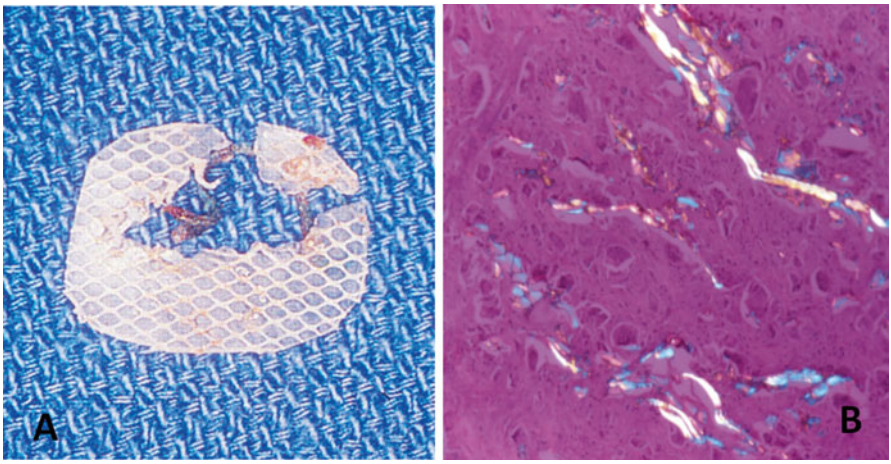


**Fig. 3.1** (**a**) Silastic TMJ implant reinforced with polyethylene terephthalate fibers that was implanted for 3 weeks in a TMD patient. (**b**) Particles of silicone rubber in the tissue

careful enthusiasm [21]. Braley cited the work of Brown et al. who reported on the use of silicone rubber to prevent the reformation of TMJ ankylosis. These surgeons removed the silicone rubber from the TMJ after healing had occurred, allowing the reactive fibrous capsule that formed around the implant to act as a deterrent to re-ankylosis [22]. In 1968, Robinson reported using Silastic (Dow Corning, Midland, MI) in a case of TMJ ankylosis [23]. The long-term results or complications of its use were seldom if ever reported [24–45].

With the implication of internal derangement of the intraarticular disc in the etiology of TMJ pain and dysfunction, the repair or removal or both in TMJ discs increased [46]. In an earlier report, Gordon had introduced the use of polyethylene caps as interpositional alloplastic implants after discectomy [47].

Silastic TMJ implants of 1–2 mm thickness were reinforced with polyethylene terephthalate fibers [48, 49]. Studies that advocated implantation of these devices in the short-term reported their ability to form a fibrous capsule of connective tissue that might act as a substitute disc [50, 51] (Fig. 3.1).

However, as early as 1983 reports began to be published that implantation of Silastic into a functioning TMJ was associated with complications in both clinical and animal studies. Small particles of Silastic were also found in the regional lymph nodes adjacent to the site of implantation of silicone rubber into the TMJ [52–59]. A local inflammatory response was also reported in animal studies [60–62].

In a 5-year follow-up clinical and radiographic study of 43 patients who had had discectomies, 22 with temporary implantation of Silastic sheeting and 21 with no implants, Eriksson and Westesson found that all patients with poor clinical outcomes had Silastic TMJ implants; and erosive changes of the condyle were seen in them all. They concluded that the use of temporary silicone rubber implants after discectomy for treatment of internal derangement should be seriously questioned [63].

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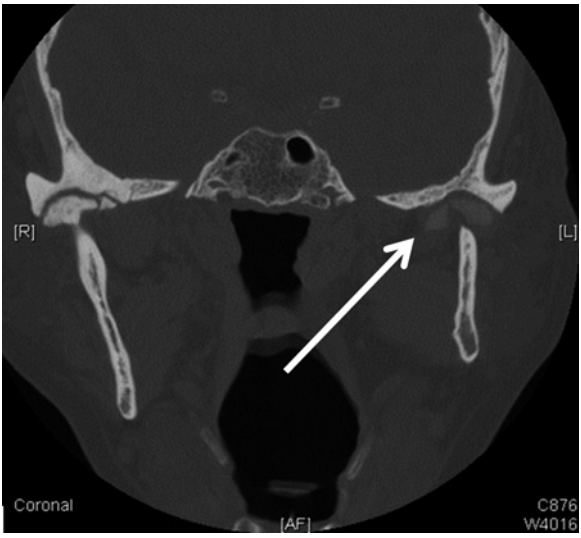


**Fig. 3.2** Coronal CT of left TMJ demonstrating fractured silicone rubber implant placed 10 years prior to manage ankylosis

Medical journals also published articles about foreign body reactions to silicone rubber that had been used as interpositional articular devices. These reactions were reported both in vivo [64–71] and in vitro [72–75].

In November 1992 the American Association of Oral and Maxillofacial Surgeons convened a 2½ days workshop to develop a consensus concerning the use of interpositional and reconstructive materials in the TMJ. The results of that workshop were published in 1993 [76].

With regard to Silastic, there was the consensus that the use of permanent Silastic implants should be discontinued, except when used to prevent recurrence of ankylosis opinions differed concerning the use of temporary reinforced Silastic sheeting after discectomy. Dow Corning Wright discontinued manufacture and distribution of Silastic HP Sheeting and Silastic TMJ Implant (Wilkes Design [77]) effective from their letter dated 25 January 1993. However, some surgeons continue to use this technique. In the light of this information the use of interpositional silicone elastomer products as disc replacements in a functioning TMJ should be questioned [15] (Fig. 3.2).

## 3.3 Teflon and Proplast

Small experimented with the use of Teflon (polytetrafluoroethylene) (DuPont, Wilmington, DE) and Silastic as materials for TMJ and mandibular reconstruction. In 1964, he reported that Teflon seemed more adaptable to restoration of large

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mandibular resections, whereas Silastic seemed better suited for replacement of the condyle [78].

In 1972, Cook reported in 2 different animal studies lack of inflammatory reaction when Teflon cloth was placed between resected condyle and residual mandible followed 1 year. Further, he reported the successful use of this material as an interpositional material in 4 human TMJ cases followed 18 months [79], despite evidence published by Charnley [80] and Scales and Stimson [81] that Teflon under functional loading underwent fragmentation resulting in foreign body giant cell reactions in the hip. However, Cook did not feel that the TMJ was a loaded joint and therefore Teflon would not fragment [79].

In the late 1970s, Proplast, the porous form of Teflon (polytetrafluoroethylene [PTFE]) was fused with vitreous carbon (Proplast I), aluminum oxide (Proplast II), or synthetic hydroxylapatite (Proplast HA) by the Vitek, Inc. (Houston, TX). The interpositional TMJ implant that resulted was a laminate of either Proplast I or II and Teflon sheeting. The Proplast component was designed to be placed against the fossa temporal bone to encourage the ingrowth of tissue to stabilize the implant. The smooth Teflon portion was designed to function against the condyle [82].

Homsy et al. demonstrated the ingrowth of fibrous tissue into PTFE-pyrolytic graphite [82]. In a 1973 publication, Homsy et al. reported the presence of giant cells around these implants, but apparently did understand their significance [83]. In a presentation to the US FDA in 1989, Homsy again recognized the presence of macrophage and macrophage polykarons around these implants but felt they were "non-morbid" and might be contributory to normal healing [84] (Fig. 3.3).

Several authors reported successful management of TMJ symptoms after implantation of the Vitek Proplast-Teflon interpositional implant (IPI) [85–90]. They all reported a high level of patient satisfaction and function; however, severe post-discectomy changes in condylar bony architecture were seen. McBride and Ware reported 71 % of their cases showed severe TMJ bony osteoarthritic changes [87, 88].

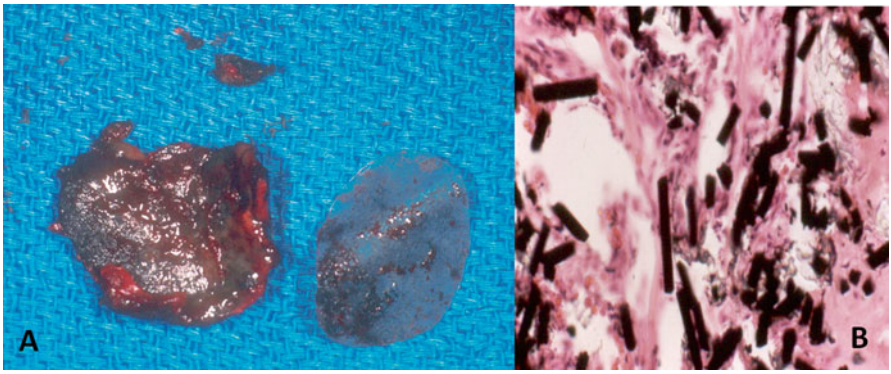


**Fig. 3.3** (**a**) Delaminated initial iteration of the proplast-Teflon interpositional TMJ implant. (**b**) Foreign body reaction to Proplast particles

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Ryan [49] reported the development of an anterior open bite in 20 % of his IPI patients on recall. He felt this was due to degeneration of the mandibular condyles; however, the etiology of this degeneration was not identified.

Timmis et al. demonstrated marked osteoclastic activity with resorption and severe bony degeneration in 46 % of the rabbit condyles where the TMJ disc was replaced with either Silastic or the IPI [60].

Lagrotteria et al. reported lymph node involvement with foreign body giant cell reaction in a patient due to the breakdown of the IPI [91]. Florine et al. reviewed tomograms of 18 IPI cases followed for more than 2 years and found 72 % had severe condylar degeneration. They concluded that this material may result in increased postoperative complications and adversely affect long-term results. These authors recommended further investigation using other techniques and correlation with their clinical findings to place these findings into proper prospective [92].

Heffez et al. reported on a 2-year follow-up CT scan study of 12 TMJs implanted with the IPI. The results revealed severe condylar, glenoid fossa, and articular eminence remodeling changes; implant migration and fragmentation; and loss of implant adaptation to the temporal bone. However, these authors reported that the patients were clinically asymptomatic. They concluded that the risk of implant displacement and fragmentation may outweigh the benefits of its use as a disc substitute [93].

Bronstein reported on the results of a retrospective study of 12 patients who had been implanted with an IPI after an average of 15 months, and 6 patients implanted with Silastic followed an average of 36 months. He found that the IPI produced a more severe bony response of flattening and sclerosis of the fossa and condylar resorption. He offered no reasons for these findings but concluded that patients with both types of implants should be closely monitored and that these implants should be removed before they become symptomatic [94].

In 1988, Morgan presented a review of the development and approval of the IPI, followed by a report of 3 cases where those implants fragmented resulting in severe pain and TMJ bony degeneration requiring total joint reconstruction. Morgan further commented that surgeons do not know the long-term effect of the IPI failures on the joint tissues after the implant has been removed [95].

The reports of radiographic changes in TMJ articular bones were not restricted to the surgical literature [55, 56]. In 1988, Kaplan et al. reported 6 patients with destructive osseous changes in the TMJ an average of 38 months after placement of an IPI. She also reported that at surgery to remove these implants, foreign body reactions were found which she stated accounted for the radiographic findings. She concluded that further studies such as MRI or CT may show abnormalities before osseous destruction [96].

Schellhas et al. reported MRI findings of 30 patients, 34 TMJs, in which there were locally destructive bone and soft tissue complications identified 4–54 months post-implantation of an IPI. They concluded that MRI was useful in detection and evaluation of these destructive complications and that tomography more accurately delineated soft tissue calcifications and cortical margins of the involved osseous structures [97]. Katzberg and Laskin, in a commentary on Schellhas' article, concluded by stating that they wished to emphasize the need for greater clinical aware-

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ness of this problem and increased attentiveness to early warning signs of IPI failures. Further, they recommended careful monitoring of patients with all types of alloplastic implants for similar problems [98].

Florine et al. retrospectively studied 55 IPI and 18 disc repair patients for 20 and 48 months, respectively. Greater than 60 % of the TMJs with an IPI demonstrated severe destructive TMJ osseous changes, whereas none of the disc repair patients showed any such changes. They speculated that the size and number of fragmented particles from a failed IPI probably exceeded the capacity of lymphatic system to remove them [99].

El-Deeb et al. investigated the use of the Proplast in non-weight-bearing areas and found fragmentation, giant cell foreign body reaction, collapse of the Proplast, and loss of the inter-bridging fibrous tissue connections. They speculated that the latter would lead to decreased stability and increased foreign body giant cell reaction. These authors suggested that a similar phenomenon might be the cause of the reactions seen when Proplast was used in the weight-bearing TMJ as part of the IPI [100].

Valentine et al. reported on the results of a light and electron microscopic study of tissue removed from 9 patients, 14 TMJs, where an IPI had been in place from 10 to 28 months. These implants were removed due to complaints of pain, occlusal changes, or radiographic changes. Evidence of gross deterioration of the implant, manifested as fracture, was present in 10 of the 14 implants, and microscopic evidence of deterioration was seen in all cases as were foreign body giant cell reactions. These authors concluded that micro-fragmentation of the IPI contributed to the induction of the foreign body reaction. Further, they stated that since multinucleated giant cells are derived from the same precursors as osteoclasts, they were considered to be osteoclasts in these cases since these cells were in the adjacent degenerating bone. Therefore, they felt it was reasonable to believe that the stimulation of these cells by the IPI failure was responsible for the bony changes found [101] (Fig. 3.4).

In December 1990, The United States Department of Health and Human Services, Public Health Service, FDA, Center for Devices and Radiological Health issued a Safety Alert to oral and maxillofacial surgeons urging them to reexamine all patients

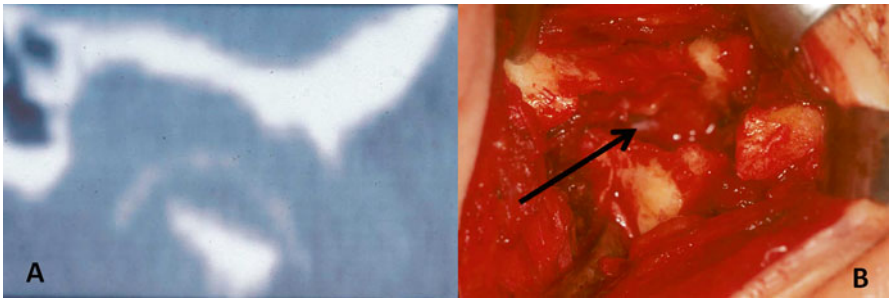


**Fig. 3.4** (**a**) Sagittal CT image of a right TMJ demonstrating the osseous damage resulting from the foreign body reaction to a failed Proplast-Teflon interpositional implant. (**b**) Intraoperative image of this joint at removal of the failed Proplast-Teflon interpositional implant. Note the loss of the zygomaticotemporal component of the zygomatic arch and perforation of the glenoid fossa into the middle cranial fossa (*arrow*)

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implanted with an IPI [102]. This Alert was based on data the agency culled from two Master's theses from the University of Iowa [103, 104] as well as a report by Wagner and Mosby [105].

Wagner and Mosby had reported that after a mean follow-up period of 36 months, 19 of the 20 patients (95 %) implanted with Proplast-Teflon interpositional TMJ implants reported severe pain. Malocclusion was found in 30 %, and 70 % demonstrated restricted mouth opening. Radiographic evaluation revealed 100 % of the condyles, and 68 % of the fossae had degenerated from presurgical levels. Tissue from all of the TMJs where these implants were removed showed histologic evidence of foreign body giant cell reactions. These authors concluded that this reaction was in response to the micro and/or macro particles of the Proplast-Teflon that had failed and that the foreign body giant cell reaction progressively destroyed both the condyle and fossa in their cases [105]. Further, these authors questioned the role of NaCl, an ingredient at the ratio of 80 % by volume in Proplast as reported by Homsy [106].

Estabrooks et al. then reported good results in a retrospective review of 301 TMJ meniscectomies with implantation of an IPI. They reported an 88.7 % success rate after an average follow-up of 33 months based on objective criteria. They presented only a 10 % failure rate but admitted that "many patients" had radiographic evidence of articular TMJ degeneration; however, they were asymptomatic [107].

Berman and Bronstein presented one case of an osteogenic rather the osteoclastic response 2 years after the implantation of a Proplast-Teflon interpositional TMJ implant. The implant was removed, an arthroplasty and temporalis flap reconstruction were performed. The tissue removed with the implant was consistent with the foreign body giant cell reaction reported in all other reports [108].

Three groups reported the complication of perforation into the middle cranial fossa with the degeneration of the fossa after failure of an IPI [109–111]. One reported a cerebral spinal fluid leak [110]. These authors concluded that these 3 perforation cases represented possible serious sequelae of the use of the IPI.

In September 1991, the United States Department of Health and Human Services, Public Health Service, Food and Drug Administration, Center for Devices and Radiological Health issued a Public Health Advisory to health professionals, hospital operating rooms, medical records and purchasing departments, and risk managers urging the recall and examination of patients implanted with an IPI [112]. In December 1991, The Food and Drug Administration's Medical Bulletin contained an item in which outlined the problems being encountered by patients and urged routine evaluations of patients implanted with these devices. They also stated that the probability of problems occurring increases the longer the implant is in place [113].

On June 4, 1992, prompted by problems resulting from the Vitek Proplast-Teflon TMJ implants, the United States House of Representatives government Operations Human Resources and Intergovernmental Relations Committee held a hearing. The focus of this hearing was whether the Food and Drug Administration and the National Institutes of Health had failed to act appropriately to protect the public from the inadequacies of the IPI. The hearing raised serious questions about these implants and their safety. As an outcome, oral and maxillofacial surgeons who contemplated continuing to implant TMJ devices were advised to carefully review

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all published data to determine whether the devices they might be using were safe and effective [114].

On August 10, 1992, the American Association of Oral and Maxillofacial Surgeons distributed by mail to all United States and Canadian fellows, members, life and retired fellows/members, candidates, residents, and affiliate members a TMJ Implant Advisory which was also published in the Journal of Oral and Maxillofacial Surgery. In that Advisory, they outlined the results of the June 4, 1992 House of Representatives hearing, outlined the FDA September 1991 Public Health Advisory, recommended recall of patients implanted with Proplast-Teflon, and outlined what the Association was presently doing internally and externally with the Food and Drug Administration to deal with this issue [115].

In October 1992, Spagnoli and Kent published the results of a retrospective study of IPI implants placed after discectomy in 680 TMJs, 465 patients, followed from 6 to 76 months. 584 of the 680 implants (85.9 %) were in place with a weighted average follow-up of nearly 32 months. 92.4 %, 540 joints, were asymptomatic. However, 224 asymptomatic (44.3 %) and 25 symptomatic (17.8 %) TMJs exhibited condylar resorption and 45 (4 %) had a malocclusion. They concluded that statistically, 54 % of the implants in the study may fail in 3 years. Since no one had reported any follow-up beyond 5 years, the long-term survival of these implants was doubtful in these authors' estimation [116]. These survival estimates were reiterated in a report by Fontenot and Kent in the same year [117].

Spagnoli and Kent recommended yearly evaluation of asymptomatic patients with tomography, CT, or MRI. Symptomatic patients were recommended to be followed every 4–6 months. They recommended removal of the implant if malocclusion were progressive and/or condylar and/or fossa degenerative changes were evident radiographically beyond the time of expected remodeling after surgery [116].

In November 1992, the American Association of Oral and Maxillofacial Surgeons' 2 ½-day workshop was attended by 23 invited participants to develop a consensus concerning the use of TMJ interpositional and reconstructive materials. The results of that workshop were published in 1993 [76].

With regard to Proplast-Teflon, it was the consensus of the participants that the use of the IPI should be discontinued because it was considered an inappropriate material for that purpose. The workshop made the following recommendations with regard to the management of patients who had received an IPI. Patients who were asymptomatic with no imaging changes should be advised of the risks of retaining/removing the implant. Removal of implant and associated affected tissue was recommended. Follow-up with MRI and/or CT at least yearly for 5 years after the time the implant was placed, and then discontinuance of imaging if continued asymptomatic. If replacement was required, autogenous tissue was recommended. If a large perforation into the middle cranial fossa occurs, repair with temporalis muscle or bone graft appeared to offer appropriate treatment.

Asymptomatic patients with imaging changes, symptomatic patients without imaging changes, and symptomatic patients with imaging changes were all recommended to have the IPI removed. Follow-up post-operatively with MRI and/or

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CT at least yearly for 5 years then discontinuance if asymptomatic and any previous changes stabilized. Reoperation was recommended if symptoms recurred.

If no imaging changes were present, replacement after implant removal was not considered necessary. If bony changes were present, reconstruction using autogenous tissue or total joint prostheses considered safe and effective by a regulatory body (FDA) was considered appropriate. If the patient refuses implant removal, it was recommended that they be followed yearly with clinical examination and MR imaging and CT scan.

In 1993, Trumpy and Lyberg reported the results of a scanning electron microscopic and energy-dispersive x-ray analysis of IPI implants removed from 12 patients who had them in place a mean of 54.6 months. In all cases there were resorptive changes as reported by others with replacement of articulating bone by granulation tissue. All of the implants removed showed significant signs of wear, such as thinning, cracks, and tears. Overt perforations were seen in 5 cases. Microfragments were demonstrated with scanning techniques by their aluminum content. They suggested that besides the foreign body giant cell reaction to the materials, there were toxic and hypersensitivity reactions to aluminum in the pathogenesis of the bone destruction [118]. Choung, Piper, and Boland, in the same year, presented the first report of a recurrent foreign body giant cell reaction in 4 of 112 TMJs where Proplast-Teflon had been previously removed [119].

Papers began to appear in the oral and maxillofacial surgery literature concerning the management of TMJs affected by Proplast-Teflon device failures. Lorge et al. presented 24 patients who had previously been implanted with those devices an average of 7.3 years prior to removal. All patients underwent removal of these implants under magnification with minimal osteoplasty. No other TMJ reconstruction was performed. Active postoperative physical therapy emphasizing range of motion was prescribed for 6 weeks. All but one patient who developed ankylosis were reported to be doing well after a mean follow-up period of 17 months [120].

Henry and Wolford presented a retrospective study of 107 patients with 163 TMJs previously implanted with Proplast-Teflon devices. The average time these devices were in situ was 59.8 months. Only 12 % of these joints demonstrated no significant bony changes on radiographs. TMJ reconstruction with autogenous tissue was performed in all cases. Success rates using autogenous tissues were reported as follows: 31 % with free temporalis fascia and muscle graft with and 13 % without sagittal split osteotomy; 12 % costochondral grafts; 8 % dermal grafts; 25 % conchal cartilage; and 21 % sternoclavicular grafts. A foreign body giant cell reaction was present an average of 40 months after implant removal and also after an average of 4.5 additional surgeries. Results with a total alloplastic CAD/CAM device (Techmedica, Camarillo, CA now TMJ Concepts, Ventura, CA) reconstruction yielded statistically significant better results than autogenous tissue reconstructions at 25 months follow-up. Therefore, these authors concluded that the use of total alloplastic TMJ prosthesis may be indicated to achieve successful reconstruction in these cases [121].

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Kearns et al. in a mean follow-up period of 38.3 months retrospective study reported the results of removal of 24 failed Proplast-Teflon, 11 failed Silastic, and 7 failed Christensen fossa/eminence interpositional TMJ implants. These surgeons performed aggressive debridement and placed pedicled temporalis muscle/ fascia flaps for lining of the TMJ in 27 patients (47 joints). They reported that pain was well controlled in 88.9 % (24/27) of the patients. Seven patients (25.9 %) required orthognathic surgery to manage loss of posterior vertical dimension due to severe condylar degeneration caused by these implants [122].

Investigators began looking at the cellular tissue response to Teflon-Proplast. In 1996, Trumpy et al. reported the results of a morphologic and immune-histochemical analysis of explanted Proplast-Teflon implants. These authors concluded that the tissue reaction induced by the failure of these implants was not due to any toxic or immunologic pathology. They concluded that mechanical stress seemed to be important in the fragmentation of these implants, and this fragmentation was what induced the foreign body giant cell reaction [123].

Zardeneta et al. reported on the nature of protein interactions with particulate Teflon. These authors found that the smaller the particle size, the greater the biological response. They therefore concluded that the severity of the biological response to this material appeared to be directly dependent on the size of the debris particles [124]. Milam presented a review of alloplastic TMJ reconstruction and a further discussion of Zardeneta's results [125].

As a result of the clinical issues with Proplast-Teflon containing TMJ implants, the following statements concerning the use of alloplastic materials in TMJ disease came from a technology assessment conference on the management of TMJ disorders held at the National Institutes of Health in the spring of 1996. The conference report recommends "rigorous investigation with utmost caution regarding the use of any new implants. At the same time, it is recognized that certain patients are in need of these procedures." The report goes on to say, "…evidence indicates that the probability of success decreases with each additional surgical intervention" [126].

Raphael et al. studied the general health consequences of exposure to failed Proplast-Teflon interpositional TMJ implants and the subsequent foreign body giant cell reactions. 44 of the 64 patients who had received these implants had had them removed. 22 unexposed TMJ patients served as the control group. These investigators found that although the exposed patients did not report more systemic health conditions than the controls, those with removed implants reported more conditions and were more likely to be seen by clinicians. The authors felt that this finding may lead to a bias in the general perception regarding the systemic health status of the exposed patients. The authors stated further that any effects may be secondary to high levels of pain and dysfunction among patients with removed implants, rather than implant exposure itself [127, 128].

In 2002 Fricton et al. published a long-term study of outcomes of operations on the TMJ after Silastic and Proplast-Teflon implantation compared with non-implant operations on the TMJ and nonsurgical rehabilitation for painful displacement of the TMJ disc. The results of this study suggested that the use of interpositional disc

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implants in the TMJ was not associated with improved outcomes when compared with non-implant surgery or nonsurgical rehabilitation [129].

## 3.4 Acrylics

In 1950 Judet [130] developed a total hip prosthesis using a fixed metal acetabulum component articulating against an acrylic, polymethylmethacrylate (PMMA) mobile femoral head component. Failure due to PMMA wear under load with particulate debris foreign body reaction, led to the demise of this device [131].

In 1954, Healy [132] reported on the use of acrylic implants to reconstruct the mandible after ablative surgery, and in 1975, Kameros and Himmelfarb [133] offered the use of interpositional methylmethacrylate acrylic in the treatment of TMJ ankylosis.

Boyne and colleagues [134, 135] reported the use of a polyoxymethylene (Delrin) condylar replacement in the management of ankylosis, and Szabo et al. [136] presented results with the prototype of a ceramic condyle. Hahn and Corgill [137], in 1970, first reported the use of a ramus-condyle hemiarthroplasty prosthesis for the treatment of ankylosis. The condylar component was fashioned from dental polymethylmethacrylate. The ramus component was stainless steel wire mesh. None proved to be effective clinically.

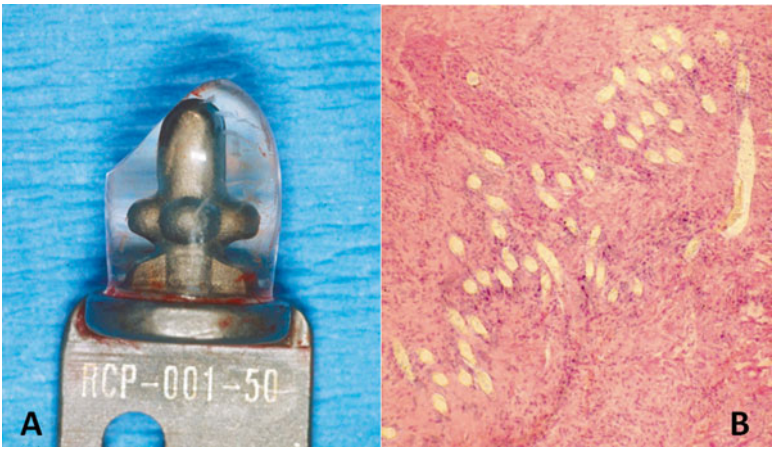


**Fig. 3.5** (**a**) Polymethylmethacrylate (PMMA) head of a failed TMJ Inc. condylar component demonstrating wear 3 years after implantation. (**b**) Particles of PMMA demonstrated in the peri-articular tissue

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In 1963 [138], 1964 [139], and 1970 [140] Christensen reported the use of a thin cast vitallium fossa-eminence hemiarthroplasty prosthesis for management of TMJ ankylosis. A cast vitallium ramus-condyle component with a PMMA head was later added to create a total joint prosthesis. Due to wear under functional loading, this bearing surface geometry was abandoned in the late 1990s [141] (Fig. 3.5).

## 3.5 Hemiarthroplasty

In 1933, Risdon [142] reported management of a TMJ ankylosis patient by interposing gold foil between the bony surfaces after gap arthroplasty. Eggers [143] in 1946 and Goodsell [144] in 1947 reported the use of tantalum foil in cases of TMJ ankylosis. In 1951, Castigliano [145] and Kleitsch [146] resurfaced the bone in TMJ ankylosis cases with vitallium. In 1952, Smith [147] reported hemiarthroplasty for ankylosis using stainless steel. Ueno [148] in 1955 reported experimental and clinical results with zirconium in TMJ ankylosis.

In 1960, Henry [149] described replacement of an ankylosed temporomandibular joint with a stainless steel prosthesis, and Robinson [150] reported correction of a TMJ ankylosis by creating an artificial stainless steel fossa. Hellinger [151] in 1964 reported the use of tantalum foil in such cases and was the first to stress the importance of physical therapy in the rehabilitation of these patients.

In 1963 [138], 1964 [139], and 1970 [140] Christensen reported resurfacing of the glenoid fossa with a thin cast vitallium fossa-eminence hemiarthroplasty prosthesis for management of TMJ ankylosis. A number of reports on the use of the fossa-eminence hemiarthroplasty device followed [12, 152–161].

In 1971 [162], 1975 [163], and 1977 [164] Morgan presented another form of fossa resurfacing device which consisted of a thin cast vitallium eminence prosthesis with a Silastic articulating component [165].

In 1972, Taurus reported the use of a custom-made cast gold ramus-condyle hemi-articulation in reconstruction of a TMJ [166].

Hinds, Homsy, and Kent reported the use of a ticonium alloy condylar prosthesis, the shank of which was coated with Proplast I [167]. Three cases were included in the original report followed by 6 more cases in a 1974 report [168]. Further reports followed in 1980 [169] and 1981 [170]. In 1983, Kent et al. published a 10-year report on the use of this hemi-articulation reconstruction of the TMJ in 80 patients, 109 TMJs. The authors considered this device successful by the subjective and objective criteria they developed in 87.3 % of the cases followed and average of 25.4 months [171].

Between 1974 and 1978, there were a number of other reports of partial and total temporomandibular joint alloplastic devices composed of both metal and nonmetallic components [133, 172–176].

Rooney et al. reported three cases of rapid TMJ condylar degeneration after insertion of PTFE glenoid fossa components against natural condyles. Histologically,

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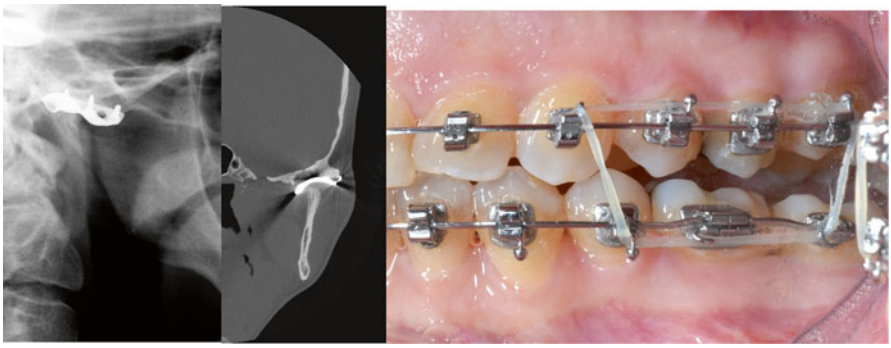

**Fig. 3.6** Right fossa-eminence device in place for 2 years resulting in ipsilateral loss of condylar height, pain, and contralateral open bite that was attempted to be managed orthodontically

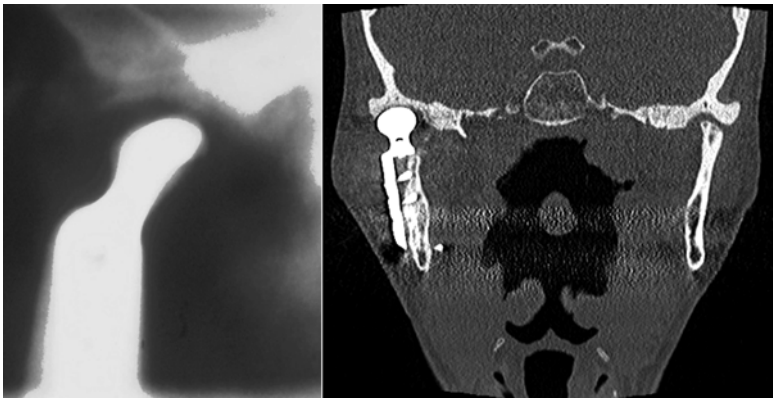

**Fig. 3.7** Ramus components of TMJ TJR devices implanted without fossa component eroding into the glenoid fossa bone

the surrounding tissue removed with these devices at surgery demonstrated a foreign body giant cell reaction with bi-refringent PTFE particles. It was concluded that these cases strongly supported warning that this prosthesis should be used with caution against the natural condyle [176] (Fig. 3.6).

Hemiarthroplasty, a metallic bearing surface articulating with normal articular cartilage, is frequently utilized in orthopedic surgery for fractures of the hip and shoulder in geriatric patients. The surgery can be quite successful in such cases where functional demands are low; however, over time the metallic component against the articular cartilage causes cartilage wear and may cause pain, requiring total joint replacement. For this reason, hemiarthroplasty is generally not performed in young patients or in patients with preexisting degenerative joint disease [177, 178].

The use of alloplastic condylar components against the bone of the glenoid fossa had been advocated [133–136, 166–175]. Marx et al. reported a 7.8-year follow-up

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of 131 patients (132 joints) who had undergone alloplastic replacement of the mandibular condyle with a metallic condyle on a rigid reconstruction plate functioning against a natural disc or a soft tissue graft without alloplastic replacement of the fossa after disarticulation for pathology or trauma provides long-term stability with minimal complications (10.6 %). They report no erosions through the glenoid fossa but one condylar head erosion into the external auditory canal [179].

Advocating placing a functional alloplastic condylar component against either the glenoid fossa or the articular eminence has been discouraged in the literature due to concern for erosion of these devices into the middle cranial fossa [180–182] (Fig. 3.7).

## 3.6 Total TMJ Replacement

As a result of the bony condylar changes see with the fossa-eminence device, Christensen developed a cast vitallium ramus-condyle component with a PMMA head to create a total joint prosthesis [183]. Due to wear under functional loading, this bearing surface geometry was later abandoned [140] for a metal-on-metal bearing surface [184].

Morgan added a variation of the Hahn and Corgill ramus-condyle component with a polyoxymethylene condyle to make a total joint prosthesis. In 1984, House et al. [185] reported the results of the use of the Morgan devices. In 1992, Morgan [11] reviewed the development of alloplastic materials for TMJ prostheses with an emphasis on his prostheses.

Kiehn applied the principles used in total hip reconstruction to the TMJ, utilizing a Howmedica (Kalamazoo, MI) vitallium mandibular fossa plate reinforced on its temporal side with polymethylmethacrylate and a vitallium-modified Cargill-Hahn ramus/condyle prosthesis. In 1979, he and his coauthors reported follow-up of 27 patients who had undergone total TMJ reconstruction with this device in the management of TMJ ankylosis, arthritis, neoplasia, infection, or refractory pain. They described 23 successful cases with a 1–3-year follow-up. Success was defined as being the ability to open the mouth to eat without pain [186].

Kammoona [187] reproduced Kiehn's work in the lab using 6 monkeys. After 9–10 months of function, half of the devices were reported unsuccessful due to condylar component failure. Microscopically, there was a minimum of inflammatory cells, no evidence of infection, and well-organized granulation tissue and collagen fibers with fibrous tissue beneath the cement and condylar component. Collagen fibers ran parallel to the implant. The bone in the surrounding area was vital and healthy, and in some areas the fibrous tissue had turned to bone. Microradiographs demonstrated tolerance of the metallic joint and bone cement, with incorporation by healthy granulation tissue, collagen fibers, and new bone to such an extent as to justify complete biological acceptance of the implant by the natural tissue. This was the second report of animal studies with alloplastic TMJ devices after Ueno [148].

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In 1983, Kent, Block, and Homsy reported the use of a Dacron/Proplast-Teflon (VK-I) and later a Dacron/Proplast-Teflon/ UHMWPE (VK-II) fossa (PTFE) [188]. The ramus-condyle and fossa components were then reported as used as a total alloplastic TMJ reconstruction prosthetic device [189]. In the later study, 192 TMJs were reconstructed, 133 with total joints (ticonium condylar component [95] or Synthes (West Chester, PA) condyles [38]) against Vitek-Kent (VK) I/II fossa replacement and 59 with hemiarthroplasty with only PTFE-I (6) or VK-II (56) fossa components. Follow-up was 46 months, and a 91.51 % success rate was reported. Failures were reported as early infections, fossa erosions, anterior dislocation of the natural or prosthetic condyles, or ankylosis. The authors warn that unfavorable remodeling of the natural condyle may be anticipated when it is articulated against the dry glenoid fossa prosthesis. The VK-I glenoid fossa replacement prosthesis was discontinued due to reports of articular surface wear.

In 1985 [190] and 1990 [191] Schonnenberg and Schonnenberg reported the use of a total TMJ device which consisted of a chromium–cobalt–molybdenum (Cr-Co-Mo) ramus-condyle component which articulated against an ultra-high molecular weight polyethylene (UHMWPE) fossa. This mimicked the materials and geometry used in the design of alloplastic joint prostheses by orthopedic surgeons.

In 1993, Kent et al. reported the long-term follow-up of the Vitek (Houston, TX) partial and total TMJ reconstruction prostheses [192]. 262 partial and total TMJ reconstructions were followed up to 10 years. VK-I total joint cumulative success rate was 44 % at 6 years and 20 % at 10 years. While VK-II cumulative success rate was 80 % at 6 years. Material wear was reported as the most common reason for

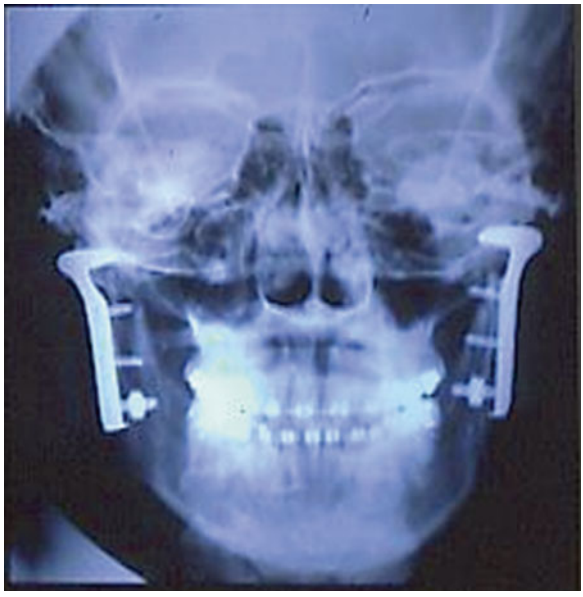

**Fig. 3.8** Bilateral Kent-Vitek TMJ TJR devices

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failure with the VK-I system with foreign body giant cell reactions seen in the surrounding tissues. The authors reported that they had not seen evidence of wear of the ultra-high molecular weight polyethylene (UHMWPE) surface of the VK-II glenoid fossa component in total TMJ reconstruction cases reoperated for release of ankylosis or device removal (Fig. 3.8).

As interest and need for an alloplastic TMJ replacement system grew in light of the material failure of Proplast-Teflon, a number of reports were surfacing in the literature related to the development, utilization, and outcomes of TMJ TJR devices [136, 193–198].

In 1995, Mercuri et al. reported on preliminary results with the use of the Techmedica (Camarillo, CA) patient-fitted (custom) CAD/CAM total alloplastic TMJ reconstruction prosthesis in a prospective-limited clinical study [199]. Based on this study TMJ Concepts (Ventura. CA) received FDA approval to manufacture and market this device in 1999.

In 2000, Quinn introduced the stock Biomet Microfixation TMJ TJR device [200]. This system received FDA approval to manufacture and market a stock TMJ TJR device based on a clinical study published later by Giannakopoulos et al. [201].

A description of the presently available custom and stock devices, their Indications, contraindications, and outcomes will be presented in the following chapters.

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# Part II
# TMJ TJR Devices

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# Chapter 4
# Stock Prostheses for Total Reconstruction of the Temporomandibular Joint

**Peter Quinn and Eric J. Granquist**

Alloplastic total joint replacement (TJR) is a universally accepted procedure in orthopedics. According to recent estimates, the global market for orthopedic implants is projected to reach 46.5 billion dollars by 2017 [1]. The growth has been fueled by the overall safety and efficacy of orthopedic implants and the fact that a steadily increasing aging population and a younger population with higher expectations continue to seek relief from pain and physical independence and maintain mobility and quality of life. In the United States alone, in 2010, there were approximately 330,000 hip replacements and 720,000 knee replacements [2]. In addition to continually improving the safety and performance of alloplastic implants, orthobiologics have also been improving the overall success of orthopedic interventions. Growth factors, synthetic tissue grafts, bioengineered tissue implants, and viscosupplementation substances are only a few of the recent advances.

Unfortunately, progress in the use of alloplastic implants in temporomandibular joint (TMJ) surgery was significantly adversely affected by widely published failures with TMJ alloplasts, including polytetrafluoroethylene, silicone rubber, and poor-quality metallic implants. Partially, this failure was caused by the inattention of the oral and maxillofacial surgical community to lessons that had been learned in earlier orthopedic trials. In the past 25 years, we have made incredible progress in reversing these failures with well-designed, pre-market approval trials for both patient-fitted and stock TMJ TJR devices.

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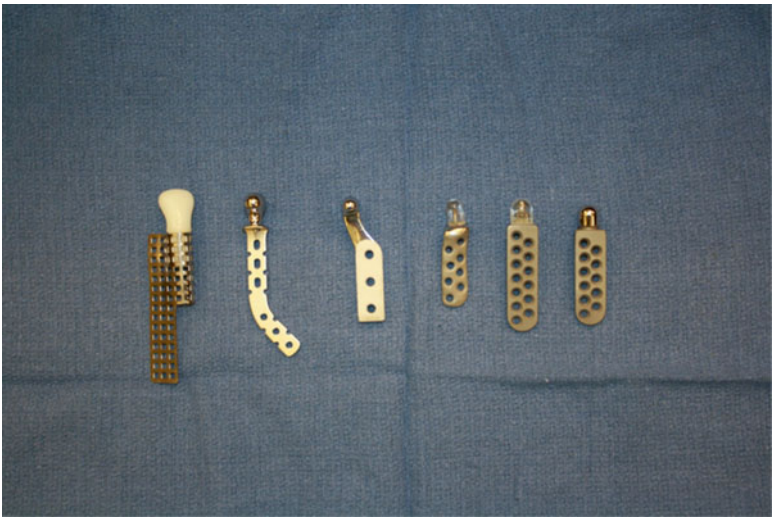

**Fig. 4.1** Previous iterations of stock alloplastic temporomandibular replacement systems, mandibular components. *From left to right*: Delrin-Timesh, Synthes, Kent-Vitek, Christensen type 1, Christensen type 2, Christensen metal-on-metal system

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It is estimated that approximately 12 % of the general population suffers from TMJ and associated orofacial pains. Even though the majority of these disorders are muscular in nature, the TMJ itself is affected by the same pathology as every other joint in the body—arthritis, trauma, benign and malignant tumors, infection, and developmental abnormalities. As such, oral and maxillofacial surgeons must have a full spectrum of both nonsurgical and surgical management options in order to deal with disorders of the TMJ. This clearly includes the availability and utilization of patient-fitted and stock TMJ TJR devices.

In the early 1990s, surgeons had experience with stock TMJ devices. However, these had not undergone stringent, pre-market approval for materials testing and/or clinical trials. These included the Morgan prosthesis, the Christensen prosthesis, the Kent-Vitek prosthesis, the Osteomed prosthesis, and the Delrin-Timesh condylar prosthesis (Fig. 4.1). Most of these developed device-related mechanical failures are caused by particulate wear, mechanical loosening, and metal fracture. Mercuri et al. [3] published their preliminary multicenter report in 1995 proving the safety and efficacy of a patient-fitted CAD/CAM total temporomandibular joint system (Techmedica, Camarillo, CA).

Believing surgeons should have the option of both patient-fitted and stock TMJ TJR devices, in 1995, Biomet (Jacksonville, FL) began clinical trials with a stock TMJ TJR device initially named the Lorenz Total Temporomandibular Joint Implant. This device was rebranded as the Biomet Microfixation TMJ Replacement System (Biomet Microfixation, Jacksonville, FL) and was granted FDA approval in 2005.

In 2012, Giannakopoulos et al. [4] reported outcomes for 442 Biomet Microfixation TMJ Replacement System TMJ TJR devices implanted in 288

patients. The results demonstrated a statistically significant improvement in pain, jaw function, and interincisal opening. Reported complications (infection and heterotopic bone formation) required the removal of 14 of 442 implants (3.2 %), but there were no reported device-related mechanical failures.

There was understandable skepticism about any new TMJ implant due to prior TMJ material failures. In a 2004 paper, Dimitroulis [5] opines that, "Despite the disasters (i.e. implant failures) prominent surgeons continue to advocate the use of alloplastic joint reconstruction for a wide variety of TMJ disorders such as ankylosis, inflammatory joint disease such as rheumatoid arthritis and also the multiply-operated patients with mutilated joints." Ten years later, in February of 2014 [6], the same author compared condylectomy, costochondral grafts, and the Biomet TMJ TJR stock device. Although the condylectomy group demonstrated the best mandibular range of motion, 43 % of rib graft patients experienced complications necessitating a return to the operating room. The stock TMJ TJR device group recorded the best mean-aggregate quality of life score. Therefore, the Biomet TMJ TJR stock device has demonstrated in a well-designed, pre-market approval clinical study to be a safe and effective option for the patient who has end-stage TMJ pathology.

In 2000 [7], the Biomet initial multicenter clinical trial results were reported when it was called the Lorenz prosthesis. Outside of the United States, the Biomet Total TMJ Replacement System is available in a custom-fitted version, and several authors have reported statistically significant success using that device [8, 9]. This chapter will discuss only the 2005 US FDA-approved stock Biomet Microfixation Total TMJ System.

Since that time, there have been several international publications detailing successful trials. The largest of these was a 10-year follow-up of 300 patients reported by Lobo Leandro et al. [10]; Machon et al. [11] reported experience with 27 patients (38 joints) in the Czech Republic and Slovakia. Their conclusion was that "total alloplastic TMJ replacement appears to be a safe and effective method of reconstruction." In 2010, Westermark [12] reported good outcomes with up to 8 years follow-up in 12 patients. Sanovich [13, 14] detailed the use of the Biomet prosthesis at the University of Florida. In the first study, a retrospective chart review of 37 patients (17 patients had TMJ replacement with Biomet prostheses and 20 were reconstructed with patient-fitted TMJ TJR devices), "both TMJ reconstructions demonstrated similar outcomes (pain reduction, improvement in interincisal opening) with a low incidence of complications."

In all five of these studies, there was significant decrease in pain, increase in interincisal opening, and improvement in diet. Van Loon et al. [15] and Quinn et al. [16] provide comprehensive reviews of the previous history of stock implants.

The Biomet Microfixation TMJ Replacement System is based on the following assumptions:

1. In the skeletally immature patient, an autogenous joint replacement or distraction osteogenesis is the preferred method of reconstruction.
2. In the skeletally mature patient with an acceptable indication for alloplastic joint reconstruction (see Table 4.1), a safe and effective stock prosthesis should be available for reconstructing the non-mutilated joint.

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**Table 4.1** Indications and contraindications for stock TMJ TJR

| Indications for alloplastic joint reconstruction | Contraindications to alloplastic joint replacement |
|---|---|
| Late-stage degenerative joint disease (osteoarthritis, rheumatoid arthritis, traumatic arthritis, etc.) | Allergy to any of the prosthetic materials |
| | Chronic infection |
| Recurrent ankylosis | Systemic disease with increased susceptibility to infection |
| Irreparable condylar fracture | Skeletal immaturity |
| Revision procedures for failed alloplastic or autogenous reconstruction | |
| Avascular necrosis | |
| Neoplasia requiring extensive resection | |
| Congenital disorders, e.g., hemifacial microsomia, Treacher Collins syndrome | |

3. In the patient who has undergone multiple operations with significant anatomic mutilation, or has a severe anatomic deficit following tumor surgery, a custom joint prosthesis designed with CAD/CAM technology from a 3D CT scan may be indicated.

As to the last contraindication listed, as more experience and success with TMJ TJR devices are gained and reported, the indications may include patients who have not achieved complete skeletal maturity. In some adolescent patients who have had severe ankylosis and multiple procedures, there is no potential continued growth in the site on the ankylosed or mutilated joint as reported by Mercuri and Swift [17]; therefore, TMJ TJR may be beneficial in limited cases such as children or adolescents with severe deformities.

The major disadvantages of TMJ TJR devices are:

- The potential for wear debris and the associated biologic responses
- Mechanical failure due to component fracture, loosening of screw fixation, and metal fatigue
- Cost of the device
- Unpredictable need for revision surgery since long-term data on the longevity of these devices is as yet unknown

The Biomet stock prosthesis is composed of an ultrahigh molecular weight polyethylene (UHMWPE) fossa which is available in three different sizes where the only variability is in the anterior-posterior length of the zygomatic flange allowing for multiple screw fixation sites. The articulating surface of the fossa has the same geometric configuration and dimensions in three sizes (Figs. 4.2 and 4.3). The UHMWPE is Biomet's ArCom® polyethylene specifically designed for use in articulating orthopedic joint designs. This material has increased tensile and shear strength and a low coefficient of friction. It is gamma-radiated to increase the cross-linking to decrease wear. Stabilization of the fossa component relies on accurate

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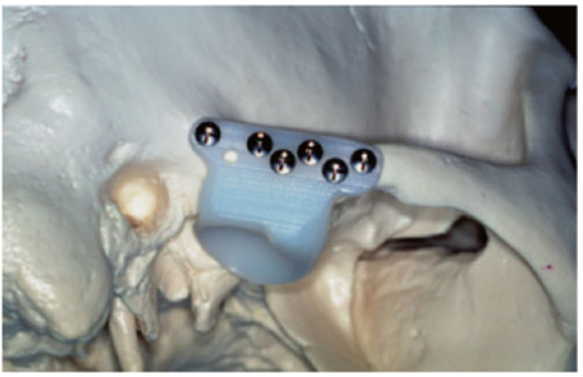

**Fig. 4.2** Ultrahigh molecular weight polyethylene fossa component for the Biomet stock system. The fossa component should parallel the zygomatic arch as shown

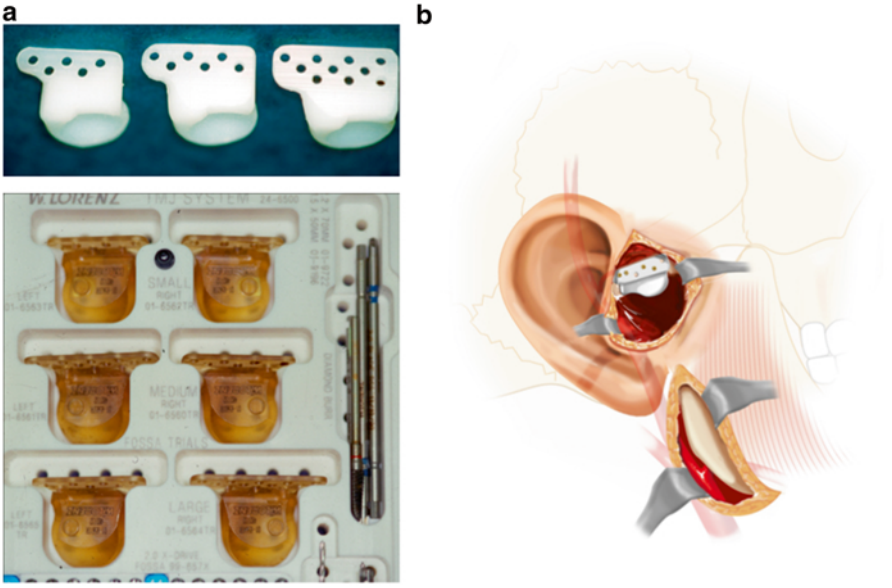


**Fig. 4.3** (**a**) *Top image* shows the three sizes of the fossa component. Note that the articulating surface and thickness are the same for all three sizes. The flange increases in size to allow more pre-drilled holes for screw placement into sound zygomatic bone. *The lower image* shows the trial sizers. These trial sizers should be utilized to confirm not only the appropriate size but also the orientation and stability of the implant. Once this is achieved, the fossa implant should be placed. (**b**) Diagram showing correct placement and orientation of the fossa component. The mandible can be placed in the closed-mouth position to ensure appropriate clearance of the fossa component before the surgeon places the patient in intermaxillary fixation

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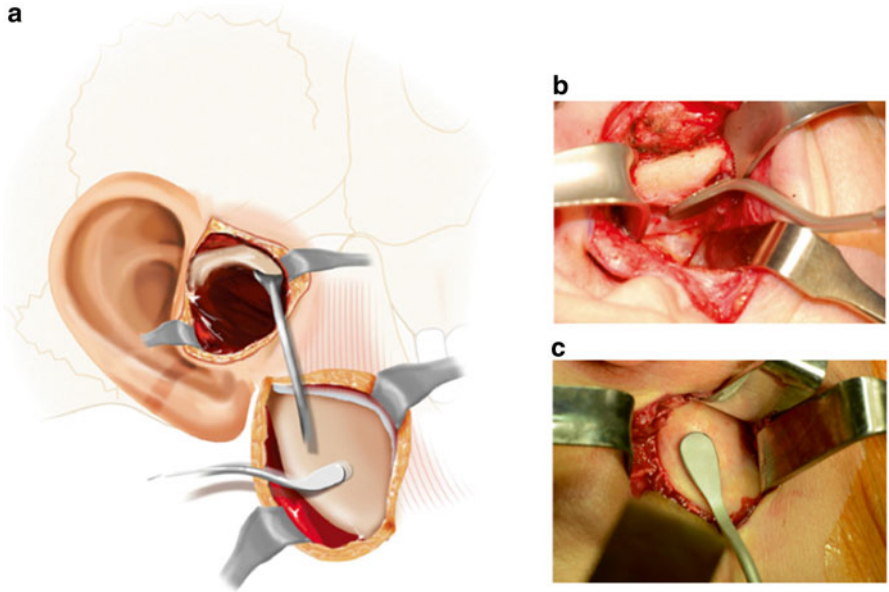


**Fig. 4.4** (**a**) Diagram showing the use of the diamond rasp to perform the eminoplasty and, if necessary, remove a small amount of bone along the lateral aspect of the ramus to ensure a stable seating of the mandibular component and proper prosthesis orientation. (**b**) Intraoperative image showing the use of the diamond rasp for the eminoplasty. Bone is carefully removed to ensure tripod stability of the prosthesis and correct orientation. (**c**) Intraoperative image showing the diamond rasp removing a small amount of bone along the lateral aspect of the mandible

alteration of the articular eminence to remove the variability in the shape of the eminence by surgically flattening it to achieve tripod stability of the component (Fig. 4.4).

The mandibular component is manufactured from a cobalt-chrome alloy (ASTM type F799) plasma-sprayed with a roughened titanium coating on the medial host bone side of the ramal plate. This is a wrought alloy with improved tensile strength compared to older cast alloys. The mandibular ramus component comes in lengths of 45 mm, 50 mm, and 55 mm. There are two separate mandibular component designs, one a "narrow" and the more commonly used "standard" which has a broader ramal plate which provides more fixation screw options, especially in patients who have had previous rib grafts or failed alloplastic implants (Fig. 4.5). There is also an offset condylar component that is only available in the 50 mm length. In this component, the angulation of the condylar head is the reverse of the standard medially angulated head providing a laterally angulated condylar head for cases where the ramus is medially offset (Tables 4.2 and 4.3).

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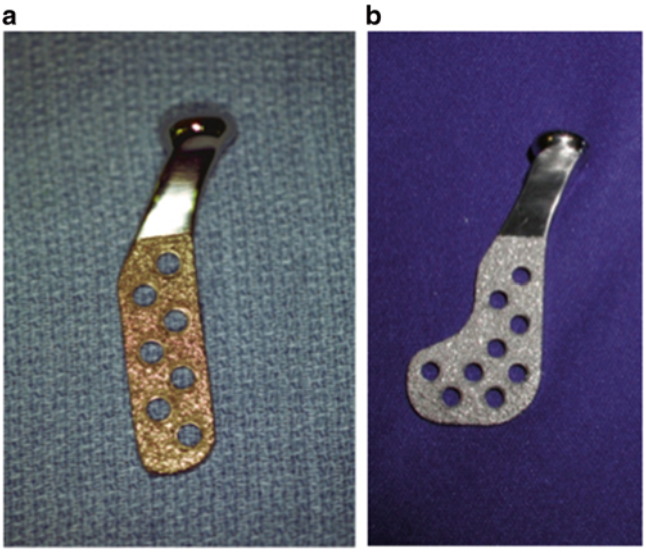


**Fig. 4.5** (**a**) Narrow Biomet mandibular component, medial surface. Note the titanium plasma spray for improved prosthesis-bone integration. (**b**) Standard Biomet mandibular component. The standard component has an enlarged foot plate for increased screw hole availability. Staggering the screw placement allows for increased stability of the mandibular implant and flexibility to avoid injury to the inferior alveolar nerve

**Table 4.2** Stock prostheses

| Advantages | Disadvantages |
|---|---|
| Fit flexibility | Limited potential for anterior-inferior movement of mandible |
| Immediate availability (e.g., irreparable trauma, tumor resection) | Surgeon experience with multiple joint reconstructions required to manage variability of fit |
| Lower cost | |

**Table 4.3** Custom prostheses

| Advantages | Disadvantages |
|---|---|
| Patient-matched; anatomically stable | Higher cost |
| Addresses distorted anatomy | Potential for two-stage surgeries (e.g., removal of failed previous metallic implant) |
| Excessive anterior-inferior movements possible | Time for fabrication of custom implant (8–12 weeks) |
| | Limited flexibility (must replicate model surgery exactly) |
| | Potential for two-stage surgeries (e.g., removal of failed previous metallic implant) |

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## 4.1 Planning and Preparation

Preoperative planning should include a detailed discussion with the patient of potential complications, which can include, but not limited to, infection, temporary and/or permanent damage to the facial nerve, damage to the inferior alveolar nerve with permanent numbness or dysesthesia, foreign body reaction to polymeric or metallic debris, heterotopic bone formation with ankylosis, dislocation of the prosthesis, malocclusion, continued pain requiring pain management, facial swelling, material hypersensitivity and potential need for future revision, and or replacement of the device [18–23].

Patients with a history of multiple previous surgeries and chronic central or neurogenic pain will benefit from an evaluation by a pain specialist for continued pain management postsurgery [24].

Patients who are being implanted to manage end-stage TMJ arthritic conditions may be on immunosuppressant medications, and the surgeon must coordinate temporary cessation of these medications (e.g., disease-modifying biologics, anti-cytokine medication, and glucocorticoids) with the patient's primary and/or specialist physicians.

Panorex, computed tomography, and 3D reconstructive images can provide valuable preoperative information concerning bone quality as well as aid in determination of the proximity of adjacent vital anatomic structures.

## 4.2 Preparation and Surgery

After anesthesia has been obtained, the hair is shorn to the top of the helix and the remaining hair is taped out of the surgical field. A head wrap is applied and the skin is prepped. We have also used a sterile urologic rectal condom as a way of allowing manipulation of the mandible during surgery to determine the position of the lateral pole of the condyle (Fig. 4.6).

Copious irrigation of the external ear canal is vitally important prior to incisions. Clindamycin solution is recommended for this and as a surgical irrigant during the entire case. As prophylaxis, patients are given parenteral cefazolin and metronidazole 1 h prior to the surgery. Strict attention to separating the sterile surgical site from the oral cavity is extremely important to avoid contamination of the device components.

The fossa and ramal components are implanted through a combination of a superior endaural pre-auricular incision and a posterior mandibular incision, respectively (Fig. 4.7). It is important to complete the surgical dissections for both the superior and inferior incisions before any bony surgery is performed, especially in ankylosis cases, to allow for optimal visualization and control of potential hemorrhage from branches of the external carotid artery, should it occur during the procedure.

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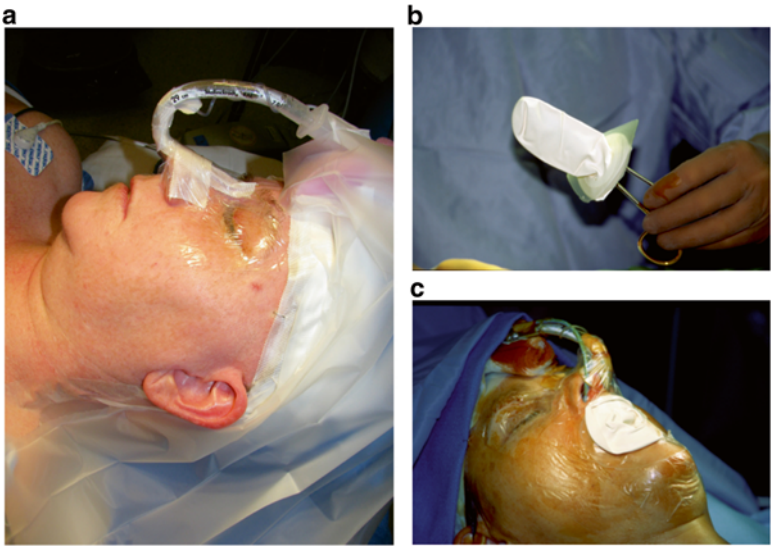


**Fig. 4.6** (**a**) Patient draped. (**b**) Modified urologic rectal sterile dressing. (**c**) Urologic dressings allowing sterile manipulation of the mandible intraoperatively. Also note Tegaderm covering the nares to limit contamination

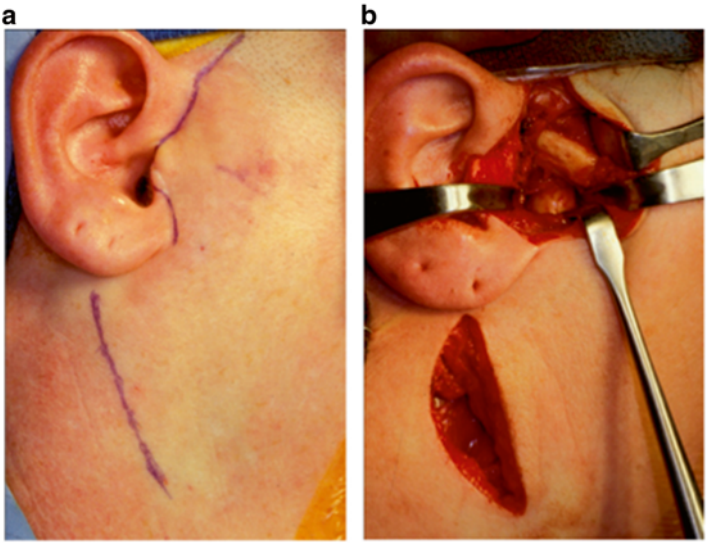


**Fig. 4.7** (**a**) Incision design. The endaural incision is chosen for its improved cosmesis and "stepped" dissection over the implant improving tissue coverage and moving the incision away from the prosthesis. (**b**) Dissections complete. The retromandibular incision is completed prior to the condylectomy. This allows access to the vasculature if hemorrhage is encountered

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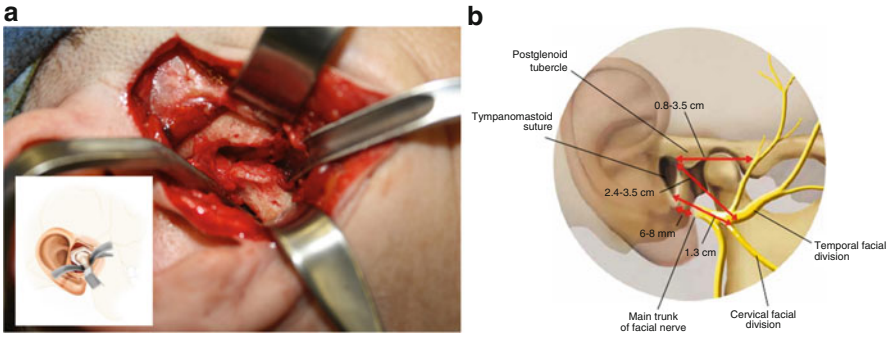


**Fig. 4.8** (**a**) Patient with severe condylar degeneration prior to condylectomy. Note the placement of the Dunn-Dautrey retractors. These retractors avoid injury to vessels medial to the condyle. Diagram of proper retractor placement (*insert*). (**b**) Diagram showing the branches of the facial nerve in relation to the external auditory canal

The superior endaural pre-auricular incision dissection is carried down to the posterior root of the zygomatic arch keeping the dissection as far posteriorly as possible to avoid any damage to the branches of the facial nerve (Fig. 4.8). The upper trunk of the facial nerve courses between 8 and 35 mm in front of the most anterior portion of the bony ear canal (Fig. 4.8b).

Carrying the dissection deep to the periosteum, an adequate portion of the zygomatic arch should be exposed to secure three to four 2.0 mm polyethylene fossa fixation screws in place. Condylar retractors are used to isolate the neck of the condyle to avoid potential damage to the internal maxillary artery as it courses behind the neck of the condyle (Fig. 4.9). Once the condyle is isolated, no further dissection is done until the inferior-posterior mandibular incision is completed. This incision is placed approximately one fingerbreadth behind the posterior border of the mandible curving anteriorly approximately 4–5 cm under the inferior border. The dissection is largely in a vertical plane anterior to the sternocleidomastoid muscle and posterior to the submandibular gland. It is usually not necessary to ligate the facial artery itself if it is retracted anteriorly and the retromandibular vein is retracted posteriorly.

The dissection is carried inferior to the mandible until the digastric tendon is visualized, isolating the inferior border of the mandible (Fig. 4.10). A No. 15 blade is used to incise the pterygomasseteric sling, and the masseter muscle is stripped superiorly. This allows communication between the inferior-posterior mandibular incision and the superior endaural pre-auricular incision. During the posterior mandibular dissection, a nerve stimulator is used to find the marginal mandibular branch of the facial nerve and make sure that the dissection is below that nerve.

The neck of the condyle is then isolated with Dunn-Dautrey retractors through the superior endaural pre-auricular incision. Specifically designed condylar neck retractor and zygomatic retractors (PDQ retractors) are used to isolate the condylar neck and zygoma in preparation for the condylectomy.

Adequate soft tissue dissection medial to the neck of the condyle is important to avoid hemorrhage from the internal maxillary artery and branches most commonly

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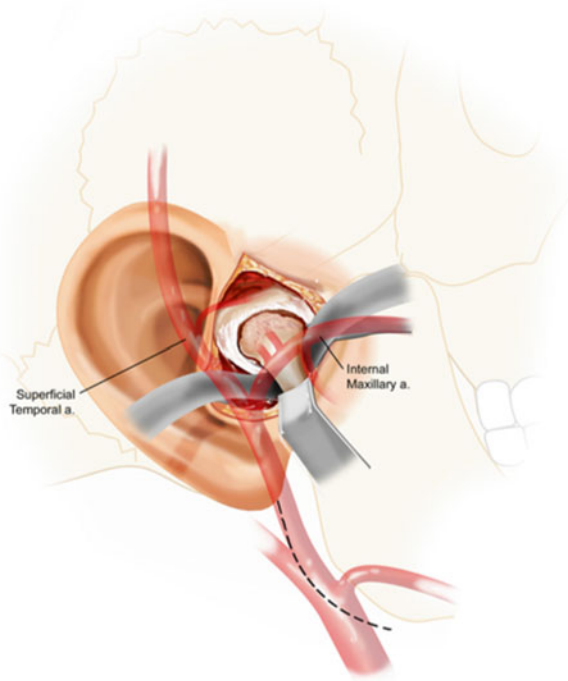


**Fig. 4.9** Diagram of the vasculature medial to the mandible. The internal maxillary artery runs as close as three millimeters from the mandible at the inferior portion of the coronoid notch

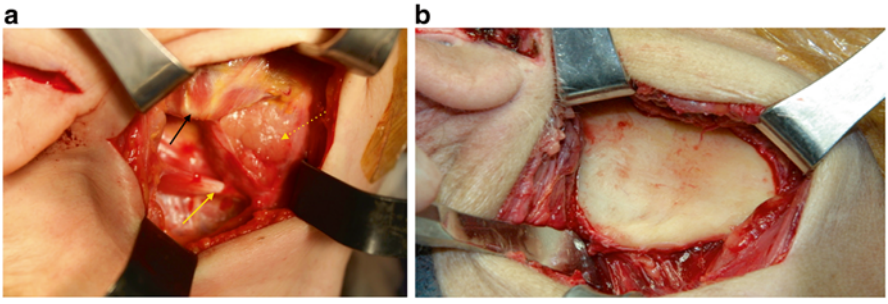


**Fig. 4.10** (**a**) Retromandibular incision with exposure of the posterior digastric muscle (*solid yellow arrow*), submandibular gland (*dashed yellow arrow*), and masseter muscle (*black arrow*). The masseter muscle can be dissected free from the mandible by making an incision along the raphe of the inferior border of the mandible. (**b**) Exposure of the lateral aspect of the mandibular ramus through the retromandibular incision. The facial artery can be safely retracted anteriorly, avoiding the need to sacrifice this vessel

involved with bleeding during TMJ TJR surgery (i.e., middle meningeal artery and the deep temporal artery). This is especially important in the multiply operated patient where scarring and fibrosis may bring these vessels in closer proximity to the condylectomy cuts.

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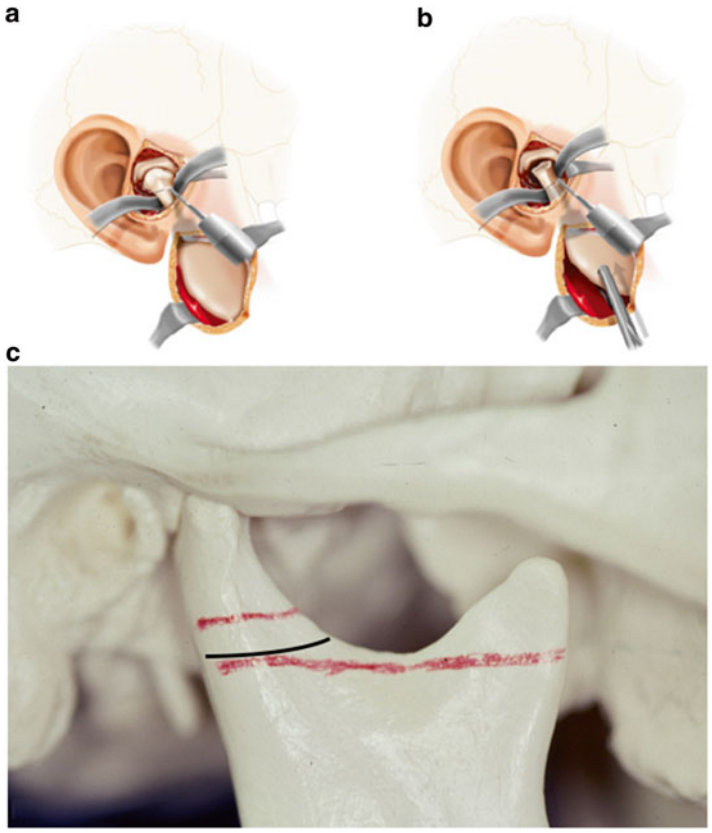


**Fig. 4.11** Two-step osteotomy. (**a**) Initial condylectomy with the use of Dunn-Dautrey retractors to protect deeper structures. (**b**) Superior repositioning of the ramus to allow for improved access to the second-stage osteotomy and increased distance from the internal maxillary artery. (**c**) Location of two-step osteotomies; note curvilinear shape of black line if coronoidectomy is not required

### *4.2.1 Two-Step Condylectomy*

A two-step condylectomy has been developed and advocated to minimize the risk of injury to the internal maxillary artery and ensure adequate bone removal for the fossa component as the thickness of all UHMWPE implants necessitates adequate removal of bone, usually 2–3 mm below the sigmoid notch, to provide space for its anterior lip. A 1 mm fissure bur is used to perform a condylectomy in the midportion of the condylar neck. This can be placed more inferiorly in the ankylosis patient. The initial goal is simply to remove the condylar head. After 90 % of the condylar cut is performed with the fissure bur, a T-bar osteotome is used to complete the condylectomy (Fig. 4.11). The condyle is then grasped with a bone-holding forceps and the lateral pterygoid is carefully dissected free. At this point, significant

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bleeding may occur and the surgeon should be ready to control any hemorrhage with the aid of pressure to the superior portion of the wound or hemostatic agents including thrombin and collagen. Once the condyle is removed, this creates space and allows the surgeon to superiorly reposition the ramus with bone-holding forceps from below. This maneuver allows better visualization by the surgeon and easier access to make the second part of the condylotomy by placing the second bone cut higher in the pre-auricular incision and further away from the medial internal maxillary artery. To ensure sufficient space for the fossa, bone should be removed just below the most inferior point of the coronoid notch by making a curvilinear-shaped ostectomy, and great care should be taken to protect the deep soft tissue structures with the aid of the Dunn-Dautrey retractors considering that the internal maxillary artery normally runs approximately 3 mm medial to the mid-sigmoid notch. In cases of long-standing ankylosis, this cut can be extended anteriorly to include the coronoid, if the coronoid is to be removed at the same time.

## 4.3 Fossa Placement

Secure and stable fossa placement requires tripod stability of this component in situ. This is achieved by flattening the articular eminence with a reciprocating diamond rasp specifically designed for this procedure. The depth of the cutting surface of the rasp matches the width of the three available UHMWPE fossa component sizes. The surgeon should remember that the more anterior the periosteum has to be stripped to accommodate a larger flange of the fossa, the higher the risk of temporary and/or permanent damage to the upper trunk of the facial nerve. The Biomet kit includes fossa sizers to determine the appropriate size of the fossa component prior to opening the sterile package.

Correct angulation of the fossa is critical to minimize dislocation and allow maximal opening. The fossa component should be parallel to the Frankfort horizontal plane or have slightly inferior position of the anterior lip compared to the posterior lip to avoid potential anterior dislocation. The fossa is then secured with two 2.0 mm screws into solid zygomatic arch bone. Correct alignment should be checked before any additional screws are placed. The tip of a nerve stimulator can be used to determine whether there is adequate bone under the fossa screw hole along the zygomatic arch before the final screws are placed.

At this point, copious irrigation of this wound with the aforementioned clindamycin solution is recommended. The external ear canal is irrigated with the antibiotic solution again to ensure that any bacterial-laden cerumen within the ear canal is flushed out prior to final component placement. Antibiotic-soaked sponges are used to cover the exterior wounds and the patient is positioned for intermaxillary fixation.

Prior to making any extra-oral incisions, Erich arch bars, Ivy loops, or IMF screws are placed to allow for intermaxillary fixation after placement of the fossa prosthesis. Again, great care should be taken not to contaminate the sterile field or

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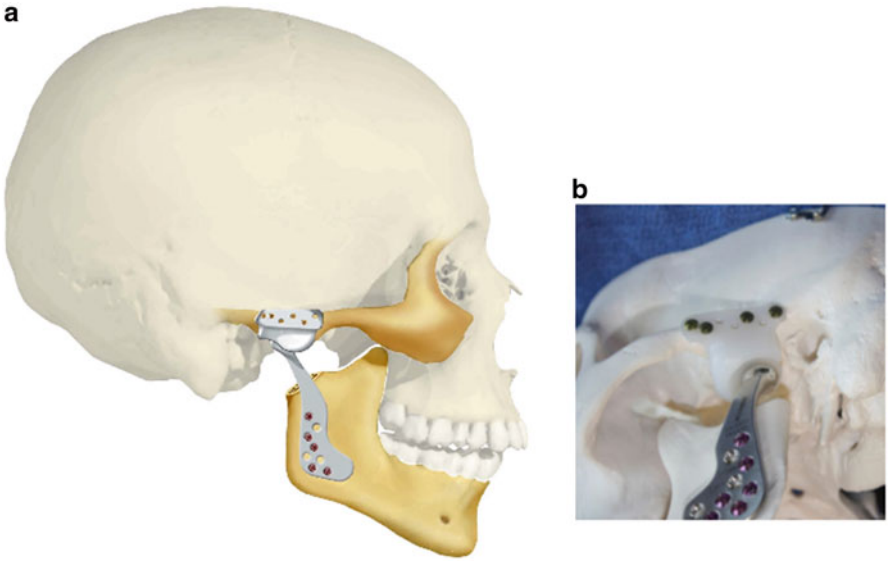


**Fig. 4.12** (**a**) Diagram showing the fossa and mandibular components placed. (**b**) Two screws for each component can be utilized and the mandible functioned to ensure proper component fit before the final screws are placed

instruments during the intraoral approach. A separate Mayo stand is set up for the intraoral instruments. A sterile layer of four towels and a body sheet are employed to cover the patient during the intraoperative occlusion setting procedure. Orthodontic brackets also can provide a useful and time-saving method for intraoperative intermaxillary fixation. After the occlusion is secured, the surgical team must change their gowns and gloves before returning to the sterile field.

With the patient in intermaxillary fixation, one of the three condylar sizers (45, 50, and 55 mm) are used to ensure that there is appropriate mating of the fossa prosthesis and the condylar prosthesis. The diamond reciprocating rasp can be used to remove any irregularities from the lateral surface of the ramus that would cause the prosthesis not to have a "flush fit" against the ramus (Figs. 4.12 and 4.13).

It is extremely important to position the head of the condyle in the fossa as far posterior as possible so that there will be some degree of "pseudo-translation" of the condylar head in the fossa as the patient opens to the expected range of 32–35 mm [25] (Fig. 4.14). Positioning the condyle too far anteriorly in the closed position, as shown in Fig. 4.15, could lead to dislocation of the condyle anterior to the fossa. Again, also note that the fossa is parallel to the Frankfort horizontal plane and is not tipped in an "open" anterior position. At this point, if the condylar head seems to seat too far laterally in the fossa, bone can be removed from the superior edge of the ramal cut with the reciprocating diamond rasp to allow more medial seating of the condylar head. In the rare occasion that the condylar head seats too medial in the UHMWPE fossa, the offset 50 mm ramal component should be used to position the condylar head more laterally in the fossa.

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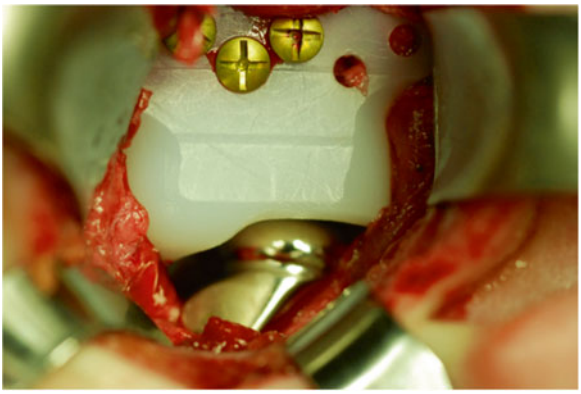

**Fig. 4.13** Intraoperative view of total joint prosthesis in position

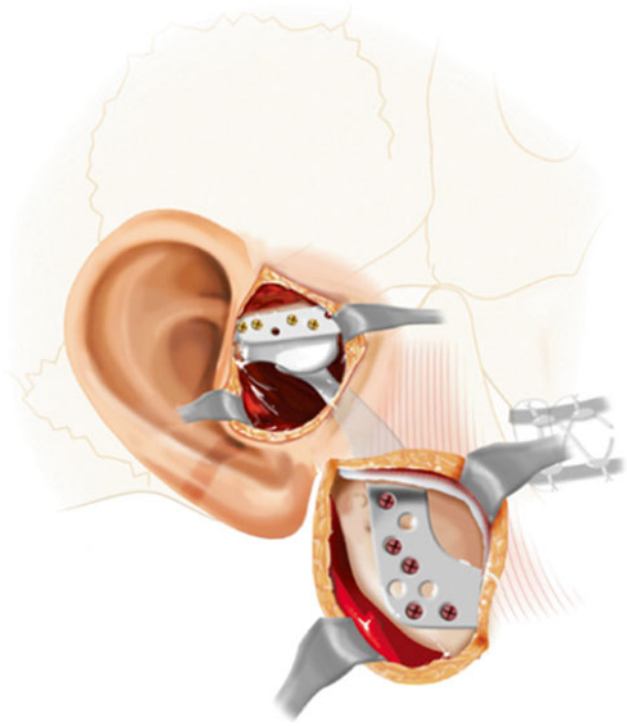

**Fig. 4.14** Demonstration of pseudo-translation of the prosthesis. This occurs when a unilateral prosthesis is placed and the patient has a functioning lateral pterygoid on the contralateral side

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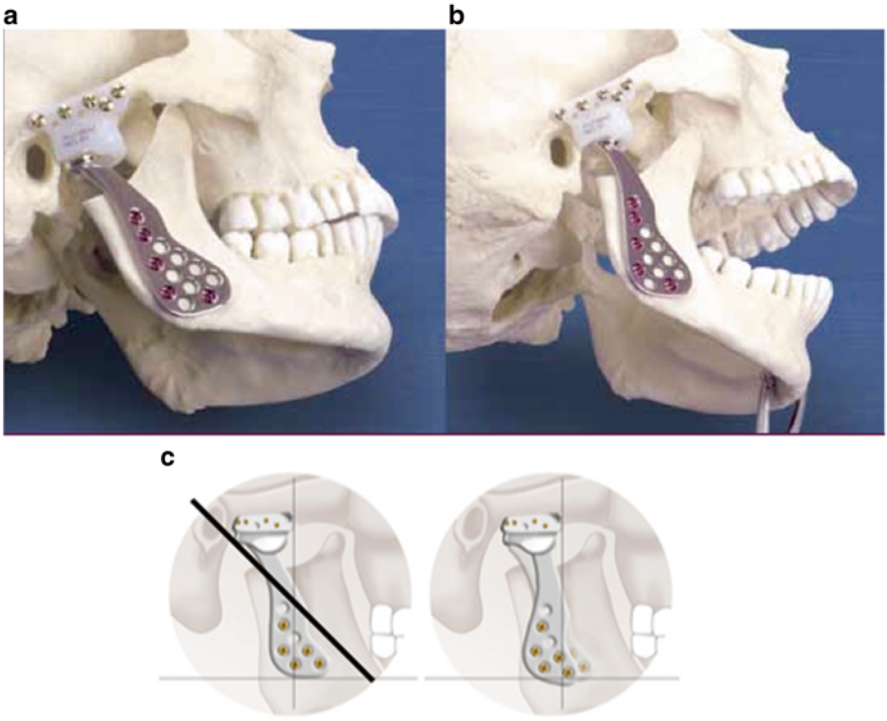


**Fig. 4.15** *The left diagram* shows correct posterior position of the prosthesis while the *right diagram* shows incorrect positioning

Once the surgeon has placed the ramal component in the appropriate position, it can be secured with two 2.7 mm screws, usually along the posterior border of the mandible to engage bicortical bone and also to avoid the inferior alveolar nerve (Figs. 4.16, 4.17, and 4.18).

Again, the wound is irrigated, sterile drapes are placed over the wound and the body of the patient, and the surgeon and surgical assistant now return to the oral cavity to remove the intermaxillary fixation and move the mandible in an acceptable range of motion to ensure that there is no mechanical obstruction, malocclusion, or anterior dislocation of the device. If the prosthesis needs to be repositioned, the two screws can be removed, and again, with intermaxillary fixation, the condyle can be positioned until the surgeon is satisfied with the mandibular function.

The standard design with the expanded "foot plate" was designed to allow some flexibility in screw placement, especially in patients who have had previous alloplastic implants or rib grafts. It is preferable to use the heavier bone along the inferior-posterior ramus, if possible, for screw placement. The implant should never be bent and great care should be taken to avoid any scratching of the mandibular component.

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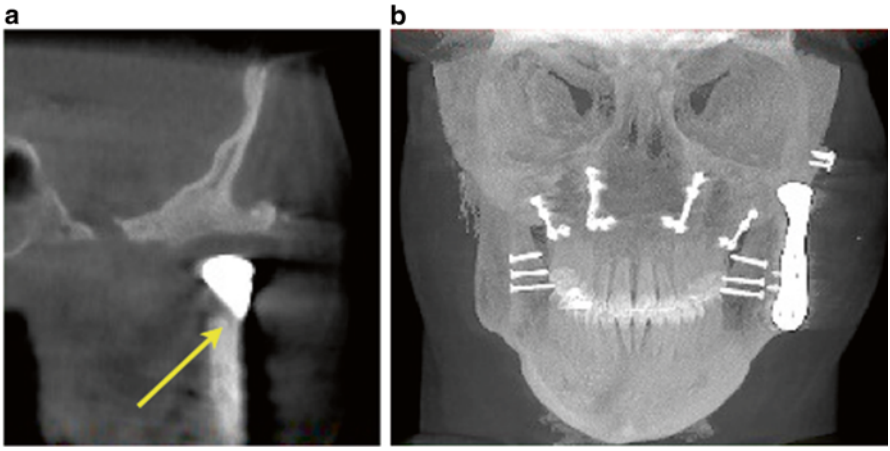


**Fig. 4.16** (**a**) Well-adapted mandibular component. Note the need for sufficient mandibular bone reduction to accommodate the "swan neck" of the prosthesis. (**b**) The same patient with well-positioned prosthesis

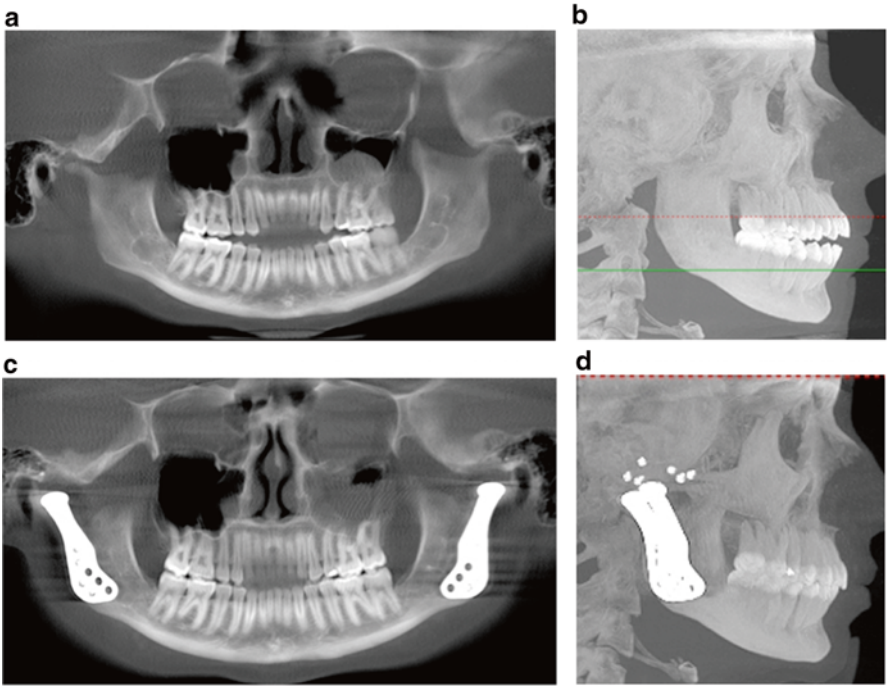


**Fig. 4.17** (**a**) Patient with juvenile idiopathic arthritis, severe bilateral condylar resorption, and a loss of vertical height. (**b**) Note the anterior open bite in this patient. (**c**) Same patient following bilateral stock alloplastic joint reconstruction with the Biomet system. (**d**) Note how the mandibular height is restored and the open bite closed

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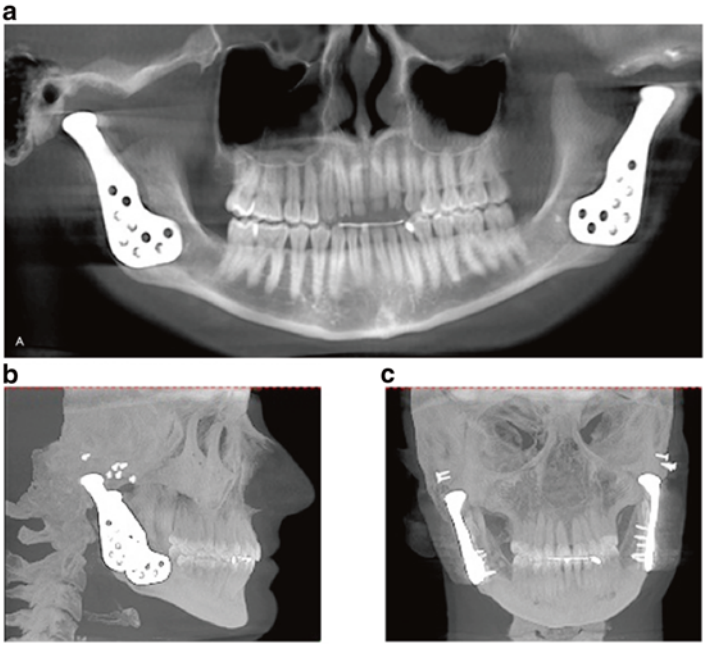


**Fig. 4.18** (**a**) Patient with a history of rheumatoid arthritis and severe temporomandibular joint pain. The patient underwent bilateral joint reconstruction and was able to achieve a pain-free opening of 40 mm. (**b**) Lateral view of the same patient showing good orientation of the prosthesis. (**c**) Anterior view showing well-adapted prosthesis with good bony interface

In 2011, Abramowicz et al. [26] coordinated a study where they retrospectively looked at stereolithic models of patients who had been managed using patient-fitted TMJ TJR devices. They reported that 77 % of the stock TMJ system components fit the stereolithic models with "3 mm or less" of bone modification necessary to achieve an acceptable fit.

Several studies have employed virtual surgical planning (VSP) as part of TMJ TJR. Using VSP, Chandran et al. [27] predetermined how much host bone modification was required to place stock TMJ TJR components and then mimicked this with the use of cutting guides provided by the VSP company that they secured in place with bone screws to assist in an ankylosis case. This computer-assisted planning and intraoperative navigation can lead to improved preoperative planning and more precision surgery in the placement of the stock TMJ TJR device components (Figs. 4.19, 4.20, 4.21, and 4.22).

In 2014, Bai et al. [28] published a study of six patients who underwent total alloplastic joint replacement surgery from November 2013 to March 2014. They used VSP-generated templates as guides for bone alteration in the placement of Biomet stock TMJ TJR components. Their conclusion was that "Digital templates can accurately guide the bone trimming required for placement of Biomet total

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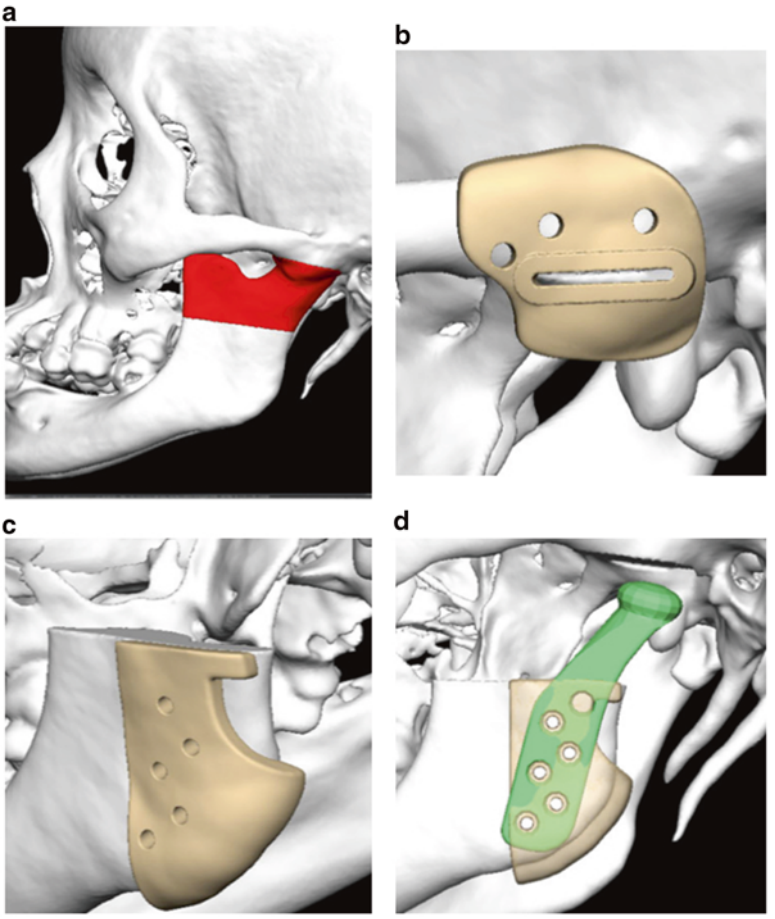


**Fig. 4.19** (**a**) Preoperative CT showing the planned osteotomy. (**b, c**) Computer-designed cutting guides based on the preoperative plan. (**d**) Mandibular cutting guide with the stock Biomet prosthesis component positioned. The cutting guide can be used to drill the screw holes prior to implantation. This ensures correct position and orientation (Images courtesy of Dr. Ron Caloss)

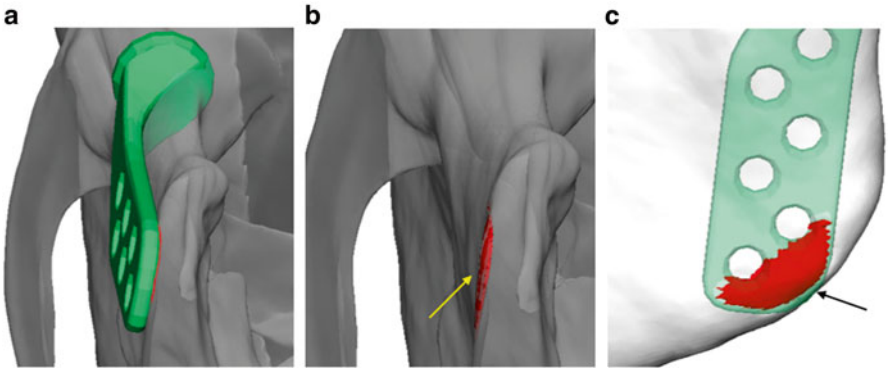


**Fig. 4.20** (**a**) Computer-generated surgical plan with stock prosthesis in place. (**b**) Utilizing this software allows for the evaluation of potential interference (*yellow arrow*). (Images courtesy of Dr. Ron Caloss)

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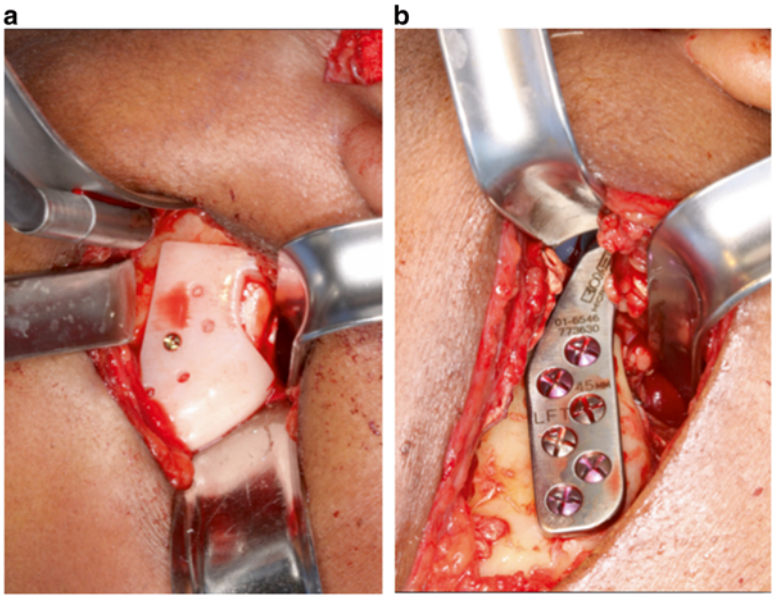


**Fig. 4.21** (**a**) Intraoperative image with cutting guide secured in place. (**b**) Intraoperative image with the mandibular component secured in the correct position (Images courtesy of Dr. Ron Caloss)

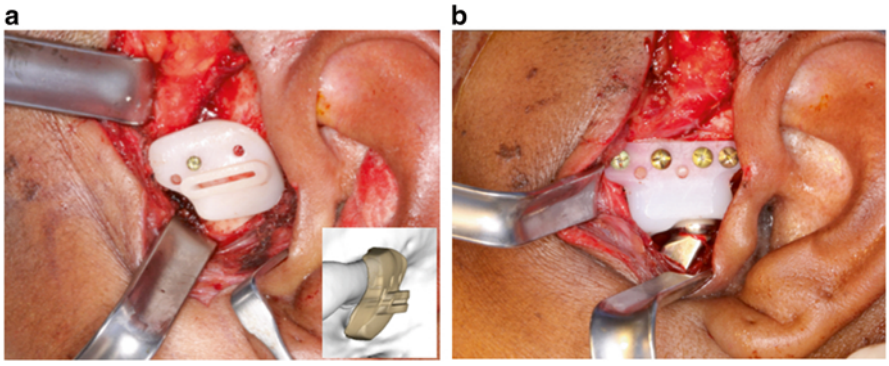


**Fig. 4.22** (**a**) Intraoperative image with the fossa cutting guide secured in place. Computer-generated plan of fossa cutting guide (*insert*). (**b**) Intraoperative image showing excellent matching and position of the prosthesis (Images courtesy of Dr. Ron Caloss)

alloplastic joints. Likewise, these templates help place the prostheses in the desired locations, enhance stability, and avoid damage to the skull base and inferior alveolar neurovascular bundle." Further improvements in this technology may allow this "hybrid approach" to fit the stock prostheses.

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There should be reasonable expectations for success with TMJ TJR especially in patients with functional mandibular problems and continued chronic pain management. A postoperative interincisal opening of 30–35 mm, with a reduction of approximately 60–70 % of preoperative pain levels, and functional diet capability of approximately 75 % of a normal diet are achievable goals with proper placement of TMJ TJR devices. In 1994, McBride [29] stated "As improved biomaterials in new total joint implant systems become available and additional experience is gained with total joint implants, the quality of results obtained will continue to improve to the point where total joint reconstruction will become the treatment of choice for severe temporomandibular joint degeneration."

In the twenty-odd years since that statement was made, there is encouraging reports that that landmark appears to have been achieved. Therefore, in those select patients where a TMJ TJR is indicated, there are proven safe and efficacious options for TMJ TJR with a stock prosthesis.

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# Chapter 5
# Custom TMJ TJR Devices

## Description, Indications and Contraindications, Surgical Technique, and Outcomes

**Louis G. Mercuri**

## 5.1 Introduction

End-stage temporomandibular joint (TMJ) pathology resulting in anatomical architectural form distortion and physiological dysfunction dictates the need for total joint replacement (TJR). The complex nature of the TMJ's functional relationship with the local anatomy and masticatory muscles and the technical requirements of implanting a replacement mean that it is unreasonable to expect the replaced joint to return to its premorbid, fully functional condition.

The essential life functions of mastication, speech, airway support, and deglutition are supported by proper TMJ function and form. This puts the TMJ complex under more cyclical loading and unloading than any other body joint over a lifetime. Therefore, to provide long-term effective outcomes, the TMJ TJR device chosen must be capable of managing the anatomical, functional, and esthetic discrepancies that influenced its choice.

The surgeon should review the pertinent literature and use the TMJ TJR system that best meets the functional and form needs of each patient, based on reported long-term outcomes.

Based on the available refereed and edited literature, this chapter will present the well-accepted orthopedic criteria for the development and utilization of successful TJR devices to establish a rationale for the use of custom TMJ TJR devices in the long-term management of end-stage TMJ disorders.

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## 5.2 Goals of TMJ Reconstruction

Regardless of whether the TMJ is reconstructed using an alloplast, allogenic, or autogenous materials, the following should be the management goals [1]:

1. Improve mandibular function and form
2. Reduce suffering and disability
3. Contain excessive treatment and cost
4. Prevent morbidity

Severe pathology with functional and anatomical distortion dictates the need for total joint reconstruction. Due to the complex nature of joint function and its related muscle function, it is not a reasonable expectation that a reconstructed joint can be returned to "normal" premorbid function. Therefore, there will always be some functional disability involved in any reconstructed joint. In the multiply operated, anatomically distorted joint reconstruction patient, chronic neuropathic pain will be a major component of that patient's disability. Therefore, it is important for both the surgeon and the patient to understand that the primary goal of any type of TMJ reconstruction is the restoration of objective mandibular function and form. Any subjective pain relief gained can only be considered as of secondary benefit [2].

## 5.3 Indications for Total Alloplastic TMJ Replacement

Alloplastic total TMJ reconstruction salvage procedures should be considered for the management of end-stage TMJ pathologic conditions [3]:

### *5.3.1 Inflammatory Arthritis Involving the TMJ Not Responsive to Other Modalities of Treatment*

Since inflammatory arthritis involves a local synovially mediated destructive systemic disease process, and complete synovectomy is not achievable, the orthopedic literature opts for an alloplastic joint replacement in these cases since the results are very predictable [4].

In the TMJ, alloplastic reconstruction has been discussed at length [1–11]. All of these authors agree that when the mandibular condyle is extensively damaged, degenerated, or lost, as in arthritic conditions, replacement with either autogenous graft or alloplastic implant is an acceptable approach to achieve optimal functional and symptomatic improvement.

However, dissatisfaction with some of the aspects of autogenous costochondral grafting, particularly in patients with high-inflammatory arthritic disease (e.g., rheumatoid arthritis) and ankylosis, led to the development and use of total alloplastic TMJ replacement (TMJ TJR) devices with data that can be evaluated to support good results.

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Stern et al. [12] published a case report specifically dealing with the use of an alloplastic total TMJ system (Vitek II—Kent, Houston, TX). While this paper discussed using this modality to manage arthritic TMJ conditions, it was not until 1986, when Zide et al. [13] and Kent et al. [14] published their comprehensive review of rheumatoid arthritis and its surgical management that the subject was specifically addressed.

In 1994, Kent and Misiek provided a comprehensive review of partial and total temporomandibular joint reconstruction. They concluded that when there is a major vertical dimension problem, loss of disc and entire condylar head with chronic pain, hypomobility, malocclusion, such as in advanced arthritic conditions, total joint replacement with an alloplastic prosthesis, is indicated [7].

In 2000, Speculand et al. published a report of 86 total alloplastic joints (27 VK II (Houston, TX) and 59 TMJ, Inc. (Golden, CO)) used to reconstruct degenerative joint disease and rheumatoid arthritis with a median follow-up of 14.5 months (range 1–120 months). Using the subjective (pain and diet) and objective (interincisal opening) criteria they established for this study, they reported an overall success rate of 94 %. However, four patients required replacement of the VK II devices due to foreign body giant cell reactions [15].

Saeed et al. in a 2001 publication reported on a series of seven patients with rheumatoid arthritis whose TMJs were replaced with TMJ, Inc. (Golden, CO) devices. After the mean follow-up of 30 months (range 8–50 months), they report improved subjective (pain and diet) and objective (interincisal opening) scores in these patients and concluded that patients with severe rheumatoid arthritis affecting the TMJ should consider alloplastic total TMJ reconstruction to restore some normal function and appearance [16].

Mishima et al. reported on 6 rheumatoid patients on whom they performed total alloplastic TMJ reconstructions to improve respiratory status and correct occlusal discrepancies. They reported that after surgery, symptoms of daytime sleepiness and nighttime snoring improved, and each patient's ability to masticate solid foods improved significantly. Postoperative cephalograms revealed that both posterior airway space and ramal height were significantly improved as did the dental occlusion. Mean oxygen saturation significantly improved 1 month post reconstruction, whereas apnea–hypopnea indices did not change significantly [17].

Wolford et al. in 1994 reported on the long-term results in 38 cases, followed for a mean of 45 months (range 10–84 months), with the use of autogenous sternoclavicular grafts in 3 groups of patients, one of which ($n = 10$) included patients with documented inflammatory arthritis. The results of this study showed that autogenous sternoclavicular joint TMJ reconstruction had excellent subjective and objective results when used to manage joints not affected by prior failed TMJ alloplastic devices (Proplast-Teflon or Silastic) or joints affected by inflammatory arthritis. In the later, the procedure was successful by the subjective and objective criteria used for the study in only 50 % of the patients with inflammatory arthritis. Ankylosis requiring reoperation and replacement with an alloplastic total TMJ prosthesis was the typical sequelae in these failed inflammatory arthritis cases [18].

Freitas et al. reported on 12 arthritic nongrowing patients (24 joints) requiring total TMJ reconstruction. Six were managed with autogenous sternoclavicular or

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costochondral grafts and six with total alloplastic TMJ prostheses. Each group was followed for a mean of 48.8 months and 58.5 months, respectively. The authors reported that based on the criteria established for the study, the alloplastic TMJ replacement patients had statistically significant better subjective and objective results than did those reconstructed with autogenous bone. They concluded that in the light of these results and the fact that the alloplastic replacement avoided the need for another operative site and potential morbidity decreased operating room time and allowed for simultaneous mandibular advancement with predictable long-term results and stability that alloplastic TMJ replacement was more appropriate for total TMJ reconstruction in patients with low-inflammatory or high-inflammatory arthritic conditions [19]. They also reported long-term stability of the orthognathic component of management of these cases [20, 21].

In the late stages of the other inflammatory arthritic diseases such as psoriatic arthritis, juvenile idiopathic arthritis, systemic lupus erythematosus, Reiter's Syndrome, gout, and pseudo-gout, or when severe condyle, articular eminence and glenoid fossaosteolysis result in functional and/or occlusal-facial dysfunction or ankylosis, TMJ TJR is indicated [4].

In light of these published experience in both the orthopedic and oral and maxillofacial surgery, and the literature comparing autogenous versus alloplastic total TMJ replacement in arthritic conditions, it appears that TMJ TJR is appropriate for the management for advanced stage arthritic disease of the temporomandibular joint (Fig. 5.1).

### *5.3.2 Recurrent Fibrosis and/or Bony Ankylosis Not Responsive to Other Modalities of Treatment*

The traditional management of complete bony TMJ ankylosis has been gap arthroplasty with autogenous tissue graft or alloplastic hemiarthroplasty reconstruction [2]. While the autogenous grafting techniques develop form, mandibular function is typically delayed. Since autogenous graft mobility during healing will compromise its incorporation into the host environment or compromise its blood supply, early mandibular mobilization often leads to graft/host interface failure [9]. Matsuura et al. reported a high incidence of failure and ankylosis of autogenous costochondral grafts in sheep after condylectomy if the jaws were only partially immobilized [22].

Saeed and Kent reported a high incidence of re-ankylosis in patients with ankylosis who underwent autogenous costochondral TMJ reconstruction and advised caution in using this technique in this group of patients [23].

For the patient with re-ankylosis, placing autogenous tissue such as bone into an area where reactive or heterotopic bone is forming intuitively makes no sense. Orthopedic surgeons will typically opt for total alloplastic joint replacement in similar situations with other joints [24].

In the light of the biological considerations and the orthopedic experience, total alloplastic reconstruction should be considered in the management of these cases involving the TMJ (Fig. 5.2).

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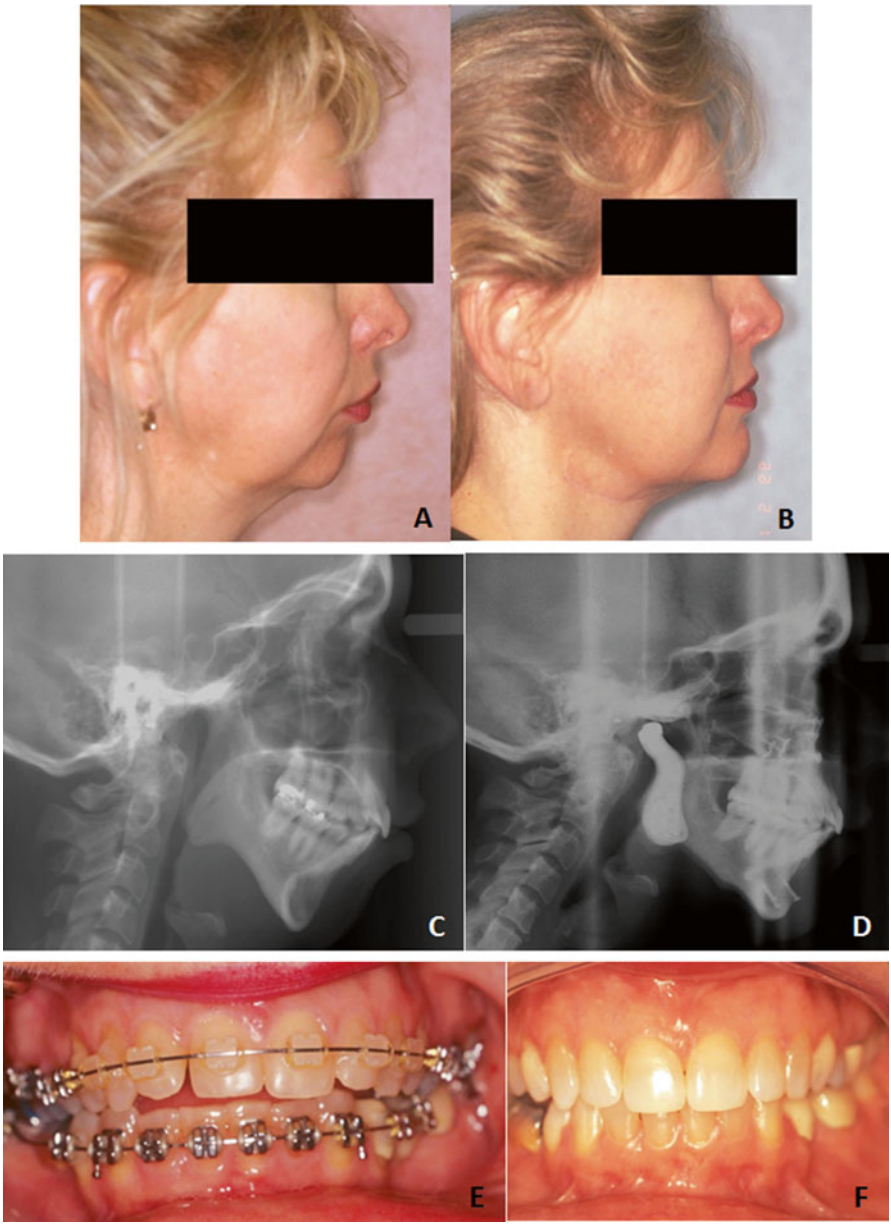


**Fig. 5.1** (**a**) High inflammatory arthritis patient (RA) preoperative. (**b**) Postoperative custom TMJ TJR (**c** and **e**). Preoperative. (**d** and **f**). 16 years postoperative

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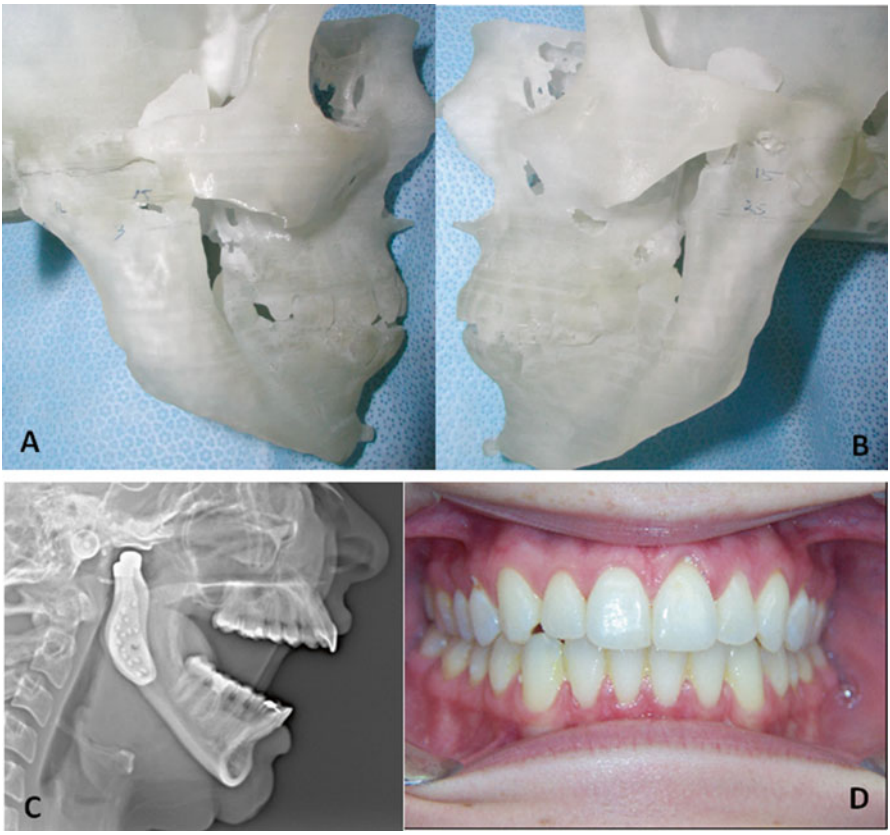


**Fig. 5.2** (**a** and **b**) Bilateral TMJ ankylosis in adult patient preoperative. (**c** and **d**) 5 years postoperative bilateral custom TMJ TJR

#### 5.3.2.1 TMJ Ankylosis in Growing Subjects

Classically, pathologic, developmental, and functional disorders affecting the TMJ in growing patients have been reconstructed with autogenous tissues. Autogenous costochondral grafts (CCG) are reported as the "gold standard" for these TMJ reconstructions [25–30].

In growing patients, theoretically autogenous (e.g., CCG) allografts will "grow with the patient." However, often this so-called "growth potential" has been reported to be unpredictable or to result in ankylosis. These complications can occur either as the result of the allograft and/or fixation failure or because of the uncooperative nature of the young patient with physical therapy after reconstruction [25, 26, 30–32].

Studies have even questioned the necessity for using a cartilaginous graft to restore and maintain mandibular growth [33, 34]. Long-term reports of mandibular growth in children whose TMJs were reconstructed with CCG show that excessive

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growth on the treated side occurred in 54 % of the 72 cases examined, and growth equal to that on the opposite side occurred in only 38 % of the cases [35–40].

Furthermore, Peltomäki et al. reported investigations of mandibular growth after CCG, supported previous experiments with regard to the inability of the graft to adapt to the growth velocity of the new environment [41–43].

On the basis of the problems that have been reported with CCG TMJ reconstruction in children, such as graft failure, unpredictable growth, ankylosis, and potential for donor-site morbidity, and the orthopedic experience and success reported with alloplastic TJR in improving the quality of life of growing patients with severe anatomic and functional joint disorders, it seems reasonable to consider examining the feasibility of alloplastic TMJ TJR for the following conditions in children:

1. High inflammatory TMJ arthritis unresponsive to other modalities of treatment
2. Recurrent fibrosis and/or bony ankylosis unresponsive to other modalities of treatment
3. Failed tissue grafts (bone and soft tissue)
4. Loss of vertical mandibular height and/or occlusal relationship because of bony resorption, trauma, developmental abnormalities, or pathologic lesions

To continue to reoperate in children with failed, overgrown, or ankylosed CCG, with either autogenous bony or soft tissue replacements (or both), using the same modalities that failed previously, when there may be a more appropriate solution available, seems myopic. These patients would be better off undergoing alloplastic TMJ TJR knowing that, depending on growth, revision and/or replacement surgery may likely be required in the future, rather than incurring continued CCG failures that will also very likely require further surgical intervention in the future [44] (Fig. 5.3).

### *5.3.3 Failed Tissue Grafts (Bone and Soft Tissue)*

Ioannides and Maltha [45] reported the use of autogenous auricular cartilage led to the use of this technique in autogenous disc replacement. However, Takatsuka et al. investigated histologically auricular cartilage after discectomy in the rabbit TMJ and found fibrous adhesion of the grafted auricular cartilage to the condyle and the presence of a fibrous layer containing fragmented cartilage on the articular surface. They concluded that auricular cartilage did not appear to be an ideal material for disc replacement [46]. Other investigators reported similar results [47, 48].

The biology of autogenous tissue grafting success requires that the host site have a rich vascular bed. Unfortunately, the scar tissue always encountered in the multiply operated patient does not provide an environment conducive to the predictable success of free and occasionally vascularized autogenous tissue grafts. Marx reports that capillaries can penetrate a maximum thickness of 180–220 μm of tissue, whereas, scar tissue surrounding previously operated bone averages 440 μm in thickness [1]. This may account for the clinical observation that free autogenous tissue grafts, such as cartilage, costochondral, and sternoclavicular grafts often fail

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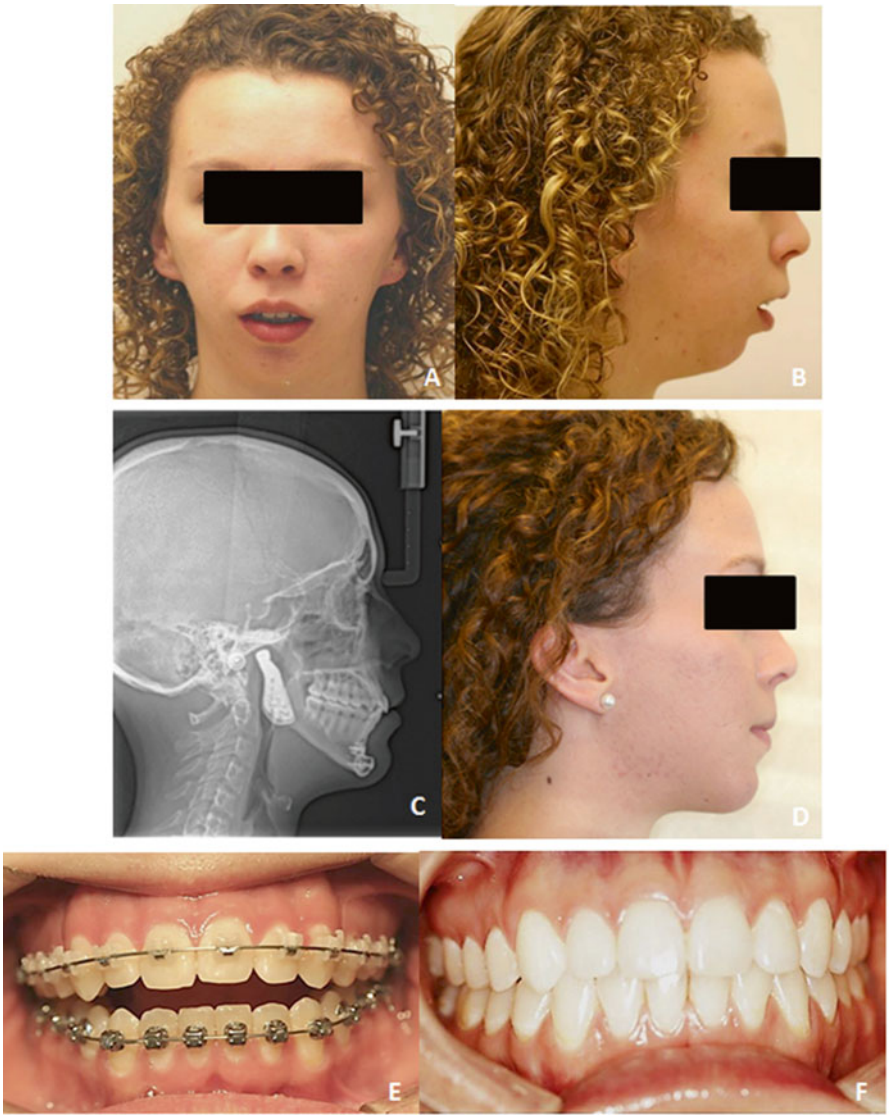


**Fig. 5.3** 13-year-old ICR patient (**a, b,** and **e**). Preoperative bilateral custom TMJ TJR. (**c, d,** and **f**). 5 years postoperative (*Courtesy of Dr. Donald Kalant, Sr. Naperville, IL*)

in cases of multiply operated patients or those with extreme anatomical architectural discrepancies resulting from pathology (e.g., failed autogenous materials).

The CCG has been the most frequently recommended autogenous bone for the reconstruction of the TMJ due to its supposed ease of adaptation to the recipient site, its gross anatomical similarity to the mandibular condyle, reported low morbidity rate at the donor site, and its growth potential in juveniles [25–30].

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Reitzik reported that in an analogous situation to autogenous costochondral grafting, cortex-to-cortex healing after vertical ramus osteotomy in monkeys requires 20 weeks and probably 25 weeks in humans [49]. Typically in patients reconstructed with CCGs, maxillomandibular fixation is maintained for only 4–6 weeks in order to return the mandible to function and prevent ankylosis. Despite screw/plate fixation, micromotion of these free grafts will invariably occur with the early mandibular function resulting in shear movements of the graft leading to poor vascularization, nonunion, and/or potential failure [50]. This fact along with the compromise in vascularity discussed above undoubtedly account for autogenous CCG failures seen in these cases. Therefore, in light of the fundamental biological issues discussed and reported, TMJ cases involving multiply operated, failed prior alloplastic materials, anatomically distorted, and severe intra-articular pathology should be replaced with a total alloplastic device to achieve the optimum outcomes.

These results may along with the vascularity appear to be two reasons for failure of autogenous grafts in multiply operated TMJ patients, or those with severe anatomical discrepancies and/or end-stage TMJ pathology. Also, the work of Henry and Wolford indicates that reconstruction with autogenous materials is much less predictable than total alloplastic TMJ reconstruction, especially in the later scenario [51] (Fig. 5.4).

### *5.3.4 Failed Alloplastic Joint Reconstruction*

Due to the osteolysis around failed alloplasts and the resultant anatomical discrepancies of the host bone architecture, it is difficult to adapt and fixate autogenous materials stably to the distorted anatomical remnants of either the fossa or ramus. Further, the foreign body giant cell reactions associated with failed or failing materials or devices provide a poor environment for the introduction of an autogenous graft as discussed above. Henry and Wolford's results confirm this as they reported that reconstruction with autogenous materials was much less predictable than alloplastic replacement in these cases [51].

Mercuri and Giobbe-Hurder discuss this issue at length in a report where they evaluated long-term outcomes with total alloplastic TMJ reconstruction in patients with prior exposure to failed Proplast-Teflon and/or silicone rubber. They found that while the TMJ TJR devices remained functional long-term (60.2 months mean), the patients exposed to failed materials had lower subjective improvement scores (pain, jaw function, diet consistency) when matched to a group of patients never exposed to a failed TMJ alloplast. Therefore, based on the available literature, it appears appropriate to reconstruct TMJ affected by prior failed alloplastic material with TMJ TJR devices rather than autogenous tissues [52].

Orthopedists and biomedical engineers have been studying the effect of failed and failing devices on the long-term outcomes of future implanted alloplastic devices. There is now a question, yet to be answered, as to whether failure of a prior

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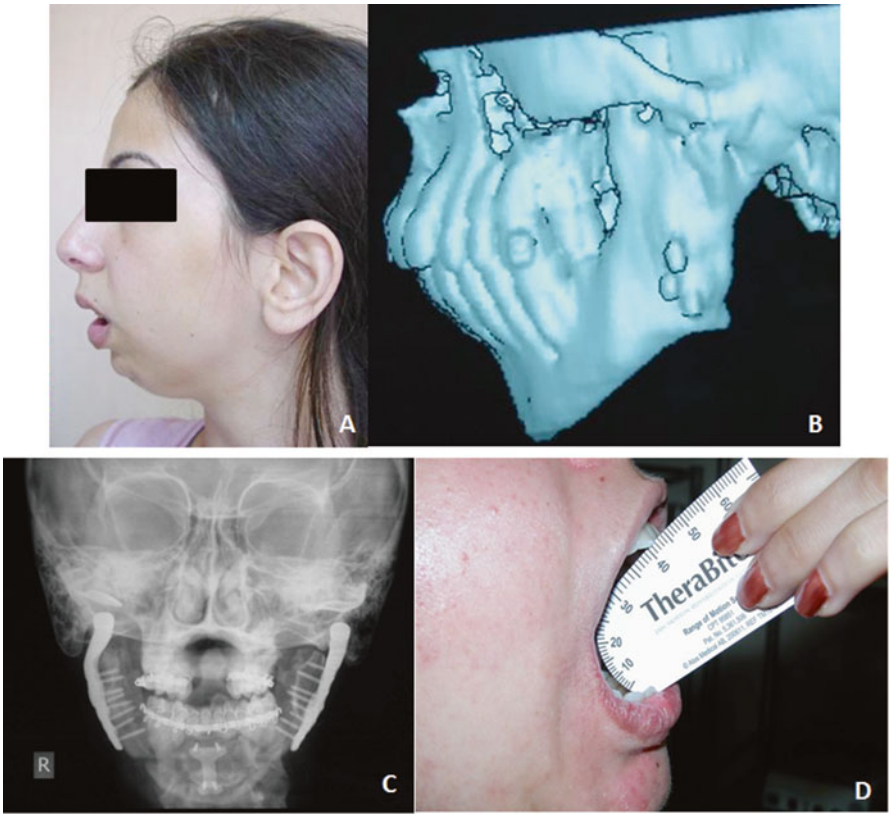


**Fig. 5.4** (**a** and **b**) Bilateral traumatically induced TMJ ankylosis preoperative status post 2 re-ankylosis after bilateral costochondral grafts. (**c** and **d**). 5 years postoperative bilateral custom TMJ TJR (*Courtesy of Dr. Michael Bowler*, *New Castle*, *NSW*, *Australia*)

implanted device results in a cell-mediated immune response that negatively affects the outcome with any future implanted alloplast. This topic is discussed in detail in Chap. 9 (Fig. 5.5).

### *5.3.5 Loss of Vertical Mandibular Height and/or Occlusal Relationship Due to Bony Resorption, Trauma, Developmental Abnormalities, or Pathologic Lesions*

Loss of posterior mandibular vertical dimension due to developmental abnormalities, pathology, or traumatic injury all result in a discrepancy in the occlusion of the teeth. This is manifested as either an anterior (bilateral loss) or lateral (unilateral

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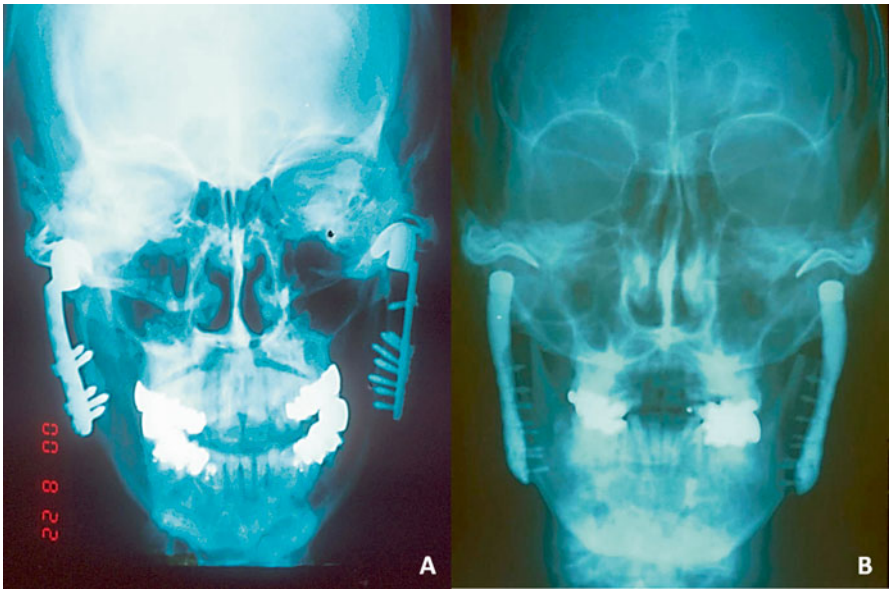


**Fig. 5.5** (**a**) Bilateral failed stock metal-on-metal TMJ TJR devices. Note the loose fixation screws and bilateral fractured fossas. (**b**) Bilateral custom TMJ TJR 12 years postoperatively

loss) open bite deformity. These situations can be managed by diagnosis of the etiology of the problem and correction at the site of the pathology. In the case of primary TMJ etiology, joint reconstruction rather than osteotomy should be considered. Once again, the reconstructive surgeon must take into consideration the nature of the pathology, the patient's prior local surgical history, and the state of the host bone architecture before deciding on the type of TMJ reconstruction. Discussion of management of extensive and complex mandibular segmental defects is in Chap. 6 (Fig. 5.6).

## 5.4 Relative Contraindications for Total Alloplastic TMJ Replacement

### *5.4.1 Age of the Patient*

Since total alloplastic TMJ reconstruction prostheses have no potential for growth, the benefits of their use in growing patients over autogenous tissue must be considered carefully before using them in such cases. This issue is discussed at length above (5.3.2.1).

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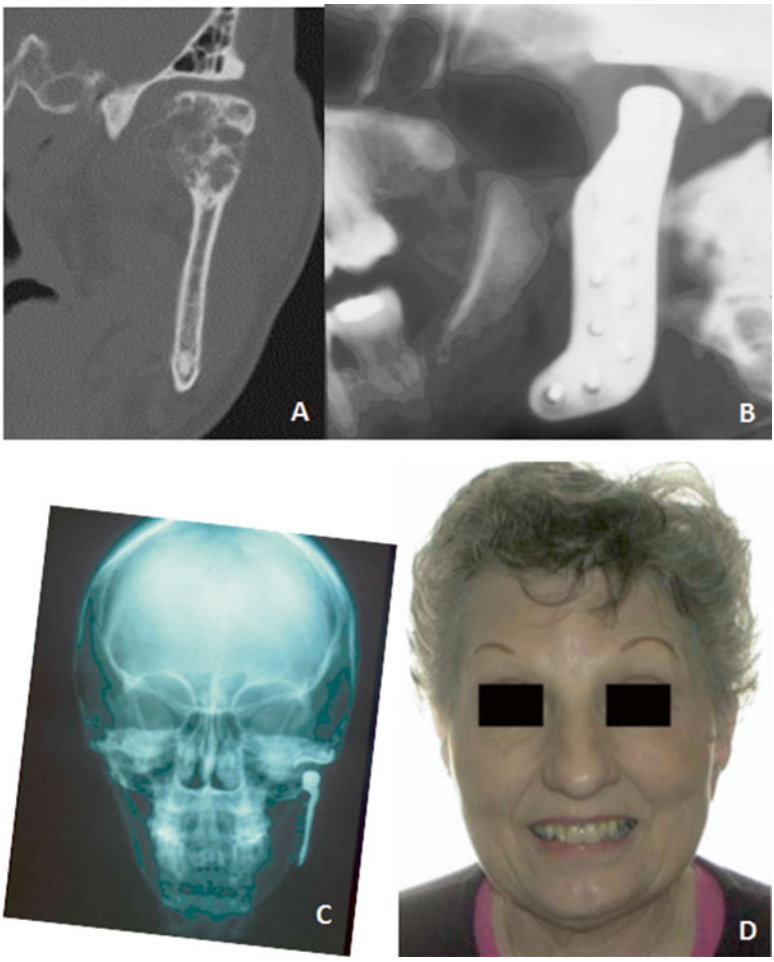


**Fig. 5.6** (**a**) *Left* TMJ low-grade fibrosarcoma preoperatively (**b–d**). Postoperative left custom TMJ TJR

### *5.4.2 Mental Status of the Patient*

Is the patient psychologically prepared to handle the permanent loss of a body part with the understanding that revision and/or replacement surgery in the future may be required? Does the patient have unrealistic expectations of complete relief of pain and normal jaw function after alloplastic TMJ reconstruction? Is the patient willing and able to do the post-implantation physical therapy required to obtain maximum functional benefit from the procedure? Many of the multiply operated, functionless TMJ patients require pre-reconstruction psychological counseling in order for them to accept the limitations of further surgery, should they choose to proceed.

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### 5.4.3 Uncontrolled Systemic Disease

As with any form of an alloplastic implant in these situations, once the disease process in under control and the risk/benefit ratio is determined for the individual patient, implantation can proceed. This is also a relative contraindication for autogenous or allogenic implantation as well.

### 5.4.4 Active Infection at the Implantation Site

As with any alloplastic material, introduction into an infected or contaminated area can result in failure of the device to stabilize, leading to its failure under function. This is due to the unpredictability of the initial fixation of the device to infection-compromised hard and/or soft tissue. While this is true of all alloplasts, it is of particular concern with implants that have a planned function under load, such as any TMJ implant would. This is discussed in more detail in Chap 8.

### 5.4.5 Documented Allergy to the Implant Component Materials

Documented allergy to commercially pure (CP) titanium, titanium alloy, cobalt–chrome–molybdenum alloy, and ultrahigh molecular weight polyethylene (UHMWPE) is rare. Although 12–15 % of the population can be sensitive to the nickel alloy in cobalt–chrome–molybdenum components, far fewer reports of such allergic reactions have been reported in the orthopedic literature in total alloplastic joint patients. Patients with documented allergy to the component metals of any device should not be exposed to that material in any new device. This is discussed in more detail in Chap 9.

## 5.5 Established Criteria for Successful Alloplastic TJR Devices

After years of use, orthopedic surgeons developed accepted criteria for successful TJR device utilization [1]. Applying these well-accepted criteria to TMJ TJR long-term successful utilization, a rationale can be established for the use of custom TMJ TJR devices.

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### 5.5.1 *The Components of Any TJR Device Must Be Stable In Situ at Implantation*

All implanted alloplastic devices depend on the principle of fixation component biointegration (screws in the case of TMJ devices) for their stability and longevity. Biointegration implies the direct incorporation of the fixation components by bone without the preliminary phase of fibrous tissue ingrowth. The requirements for biointegration are essentially the same as for primary fracture healing; basically the transmission of forces from the implant to the bone and vice versa must occur without relative motion or without intermittent loading. To assure long-term success, the most important principle in TMJ TJR must include the primary stability of the components at implantation [53].

The need for custom components in orthopedic TJR is uncommon. The bony anatomy of the pelvis, femur, and tibia affords the use of modular stock components that can be stabilized initially with screws, press-fitting, or cementation. The bony anatomy of the mandibular ramus and the temporal glenoid fossa do not provide such options for TMJ TJR. Therefore, all TMJ TJR devices must utilize screw fixation for initial fixation and stabilization of both the fossa and ramus/condyle components.

Compounding the anatomical and stability issues is the fact that most patients presenting with indications for TMJ TJR have deformed local bony anatomy. This may be the result of numerous failed prior surgical interventions, failed materials, as well as systemic primary or secondary end-stage disease pathology. Attempting to make stock TMJ TJR components fit and remain stable in these situations confronts the surgeon with a difficult challenge.

At implantation, to make stock TMJ TJR components fit, it is often the case that precious host bone must be sacrificed to create stable component-to-host-bone contact. To achieve a fit in complex cases, the surgeon may have to consider bending a stock component or shimming it with autogenous bone, bone substitute, or alloplastic cement. These tactics can lead to component or shim material fatigue and/or overload fostering early failure under repeated cyclical functional loading (Fig. 5.7).

Of more concern is the potential for the development of micromotion of any altered or shimmed component. Micromotion interferes with screw fixation biointegration which is necessary for component stability. Micromotion leads to the formation of a fibrous connective tissue interface between the altered component and the host bone. This can result in early loosening of the screw fixation leading to component mobility and potential early catastrophic or certain later premature device failure (Fig. 5.8).

Custom TMJ TJR components are designed and manufactured to each patient's specific anatomical condition on a stereolaser (SL) model developed from a protocol-computed tomography (CT) scan. Therefore, the fossa and ramus components can be designed and manufactured to conform to any unique or complex anatomical host bone situation.

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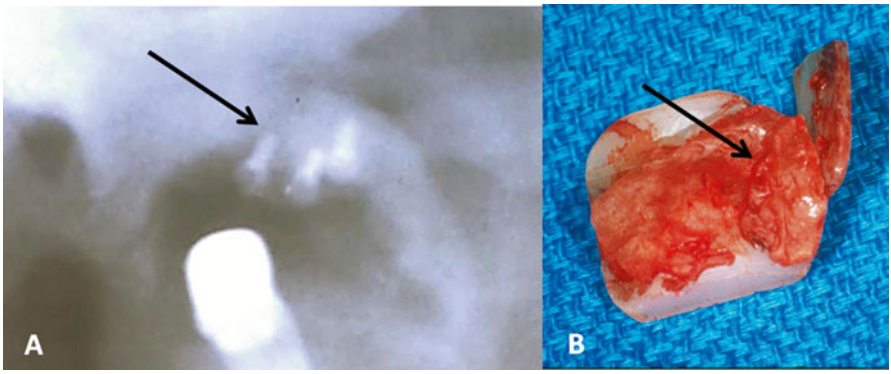


**Fig. 5.7** (**a**) Failed stock TMJ TJR fossa fixation screw due to osteolysis resulting from cracked thin layer of PMMA shim (**b**)

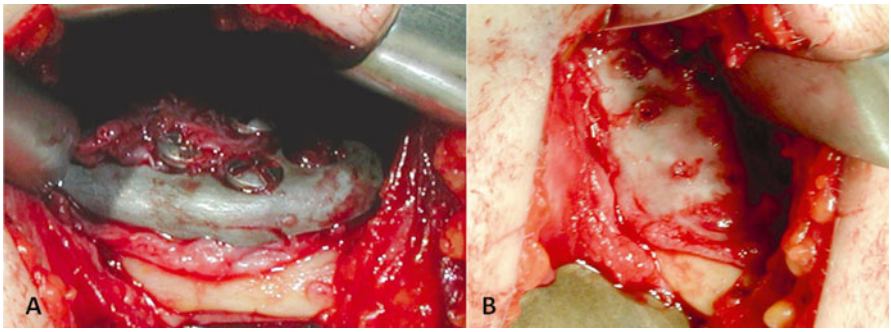


**Fig. 5.8** (**a**) Failed right stock ramus component due to loose fixation screws resulting in micromotion. Note the development of the thick fibrous connective tissue mantle between the device component and the host bone as the result of micromotion (**b**)

At implantation, neither the custom TMJ TJR components nor the host bone requires alteration or shimming to achieve initial component screw fixation and stability. The screw fixation secures the components intimately to the host bone mitigating the potential for micromotion and maximizing the opportunity for fixation screw biointegration.

### *5.5.2 The Materials from Which TJR Devices Are Manufactured Must Be Biocompatible*

In 1960, Sir John Charnley reported the use of a total alloplastic prosthetic hip replacement system. He developed a metal-backed polyethylene polymer acetabular cup which articulated with a stainless steel femoral head component that was cemented in place with polymethylmethacrylate [54].

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Modifications of this device using titanium (Ti), titanium alloy (Ti–6Al–4V), cobalt–chromium–molybdenum (Co–Cr–Mo), and ultrahigh molecular weight polyethylene (UHMWPE) have led to these materials becoming the gold standard for low friction orthopedic TJR. Acceptance of this management option for end-stage joint disease has made the modern practice of orthopedic surgery impossible without the availability of TJR devices [55, 56].

Employing the most advantageous physical characteristics of biocompatible materials is an essential consideration in the design and manufacture of any TJR device. Wrought, unalloyed titanium was originally chosen for endosteal implants and bone plates because of the rapid reaction of elemental titanium with oxygen in the air to form a thin chemically inert titanium oxide layer. This layer provides a favorable surface for biointegration of implant components with bone. Titanium also has properties of strength, corrosion resistance, ductility, and machinability. The extensive literature demonstrating its biocompatibility and biointegration makes titanium the metal of choice for the manufacture of the major components of TJR devices to date [57].

Co–Cr–Mo with its relatively high carbon content contributes to its strength, polishability, and biocompatibility. Its excellent wear characteristics when articulated against an UHMWPE presently make it the standard for the bearing surface for most orthopedic TJR devices [57].

Cast Cr–Co, often employed in the manufacture of stock TMJ TJR devices, is physically inferior to any wrought alloy. Metallurgical flaws such as inclusions and porosity found in cast Cr–Co components have been associated with the fatigue failure of metal-on-metal prostheses. These flaws may also lead to the failure of Cr–Co TJR components resulting in noxious metallic debris (metalosis) found in adjacent tissues [58] (Fig. 5.9).

UHMWPE is a linear unbranched polyethylene chain with a molecular weight of more than one million. Testing over four decades of use in orthopedic TJR has led to the conclusion that UHMWPE is considered to have excellent wear and fatigue resistance for a polymeric material [59]. To date, no cases of UHMWPE particulation-related osteolysis have been reported in the TMJ TJR literature [60–63] (Fig. 5.10).

TMJ TJR materials are discussed in detail in Chap. 2 and 10, and their possible effect on Periarticular tissues is discussed in Chap. 10

### *5.5.3 TJR Devices Must Be Designed to Withstand the Loads Delivered over the Full Range of Function of the Joint to Be Replaced*

An important advantage afforded by a custom TMJ TJR is that the components can be specifically designed to manage the loads posed in the face of unique anatomic situations. For example, the center of rotation of the condyle of a custom TMJ TJR can be moved vertically to accommodate closure of the open bite deformity; or the ramus component can be shaped to accommodate the amount of available

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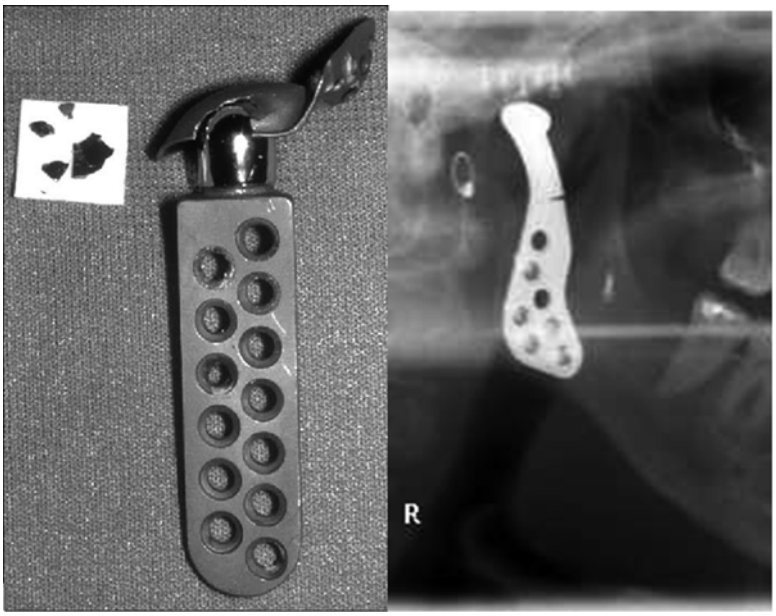


**Fig. 5.9** (**a**) Failed right TMJ stock thin cast Cr–Co fossa. (**b**) Failed right TMJ stock cast Cr–Co ramus component

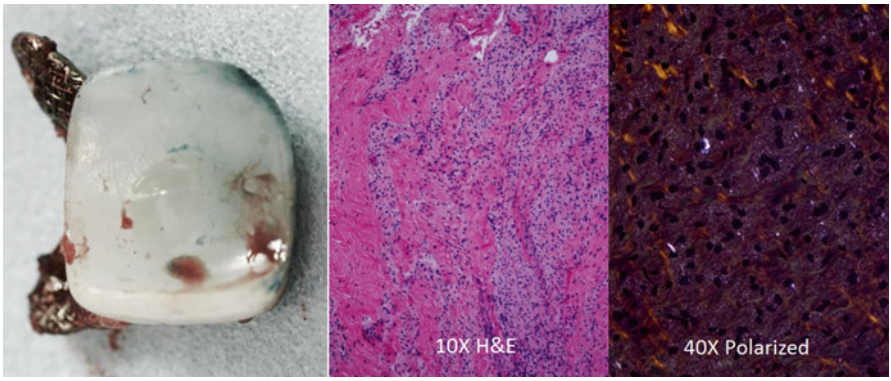


**Fig. 5.10** (**a**) Right custom TMJ fossa component retrieved after 12 years. Note the "dimple" indicating cold flow. (**b**) Light and (**c**) Polarized microscopy demonstrating little particulation

mandibular host bone. This ability to vary the design to cope with the existing anatomy leads to a more predictable result in any complex clinical situation [53] (Fig. 5.11).

Custom TMJ TJR design from anatomically accurate SL models will maximize screw fixation position options for initial component stability. The positions of the

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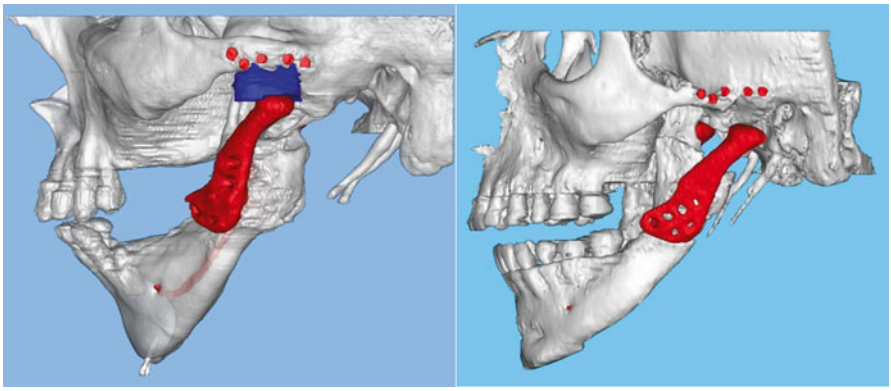

**Fig. 5.11** Examples of the inability of stock components to deal with the variations in ramus anatomy caused by pathology resulting in the need for revision and replacement with custom components

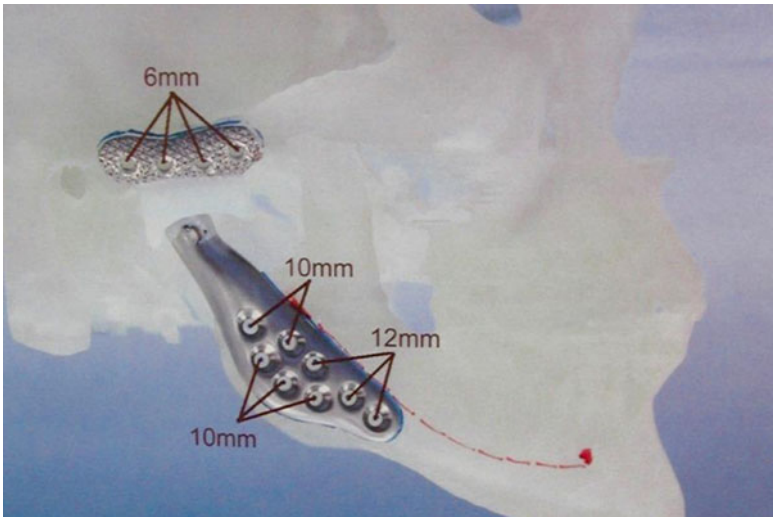


**Fig. 5.12** Custom TMJ TJR device on SL model with exact fixation screw lengths indicated

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screw holes can be designed to avoid the inferior alveolar canal, thereby eliminating potential injury to its contents during fixation (Fig. 5.12).

Proper bicortical screw length can be predetermined and prescribed. This eliminates time consuming and frustrating intraoperative screw hole "probing" to determine the appropriate fixation screw length. Knowing the proper screw length eliminates the potential for placing screws that are too long, which may be the cause of functional pain. In the case of the fossa component, if the sharp tips of the

fixation screws penetrate beyond the medial cortex of the zygoma they can irritate the temporalis muscle. In the case of the mandibular component, too long screw tip impingement on the medial pterygoid is the concern.

### *5.5.4 The Implantation Surgery Must Be Performed for the Proper Indications and Aseptically*

As with any surgical technique, outcomes are only predictable when the procedure chosen is performed correctly and aseptically, for the proper diagnosis, at the appropriate time, for the right patient, and with the right equipment.

Schmalzried and Brown report that the major causes of orthopedic TJR failures are the result of failure of the surgeon's implantation technique or the limitations of the device implanted to properly manage the posed anatomical situation. A custom TMJ TJR device mitigates both issues [64].

Ravi et al. reported the after primary total hip and knee replacements, the risks for dislocation and early revision in patients whose surgeons had carried out less than 35 procedures were 48 and 44 % higher, respectively, than in patients whose surgeons had carried out greater than 35 procedures [65].

In a prospective study to determine the risk factors related to total knee replacement surgical site infections, Levant et al. determined that of the factors studied, the time it took to complete the surgery was statistically significant [66]. Despite the fact that surgical site infection is low in TMJ TJR (See Chap. 8), it would appear that the surgical time it takes to make a stock TMJ TJR device fit is necessarily longer than placing a custom TMJ TJR device that is made to fit.

## 5.6 Relative Disadvantages of Custom TMJ Devices

### *5.6.1 Cost*

Custom TMJ TJR is thought to be more costly than stock TJR or autogenous tissue for TMJ reconstruction, but the extra operating room time, personnel, and resources must be considered in the latter scenarios. Also, in view of the potential for increased autogenous tissue donor-site morbidity resulting in an increased length of hospital stay and the unpredictable nature of the results of autogenous tissue grafting, the economic impact of TMJ TJR is likely less overall. Since custom TMJ TJR components are designed "made to fit," manipulation and implantation time will be reduced. In contrast, with stock TMJ TJR components, the surgeon must "make them fit" requiring increased time and incurring added expense.

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### 5.6.2 *Two-Stage Procedure Required for Ankylosis Cases*

The protocol, CT scan generated, SL model from which custom TMJ TJR components are designed and manufactured has a reported mean dimensional accuracy of 97.9 % [67]. Therefore, in the case of ankylosis/re-ankylosis a two-staged protocol is recommended.

In the first stage, the surgeon must perform an adequate gap arthroplasty (2–2.5 cm) and insert a spacer or "place holder" (carved to fit silicone rubber block, ocular prosthesis, etc.) to prevent the reformation of tissue and/or bone while the custom device is designed and manufactured. The patient must be placed into maxillomandibular fixation (MMF) to prevent movement of the spacer or change in bony architecture and/or occlusion. A postoperative protocol CT scan is then made and the SL model developed. The custom TMJ TJR components are designed and manufactured from that model to the specific anatomical circumstances of the specific case (Fig. 5.13).

In the second stage, the spacer is removed and the custom TMJ TJR components are fixated. An autogenous abdominal fat graft is placed around the articulation to inhibit formation of heterotopic bone and re-ankylosis. The patient then begins active postoperative physical therapy.

Pearce et al. described the use of preoperatively created templates to obviate the two-stage protocol described above [68]. Virtual Surgical Planning (VSP) can supply templates to assist in doing this in one-stage as well. However, many surgeons believe in order to realize all of the benefits afforded by a custom TMJ TJR device; the best fit for the components will be achieved and assured by using the two-stage protocol. The concern often raised about maintaining MMF between stages is moot since ankylosis patients cannot open their mouths before the first-stage procedure.

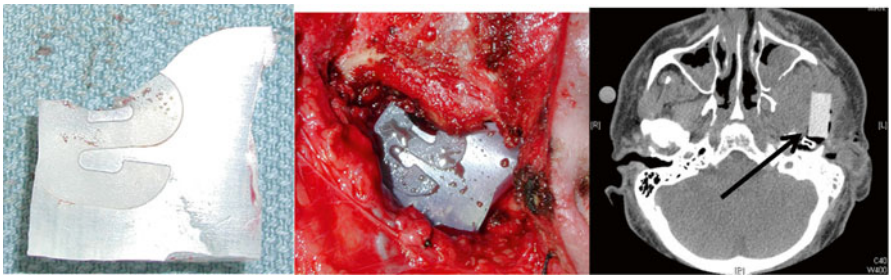

**Fig. 5.13** (**a**) Carved-to-fit silicone rubber spacer. (**b**) Spacer in place. (**c**) Axial view of spacer in protocol CT scan

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### 5.6.3 *Material Wear, Design, and Long-Term Stability*

TMJ TJR is a biomechanical rather than a biological solution to end-stage TMJ disease. Therefore, as with any implanted functioning biomechanical device, revision surgery may be necessary in the future to remove scar tissue from around the articulating components. Replacement of one or both TMJ TJR components over time due to material wear and/or failure is also a prospect.

It has been demonstrated that the use of appropriate biomaterials and design parameters can decrease material wear and increase the longevity of TJR devices [69]. Proper choice of biomaterials based on their characteristics is presented above. Design and material wear characteristics related to longevity must be considered. Stock TMJ TJR systems with multiple "make fit" choices, designed and manufactured from either thin cast Co–Cr fossa or all UHMWPE fossa components, utilizing cast Cr–Co ramus/condyle components, can pose multiple design and material issues.

Metal-on-metal design geometry can only be applied theoretically to a TJR hip where rotation is the major functional movement. For a metal-on-metal TJR hip to be successful, it requires tightly constrained radial clearances of less than 200 μm between the all metal acetabular cup and the all metal femoral head. If this conformity is not achieved at surgery due to host anatomical conditions or surgeon fit miscalculation, wear associated metal particulation will lead to metalosis, osteolysis, loosening, and micromotion resulting in device failure [64, 70].

In orthopedics, metal-on-metal devices would never be designed for a non-constrained joint. The TMJ, even after TJR, has functional movements that are unconstrained. Stresses and strains directly or eccentrically vectored against an incomplete or inadequate component-to-host-bone interface during TJR create wear. Unstable, thin, cast Co–Cr fossa cyclically loaded by the metal condylar head can lead to micromotion, galling, fretting corrosion, component screw loosening, and/or thin cast metal fossa component fatigue and fracture (Fig. 5.9a).

Cold flow is the property which allows UHMWPE under loading to develop alteration of shape rather than particulation [59] (Fig. 5.10a). In orthopedic TJR this property dictates that the stable component of a TJR articulation (i.e., the fossa) is held in position and stabilized by a stronger material (metal). Custom TMJ TJR fossa components are designed and manufactured to that material specification. Further, the metallic component of a custom fossa offers solid structure through which the zygomatic arch fixation screws pass.

Stock TMJ TJR devices with an UHMWPE flange screw fixation design have the potential to develop material cold flow around the screw holes or fracture should micromotion occur if the surgeon cannot or does not make the fossa component fit properly. Cold flow of the resultant screw fixation hole can lead to loosening of the stock fossa fixation screws and increased micromotion under repetitive loading resulting in device failure.

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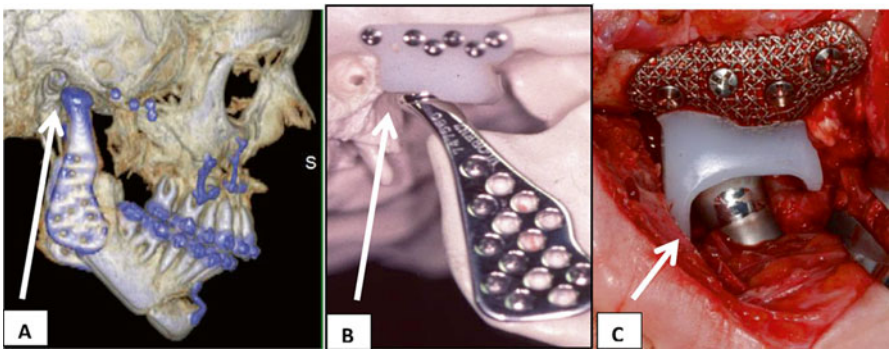


**Fig. 5.14** (**a**) Right stock TMJ TJR device condylar head displaced into the auditory canal after bi-maxillary orthognathic surgery due to lack of posterior stop as demonstrated in frame (**b**). (**c**) Custom TMJ TJR fossa exhibiting posterior stop that will prevent posterior condylar head displacement

Hallab listed eight reasons why an unbacked all-UHMWPE fossa component is not favored in orthopedics, especially when placed against host bone: increased back-side wear (component-to-host bone) under function; poor surface for bone fixation (hydrophobic UHMWPE vs. hydrophilic bone); decreased bone remodeling on the surface of the UHMWPE; no macro-texturing to enhance short and long-term bone attachment strength; can lead to increased potential for biofilm infection (due to decreased cell attachment); increased chance of "cold flow" and UHMWPE fracture; less control over host bone side implant orientation due to greater likelihood of osteolysis on the host bone side over time; and a poor surface for cementing which will probably result in high wear and micromotion [53].

Stock fossa components are designed without a posterior stop to prevent the TMJ TJR device condyle from displacing posteriorly. Should the stock condyle not be perfectly aligned in the center of the stock fossa mediolaterally and/or anteroposteriorily, the condyle can displace posteriorly and impinge on the tympanic plate and/or the auditory canal. This can result in pain and mandibular dysfunction, malocclusion, and facial deformity. There is also the potential for infection should there be a pressure-related perforation associated with the auditory canal. This is of special concern when using a stock TMJ TJR in combination with orthognathic surgical procedures [71]. The custom TMJ TJR fossa has a posterior stop, alleviating this concern (Fig. 5.14).

Since the components of a custom TMJ TJR interface so well with the host bone and the screw fixation is stable from implantation, mandibular function can begin immediately after implantation. This is essential in severe anatomical joint disease because masticatory muscle function has been compromised over time making physical rehabilitation difficult if delayed.

Salter in his work on continuous passive motion after orthopedic joint surgery demonstrated the importance of early active physical therapy to the long-term functional results of joint surgery [72].

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## 5.7 TMJ Concepts Custom TMJ TJR Surgical Technique [73]

### *5.7.1 Preparation for Surgery*

The avoidance of contamination of the surgical site during any alloplastic TMJ replacement surgery is important, therefore, it is essential that complete sterility be maintained at the implantation sites throughout the procedure. The following patient preparation should be considered:

(a) The patients should be directed to thoroughly wash and rinse their hair the night before surgery with a mild shampoo and avoid the use of hair spray or styling gels the day of the surgery.
(b) As with any presurgical antibiotic prophylaxis regimen, IV antibiotic (e.g., cefazolin 1 g, clindamycin 600 mg) is begun 1 h preoperatively and maintained on appropriate dosing schedule IV during the postoperative hospital course. This is followed on discharge by 1 week of oral antibiotic (e.g., cephradine 500 mg, clindamycin 300 mg) at the appropriate dosage.
(c) Anti-inflammatory steroid therapy to minimize edema may be started pre-incision (8–10 mg IV dexamethasone) and continued postoperatively as with other reconstruction or orthognathic surgery.
(d) Anesthesia—the naso-endotracheal tube can be sutured to the nasal septum (2–0 silk) and the anesthesia tubing and equipment are brought toward the patient's feet. This allows for the draping that follows to decrease the potential for contamination as well as permitting easier head movement in bilateral cases. (Fig. 5.15)
(e) After the patient is anesthetized and the airway secured, the eyes should be lubricated and protected to prevent corneal abrasion, etc. (Fig. 5.16).
(f) Any hair that could become involved in the surgical field should be carefully arranged and/or parted to facilitate the skin incision. If the hair is to be sheared, care should be taken to avoid cutting or nicking of the skin in the area of the surgical incision.
(g) After shearing the hair above the ear, pull the remaining hair away from the preauricular and surrounding areas and up toward the crown of the head.
(h) Using foam tape, wrap the head circumferentially (forehead—above the ear—occiput) so that the hair is under the tape and off the skin over the preauricular incision site(s) (Fig. 5.17).
(i) The auditory canal(s) and tympanic membrane(s) should be inspected with an otoscope to ensure there is no preoperative infection and to document any pre-surgical pathology.
(j) Occlude the external auditory canal on the surgical side. A cotton pledget moistened with sterile mineral oil is one option that can be utilized.
(k) Intermaxillary fixation appliances (arch bars, Ivy loops, MMF screws etc.) should be applied prior to skin preparation and draping.
(l) Retain all non-sterile fixation appliance application instruments on a separate Mayo stand to use later in the procedure when the patient is placed in the final

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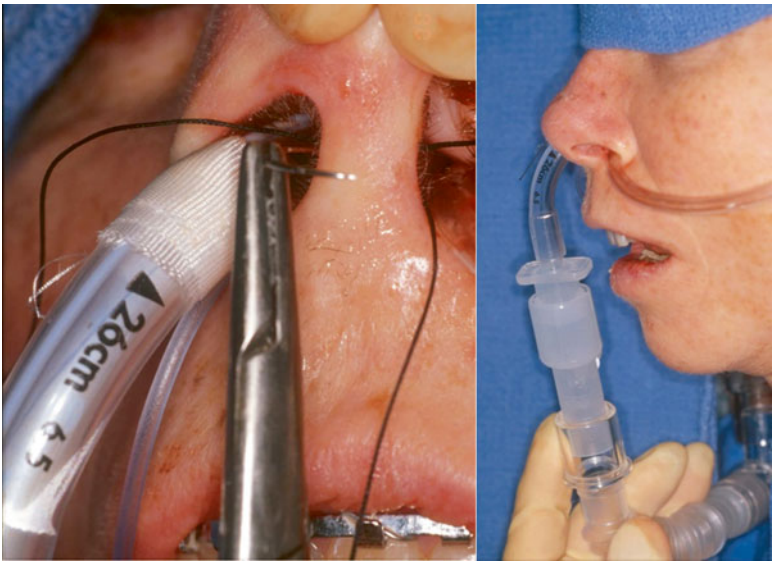


**Fig. 5.15** Naso-endotracheal tube secured to nasal septum with 2–0 silk suture and brought inferiorly away from the surgical sites

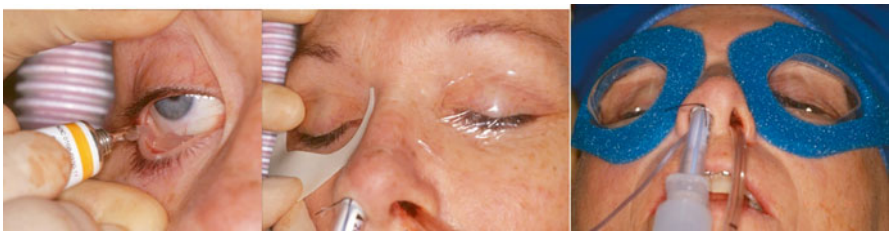

**Fig. 5.16** Lubrication of the eye. Taping the eyes shut. Application of plastic goggles to protect the eyes during surgery

occlusion for implantation of the device components. It is essential that there never be cross contamination between the mouth and the surgical wounds throughout the procedure.

(m) After appropriate skin preparation in unilateral cases, a plastic adhesive isolation drape (e.g., 1010 Steri-drape [3-M, St. Paul, MN]) is placed from the contralateral submental area to the ipsilateral temporal area to isolate the mouth from the sterile surgical field. This type of draping allows for access to the oral cavity while maintaining sterility of the implantation sites during application of intermaxillary fixation later in the procedure.

(n) In bilateral cases, first seal the mouth with a plastic adhesive occlusive dressing (Tegaderm, 3-M [St. Paul, MN] or Opsite [Smith and Nephew, London, UK]) (Fig. 5.18).

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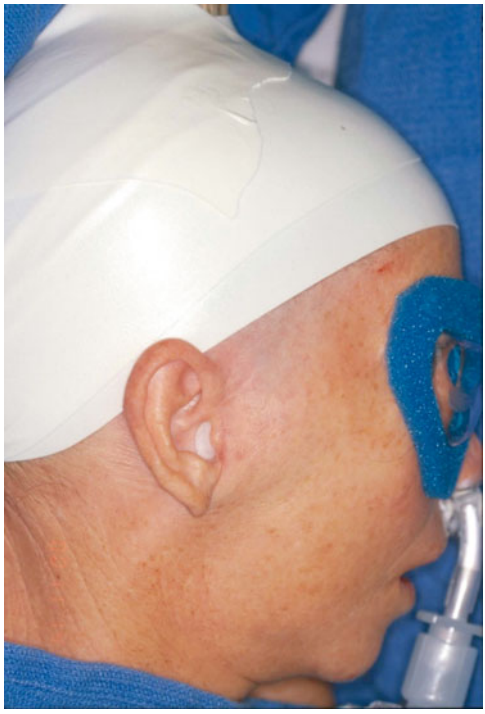

**Fig. 5.17** Using foam tape, wrap the head circumferentially (forehead—above the ear—occiput) so that the hair is under the tape and off the skin over the preauricular incision site. Note the sterile mineral oil-cotton occlusive dressing in the external auditory canal

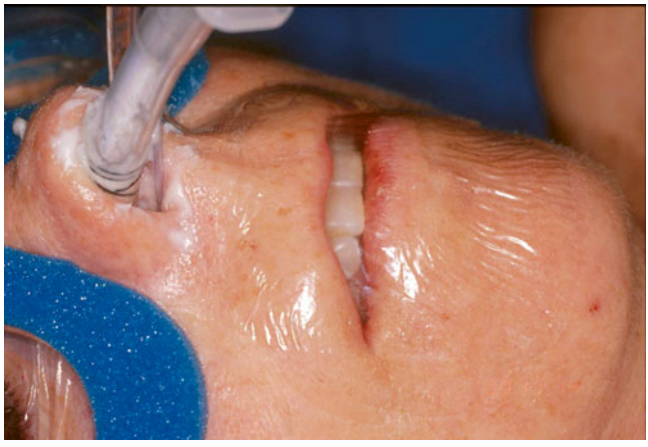

**Fig. 5.18** The mouth isolated with a plastic adhesive occlusive dressing (Tegaderm, 3-M [St. Paul, MN] or Opsite [Smith and Nephew, London, UK])

(o) The nasotracheal tube and the nose can be further isolated using bilateral 1010 Steri-drapes as described above, then folding the loose ends together over the nasotracheal tube and nose in a sterile fashion and finally sealing them together with Steri-strips (3-M, St. Paul, MN).

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### 5.7.2 *Incisions*

Standard preauricular and retromandibular incisions needed to access the TMJ area and the mandibular ramus respectively.

#### 5.7.2.1 Preauricular (Modified Al-Kyatt [74]) Incision for Exposure of the TMJ Fossa

(a) Find the crease between the helix and the preauricular skin and mark a line from the top of the helix to the lobe. In previously operated patients, use the scar to make this incision. In patients with multiple scars, excise the scarred tissue with the initial incision and revise the scar at closure. The superior aspect of the incision should be extended anteriorly and superiorly 4 cm at a 45° angle to the zygomatic process of the temporal bone.
(b) Inject a vasoconstrictor (e.g., 1:200,000 epinephrine solution) along the line to be incised to decrease bleeding. Wait for its effect (3 min).
(c) Apply traction to each end of the incision line with single-ended skin hooks.
(d) With a #15 blade, incise the skin and subcutaneous tissue along the incision line.
(e) At the superior aspect of the incision, spread the tissue with a curved mosquito hemostat to find the superficial layer of the temporalis fascia. This is the very obvious tough, shiny, white, and sinewy appearing dense tissue (Fig. 5.19).

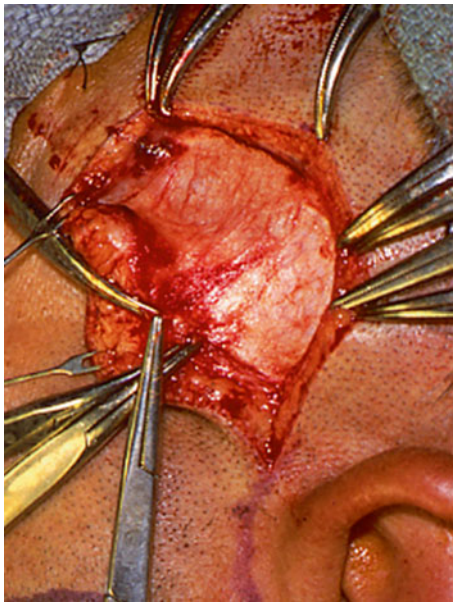

**Fig. 5.19** Exposure of the superficial layer of the temporalis fascia

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(f) Once this layer has been found, slide the hemostat inferiorly along the top of this fascia to the area of the zygomatic arch.
(g) Deepen the remainder of the incision to this plane using dissecting scissors remembering to stay close to the auricular cartilage posteriorly in the avascular plane. In the multiply operated patient, this is more difficult due to the scar tissue. Care must be taken to avoid cutting or nicking the auricular cartilage to avoid a postoperative chondritis.
(h) Using blunt retractors, retract the skin flaps. Care must be taken to avoid penetration of the parotid capsule at the inferior aspect of the incision as this may lead to persistent bleeding.
(i) At the tragus, in previously unoperated patients, just above the parotideomasseteric fascia, is the tragal ligament beneath which are found the auriculotemporal nerve and the transverse facial artery, both of which can be sacrificed.
(j) Once the parotideomasseteric and superficial temporal fascias have been exposed, make an incision approximately 2 cm long at a 45° angle through the superficial layer of the temporalis fascia. The deep temporal vein crosses the zygomatic process of the temporal bone and can be cauterized at this point to avoid persistent bleeding. Extend this fascial incision across the posterior aspect of the temporal bone inferiorly along the posterior aspect of the condyloid process (Fig. 5.20).
(k) Reflect this fascial flap anteriorly along the zygomatic process of the temporal bone exposing the lateral aspect of the fossa and the articular tubercle (Fig. 5.21). Care must be taken not to tear this tissue as branches of the facial nerve course through it in this area. Electrocautery and retraction should also

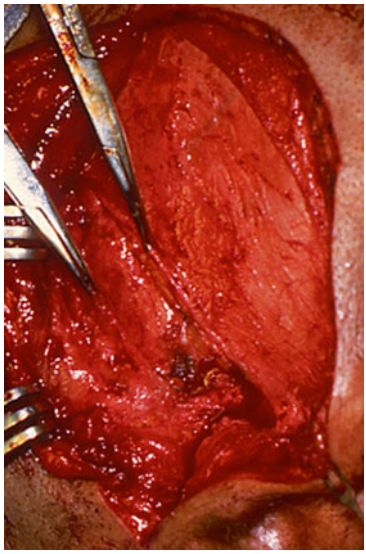

**Fig. 5.20** 45° angle incision through the superficial layer of the temporalis fascia

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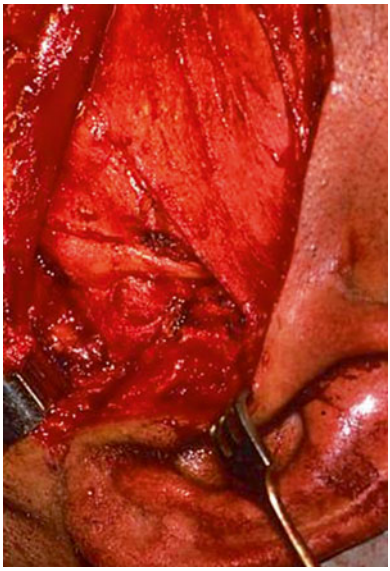

**Fig. 5.21** Exposure of the zygomatic arch and the lateral ligament of the TMJ

be done in a judicious manner to avoid injury to these nerves as well. In the multiply operated patient, this step is made more difficult due to scar tissue. This flap may have to be elevated with the assistance of dissecting scissors cutting the scar tissue away from the temporalis muscle above the zygomatic process of the temporal bone as the flap is elevated. To assist in determining the anterior extent of dissection, refer to the anatomical bone model that should be available in the operating room. Sterilizing the anatomical bone model and handling during surgery in the sterile field are specifically not recommended.

(l) The fossa can be entered through the superior aspect of the capsule if present. If there is an articular disc, it can be seen as the fossa is entered.

(m) With a Freer periosteal elevator, separate the capsular tissue from the lateral aspect of the condyle and make a vertical incision through that tissue directly over the instrument, opening this tissue to expose the lateral aspect of the condyle and condyloid process (Fig. 5.22). This step is also made more difficult in the multiply operated patient due to scar tissue.

(n) The condylar resection can be performed at this point if desired. If the remnant of the condyle or condyloid process is too small to be seen, felt, or reached from the preauricular incision, proceed to the submandibular incision and dissect up to the fossa area from below along the posterior mandibular ramus to find the bone for resection.

(o) Control all bleeding, irrigate, and pack the area with moist gauze, and direct attention to the submandibular incision.

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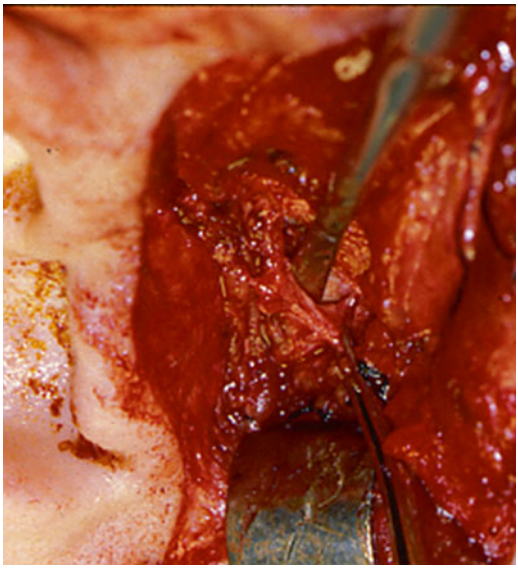

**Fig. 5.22** Freer elevator in the lateral aspect of the TMJ capsule

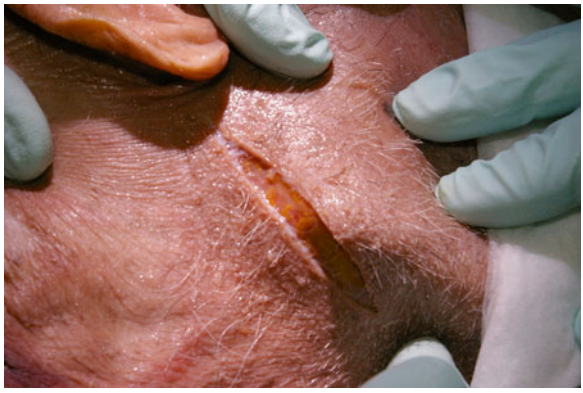

**Fig. 5.23** Retromandibular incision

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#### 5.7.2.2 Retromandibular (Modified Risdon [75]) Incision for Exposure of the Mandibular Ramus

(a) Mark a 5 cm line along one of the skin creases, one finger-breath below the earlobe and 2 cm posterior to the most inferior aspect of the mandibular angle.
(b) Inject a vasoconstrictor (e.g., 1:200,000 epinephrine solution) along the line to be incised to decrease bleeding. Wait for its effect (3 min).
(c) Apply traction to each end of the incision line with single-ended skin hooks.
(d) With a #15 blade, incise the skin and subcutaneous tissue along the incision line down to the platysma (Fig. 5.23).

(e) Incise through this muscle, carefully testing for the marginal mandibular branch of the facial nerve with a nerve stimulator.
(f) The next layer encountered in the previously unoperated patient will be the superficial layer of the deep cervical fascia. Palpate the cleft between the parotid gland and the masseter muscle.
(g) Using a mosquito clamp, open this fascial layer vertically along the cleft in front of the parotid gland. Using either a retractor (e.g., Army-Navy) or finger, gently retract the parotid posteriorly exposing the masseter and the pterygomasseteric sling at the angle and inferior border of the mandible. The structures to be avoided are the retromandibular vein posteriorly and branches of the facial nerve. The facial vein and artery rarely are encountered anteriorly with this incision. The marginal mandibular and buccal branches of the facial nerve lie in the cleft fascia. After it is opened vertically and retracted posteriorly with the parotid gland and held inferiorly with a ribbon retractor and superiorly with a retractor, these nerves are protected. However, retesting for both with a nerve stimulator is recommended before proceeding to the next step (Fig. 5.24).
(h) Identify and incise the pterygomasseteric sling and the periosteum at the angle and inferior border of the mandible along the length of the incision. Then using a periosteal elevator expose the whole lateral aspect of the ramus of the mandible, the coronoid process, and the sigmoid notch. Placing a "toe-out" retractor in the sigmoid notch after it is exposed provides for excellent exposure of the lateral ramus of the mandible (Fig. 5.25).

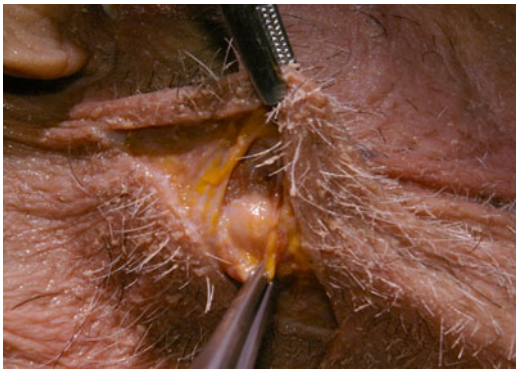

**Fig. 5.24** Cleft between the parotid gland and the masseter muscle

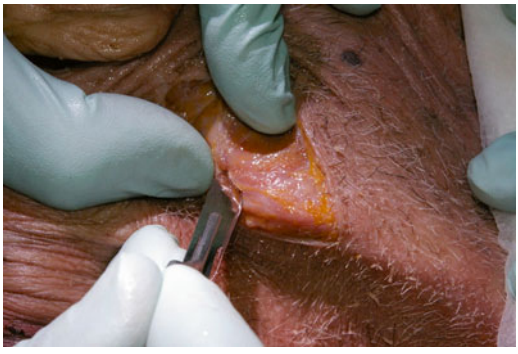

**Fig. 5.25** Incision through the pterygomasseteric sling

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(i) Connect the preauricular dissection with this one by following the posterior border of the mandible up to the condyloid process resection. Passing the blunt end of a periosteal elevator from below up into the area of the resection will allow it to be seen in the fossa through the preauricular incision (Fig. 5.26).

### *5.7.3 Condylar Resection*

(a) There must be a minimum of 15 mm between the mandibular condylar resection and the height of the articular eminence area to accommodate the anterior flange of the fossa component of the TMJ Concepts (Ventura, CA) device (Fig. 5.27).
(b) Measurement from a known point at the inferior border of the mandible (e.g., antegonial notch) to the resection line can be made on the SL model and transferred to the patient. It is important that this measurement and cut are made accurately so as not to remove more mandibular bone than necessary or involve the inferior alveolar neurovascular bundle.
(c) The superior level on the ramus for resection of the condyle is determined preoperatively on the anatomical bone model during the work-up. A template can be fashioned prior to surgery (e.g., suture pack foil, tongue blade, ruler). This can be useful to assist at surgery to assure proper the location of this cut.
(d) The model will also assist the surgeon in the determination as to whether the coronoid process is elongated and therefore would interfere with postimplantation mandibular function. If this is the case, the elongated coronoid can be removed as well at this stage of the procedure.
(e) Mark the position of this ramus cut using a marking pen and using a short-blade oscillating saw with copious irrigation separate the proximal segment containing the condyloid processes (and hyperplastic coronoid, if necessary) from the ramus.
(f) Once the proximal condyloid process segment (and coronoid) is/are separated, bring the proximal segment lateral to the ramus with a Seldin elevator. Carefully

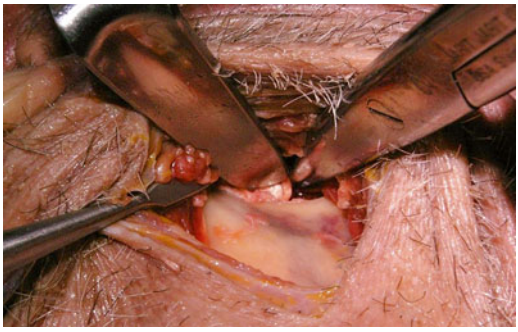

**Fig. 5.26** Retractor in the sigmoid notch through the retromandibular incision allowing access to the ramus of the mandible

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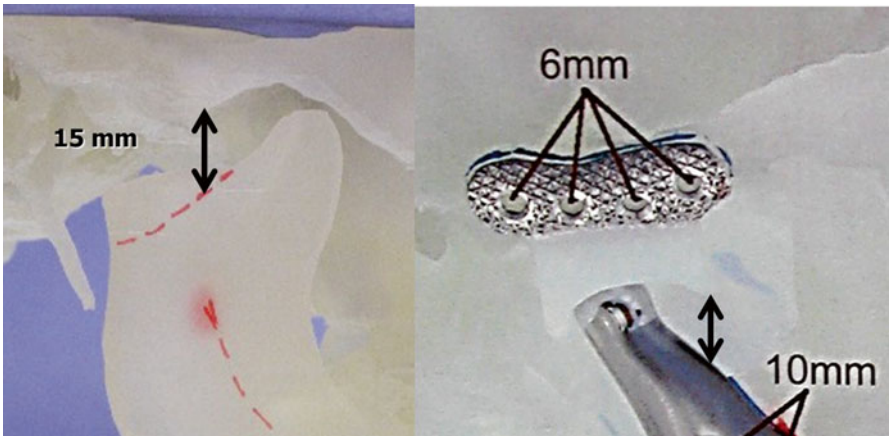


**Fig. 5.27** There must be a minimum of 15 mm clearance between the mandibular resection and the height of the eminence to accommodate the anterior flange of the fossa with the TMJ Concepts (Ventura, CA) custom device

remove any remaining lateral pterygoid muscular attachment from the condyle (and temporalis muscle from the coronoid) before attempting to deliver from the wound. To avoid excessive muscle oozing, use of an electrocautery needle tip against the pterygoid fovea bone of the condyle (and the coronoid process) will strip the muscle attachments easily.

### *5.7.4 Fossa Preparation*

Thoroughly debride the residual fossa of all soft tissue posteriorly to the tympanic plate, anteriorly to the remnant of the articular eminence of the temporal fossa, and medially to the medial ridge of the fossa where the medial capsule attaches superiorly to the temporal bone. This is extremely important in order to assure that the fossa component lies in direct contact with the remnant fossa bone, especially medially, to assure proper device condylar-fossa relationship on implantation.

### *5.7.5 Setting the Occlusion*

(a) Care must be taken not to contaminate the surgical sites during this procedure. It is recommended that the individuals applying the MMF change their gown and gloves before returning to the sterile field.
(b) Care must also be taken that none of the instruments used intraorally find their way back to the sterile field. Having a separate Mayo stand with dedicated

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MMF instrumentation and suction, as mentioned above, precludes such problems.

(c) Place the patient in tight MMF at the desired occlusion using 25 gauge box wires bilaterally posteriorly and anteriorly.

### 5.7.6 *Component Fixation*

(a) Use the fossa seating tool (TMJ Concepts, Ventura, CA) to seat and confirm the passive positioning of this component without any movement, and use this tool to stabilize the implant during fixation (Fig. 5.28). Use the ramus component clamp (TMJ Concepts, Ventura, CA) to assist in orientation and stabilization of that component on the ramus (Fig. 5.29).

(b) Once the fit of both components and their articulating relationship have been confirmed as correct, fixate the fossa and ramus components using the predetermined size and length screws.

(c) The drill guide must be used when placing each screw hole in the host bone of the temporal and mandibular bones. Use slow speed and copious irrigation so as not to overheat and potentially devitalize the bone which can lead to screw loosening. The recommended length is 2 mm diameter, self-tapping, bicortical screws should be placed after each hole is drilled with copious irrigation (Fig. 5.30).

(d) A percutaneous technique may be required for the most superior screw(s) in the ramus component.

(e) All of the screws should be placed unless the quality of the host bone prohibits and/or the 2.3 mm diameter rescue screw does not securely go to place tightly. Loose screws should not be left in place.

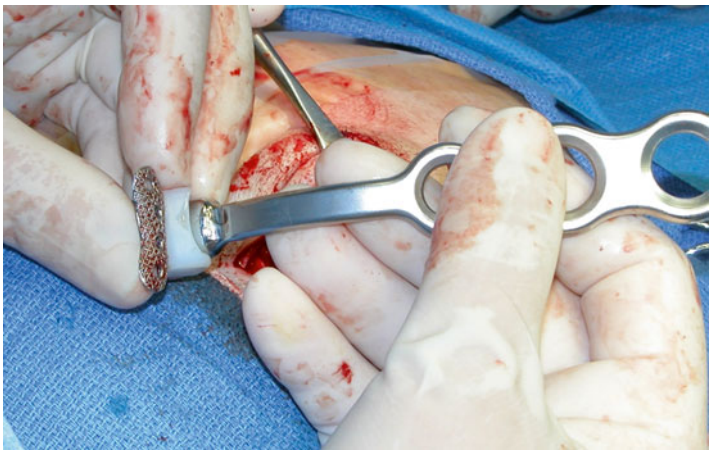

**Fig. 5.28** TMJ Concepts fossa seating tool used to assure stability of the fossa component

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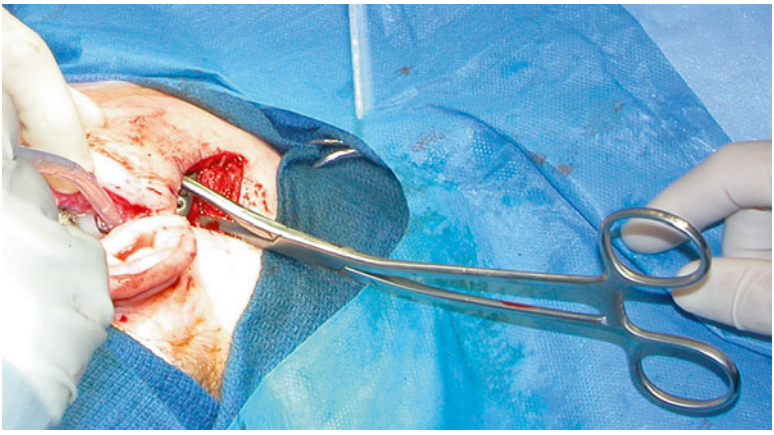

**Fig. 5.29** TMJ Concepts ramus component stabilizing clamp

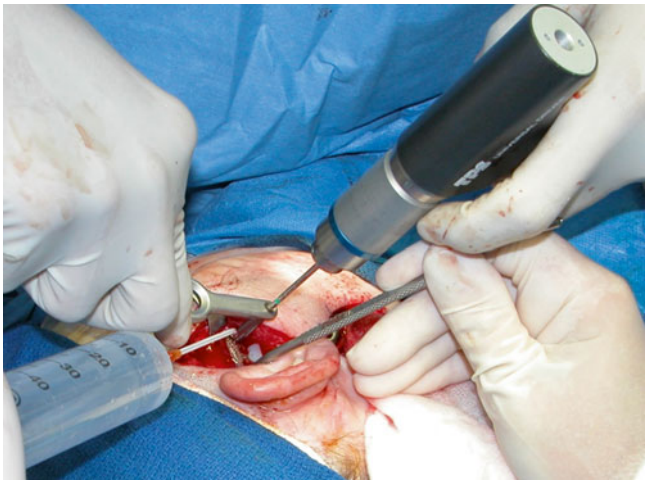

**Fig. 5.30** Drill guide and copious irrigation essential for proper screw pilot hole placement and to assure bone viability

(f) Once all the screws are in place, return to each screw and assure that it is tight.
(g) In bilateral cases, repeat the fixation protocol on the other side before closure.

### *5.7.7 Confirmation of Occlusion, Function, and Position*

(a) MMF is released and the mandible functioned, maintaining sterility of the operative field. The joint articulation is directly observed to ensure proper movement with function. While the patient is in occlusion, the condylar head of the

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ramus component should be centered on the fossa bearing in the M/L direction and seated against the fossa's bearing surface's posterior lip (TMJ Concepts, Ventura, CA) (Fig. 5.31).

(b) Training elastics are placed for immediate postoperative comfort. Once again, care must be exercised so as not to cross and contaminate the surgical sites from the oral cavity.

(c) Imaging confirmation of component alignment, position, and fixation can be confirmed by obtaining an intraoperative anterior–posterior skull x-ray (Fig. 5.32).

(d) Close the wounds after careful and copious irrigation. Irrigation containing an antibiotic is recommended.

### *5.7.8 Postoperative Auditory Canal Examination and Pressure Dressing*

(a) The auditory canal(s) and tympanic membrane(s) should be re-inspected with a speculum to ensure there was no intraoperative tear, and this inspection should be documented. Carefully remove any clots with gentle, warm irrigation and suction.

(b) Instill ofloxacin otic drops and occlude the external auditory canal(s) with cotton.

(c) Apply a Barton-type pressure dressing for a minimum of 8–12 h.

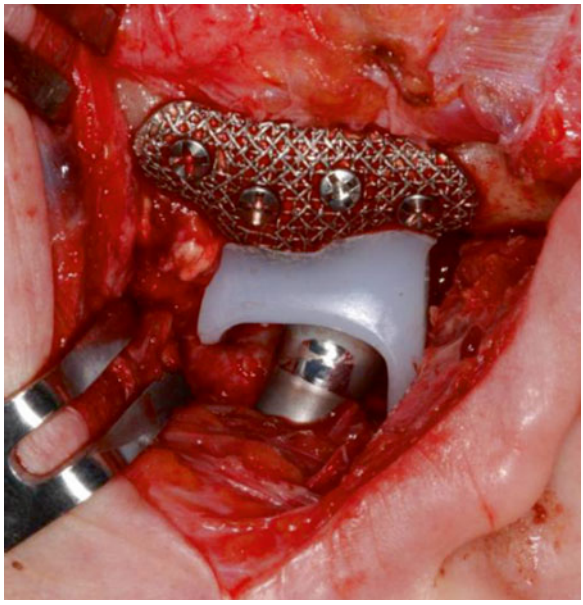

**Fig. 5.31** Proper position of the TMJ Concepts condylar head at the posterior aspect of the bearing surface of the fossa component

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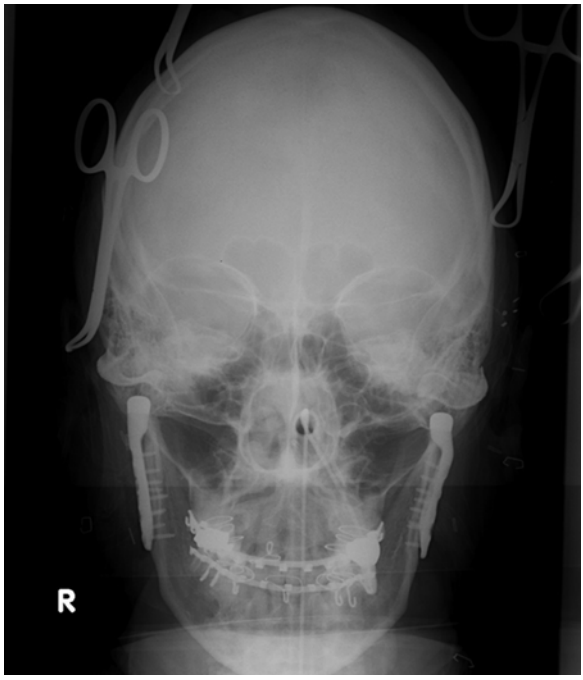


**Fig. 5.32** Intraoperative imaging to assure proper alignment and fixation of bilateral TMJ Concepts custom TMJ TJR

### *5.7.9 Postoperative Management*

(a) Limit early postoperative opening to avoid dislocation particularly in patients who have significant soft tissue laxity due to coronoidectomies and/or extensive dissection performed to regain opening or reposition mandible. The use of training elastics in the immediate postoperative period can reduce the potential for dislocation. Dislocation is typically only of concern for the first week post-op.
(b) When it is considered that the potential for dislocation is low, the training elastics can be released when the pressure dressing is removed after 8–12 h, and the patient can begin using a jaw-exercising device (e.g., Therabite—Atos Medical, Milwaukee, WI).
(c) Should the patient require the assistance of a physical therapist to increase and maintain mandibular range of motion postoperatively, two to three visits per week for a minimum of 3 months is appropriate.
(d) One week of antibiotic therapy should follow as described above.
(e) The patients should be encouraged to chew a soft diet and advance their diet as tolerated.
(f) Long-term follow-up.

Complications, their avoidance and management are discussed in detail in Chap. 8

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# Part III
# Advanced TMJ TJR Techniques

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# Chapter 6
# Concomitant TMJ Total Joint Replacement and Orthognathic Surgery

**Larry M. Wolford**

## 6.1 Introduction

Temporomandibular joint (TMJ) disorders/pathology and dentofacial deformities commonly coexist. The TMJ pathology may be the causative factor of the jaw deformity or develop as a result of the jaw deformity, or the two entities develop independent of each other. This chapter will focus on the most common TMJ pathologies that are indicated for total TMJ replacement (TMJ TJR) as well as orthognathic surgery. The health and stability of the TMJ are dependent on the structural integrity, position, and presence or absence of disease or injury affecting the articular disk, condyle, fossa, and associated soft tissues. The TMJ hard and soft tissue components may become degenerated, arthritic, and non-salvageable with any of these following TMJ pathologic conditions: (1) long-standing articular disk dislocation, (2) adolescent internal condylar resorption (AICR), (3) reactive arthritis, (4) ankylosis, (5) congenital deformation or absence of the TMJ, (6) trauma, (7) connective tissue and autoimmune diseases, (8) previously failed TMJ surgery, and (9) other end-stage TMJ disorders [1–4]. All are often associated with dentofacial deformities, malocclusion, TMJ pain, headaches, myofascial pain, TMJ and jaw functional impairment, ear symptoms, sleep apnea, etc. Patients with these conditions may benefit from corrective surgical intervention including TMJ reconstruction with TMJ TJR devices, orthognathic surgery, and other adjunctive procedures. Many clinicians may have difficulty identifying the presence of a TMJ condition, diagnosing the specific pathology, and selecting the proper management for the

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condition. This chapter should improve the clinician's diagnostic and management planning skills particularly in the end- stage TMJ conditions requiring TMJ TJR.

Although most TMJ patients have associated symptoms, approximately 25 % of patients with significant TMJ pathology/disorders may be asymptomatic. These patients pose a diagnostic challenge when undergoing orthognathic surgery because the TMJ pathology may not be recognized or managed appropriately, resulting in a poor outcome with potential redevelopment of the skeletal and occlusal deformity resulting from condylar resorption or overdevelopment. Further, there can be worsening pain, headaches, TMJ and mandibular dysfunction, as well as other TMJ symptoms [5]. However, there are clinical and imaging factors that can indicate the presence of TMJ pathology in the asymptomatic patient.

Many clinicians choose to ignore the TMJ pathology and perform only orthognathic surgery in these types of cases. But this management philosophy can result in continuation or exacerbation of the presurgery TMJ pathology and reproduce the original deformity with worsening occlusion, jaw dysfunction, facial imbalance, and pain. Clinicians who address the dentofacial deformities and TMJ pathologies that require TMJ TJR can perform the surgery in one stage or two separate stages. The two-stage approach requires the patient to undergo two separate operations and anesthesia, significantly prolonging the overall treatment. However, performing concomitant TMJ and orthognathic surgery in these cases significantly decreases treatment time, provides better outcomes, but requires careful treatment planning and surgical proficiency in both surgical techniques.

In the author's 25-year experience of using patient-fitted TMJ TJR devices, approximately two-thirds of patients requiring TMJ TJR can benefit from concomitant orthognathic surgery for improvement in function, airway and breathing capabilities, better aesthetic outcomes, and decreased or elimination of pain.

## 6.2 Patient Evaluation

It is important to know the patient's complaints, concerns, history, symptoms, and treatment expectations. Detailed information on patient evaluation for orthognathic, TMJ, and sleep apnea surgery including clinical, radiographic, MRI, and dental model analyses have been previously published [1–4, 6], so this information will not be reproduced here.

However, it is important to realize that these patients are sometimes misleading in their clinical presentation because their "natural head position" may posture their head hyperextended and lower jaw and chin tipped upward and forward to make the chin appear more prominent. But more specifically, this head position helps to open their oropharyngeal airway and thereby improve their ability to breathe. If the patients are not evaluated in a proper corrected head position, the amount and degree of maxillary and mandibular retrusion and asymmetry may be missed, thus the

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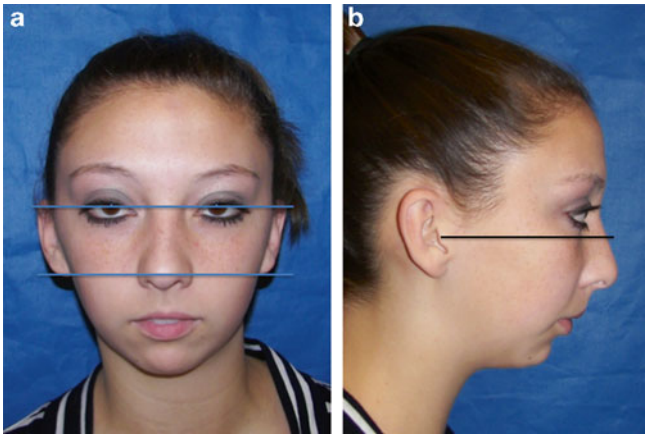


**Fig. 6.1** Patients should be evaluated in the frontal view (**a**) with the pupillary plane and the ear plane parallel to the floor, and in profile (**b**) evaluated with clinical Frankfort horizontal plane (a line from the tragus of the ear through the bony inferior orbital rim) parallel to the floor

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importance of evaluating the patient with the pupillary plane and ear plane parallel to the floor in the frontal view (Fig. 6.1a) and clinical Frankfort horizontal plane (a line drawn from the tragus of the ear through the bony infraorbital rim) parallel to the floor in the profile view (Fig. 6.1b). Obviously, there will be some variance in some individuals, but this is a basic guide.

Common factors frequently overlooked by clinicians in patients requiring TMJ TJR are AP deficient maxilla and mandible, decreased oropharyngeal airway, nasal airway obstruction, and sleep apnea issues. Patients with TMJ issues, particularly those with condylar resorption or degeneration, may experience progressively worsening breathing and sleep apnea issues. Patients with sleep apnea symptoms may be indicated for a sleep workup including polysomnography.

Many sleep apnea patients also have TMJ issues that should be addressed at the same time or before the orthognathic surgery is performed to provide a stable, predictable outcome and decrease preexisting pain. Advancing the maxillary and mandibular complex in a counterclockwise direction improves facial balance, and the oropharyngeal airway opens significantly, to improve the airway. Studies have shown that with double jaw surgery with counterclockwise rotation of the maxillomandibular complex, with the first 10 mm of advancement, the oropharyngeal airway opens up 65–70 % of the amount of mandibular advancement [7–11]. With 10–15 mm of advancement, the oropharyngeal airway continues to open, but at a lesser degree—55–60 % of the mandibular advancement. When the mandible is advanced 15–20 mm, the oropharyngeal airway continues to open, but to only 40–45 % of the amount of mandibular advancement [1].

## 6.3 Imaging

Radiographic evaluation is helpful to the diagnostic process. Cone beam computed tomography (CBCT) technology makes low-cost, low-radiation scans accessible. With CBCT imaging, the oropharyngeal and nasal airways can also be evaluated along with the lateral and anteroposterior cephalometric images, TMJ tomograms, and panoramic images. The lateral cephalometric analysis can determine the severity of the jaw deformity, dental alignment, airway dimensions, etc.

One of the best diagnostic tools for TMJ disorders is magnetic resonance imaging (MRI) because it allows evaluation of TMJ disk position, morphology, mobility, extent of joint degenerative changes, and the presence of inflammation. It can aid in the diagnosis of intra-articular TMJ disorders in the "silent joint" in which disk displacement and degenerative changes can be present, may not make noise or be uncomfortable or painful, but may contribute to poor outcomes if only orthognathic surgery is performed. CT scans, bone scans, and three-dimensional (3D) imaging may be helpful in diagnosis and management planning.

### *6.3.1 MRI Evaluation*

Magnetic resonance imaging (MRI) is one of the most important diagnostic tools that we have in evaluation, diagnoses, and management planning for TMJ pathology. In general, T-1 MRIs are helpful in identifying disk position, the presence of alteration in bone and soft tissue structures, and interrelationships of the bony and soft tissue anatomy. T-2 MRIs are more helpful in identifying inflammatory responses in the TMJ. The importance of disk position cannot be overemphasized, in this author's opinion. For MRI evaluation of the TMJs, a 1.5 T or more powerful machine is recommended. "TMJ coils" are necessary to achieve diagnostic quality images of the TMJs. The basic views that are most helpful in diagnoses include (1) sagittal views in centric relation as well as in maximum opening, (2) coronal views in centric relation, and (3) dynamic views, if available. The MRI can be correlated to cone beam imaging of the TMJs for joint space and greater interpretation of bony pathology. Figure 6.2 shows a normal TMJ MRI with healthy structures and the disk in position.

### *6.3.2 Disk Displacement*

When disks are anteriorly displaced for extended time periods, they may become nonreducing and deformed with loss of the intermediate zone and thickening of the posterior and anterior bands (Fig. 6.3). Also, there may be a degenerative process developing in the disks where there is a breakdown of the cartilaginous substance

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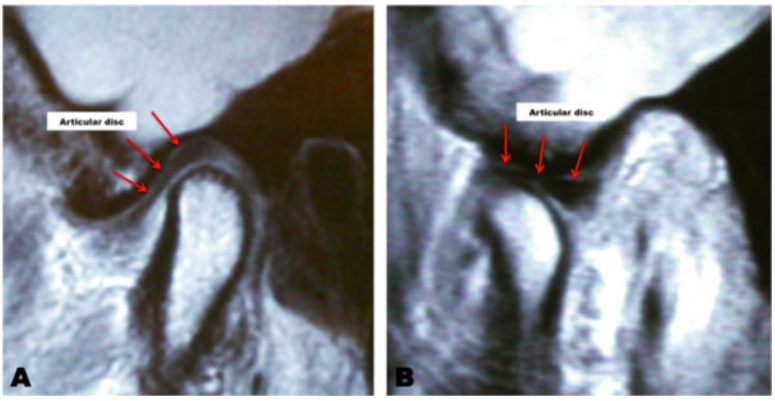


**Fig. 6.2** (**a**) MRI of a normal TMJ in *closed position* with disk in position. (**b**) *Open view* showing good translation forward of condyle and disk

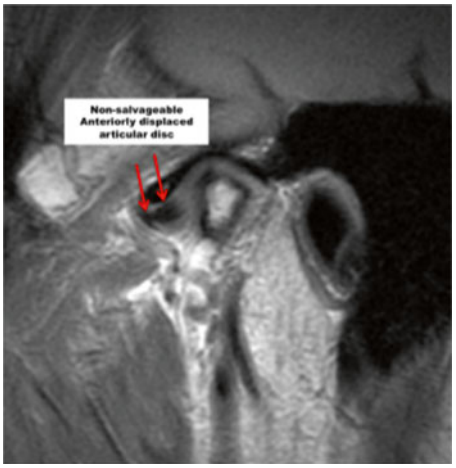


**Fig. 6.3** The articular disk is anteriorly displaced and significantly deformed, degenerated, and nonreducing rendering it non-salvageable. The condyle is arthritic

with vascular invasion and degeneration. When disks are displaced and become nonreducing, the degenerative process progresses more rapidly compared to displaced disks with reduction. When disks advance to a certain level of deformation and degeneration, they become non-salvageable. When concomitant TMJ and orthognathic surgery is indicated, in this situation, TMJ TJR devices are indicated to produce the most predictable and high-quality outcome.

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### 6.3.3 Adolescent Internal Condylar Resorption

Adolescent internal condylar resorption (AICR) is a condition that develops usually during pubertal growth between the ages of 11 and 15 years, predominantly in females (ratio 8:1 females to males) [1–4, 12, 13]. Clinically, the mandible slowly retrudes into a Class II occlusal and skeletal relationship with a tendency to an anterior open bite. These patients all have high occlusal plane angle facial morphological profiles. On the MRI, these cases present with a condyle that is slowly becoming smaller in size in all three planes of space, and the disk is anteriorly displaced similar to Fig. 6.3. In some cases, there is significant thinning of the condylar cortical bone contributing to the inward collapse of the condylar head. The articular disks are anteriorly displaced and may or may not reduce on opening. Nonreducing disks will degenerate and deform at a more rapid rate as compared to disks that reduce. Studies demonstrate that AICR is arrested if the articular disks are put back into position on top of the condyle and stabilized with the Mitek anchor technique. Results are best for AICR if the disk repositioning surgery is performed within 4 years of the onset of the pathology. After 4 years, the disks may become nonsalvageable, and condyles significantly resorbed resulting in the need for TMJ TJR to repair the TMJ and advance the mandible [1–4, 12, 13].

### 6.3.4 Reactive Arthritis

Reactive arthritis is commonly caused by bacterial or viral entities [1–4, 14–19] and may on imaging demonstrate a localized area of inflammation with erosion of the condyle and/or fossa. It also can present as a more profuse inflammatory process through the bilaminar tissues, capsule, etc. (Fig. 6.4). Surgical indication may include removal of the nidus of inflammation along with repositioning of the articular disk if salvageable. With extensive destruction of the TMJ, TMJ TJR is indicated.

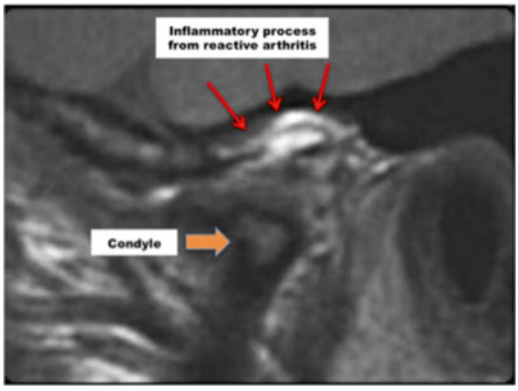


**Fig. 6.4** T-2 MRI of right TMJ with reactive arthritis and significant condylar resorption. The inflammatory process is noted to occupy a significant volume between the fossa and arthritic condyle

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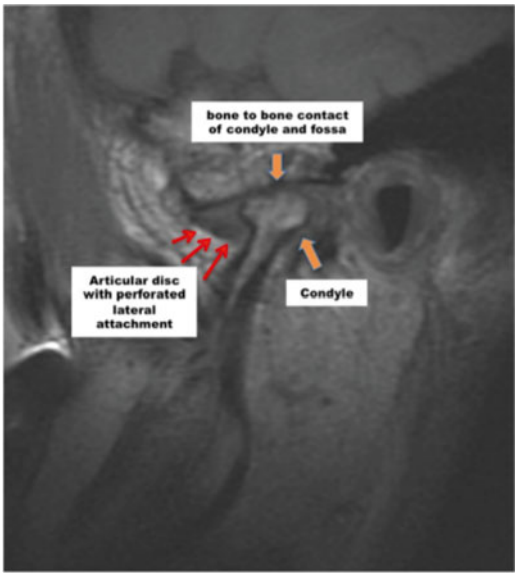


**Fig. 6.5** MRI of the left arthritic condyle with perforation of the bilaminar tissue posterior to the anteriorly displaced disk. Bone-on-bone contact of condyle and fossa is observed with crepitation on jaw function

### *6.3.5 Perforations*

Perforations can occur in the articular disk resulting in bone-on-bone contact. Perforated disks are usually anteriorly and/or medially displaced. Almost always these perforations are posterior to the posterior band of the articular disk or lateral to the disk; rarely do perforations occur through the disk itself (Fig. 6.5). Clinically, crepitation will usually be present, and the MRI will reveal evidence of bone-on-bone contact, arthritic changes in the condylar head and/or fossa, as well as an anteriorly displaced disk.

### *6.3.6 Connective Tissue/Autoimmune Diseases*

The MRI presentation of connective tissue/autoimmune disease is fairly pathognomonic. In these conditions, the articular disk often is in a relatively normal position, but there is progressive condylar resorption, "mushrooming" of the remaining condyle, and often resorption of the articular eminence, with slow but progressive destruction of the articular disk that is surrounded by a reactive pannus (Fig. 6.6) [1–4, 20–24]. This presentation almost always indicates need for TMJ TJR to manage the pathologic process in the joint. Use of autogenous tissues in this scenario likely could result in the disease process attacking autogenous tissues placed into the joint with subsequent failure.

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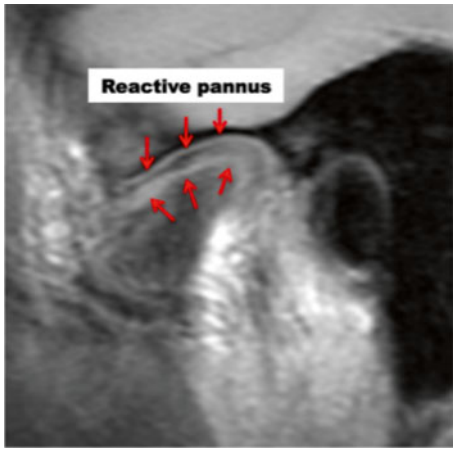
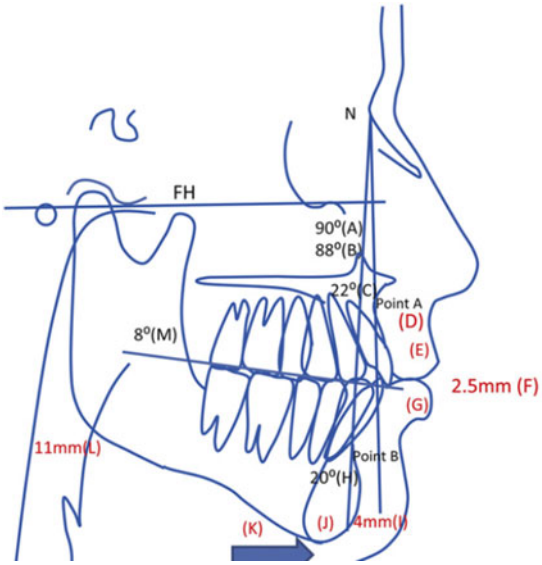



**Fig. 6.6** There are common TMJ changes in connective tissue/autoimmune disease. The disk may be in position but with a reactive pannus (*gray tissue*) surrounding the disk that destroys the disk, condyle, and articular eminence. The remaining condyle has a "mushroom" appearance




(A) Maxillary depth - Angular measuremer formed by FH and NA (normal 90 +/- 3 degrees).
(B) Mandibular depth - Angular measurem formed by FH and NB (normal 88 +/- 3 degrees).
(C) Upper incisor angulation to NA (norma +/- 2 degrees).
(D) Upper lip length (normal males 22 +/- 2 mm females 20 +/- 2 mm).
(E) NA to upper incisor tip in mm.
(F) Upper tooth to lip relationship (normal +/- 1.5 mm).
(G) NB to lower incisor tip in mm.
(H) Lower incisor angulation to NA (norma +/- 2 degrees).
(I) Pogonion projection - pogonion to NB li (normal 4 +/- 2 mm).
(J) Lower anterior dental height - perpendicular to FH, lower incisor tip to line tangent to menton (normal male 44 +/- 2 mm females 40 +/- 2 mm).
(K) Arrows indicate movements in mm.
(L) Airway (normal 11 mm).
(M) Occlusal plane angle to FH (normal 8 degrees).

**Fig. 6.7** Cephalometric tracing landmarks and measurements to aid in diagnosis and treatment planning

### *6.3.7 Cephalometric Analysis*

Cephalometric analysis is an important assessment tool for diagnosis and management planning for TMJ patients because the most dominant facial type that experiences TMJ pathology is the high occlusal plane angle facial morphology with a retruded maxilla and mandible. Normal cephalometric relationships used by the author are demonstrated in Fig. 6.7 and have been described in detail in previous publications [3, 6].

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### *6.3.8 Airway*

One of the primary factors contributing to sleep apnea is a decreased oropharyngeal airway and is commonly seen in TMJ patients, particularly those with a history of condylar resorption. The normal cephalometric AP dimension from the posterior pharyngeal wall to the soft palate and posterior pharyngeal wall to the base of the tongue should be 11 mm (±2 mm). In patients who have a retruded maxilla and mandible, their airway may be significantly decreased. Typically, accompanying the airway issue will be a high occlusal plane angle. The normal occlusal plane angle to the Frankfort horizontal plane is 8° (±4°). Commonly, with a retruded maxilla and mandible, particularly in condylar resorption, the occlusal plane is significantly increased and is a factor that must be addressed in case planning.

Three anatomical factors commonly come together in TMJ patients requiring surgical intervention. These include (1) a high occlusal plane angle facial morphology associated with retruded maxilla and mandible, (2) nasal airway obstruction related to hypertrophied turbinates and/or nasal septal deviation or spurring, and (3) TMJ pathology.

In a study of 1234 consecutive patients requiring at least maxillary osteotomies referred to the author for orthognathic surgery, there were 603 patients (49 %) with hypertrophied turbinates requiring partial turbinectomies and 278 patients (23 %) who required nasal septoplasty. For patients requiring partial turbinectomies ($n=603$), 84 % had maxillary hypoplasia, 72 % had mandibular hypoplasia, 69 % had a high occlusal plane angle, and 49 % of the patients required concomitant TMJ surgery. 67 % of the turbinectomy cases, and 73 % of concomitant turbinectomy and orthognathic and TMJ surgery cases, involved females. A strong correlation has been established between hypertrophied inferior turbinates, hypoplastic maxilla and mandible, and a steep occlusal plane [25].

These findings correlate with other studies evaluating the morphology of mouth breathing and nasally obstructed patients [26–29].Therefore, patients with the high occlusal plane angle facial morphology with a retruded maxilla and mandible should be assessed for nasal airway obstruction, decreased oropharyngeal airway, and sleep apnea, as well as TMJ pathology (even if asymptomatic).

Following completion of all of the appropriate historical, clinical, and imaging evaluations, a comprehensive diagnosis can be developed and a definitive management plan established to address the findings as well as other options that may be appropriate to the specific case. These can then be presented to the patient to allow them to make an informed decision as to how they wish to proceed.

## 6.4 Occlusal Plane Alteration

The correction of dentofacial deformities often requires surgery on both the maxilla and mandible to achieve a quality functional and aesthetic result and address airway issues. An often ignored but important cephalometric and clinical interrelationship

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in the diagnosis and treatment planning for the correction of dentofacial deformities is the occlusal plane angulation [6, 30–32]. The occlusal plane angle is formed by the Frankfort horizontal plane and a line tangent to the cusp tips of the lower premolars and the buccal groove of the second molar. The normal value for adults is 8 ± 4°. An increased (high) occlusal plane angle usually is reflected in an increased mandibular plane angle (dolichocephaly), and a decreased (low) occlusal plane angle usually correlates with a decreased mandibular plane angle (brachycephaly).

### *6.4.1 High Occlusal Plane Facial Type*

The common functional and aesthetic characteristics of the high occlusal plane facial morphology generally include the following:

- Increased occlusal plane angulation (>12°).
- Increased mandibular plane angulation.
- Anterior vertical maxillary hyperplasia and/or posterior vertical maxillary hypoplasia.
- Increased vertical height of the anterior mandible and/or decreased vertical height of the posterior mandible.
- Decreased projection of the chin (microgenia).
- Anteroposterior and vertical posterior mandibular and maxillary hypoplasia.
- Decreased angulation of maxillary incisors, although over-angulation can occur.
- Increased angulation of mandibular incisors.
- Class II malocclusion is common, although Class I and Class III malocclusions also can occur.
- An anterior open bite may be accompanied by an accentuated curve of Spee in the upper arch.
- In more pronounced cases in which the occlusal plane approaches the slope of the articular eminence, the following may occur: loss of incisal guidance, loss of canine rise occlusion, and the presence of working and nonworking dental interferences in the molar areas.
- The more severe cases may demonstrate moderate to severe sleep apnea symptoms as a result of the tongue base and soft palate displaced posteriorly and constricting the oropharyngeal airway (normal oropharyngeal airway space is 11 ± 2 mm).
- Nasal airway obstruction related to hypertrophied turbinates and/or septal deviation or spur.
- TMJ pathology.

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### 6.4.2 *Surgical Decrease of the Occlusal Plane*

In the high occlusal plane facial type, the indicated surgical correction may include a counterclockwise rotation of the maxillo-mandibular complex. In open bite cases, the maxillary occlusal plane and the mandibular occlusal plane may be different, so each should be evaluated independently. For illustrative purposes, a Class I case is used with the maxillary incisor edge as the center of rotation (Fig. 6.8). The anatomical changes that occur include the following:

- Occlusal plane angle decreases.
- Mandibular plane angle decreases.
- Maxillary incisor angulation increases (the same amount that the maxillary occlusal plane decreases).
- Mandibular incisor angulation decreases (the same amount that the mandibular occlusal plane decreases).
- Projection of the chin increases relative to the lower incisor edges.
- Posterior facial height may increase.
- Prominence of the mandibular angles may increase.
- Maxillary incisor edges move forward relative to the perinasal area.
- Incisal guidance and canine rise occlusion improves, and posterior working and nonworking interferences are eliminated.
- Oropharyngeal airway increases.

The center of rotation affects the aesthetic relationship of the jaws with the other facial structures. In Fig. 6.8, the center of rotation is at the maxillary incisor edge.

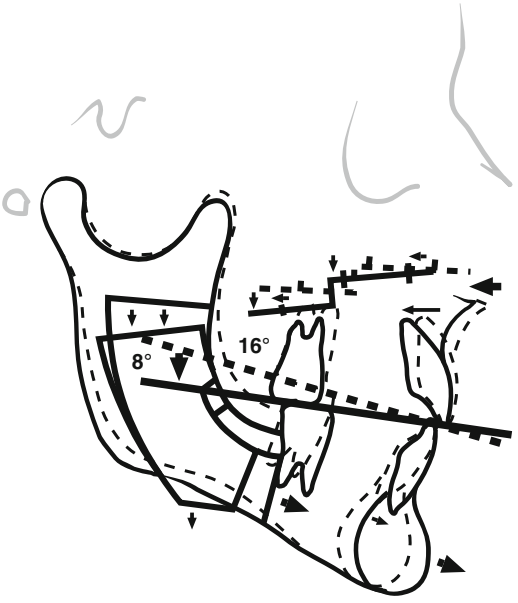


**Fig. 6.8** Surgical decrease of the occlusal plane from the dotted line to solid line (*counterclockwise rotation*) rotates the chin forward and decreased prominence of the perinasal areas, maxillary incisor angulation increases, mandibular incisor angulation decreases, and the oropharyngeal airway increases

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Counterclockwise rotation of the maxillo-mandibular complex results in the nasal tip moving posteriorly, but the mandible and chin come forward. If rotation is around point A or higher, then the perinasal area and the nose are less affected, but the maxillary incisor edges come forward, increasing the anteroposterior support to the upper lip. The mandible and chin come further forward demonstrating the significant aesthetic difference that the alteration of the occlusal plane can make [6, 30–32]. When decreasing the occlusal plane angle and advancing the mandible counterclockwise, the oropharyngeal airway increases approximately 50–70 % of the advancement measured at the genial tubercles [7–11].

## 6.5 Concomitant TMJ Total Joint Replacement and Orthognathic Surgery (C-TJR-OS)

Treatment planning for C-TJR-OS cases is based on cephalometric analysis, prediction tracing, clinical evaluation, and dental models, which provide the template for movements of the upper and lower jaws to establish optimal treatment outcome in relation to function, facial harmony, occlusion, and oropharyngeal airway dimensions. For patients who require TMJ TJR, a protocol CT scan of the maxillofacial region that includes the TMJs, maxilla, and mandible is recommended. The surgeon then has two options for model preparation to aid in the construction of a patient-fitted total joint prostheses using the TMJ Concepts System (Ventura, CA): the traditional protocol, using a stereolithic (SL) model or virtual surgical planning (VSP) [33, 34].

## 6.6 Protocol for Traditional C-TJR-OS

Using the protocol CT scan data, the SL model is fabricated with the mandible as a separate piece. Using the original cephalometric tracing and prediction tracing (Fig. 6.9a), the mandible on the SL model is placed into its predetermined position using planned measurements for correction of mandibular anteroposterior and vertical relationships, occlusal plane alteration, pitch, yaw, and roll (Fig. 6.9b). The mandible is secured to the maxilla on the SL model with quick-cure acrylic in the planned surgical position. Since many patients with concomitant TMJ pathology also require orthognathic surgery, they will benefit from counterclockwise rotation of the maxillo-mandibular complex. Repositioning the mandible into its final position requires the development of a posterior open bite on the SL model (Fig. 6.9b). Because the mandibular position on the SL model is established using hands-on measurements, the operator's manual dexterity and three-dimensional perspective play a critical role in setting the mandible in its proper and final position. This step can predispose the planning process to a certain margin of error.

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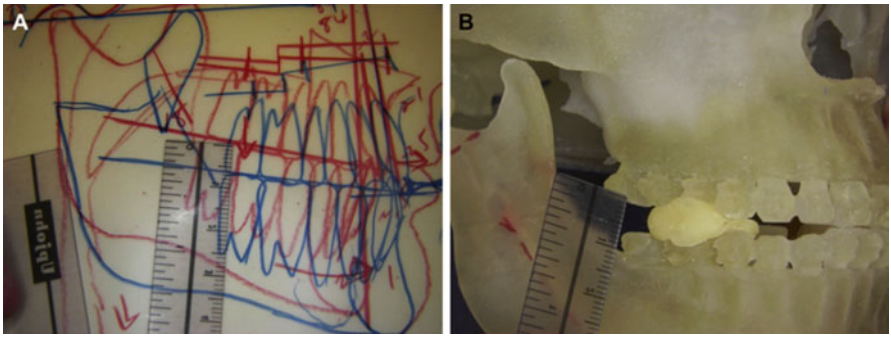


**Fig. 6.9** (**a**) Measurement of the cephalometric tracing and prediction tracing for the amount of open bite produced at the second molar after *counterclockwise rotation* of the mandible into its final position. (**b**) Duplication of the measurement obtained from the prediction tracing to the final mandibular position on the stereolithic model and fixating the mandible to the maxilla with methyl methacrylate

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As the next step on the SL model, the author recommends the required condylectomies as well as recontouring the lateral ramus to a relatively flat surface in the area where the mandibular component will be placed. The fossa requires recontouring only if heterotopic bone or unusual anatomy is present. The recontouring areas are marked in red for duplication of bone removal at surgery. Because most patients with TMJ problems requiring C-TJR-OS can benefit from counterclockwise rotation of the maxillo-mandibular complex, the SL model will likely be set with posterior open bites, because the maxilla is maintained in its original position. To accommodate the prosthesis, 20 mm of space is required between the fossa and ramus.

Once the stereolithic model is finalized, the model is sent to TMJ Concepts (Ventura, CA) for the design of the TMJ TJR device (Fig. 6.10c). The specifics of the design are sent to the surgeon for approval before manufacture of the components. The final prostheses (Fig. 6.10d) are forwarded directly to the surgeon's hospital for subsequent implantation with a schematic indicating the length of the fixation screws necessary for bicortical engagement.

Prior to surgery, the orthognathic surgical procedures are performed by the surgeon on articulator-mounted dental models. The mandible is repositioned on the articulator, duplicating the movements performed on the SL model, and the intermediate splint is constructed. The maxillary model is repositioned, segmented if indicated, and placed into the desired occlusion. A final surgical palatal or occlusal splint is constructed depending on the surgeon's preference.

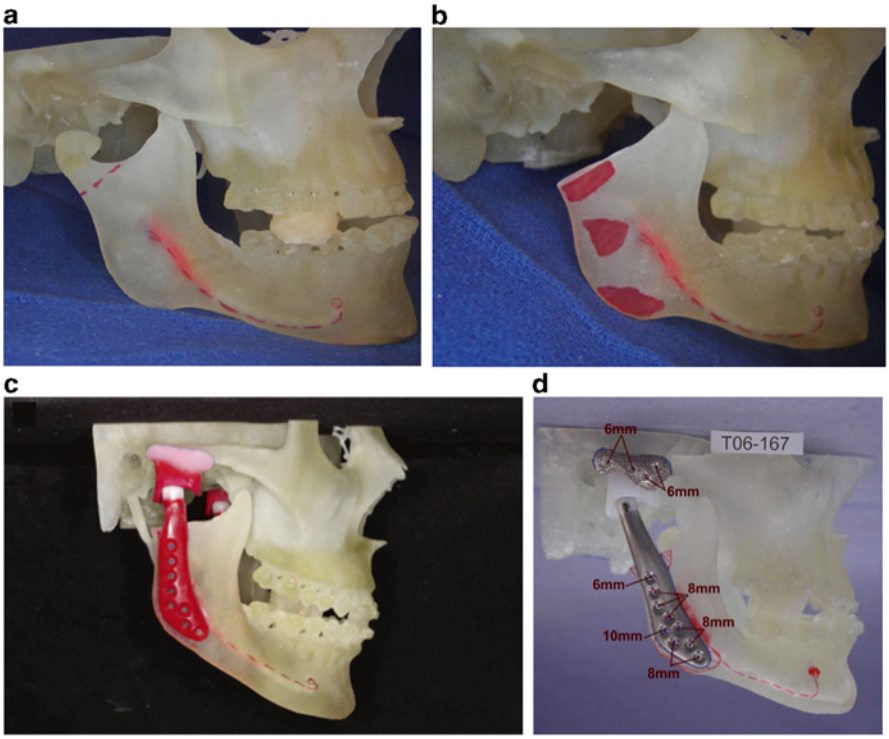


**Fig. 6.10** (**a**) Model set for ramus preparation. The level for condylectomy is marked. (**b**) The stereolithic model after condylectomy and recontouring of the fossae and rami (*marked in red*). Accommodation of the prosthesis requires 20-mm space between the fossa and ramus. (**c**) Wax-up of prosthesis is prepared for surgeon approval. (**d**) Stereolithic model with prosthesis constructed

## 6.7 Protocol for Traditional C-TJR-OS

1. Protocol CT scan including the entire mandible and maxilla, including the TMJs
2. Fabrication of SL model with the mandible separated (two-piece model)
3. Positioning of the mandible on the SL model into its final occlusion and fixating it by the surgeon
4. Removing of condyles and recontouring of the lateral aspect of the rami and fossae, if indicated
5. SL model returned to TMJ Concepts for device design
6. Approving of the design schematic by the surgeon
7. Manufacture of TMJ TJR components
8. Components and screw length schematic sent to surgeon's hospital for implantation
9. Surgery performed as planned

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## 6.8 Steps in Traditional Orthognathic Surgery and Intermediate and Palatal Splint Fabrication for C-TJR-OS

1. Acquisition of dental models
2. Mounting of maxillary and mandibular dental models on an articulator
3. Repositioning of the mandibular dental model, duplicating the positional changes acquired on the stereolithic model
4. Fabrication of intermediate splint
5. Repositioning of the maxillary dental model with segmentation if indicated
6. Construction of palatal splint (or occlusal splint if the surgeon prefers)
7. Ready for surgery

## 6.9 Virtual Surgical Planning

Virtual surgical planning (VSP) utilized computer technology to simulate the planned surgical procedures. Over the past decade, computer-assisted surgical simulation (CASS) technology has been integrated to many maxillofacial surgical applications [35, 36], including management of congenital and acquired dentofacial deformities, defects created by ablative tumor surgery, trauma, cranial defects [37], and reconstruction of the TMJ [33, 34]. CASS technology applied to orthognathic surgery can improve surgical accuracy, provide intermediate and final surgical splints, and decrease the surgeon's presurgical preparation time compared with traditional methods. VSP data for use in orthognathic surgery cases can be obtained from high-quality cone beam scans, but better-quality simulation and accuracy can be acquired from medical-grade CT scans of the jaws with 1-mm overlapping cuts.

### *6.9.1 Protocol for C-TJR-OS Using CASS*

For C-TJR-OS cases, the orthognathic surgery can be planned using CASS technology and moving the maxilla and mandible into their final position using computer simulation (Fig. 6.11a, c). Using the acquired data applied to simulation program on a computer, the anteroposterior and vertical positions, occlusal plane alteration, pitch, yaw, and roll are accurately finalized for the maxilla and mandible based on clinical evaluation, dental models, prediction tracing, and computer simulation analysis. Segmentation of the maxilla can also be simulated.

Using Digital Imaging and Communications in Medicine (DICOM) data, a virtual model is provided to the surgeon with the maxilla and mandible in the final

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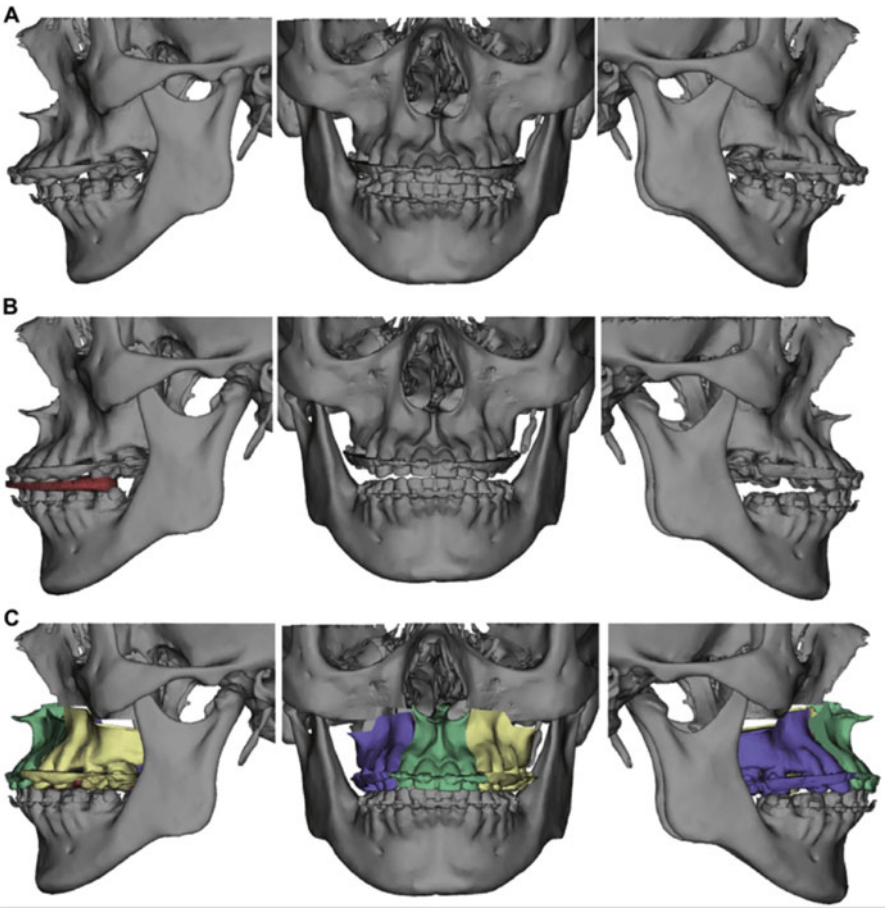


**Fig. 6.11** Staged computer-assisted surgical simulation (CASS). (**a**) Simulated preoperative position of the maxilla and mandible. (**b**) The maxilla and mandible in the simulated intermediate position, with the maxilla in its original position, but mandible in its final position with the mandibular surgery performed first for fabrication of the intermediate splint. (**c**) The final position of maxilla and mandible, after *counterclockwise* rotation-advancement of the mandible and segmented maxilla, for the production of a palatal splint or occlusal splint if the surgeon prefers

position for any specific anatomical alteration indicated. The surgically altered virtual SL model is sent to TMJ Concepts for the design of the device. Via the Internet, the design is approved by the surgeon. Then, the custom-fitted total joint prostheses are manufactured (Fig. 6.12).

Figure 6.12 demonstrates the basic design of the TMJ Concepts patient-fitted prosthesis. The black arrow points to the commercially pure titanium (cpTi) mesh framework on the underside of the fossa component that supports the ultrahigh molecular weight polyethylene (UHMWPE) bearing surface. The red arrow points

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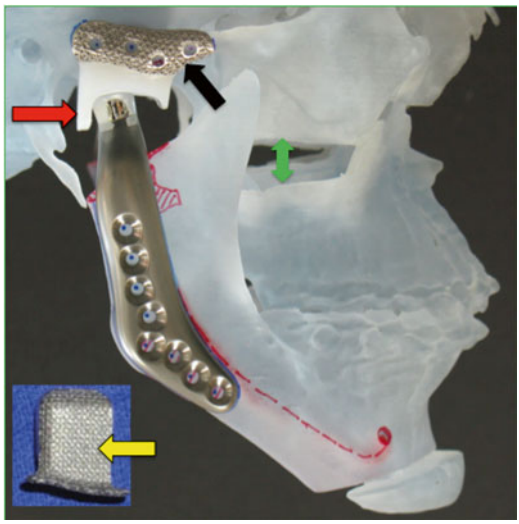

**Fig. 6.12** Stereolithic model fabricated after simulated maxillary and mandibular advancement to the final position. Condylectomy and recontouring of the lateral rami and fossae were performed and prostheses manufactured. The basic design of the TMJ Concepts patient-fitted prosthesis is observed. The *black arrow* points to the mesh framework on the underside of the custom-fitted titanium shell that secures the polyethylene articulating portion of the fossa component. The *yellow arrow* points to the mesh on the superior surface of the fossa component that allows osseointegration with the fossa bone. The *red arrow* points to the posterior stop of the fossa, a necessary component for mandibular advancement and stability. The *green arrow* shows the bony defect created from the *counterclockwise rotation* of the posterior maxilla. These defects require bone or synthetic bone grafting for stability of the maxilla

to the fossa component's posterior stop. This is an absolutely necessary component for mandibular advancement and stability in any C-TJR-OS procedure. The green arrow shows the bony defect created in the posterior maxilla from the counterclockwise rotation. These defects require bone or synthetic bone grafting for stability of the maxilla.

Approximately 2 weeks before surgery, final dental models are made, including two maxillary models if the maxilla is to be segmented or dental equilibration is required. One of the maxillary models is segmented, dental equilibration is performed, if indicated, and the segments are placed in the best occlusion with the mandibular dentition and maxillary segments fixed to each other. The dental models do not require mounting on an articulator. The three or four models (two maxillary and one mandibular or two mandibular models if equilibrations or dentoalveolar surgery is done on the mandible) are physically sent to the VSP company for scanning and simulation into the computer model. Alternatively, with an i-CAT machine, the models can be scanned and digitally sent to the VSP company. Because the author's surgical protocol routinely performs the TMJ TJR and mandibular advancement with the TMJ Concepts total joint prosthesis first, the unsegmented maxillary

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model is simulated into the original maxillary position, and the mandibular model is simulated into the mandible into its final position. The intermediate splint is constructed (Figs. 6.11b and 6.13a, b), the segmented maxillary model is simulated into the computer model in its final position in the best occlusion, and the palatal splint is fabricated (Fig. 6.13c, d). An occlusal splint can be used, if the surgeons prefer.

## 6.10 Protocol of C-TJR-OS Using CASS

1. Protocol CT scan of the entire mandible and maxilla, including the TMJs.
2. Processing of DICOM data to create a virtual computer model in CASS environment.
3. Correction of dentofacial deformity, including final positioning of the maxilla and mandible, with computer-simulated surgery.

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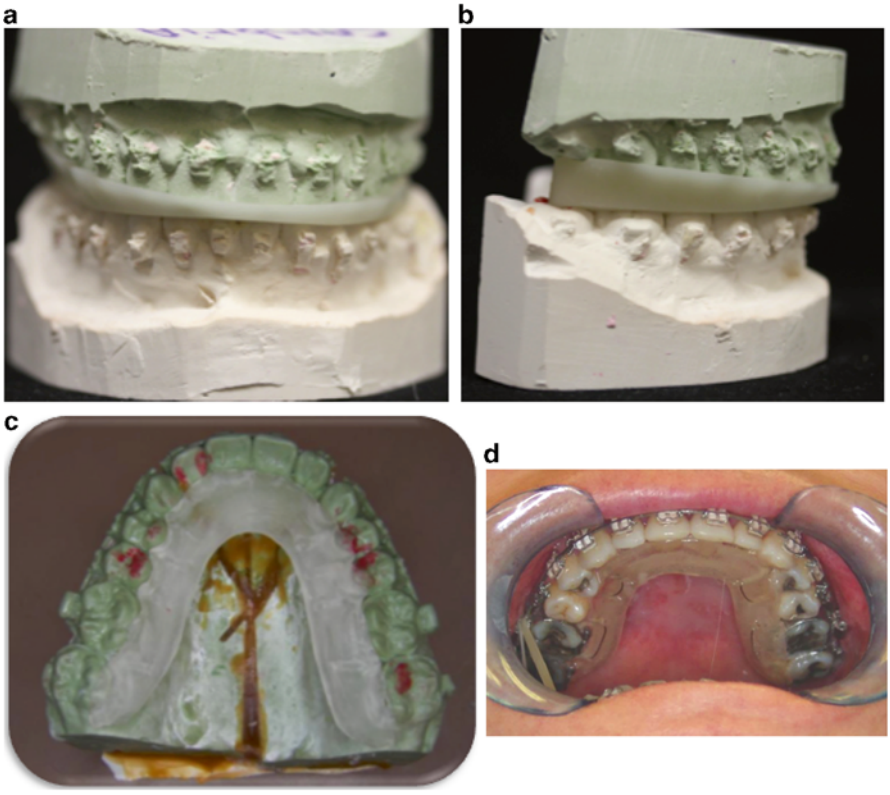


**Fig. 6.13** (**a, b**) Intermediate splint is printed from the CASS model with the mandible in the final position and maxilla in the original position. (**c**) Palatal splint printed and (**d**) inserted

4. SL model constructed with jaws in final position and sent to surgeon for condylectomy and ramus and fossa recontouring if indicated.
5. SL model sent to TMJ Concepts for device design.
6. Surgeon design approval via the Internet.
7. Components manufactured and sent to the surgeon's hospital for implantation.
8. Acquisition of final dental models, 2 weeks before surgery (two maxillary, one or two mandibular models if dental equilibrations are required); one maxillary model is segmented and models equilibrated if indicated to maximize the occlusal fit; models sent to the VSP company.
9. Models incorporated into computer-simulated surgery for construction of intermediate and final palatal splints.
10. Models, splints, and printouts of computer-simulated surgery sent to surgeon.

Using CASS technology for C-TJR-OS cases eliminates the "traditional" steps requiring the surgeon to manually set the mandible into its new final position on the SL model, thus saving time and improving surgical accuracy. Although dental model surgery is necessary only if the maxilla requires segmentation, the models do not require mounting on an articulator. This saves considerable time by eliminating the time required to mount the models, prepare the model bases for model surgery, reposition the mandible, construct the intermediate occlusal splint, and make the final palatal splint.

With CASS technology, the splints are manufactured by the VSP company.

### *6.10.1 Surgical Sequencing for C-TJR-OS*

1. Condylectomy
2. Coronoidotomy or coronoidectomy
3. Detaching the masseter and medial pterygoid muscles from the ramus
4. Mobilizing the mandible
5. Maxillo-mandibular fixation with intermediate surgical splint
6. Placement of total joint prostheses
7. Bilateral TMJ fat grafts harvested from the abdomen or buttock (Fig. 6.14)
8. Maxillary osteotomies and mobilization
9. Turbinectomies, septoplasty, etc.
10. Maxillary segmentation and application of the palatal splint if indicated
11. Maxillary rigid fixation and bone grafting
12. Adjunctive procedures such as genioplasty, rhinoplasty, UPPP, facial augmentation, etc.

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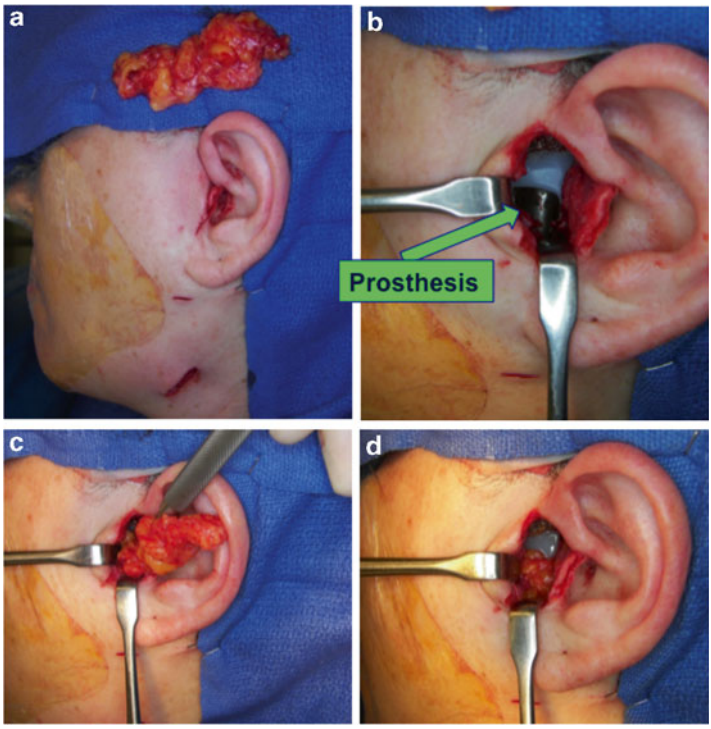


**Fig. 6.14** (**a**) Fat harvested from the abdomen for placement around the articulating area of the prostheses. (**b**) Patient-fitted prosthesis exposed via the endaural incision. (**c**) Packing the fat into the joint area. (**d**) Completion of fat packing and ready for incision closure

## 6.11 Case 1

This 22-year-old female had the onset of TMJ problems at the age of 14 secondary to adolescent internal condylar resorption (AICR). She had previous orthodontics and combined maxillary and mandibular orthognathic surgery at the age of 16; however, the TMJ pathology was not addressed and continued to worsen. She presented with a significant relapse of both the maxilla and mandible and development of a Class II anterior open bite (Figs. 6.15a–c, 6.16a–c, and 6.17a). She also had hypertrophied turbinates with nasal airway obstruction. Presurgery, her TMJ pain was 8, headaches 5, jaw function 5, diet 5, and disability 8 (0 = no pain or no limitations; 10 = worse pain imaginable or total loss of function). Incisal opening was 48 mm, but only 32 mm without significant pain. Cone beam CT (Fig. 6.18) revealed advanced arthritis with severe condylar resorption consistent with advanced

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AICR. Presurgical MRI (Fig. 6.19) demonstrated arthritic changes in the joints and severely degenerated articular non-salvageable disks.

The patient underwent the following surgical procedures: (1) bilateral TMJ TJR with patient-fitted devices and counterclockwise rotation-advancement of the mandible 18 mm, (2) multiple maxillary osteotomies to counterclockwise rotate and advance the maxilla 8 mm (Fig. 6.17b), (3) bilateral mandibular coronoidotomies, (4) bilateral TMJ fat grafts (harvested from the abdomen), and (5) bilateral partial inferior turbinectomies.

At 3 years post-surgery, she has maintained stable skeletal and occlusal relationships, improved facial balance, and good airway (Figs. 6.15d–f and 6.16d–f) with TMJ pain 0, headaches 0, jaw function 1, diet 1, disability 0, and incisal opening of 45 mm.

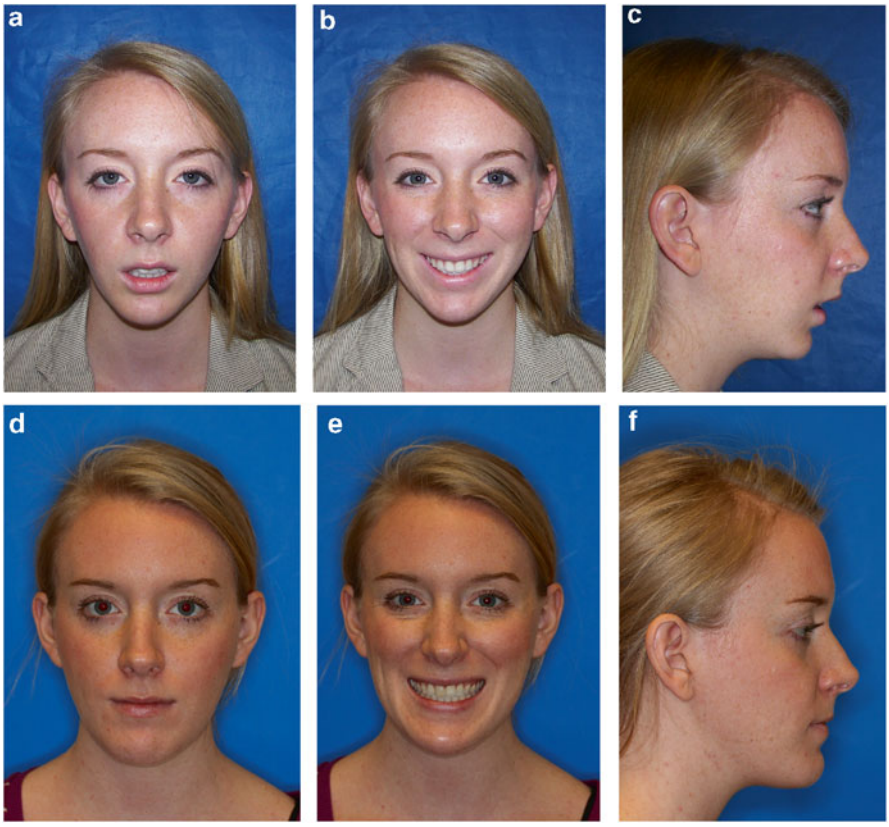


**Fig. 6.15** **Case 1**: (**a–c**) Presurgery clinical pictures. (**d–f**) Post-surgery clinical images at 3 years post-surgery

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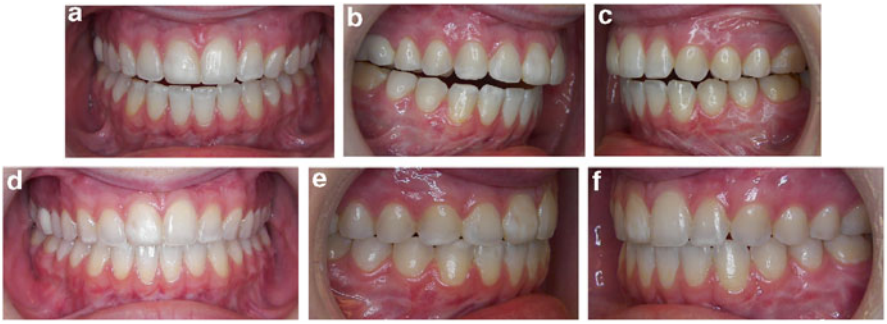


**Fig. 6.16 Case 1**: (**a–c**) Presurgery occlusion with a Class II open bite and occlusal contact only on the second molars. (**d–f**) At 3 years post-surgery, the occlusion is stable with a Class I cuspid-molar relationship

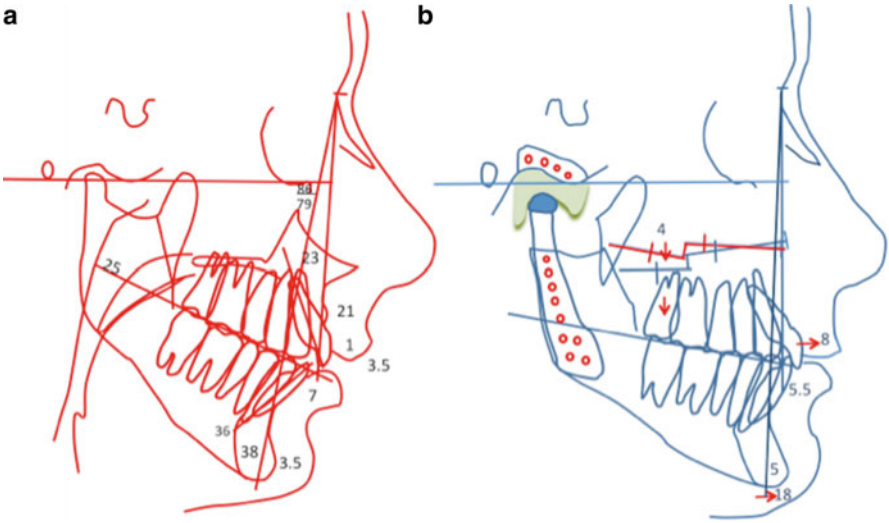


**Fig. 6.17 Case 1**: (**a**) Presurgical cephalometric tracing shows the retruded maxilla and mandible as well as the high occlusal plane angle (25°) and decreased oropharyngeal airway. (**b**) The surgical treatment objective demonstrated the planned surgical changes with *counterclockwise rotation-advancement* with the maxillary incisal edges advancing 8 mm and pogonion advancing 18 mm

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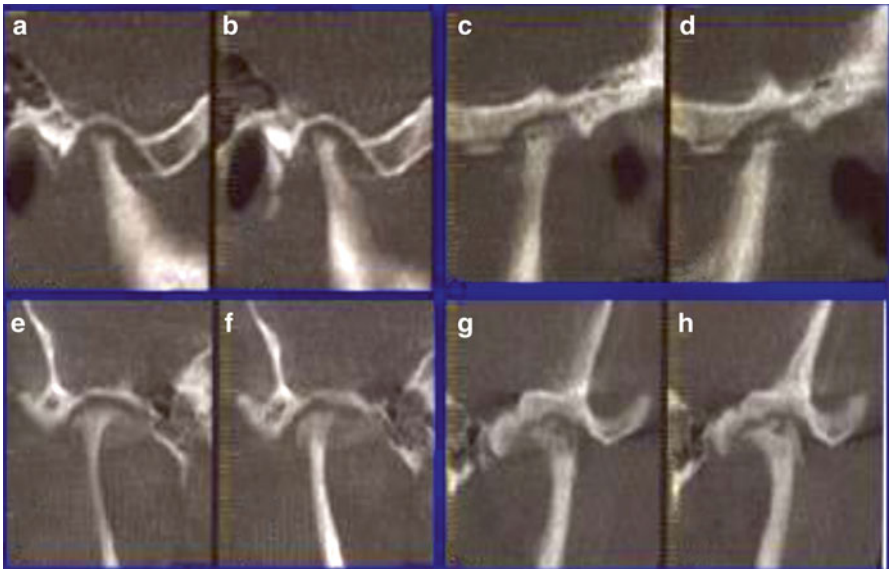


**Fig. 6.18** **Case 1**: Presurgery sagittal and coronal images (cone beam CT) of bilateral TMJs demonstrating advanced arthritis as a result of untreated AICR. (**a** and **b**) right TMJ, (**c** and **d**) left TMJ sagittal view, (**e** and **f**) right TMJ, (**g** and **h**) left TMJ coronal views

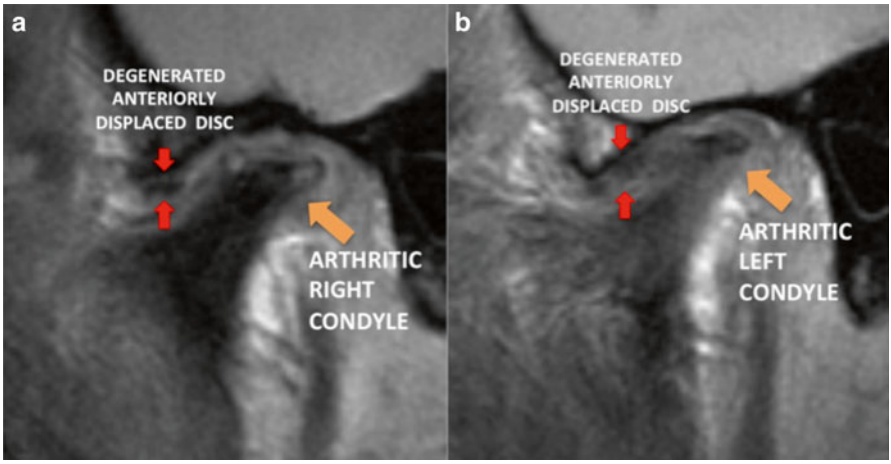


**Fig. 6.19** **Case 1**: (**a** and **b**) Presurgery sagittal T-1 images of bilateral TMJs, showing anteriorly displaced, non-salvageable disks with resorbed and severely arthritic condylar heads

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## 6.12 Case 2

A 15-year-old patient (Figs. 6.20a–c, 6.21a–c, and 6.22a) developed severe TMJ juvenile idiopathic arthritis, diagnosed at 11 years old, with milder effects in multiple other joints. She had severe sleep apnea, but was relatively pain free. Her interincisal opening was 42 mm.

The treatment plan (Fig. 6.22b) in a single-stage surgery included (1) bilateral TMJ TJR and mandibular advancement in a counterclockwise direction with patient-fitted devices, (2) bilateral coronoidectomies, (3) multiple maxillary osteotomies to move the anterior aspect upward and posterior aspect downward, (4) genioplasty with a 14-mm alloplastic implant, and (5) bilateral partial inferior turbinectomies.

One year post-surgery (Figs. 6.20d–f and 6.21d–f), the patient has good facial balance, no pain, skeletal and occlusal stability, and an incisal opening of 33 mm. Twenty-two years and 3 months post-surgery, she maintains good facial balance and stability, has no pain, and has an incisal opening of 37 mm with no dietary limitations (Figs. 6.20g–i and 6.21g–i).

### *6.12.1 Treatment Outcomes Using These Treatment Protocols*

Dela Coleta et al. [38] evaluated 47 female patients for surgical stability after C-TJR-OS with Menton advancing an average of 18.4 mm and the occlusal plane decreasing an average of 14.9°. Average follow-up was 40.6 months. Results demonstrated minor maxillary horizontal changes, while the mandibular measurements remained very stable.

Pinto et al. [39] evaluated the same 47 female patients relative to pain and dysfunctional outcomes. Patients were divided into two groups based on the number of previous surgeries: Group 1 had 0–1 previous surgeries, while Group 2 had two or more previous surgeries. Significant improvements (37–52 %) were observed for TMJ pain, headaches, jaw function, diet, and disability. Interincisal opening increased 14 %. Group 1 patients had better pain and jaw function results than Group 2 patients.

These two studies demonstrated that end-stage TMJ patients could be treated in one operation with C-TJR-OS resulting in long-term functional stability and improvement in pain and mandibular function.

### *6.12.2 Age for Surgical Intervention*

Although there are individual variations, females typically complete the majority of their facial growth (98 %) by age 15 years, whereas males by age 18 [40]. Predictability of the outcome is best when the corrective surgery is limited to the

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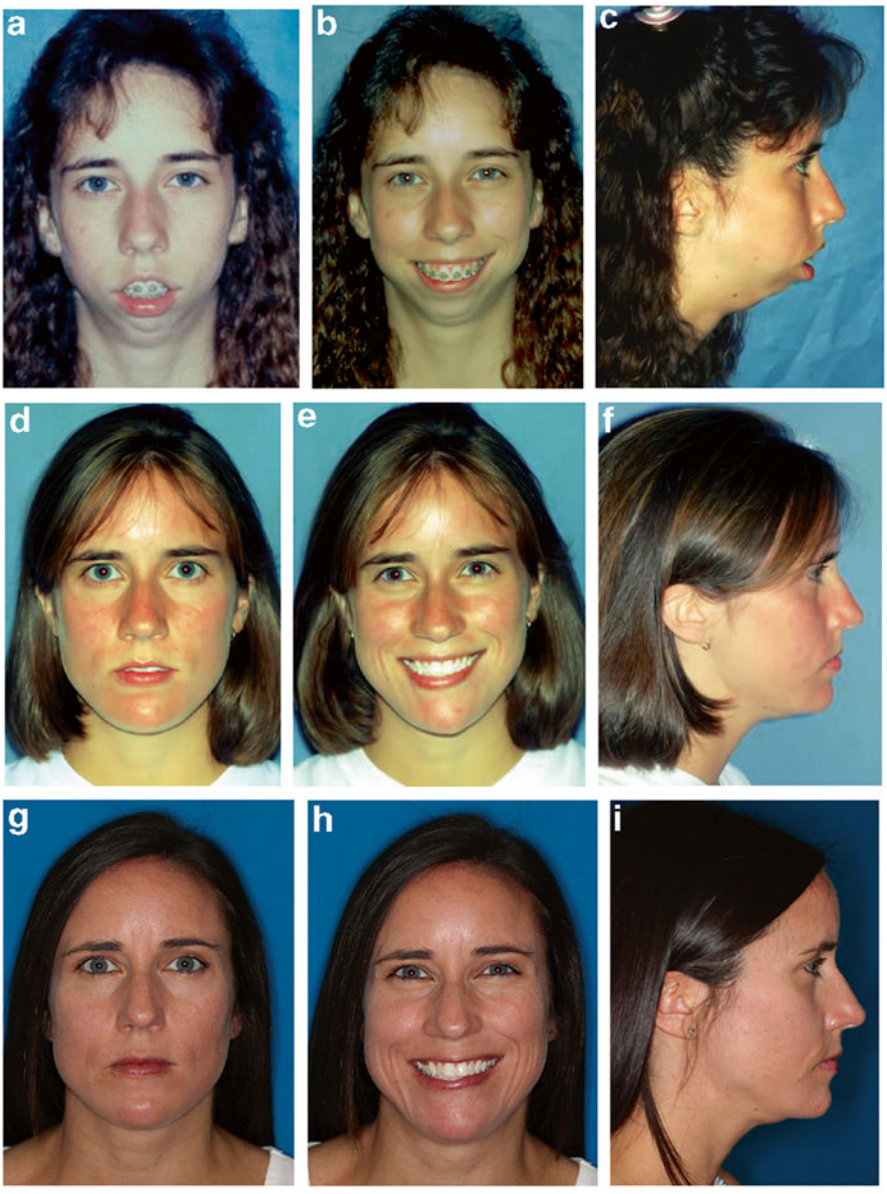


**Fig. 6.20** **Case 2**: (**a–c**) 15-year-old female with JIA and grossly resorbed mandibular condyles, severely retruded mandible, anterior maxillary vertical hyperplasia, and high occlusal plane angle facial morphology. (**d–f**) The patient is seen at 1 year post-surgery demonstrating significantly improved facial balance and function. (**g–i**) At 22 years post-surgery, the patient maintains good facial balance and function

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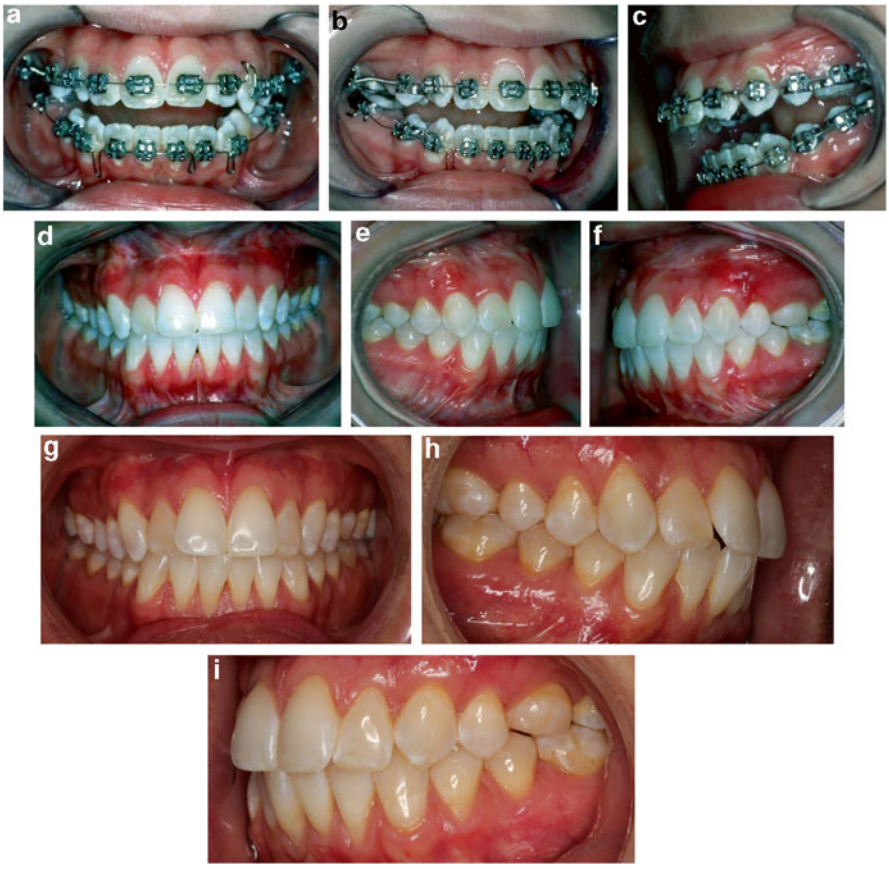


**Fig. 6.21** **Case 2**: (**a–c**) A significant anterior open bite is present and Class II occlusion. (**d–f**) At 1 year post-surgery, she demonstrates a stable Class I occlusion. (**g–i**) At 22 years post-surgery, she retains the stable occlusal result

affected jaw in one major operation by waiting until growth is relatively complete. This is particularly true if C-TJR-OS is required to manage end-stage disease in such patients.

However, there are definite indications for performing C-TJR-OS during the growing years, such as progressive TMJ deterioration, ankylosis, masticatory dysfunction, tumor removal, pain, sleep apnea, etc. Performing surgery during growth may require additional surgery at a later time to correct a resultant deformity and malocclusion that may develop during the completion of growth. Additional surgery is a greater probability with unilateral TMJ TJR and a normal contralateral TMJ if surgery is performed in a growing patient. In addition, some orthognathic surgical procedures have a profound effect on subsequent facial growth and development

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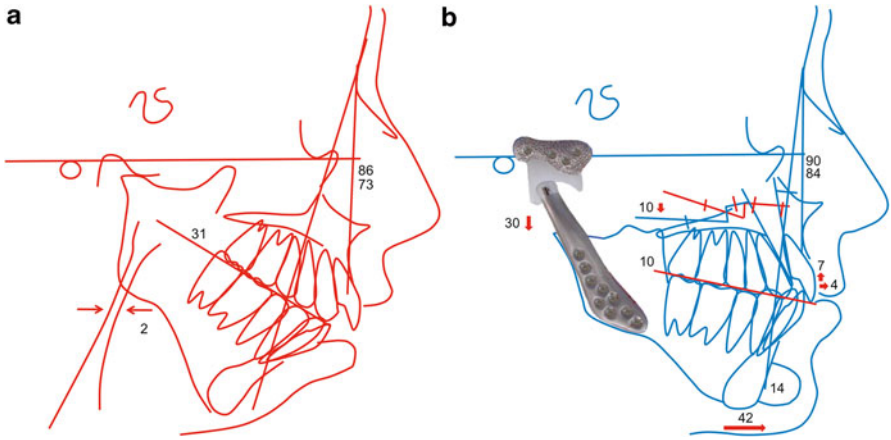


**Fig. 6.22 Case 2**: (**a**) The cephalometric analysis shows the severe jaw deformity and high occlusal plane angulation. (**b**) The prediction tracing demonstrates the *counterclockwise rotation* of the maxillo-mandibular complex. The chin is augmented with an alloplastic implant. Pogonion advanced 42 mm

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including maxillary Le Fort I osteotomies, where maxillary AP growth has ceased, but the vertical alveolar growth of the maxilla and mandible continues contributing to a downward and backward rotation vector of facial growth, but the occlusion should stay together. Therefore, bilateral TMJ TJR and maxillary osteotomies can be performed at an earlier age with predictable results.

If repeat orthognathic surgery is required at a later time, the advancement of the mandible with the TMJ TJR device can be accomplished by one of five surgical options: (1) intraoral ramus sagittal split osteotomy; (2) extraoral sagittal split ramus osteotomy (ESSRO); (3) advancement of the mandible forward relative to the prosthesis by removing the screws from the mandibular component, separation of the mandibular component from rams, advancement of the mandible along the patient-fitted prosthesis, and re-fixation of the prosthesis with bone screws to the mandible in its new position; (4) replacing the mandibular component of the TMJ TJR device with a new longer mandibular component that would be reattached to the mandibular ramus after the mandible is moved into its new position; or (5) osseodistraction.

Reports on maxillary and mandibular orthognathic surgery and the effects on growth, with guidelines for age considerations for surgical intervention [41–43] as well as TMJ surgery effects on facial growth [44], have been published. Juvenile idiopathic arthritis (JIA) patients (ages 12–14 years and older) have been managed successfully using the protocol described above in one stage with good functional and aesthetic results without requiring secondary procedures [20]. These cases are predictable when performed at age 13 years or older in females and 15 years or older in males. However, the vector of facial growth will change in younger patients to a downward and backward direction.

## 6.13 Summary

Healthy and stable TMJs are necessary for quality outcomes in orthognathic surgery. If the TMJs are not stable and healthy, orthognathic surgery results may be unsatisfactory relative to function, aesthetics, skeletal and occlusal stability, as well as pain. The surgeon should be suspicious of possible TMJ problems in the following types of patients: (1) high occlusal plane angle facial morphologies with retruded maxilla and mandible; (2) Class II high occlusal plane angle and retruded mandibular morphological type, particularly those with anterior open bites; (3) progressively worsening Class II occlusal and jaw relationship; (4) facial asymmetry, particularly with progressive worsening; and (5) patients reporting headaches, TMJ pain, myofascial pain, history of clicking and popping of the TMJs, and/or ear symptoms. The surgeon should not ignore these signs and symptoms. Patients presenting with one or more of these signs and symptoms should be evaluated for possible TMJ pathology. Advanced imaging (CT, MRI, bone scanning) can aid in identification of the specific TMJ pathology. Failure to recognize and manage these conditions can result in significant skeletal relapse, increased pain, and a greater complexity of subsequent management.

During the past 25 years, major advancements have been made in TMJ diagnostics and the development of surgical procedures to treat and rehabilitate the pathological, dysfunctional, and painful TMJ. Research has clearly demonstrated that C-TJR-OS can be safely and predictably performed at the same operation, but it does necessitate the correct diagnosis and planning, as well as requiring the surgeon to have expertise in both TMJ and orthognathic surgery. The surgical procedures can be separated into two or more surgical stages, but the TMJ surgery should be done first.

With the correct diagnosis and treatment plan, combined TMJ and orthognathic surgical approaches provide complete and comprehensive management of patients with coexisting TMJ pathology and dentofacial deformities.

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# Chapter 7
# Mandibular Replacement Utilizing TMJ TJR Devices

**Luis Vega and Daniel Meara**

Throughout this book, authors have comprehensively presented the basic principles and rationale for the use of alloplastic temporomandibular joint total joint replacements (TMJ TJR). Pearls and pitfalls of the basic surgical techniques as well as more sophisticated procedures such as combined TMJ TJR/orthognathic surgery have also been described. This chapter offers the unique perspective of using alloplastic TMJ TJR for the reconstruction of acquired mandibular defects that involved the TMJ. It is not the authors' intention to provide management protocols of the primary process that created the defect but instead to illustrate potential solutions for these challenging cases.

Mandibular defects that involved the TMJ represent a unique reconstructive challenge as the TMJ plays an important role in the function of the jaw including mastication, deglutition, phonation, and airway support. The native condyle also serves as a secondary growth center for the mandible and lower face [1]. Thus, the principles of reconstruction of mandibular defects involving the TMJ in the growing individual are different when compared to the adult. However, even in the presence of these differences, the main goals of these reconstructions remain the same: (1) stop the limitation of function, degeneration, and growth disturbance and (2) restore the form and function by providing mandibular continuity with a stable articulation.

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Although controversy still exists with the indication of alloplastic TMJ TJR in the growing patient, reports of their use can be found in the literature [2]. For the purpose of this chapter, it will be assumed that these reconstructions are being performed in skeletally mature patients.

## 7.1 Indications

Numerous surgical techniques using autogenous tissues or alloplastic materials have been described for the reconstruction of mandibular defects involving the TMJ. The indications of each technique vary depending on the severity of the problem, past medical history and age of the patient, ability to perform postoperative physical therapy, surgeon's experience, and socioeconomic factors [3]. Proper patient selection and type of reconstruction is critical for long-term treatment success of these reconstructive efforts; hence, general indications and contraindications for reconstruction of acquired mandibular defects involving the TMJ can be found in Table 7.1.

Conventionally, classification schemes and treatment algorithms have been used to aid the clinician in the decision-making process. Although these protocols exist for the management of mandibular defects, very few have been described for the reconstruction of acquired mandibular defects involving the TMJ. Potter and Dierks proposed a classification of TMJ defects and their respective management algorithms. They proposed that when discussing the reconstruction of these defects, a difference should be made in cases according to the etiology and size of the defect. They suggested that reconstruction with autogenous bone grafting or alloplastic TMJ TJR can usually be achieved in cases in which the etiology of the defect has created a residual tissue deficit that is relatively small. Furthermore, microvascular free tissue transfers were recommended in cases of large tissue deficits or irradiated or soon to be irradiated defects from malignant pathology [4]. Bredell and colleagues also suggested similar recommendations with the difference that their algo-

**Table 7.1** Indications for reconstruction of acquired mandibular defects involving the TMJ

| *Indications* |
|---|
| Posttraumatic mandibular/condylar loss or damage |
| TMJ/mandibular tumors |
| Connective tissue or autoimmune disease |
| TMJ/mandibular osteomyelitis |
| Previous failed alloplastic reconstructions |
| *Contraindications* |
| Uncontrolled systemic disease |
| Psychiatric instability |
| Active infection |
| Allergy to prosthetic components |
| Uncontrolled parafunction |

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rithm was developed taking into consideration the anatomical structures preserved during the ablation and the risk factors for complications [5].

Reconstruction of large mandibular defects that involved the TMJ with TMJ TJR devices has been successfully reported in the literature, but those descriptions are from small case series or case reports [6–11]. The paucity of scientific data for reconstruction with these devices makes management algorithms or strong recommendations very difficult. Therefore, decisions rely on the clinician's experiences and knowledge gained from routine use of TMJ TJR devices for such reconstructions. Currently, stock and custom- or patient-fitted TMJ TJR devices are available. The use of stock prostheses is limited only to defects involving the condyle and a very small amount to the mandibular ramus; nevertheless, cases of combined microvascular free tissue transfers and a stock TMJ TJR device have been reported [12]. However, custom- or patient-fitted TMJ TJR devices have the ability to normalize the anatomy by providing the necessary amount of mandibular advancement, ramus lengthening, and stability necessary to correct large and complex mandibular defects [13].

When a TMJ TJR prosthesis is being considered for reconstruction of a mandibular defect that involves the TMJ, patients can be classified based on the time in which the reconstruction is going to be performed as:

1. Immediate primary TMJ TJR reconstruction
2. Delayed primary TMJ TJR reconstruction
3. Secondary TMJ TJR reconstruction
4. Delayed secondary TMJ TJR reconstruction

## 7.2 Evaluation and Planning

Patients presenting for reconstruction of segmental mandibular defects involving the TMJ present unique risk factors for potential complications. Typically, they have faced or will be facing extensive surgery that affects the bone, soft tissues, and the dentition. Therefore, subsequent functional impairments include significant scarring, trismus, malocclusion, and facial asymmetry. Thus, the evaluation and planning of these patients will vary depending on the clinical presentation and time of the reconstruction.

### *7.2.1 Immediate Primary TMJ TJR Reconstruction*

Immediate primary reconstruction using a TMJ TJR prosthesis is a one-stage procedure that may be considered in patients that require a mandibular resection with disarticulation to address a pathological process. Typically, these defects are created after either ablation of benign pathology such as an ameloblastoma or malignant

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pathology that does not require postoperative radiation, such as osteosarcomas. Cases that require radiation are currently better managed with microvascular free tissue transfers. The role of the TMJ TJR devices in cases requiring radiation is unknown and requires further research.

Upon patient presentation, the initial efforts should be directed to determining the nature of the primary pathology. Tissue samples and imaging studies are completed to finalize a histopathological diagnosis and establish the extent of the lesion. Once the pathological diagnosis has been confirmed, the clinician will have to decide the extent of the resection and the need for immediate reconstruction. If a TMJ TJR prosthesis is considered, the protocol consists of obtaining a specified maxillofacial CT scan that is used to build a stereolithic (SL) model. The ablative surgery is then planned and carried out on this model. The model is sent back to the company that will fabricate the custom- or patient-fitted TMJ TJR device. Currently, a virtual planning surgery (VSP) protocol is also available. Using this technology, once the maxillofacial CT scan is acquired, a computer 3D model is used to perform the ablative surgery virtually. Cutting guides to assist during surgery and a SL model with the planned mandibular resection are built and sent to the company that will fabricate the prosthesis. The surgeon then approves the design and the final prosthesis is fabricated. Cases of primary TMJ TJR reconstruction with concomitant mandibular bone grafting with iliac crest bone grafting have been described in the literature [8, 10].

Another group of patients that can benefit from having a one-stage primary TMJ TJR reconstruction are patients with extremely severe bone resorption that has produced a significant condylar and mandibular defect or deformity, such as in patients with scleroderma. In these cases, the planning focuses on determining the extent of the dysfunction, malocclusion, as well as the cosmetic deformity. Similarly, a protocol-specific maxillofacial CT scan is obtained to better understand the extent of the mandibular defect or deformity. The scan is then used to fabricate a SL model that if in the presence of a malocclusion can be fabricated with the mandible and maxilla separated (two-piece model) to allow the surgeon to establish the proper occlusion. The surgically prepared SL model is then used to design the prosthesis. The prosthetic designed is then approved and the prosthesis is made. If necessary, 2 weeks before surgery, dental cast is obtained to fabricate a final splint. Care must be taken to avoid overcorrection of the cosmetic deformity in the area of the mandibular angles as lack of tissues in the area can lead to the risk of a late exposure of the device in that area.

### *7.2.2 Delayed Primary TMJ TJR Reconstruction*

A delayed primary TMJ TJR reconstruction is indicated in patients that previously underwent treatment for a primary pathology, and immediate reconstruction was contraindicated, such as cases of osteomyelitis or avulsive trauma. Patients present with facial asymmetry, malocclusion, and limitation of mandibular range of motion. The management plan in these cases will include the review of the previous records,

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if necessary, a new maxillofacial CT scan and dental models with the goal being to better understand the deformity. The fabrication of the custom- or patient-fitted prosthesis is developed by using either the traditional or the VSP protocol. If malocclusion exists, the traditional method calls for the fabrication of a two-piece SL model from a protocol CT scan. The surgeon then establishes the proper occlusion by relating and fixating the maxilla and mandible of the SL model together. Traditional model surgery with dental casts is used to fabricate a final splint. In the VSP protocol, the same process is carried out virtually, in similar fashion as previously described in the chapter describing combined TMJ TJR and orthognathic surgery. Recently, cases of custom 3D antibiotic spacers in delayed primary TMJ TJR reconstruction have been described in the literature [11].

### *7.2.3 Secondary TMJ TJR Reconstruction*

A secondary TMJ TJR reconstruction is a definitive reconstruction for patients with defects that were immediately reconstructed by placing a condyle-supported metallic reconstruction plate directly against the mandibular fossa. Although some authors have described this technique as a successful permanent reconstruction, others have recommended it as a temporary solution due to complications such as mandibular dysfunction, broken hardware, and displacement to the medial cranial fossa or the external auditory canal [14–16]. The review of the previous records and a maxillofacial CT scan are used to understand the nature of the defect and plan the reconstruction. The CT scan can be processed to digitally remove the metallic condyle-supported reconstruction plate before the SL model is developed. The patient-fitted design is completed and approved by the surgeon, and the device is fabricated.

### *7.2.4 Delayed Secondary TMJ TJR Reconstruction*

A delayed secondary TMJ TJR reconstruction is usually performed in multiple-operated patients who have undergone previous autogenous or alloplastic reconstructions that have failed. The importance of a meticulous review of the previous records cannot be overstated. Therefore, upon initial presentation, the surgeon should focus on determining the possible causes of the unsatisfactory outcome. Frequently, retained hardware will be present, so an initial failed hardware removal surgery is required to obtain the most accurate 3D model. During this surgery, the surgeon should establish the correct occlusion by repositioning the mandible in the proper relationship to the maxilla. The patient should remain in intermaxillary fixation with or without a spacer, or a temporary reconstruction with a condyle-supported metallic reconstruction plate can also be inserted. A new protocol CT scan is made, and the new reconstruction device designed, approved, and fabricated.

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## 7.3 Surgery and Its Sequence

The complexities of these cases represent a unique surgical challenge. The extent of the reconstructions will require larger or modified surgical access such as Blair or Apron approaches. Others might require the identification and preservation of the facial nerve or a combined bone graft. However, the basic surgical principles of the placement of TMJ TJR devices as described in previous chapters all apply.

The following cases illustrate the uniqueness of these reconstructions:

### *7.3.1 Case #1 Immediate Primary TMJ TJR Reconstruction (Fig. 7.1)*

A 69-year-old female presented to her general dentist complaining of pain on tooth #18. The dentist noted a limited opening and grossly decayed tooth #18. Panoramic imaging was obtained, and a significant resorption of her bilateral mandibular angles and condyles was noted. Concerned about the potential for a mandibular fracture during the extraction of tooth #18, the patient was referred to the authors' institution for further evaluation and management.

After reviewing the pattern of mandibular resorption, a working diagnosis of scleroderma was established. Past medical history and clinical examination did not reveal any typical signs or symptoms of either systemic or localized sclerosis; further serology testing was also negative. After further discussion with the patient, bilateral patient-fitted TMJ TJR device reconstruction, chin implant, and a neck lift were recommended.

Her planning consisted of:

1. Review of previous medical records
2. Plain films and protocol maxillofacial CT scan
3. Scleroderma testing (negative)
4. Extraction of compromised tooth #18 under local anesthesia
5. Fabrication of two-piece SL model to manage her malocclusion
6. Model surgery (bilateral condylectomies) and establishment of a functional occlusion
7. Bilateral patient-fitted TMJ TJR design to increase mandibular ramus height and provide some mandibular angle contour
8. Design approval and prosthesis fabrication
9. Surgical implantation of the bilateral patient-fitted TMJ TJR devices

Her surgical sequence included:

1. Placement of maxillomandibular fixation (unsterile)
2. Patient prepped and draped in sterile fashion
3. Identification of the condyles via preauricular approach

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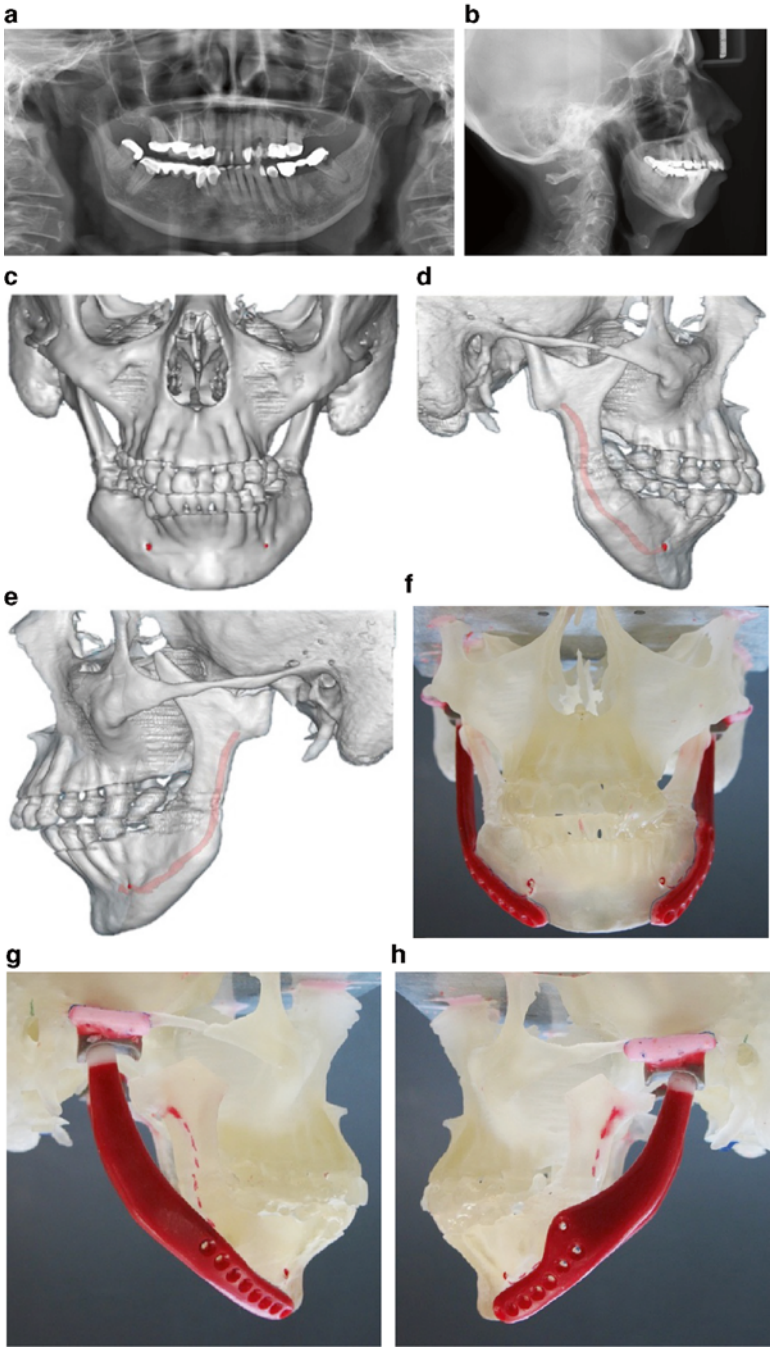


**Fig. 7.1** Case #1 immediate primary TMJ TJR reconstruction. (**a**) Preoperative panoramic x-ray; note the severe resorption of the mandibular angles as well as condyles. (**b**) Preoperative lateral cephalometric x-ray showing severe resorption of the mandibular angles, mandibular retrognathia, and microgenia. (**c–e**) Preoperative 3D renderings corroborating the findings of the plain films. (**f–h**) Prosthesis wax-up. (**i–k**) Actual total TMJ custom-made prosthesis. (**l**) Left and (**m**) right intraoperative endoscopic views of the mandibular alloplastic components in the planned location. (**n**) Before and after patient's profile. (**o**) Postoperative CT scan 3D, (**p**), panoramic, (**q**), lateral, and (**r**) anteroposterior cephalometric radiographs showing good prosthesis placement

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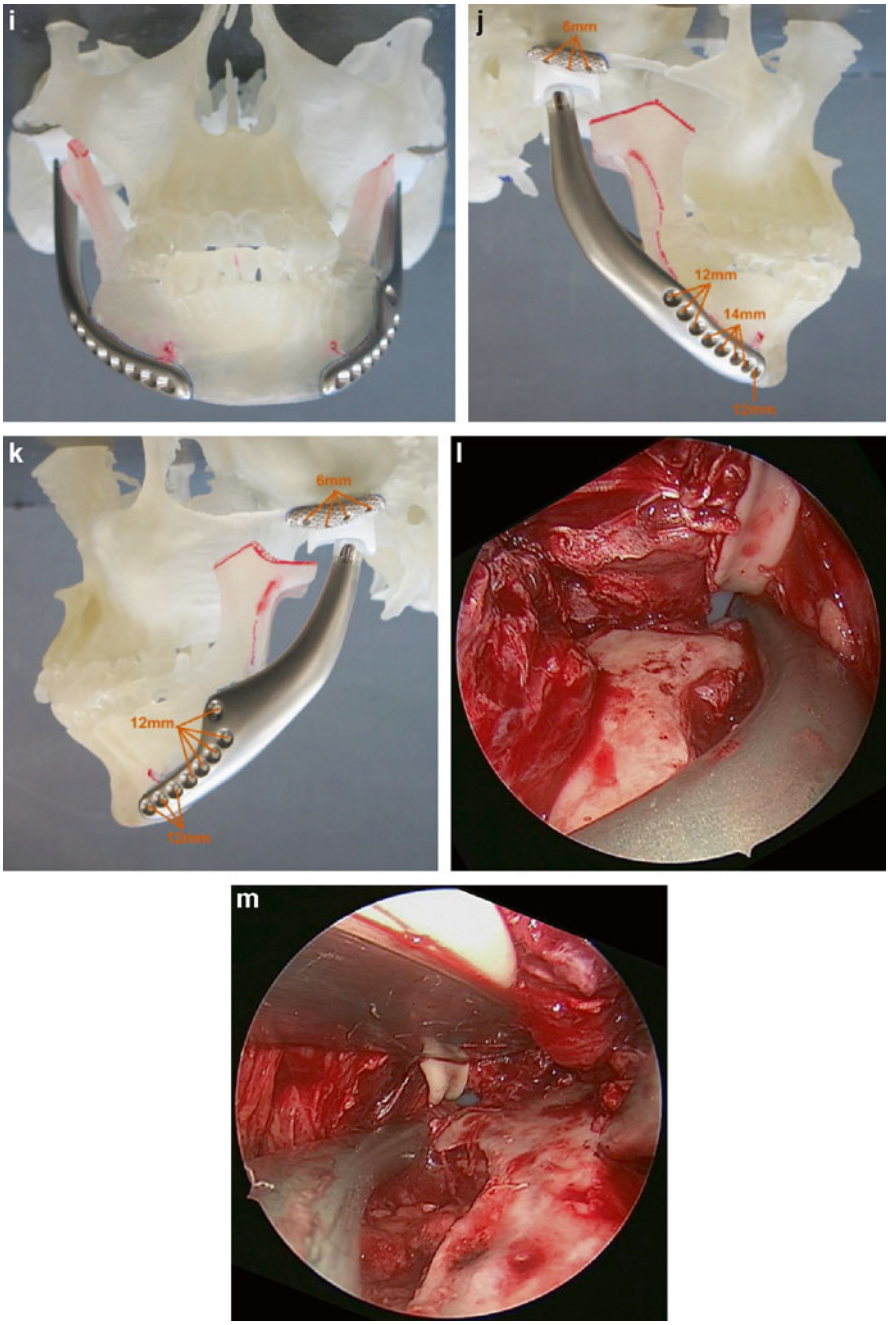


**Fig. 7.1** (continued)

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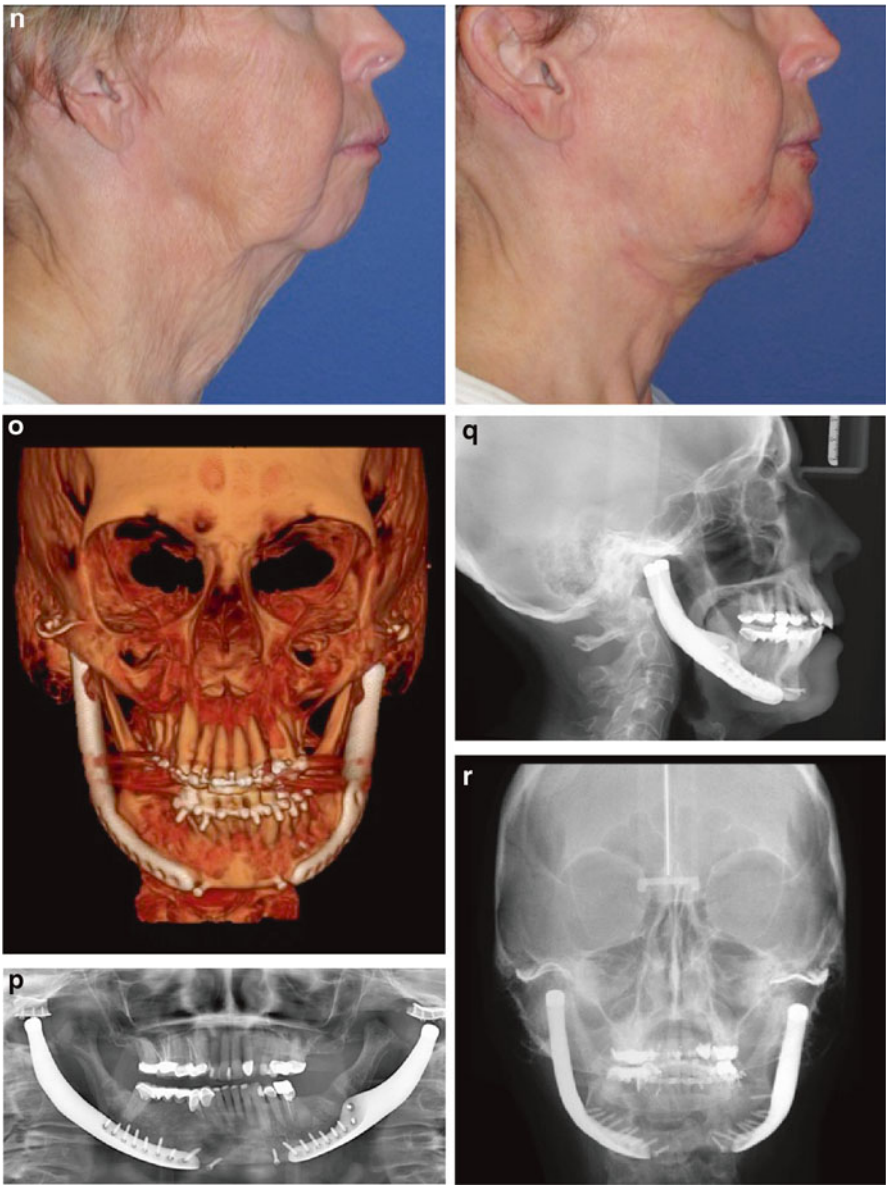


**Fig. 7.1** (continued)

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4. Submandibular approach with identification of the condyles and coronoid processes
5. Debridement and removal of the articular disk and residual soft tissues through the preauricular approach
6. Bilateral condylectomies and coronoidectomies (submandibular approach with an angled-oscillating saw)
7. Implantation of the fossa components
8. Implantation of the mandibular components
9. Check occlusion
10. Irrigation and closure of the surgical approaches (sterile)
11. Extraoral placement of chin implant (submental approach)
12. Direct neck lift with platysma plication

Three years after surgery, the patient is pain-free with a maximum interincisal opening of 33 mm.

### *7.3.2 Case #2 Delayed Primary TMJ TJR Reconstruction (Fig. 7.2)*

A 31-year-old female with long history of right TMJ pain and dysfunction after having a proximal condylar segment fracture during a right mandibular modified condylotomy in the past. The original surgeons unsuccessfully tried to stabilize the fractured segments. One year later, patient presented to the authors' institution with worsening right TMJ pain and dysfunction. CT scan 3D reconstructions demonstrated a lack of bone stock in the mandibular ramus and a significant dislocation of the condylar segment. After clinical and radiographic evaluation, it was determined that the patient required a patient-fitted TMJ TJR device that included a contoured mandibular angle.

Planning in this case included:

1. Review of previous medical records
2. Plain films and maxillofacial CT scan
3. Fabrication of two-piece SL model to manage her malocclusion
4. Model surgery (right condylectomy) and establishment of a functional occlusion
5. Device design with increase of mandibular ramus width with improved mandibular angle contour
6. Design approval and device fabrication
7. Surgical implantation of the patient-fitted TMJ TJR device

Her surgical sequence included:

1. Placement of maxillomandibular fixation (unsterile)
2. Patient prepped and draped in sterile fashion
3. Submandibular approach with identification of the dislocated condylar segment
4. Preauricular approach with identification of the dislocated condylar segment

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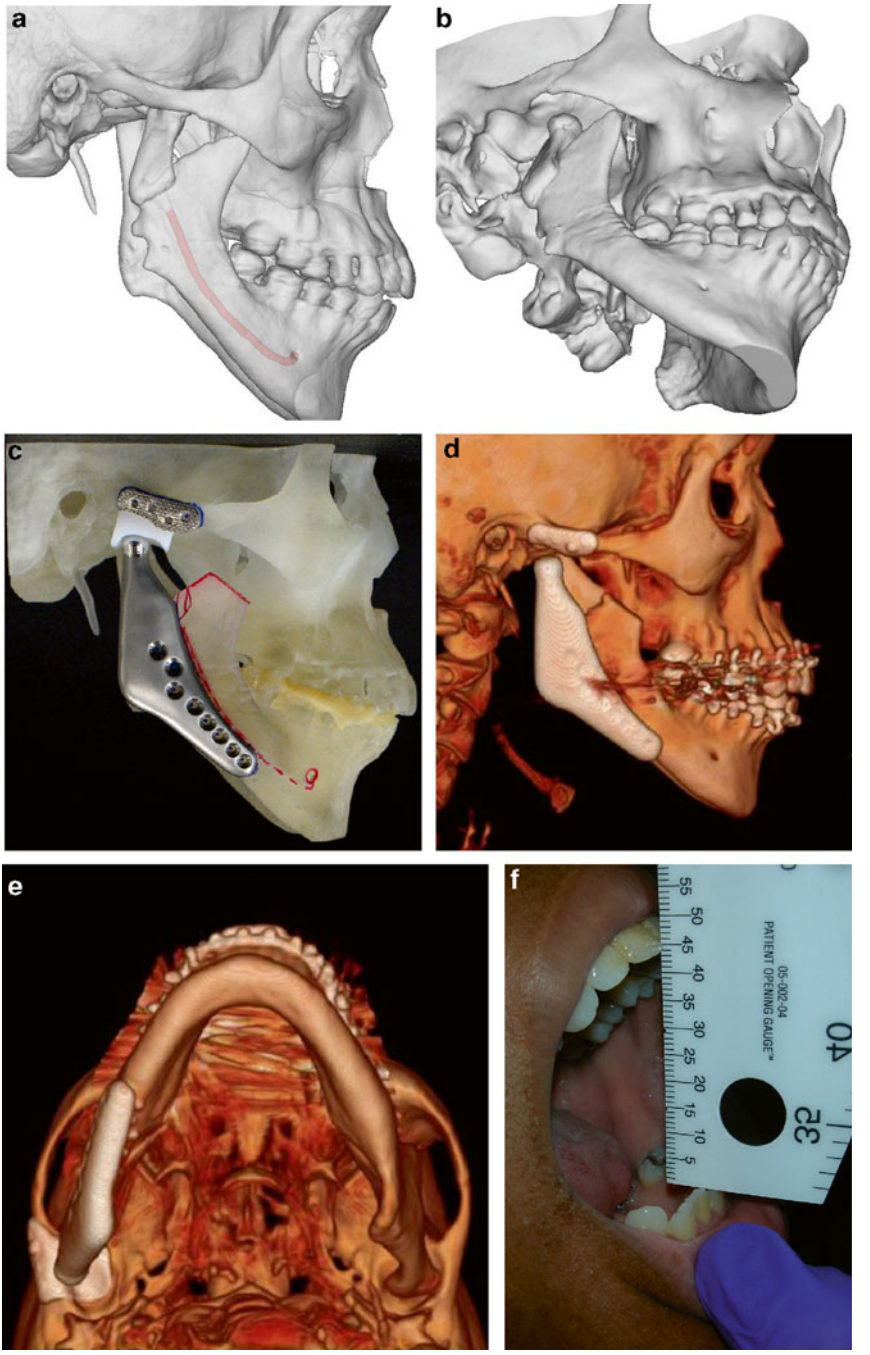


**Fig. 7.2** Case #2 delayed primary TMJ TJR reconstruction. (**a, b**) CT scan 3D reconstructions after failed modified condylotomy. Note the lack of bone stock in the mandibular ramus and the level of dislocation of the condylar segment. (**c**) Custom-made alloplastic TMJ prosthesis that included the creation of a new mandibular angle for mandibular symmetry. (**d, e**) Postoperative 3D CT scan reconstructions showing good prosthesis placement and achievement of mandibular symmetry. (**f**) Patient's postoperative maximum interincisal opening 9 months after the TMJ replacement surgery (*Source*: Vega LG, Gutta R, Louis P. Oral Maxillofac Surg Clin North Am. 2011;23(1):119–32)

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5. Condylectomy and coronoidectomy (submandibular approach with an angled-oscillating saw)
6. Debridement and removal of the articular disk and residual soft tissues (preauricular approach)
7. Implantation of the fossa component
8. Implantation of the mandibular component
9. Check occlusion
10. Irrigation and closure of the surgical approaches (sterile)

Five years after surgery, the patient is pain-free with a maximum interincisal opening of 44 mm.

### *7.3.3 Case #3 Delayed Secondary TMJ TJR Reconstruction (Fig. 7.3)*

This is the case of a 27-year-old male with a history of poorly differentiated fibrosarcoma of the left mandibular ramus and condyle. Chemotherapy was started preoperatively, and subsequently the patient underwent a left mandibular resection with left TMJ disarticulation, left total parotidectomy, left infratemporal fossa resection, and left selective neck dissection. Initial reconstruction consisted of a left pectoralis muscle flap and insertion of a condylar supported metallic reconstruction plate. The patient did well until 3 years later when he presented to the authors' institution complaining of new onset of pain and swelling in the left mandible.

After complete clinical and radiographic evaluation, it was determined that the left mandibular reconstruction plate had broken and he had developed a secondary MRSA infection. The patient was taken to the OR for debridement and after being deemed infection-free at 6 months, the decision was made to reconstruct him with a left mandibular patient-fitted TMJ TJR device.

His planning included:

1. Review of previous medical records
2. Plain films and maxillofacial CT scan
3. Surgical removal of the broken condyle-bearing reconstruction plate and long-term IV antibiotics
4. After 6 months infection-free, a new maxillofacial CT scan without the reconstruction plate
5. Fabrication of two-piece SL model to manage his malocclusion
6. Establishment of the correct functional occlusion
7. Prosthesis designed to mimic the ablated anatomy; additionally a hole was placed in the neck of the prosthetic condyle to provide anchorage to avoid potential dislocation. Additional suture holes were also done in the region of the angle to resuspend the soft tissues in the area
8. Design approval and device fabrication
9. Surgical implantation of the patient-fitted TMJ TJR device

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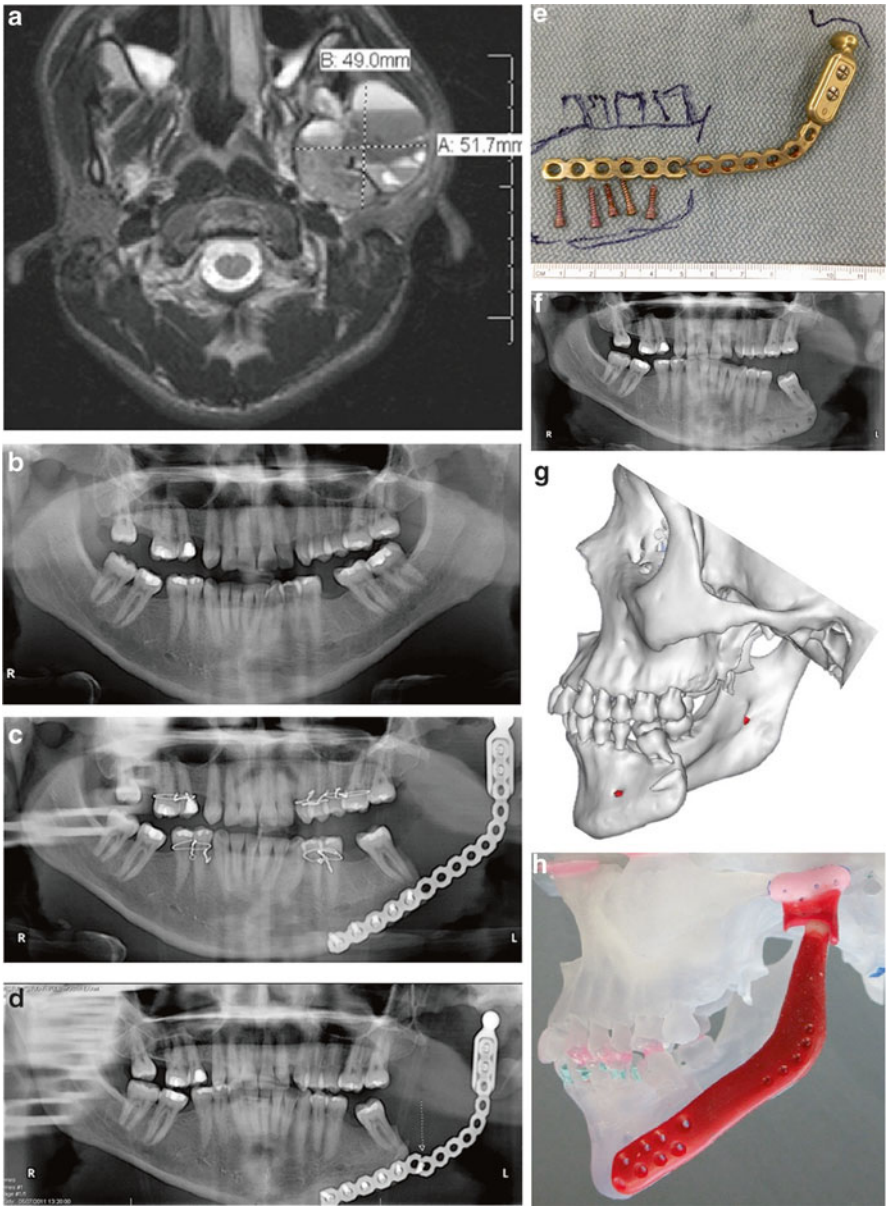


**Fig. 7.3** Case #3 delayed secondary TMJ TJR reconstruction. (**a**) Original axial MRI showing the sarcoma lesion. (**b**) Preoperative panoramic radiograph showing destruction of the left mandibular condyle. (**c**) Postoperative panoramic radiograph showing the temporary reconstruction of the left TMJ with a condyle-bearing reconstruction plate. (**d**) Panoramic x-ray showing the broken reconstruction plate. (**e**) Surgical specimen. (**f**) Postoperative panoramic after removal of the reconstruction plate. (**g**) CT scan 3D renderings corroborating the findings of the plain films. (**h, i**) Prosthesis wax-up. (**j**) Actual total TMJ custom-made prosthesis. (**k, l**) Postoperative panoramic and anteroposterior cephalometric x-rays showing good prosthesis placement

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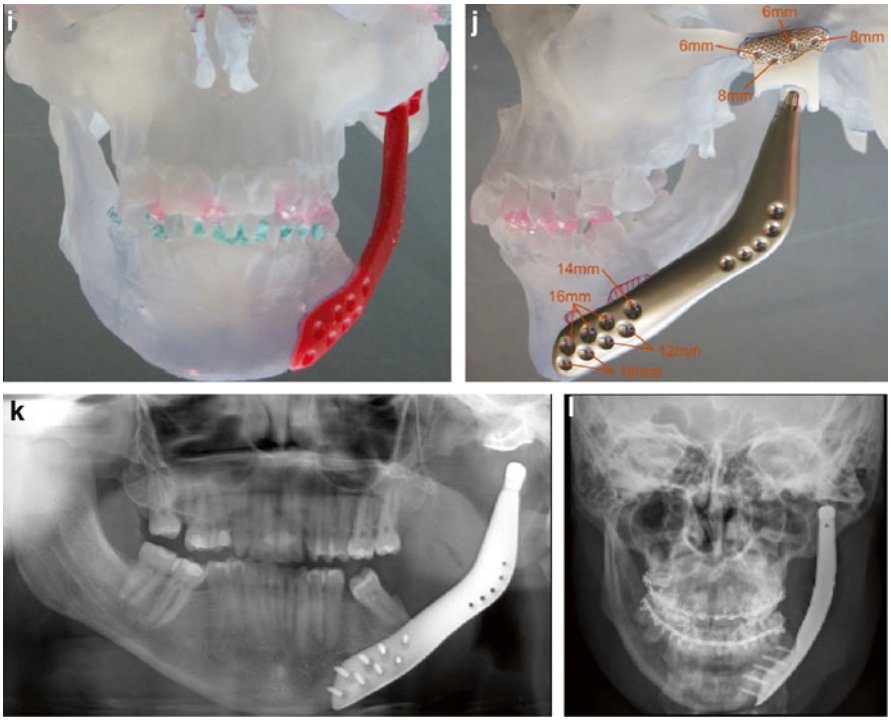


**Fig. 7.3** (continued)

His surgical sequence consisted of:

1. Patient prepped and draped in sterile fashion
2. Extended submandibular approach with tunneling of the soft tissues toward the mandibular fossa
3. Preauricular approach with debridement and removal of soft tissues
4. Placement of the fossa component
5. Placement of the mandibular component
6. Check occlusion
7. Irrigation and closure of the surgical approaches (sterile)

Two years after surgery, the patient is cancer-free and has good mandibular range of motion.

### *7.3.4 Case #4 Delayed Secondary TMJ TJR Reconstruction (Fig. 7.4)*

This is the case of a 52-year-old female with a history of a large left mandibular vascular malformation who had undergone a left mandibular resection and iliac crest bone graft reconstruction with preservation of her condyle 15 years prior to

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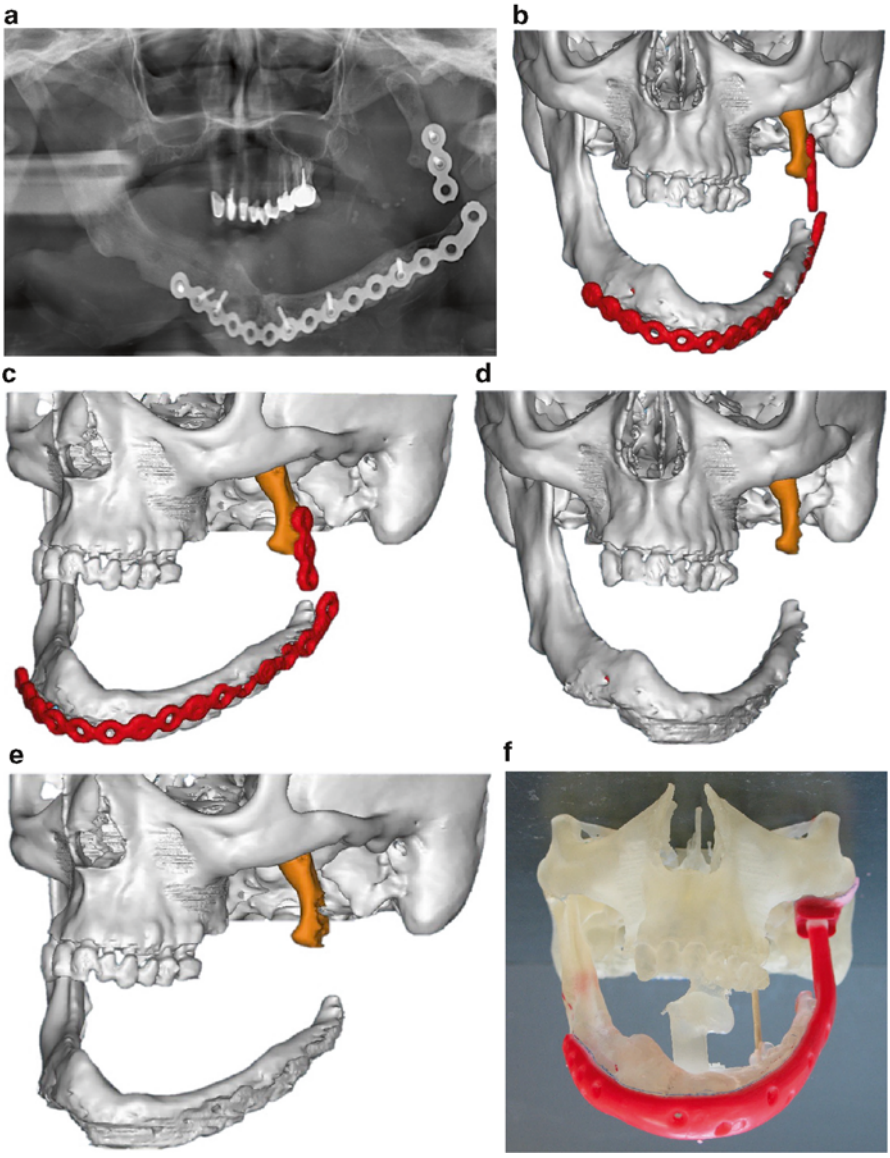


**Fig. 7.4** Case #4 delayed secondary TMJ TJR reconstruction. (**a**) Panoramic radiograph showing broken reconstruction plate. (**b, c**) 3D renderings showing the broken reconstruction plate and the residual malposition condyle. (**d, e**) Same 3D renderings after removal of the reconstruction plate digitally. (**f, g**) Prosthesis wax-up. Note the extension of the prosthesis toward the native mandibular bone. (**h, i**) Actual total TMJ custom-made prosthesis. (**j**) Intraoperative view of the previous failed hardware. Note the extended surgical access and the bone growing over the reconstruction plate. (**k**) Surgical specimen. (**l**) Intraoperative view of the prosthetic fossa (**m**) and mandibular components secured in place. (**n**) Postoperative 3D CT scan reconstruction. (**o**) Panoramic and (**p**) anteroposterior cephalometric radiographs showing good prosthesis placement (*Source*: Vega LG, Gonzalez-Garcia R, Louis PJ. Oral Maxillofac Surg Clin North Am. 2013 May;25(2):251–69)

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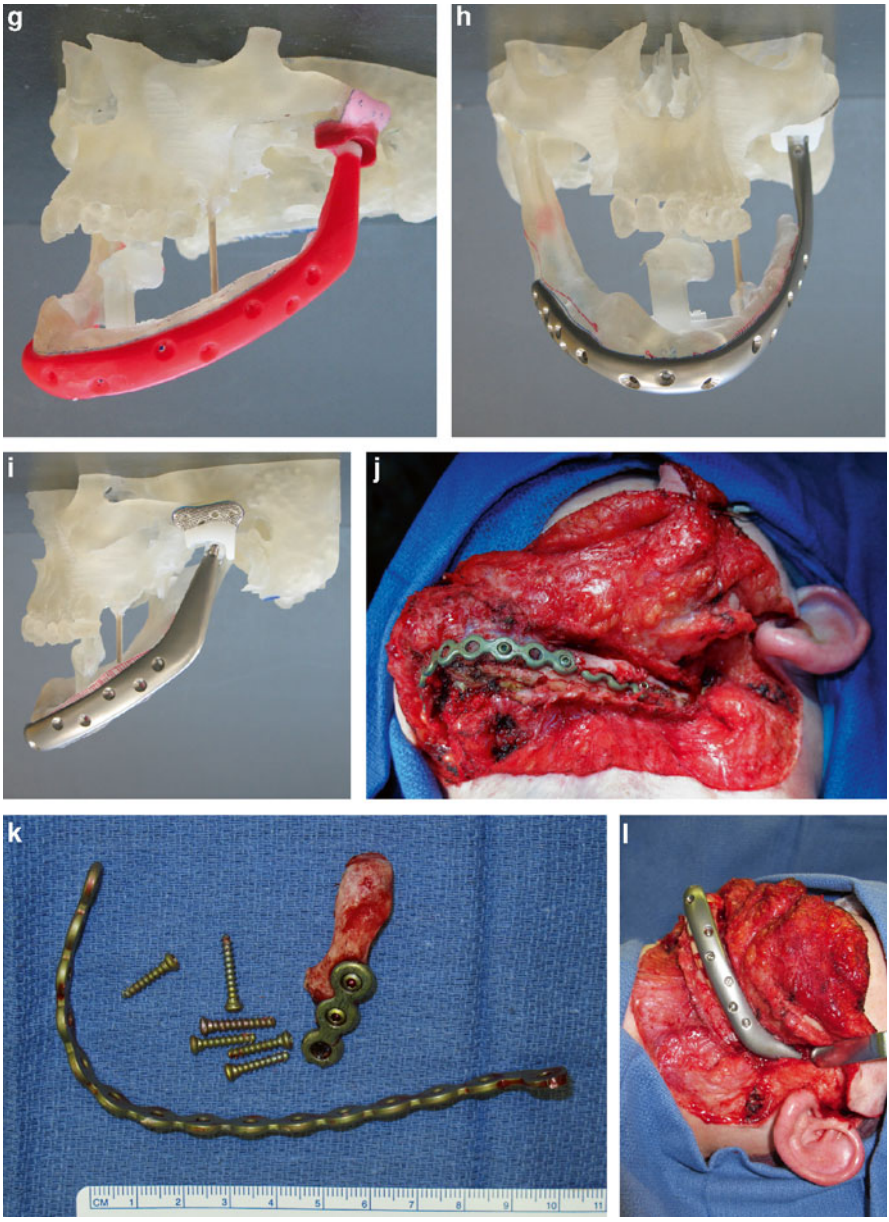


**Fig. 7.4** (continued)

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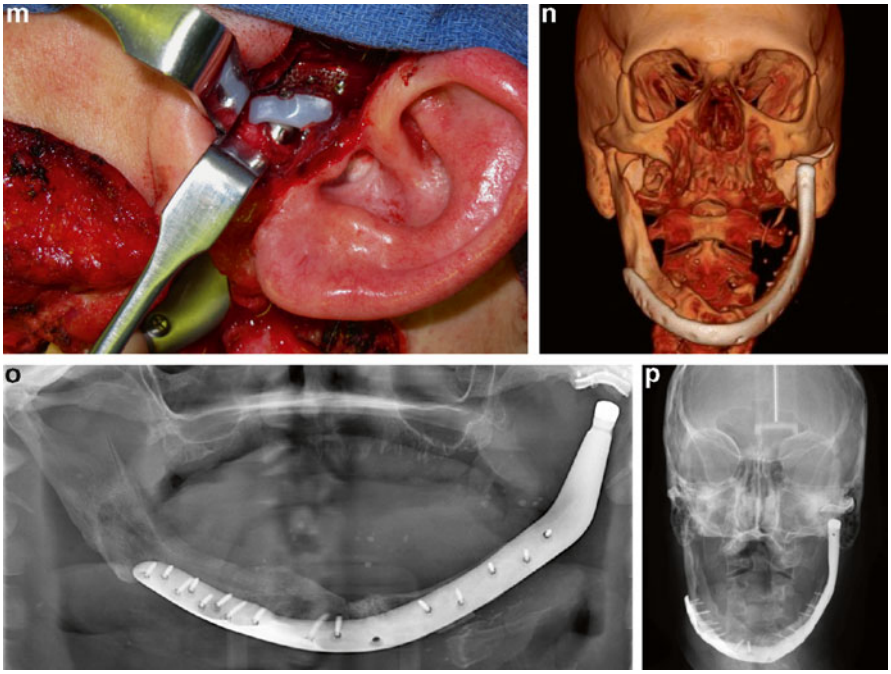


**Fig. 7.4** (continued)

presenting to the authors' institution complaining of left TMJ pain and dysfunction. Clinical and radiographic evaluation revealed the presence of a broken reconstruction plate. Due to her medical status, a microvascular reconstruction was contraindicated, so a patient-fitted TMJ TJR device was chosen to manage her large left mandibular/TMJ defect.

Her planning consisted of:

1. Review of previous medical records
2. Plain films and maxillofacial CT scan
3. Removal of reconstruction plate and condylar segment digitally
4. Fabrication of two-piece SL model (lack of mandibular dentition)
5. Model surgery (condylectomy) and establishment of the proper maxillomandibular relationship
6. Prosthesis designed to mimic the lost anatomy; additionally a hole that was placed in the neck of the prosthetic condyle to provide anchorage to avoid potential dislocation
7. Design approval and prosthesis fabrication

Her surgical sequence included:

1. Patient prepped and draped in sterile fashion
2. Extended submandibular approach with identification of the failed hardware and condylar segment

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3. Preauricular approach with debridement and removal of soft tissues
4. Hardware removal and excision of the condyle
5. Bone recontouring of the residual mandibular bone
6. Implantation of the fossa component
7. Implantation of the mandibular component
8. Irrigation and closure of the surgical approaches (sterile)

Three years after surgery, the patient is pain-free with good mandibular range of motion.

## 7.4 Summary

Reconstruction of mandibular defects involving the TMJ represents a complex challenge as the TMJ plays an important role during mastication, deglutition, phonation, and airway support. The extensive nature of these reconstructions makes them vulnerable to complications such as scarring, trismus, malocclusion, and facial asymmetry. Patient-fitted TMJ TJR devices represent an alternative to both vascularized and non-vascularized autogenous bone grafting for reconstruction of these complex defects as they can be shaped to mimic the lost anatomy and have much lower morbidity. These devices allow for stable and predictable mandibular advancement, ramus lengthening, and the ability to correct large and complex mandibular defects without the concern of relapse or fracture. Although the experience with this type of reconstructions is relatively small, early reports are very positive [3]. Further research is necessary to properly establish evidence-based clinical recommendations and management algorithms.

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# Part IV
# Complications

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# Chapter 8
# Complications Associated with TMJ TJR: Management and Prevention

**Louis G. Mercuri**

## 8.1 Introduction

As with any surgical procedure, complications can and will develop, requiring further management. Adverse outcomes may be related to, or affected by the patient's medical and/or surgical history, the surgeon's diagnosis and experience, and the patient's compliance with pre- and postoperative instructions.

The most common complications resulting in adverse outcomes associated with TMJ TJR include, but are not limited to, the following:

1. Periprosthetic joint infection
2. Heterotopic bone formation
3. Dislocation
4. Continued or increasing post-TMJ TJR pain
5. Material Hypersensitivity

## 8.2 Periprosthetic Joint Infection

The Medicare 5 % national sample administrative database documents a 1.63 and 1.55 % risk of infection within the first 2 years following primary total hip (THA) and knee arthroplasty (TKA), with an additional risk between 2 and 10 years of 0.59 and 0.46 %, respectively [1, 2]. Further studies have suggested that both the incidence and prevalence of periprosthetic joint infection (PJI) are increasing with time, with the overall infection burden expected to rise to > 6 % in the coming years [3].

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1. Presence of a sinus tract communicating with the prosthesis
2. A pathogen isolated by culture from two or more separate tissue or fluid samples obtained from the affected prosthetic joint
3. Four of the following six criteria:
    a. Elevation of serum ESR and serum CRP concentrations
    b. Elevated synovial white blood cell count
    c. Elevated synovial polymorphonuclear percentage
    d. Presence of purulence in the affected joint
    e. Isolation of a microorganism in one culture of periprosthetic tissue or fluid
    f. More than five neutrophils per high-power field in five high-power fields observed in a sample for histological analysis of periprosthetic tissue at x400 magnification

**Fig. 8.1** Musculoskeletal Infection Society (MSIS) workgroup definition for periprosthetic joint infection. Key: ESR, erythrocyte sedimentation rate; CRP, C-reactive protein

A retrospective survey of 2476 temporomandibular joint total alloplastic joint replacement (TMJ TJR) cases involving 3368 joints reported 51 (1.51 %) PJI cases occurring in that cohort over a mean of 6 months postoperatively (range 2 weeks–12 years) [4].

Despite these statistics revealing the incidence of PJI after total joint replacement to be uncommon, the clinical, psychological, and economic consequences of this complication can be substantial. Therefore, the development of management algorithms based on early diagnostic testing has been the subject of continued exploration in the orthopedic literature.

In 2011, a Musculoskeletal Infection Society (MSIS) workgroup evaluated the available literature and proposed a definition for PJI that could be universally adopted by all [5] (Fig. 8.1).

Periprosthetic joint infections present characteristic signs that can be divided into acute manifestations (severe pain, high fever, toxemia, heat, rubor, and surgical wound discharges) and chronic manifestations (progressive pain, skin fistulae, and drainage of purulent secretions, without fever). The clinical presentation depends on the virulence of the etiological organism, the nature of the infected tissue, the infection acquisition route, and the duration of disease evolution [6].

The classification system most widely used today in orthopedics is the one proposed by Fitzgerald Jr et al. [7]. This classification defines the time at which contamination occurs and establishes the likely etiological agent involved and the best management strategy (Fig. 8.2).

Early and delayed infections are thought to be due to organisms introduced at the time of surgery, whereas late infections are more likely to have a hematogenous etiology. Infecting organisms form micro-colonies on the prosthesis surface, and these elaborate exo-polysaccharides that coalesce, forming a biofilm. Once formed, organisms within the biofilm are protected from host immune responses and may display reduced susceptibility to antibiotics as a result of changes in metabolic processes and poor diffusion [8].

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**1. Acute postoperative infections occurring within 3 months of surgery**
The etiological agents are generally of hospital origin, especially *Staphylococcus aureus* and *Staphylococcus epidermidis*
**2. Late deep infections that appear between 3 months and 2 years after surgery**
The etiological agents are considered to be of nosocomial origin, since the contamination probably occurred during prosthesis implantation, and generally consist of bacteria from the normal skin flora, such as *S. epidermidis*
**3. Late hematological infections that occur more than 2 years after surgery**
The etiological agents are of community origin and are determined by the apparent source of bacteria: anaerobic bacteria, while cellulitis and skin abscesses are associated with *S. aureus* or streptococci or enterobacteria originating from the gastrointestinal and genitourinary tracts. Dental infections are associated with bacteremia due to viridans streptococci

**Fig. 8.2** Classification of orthopedic periprosthetic joint infections

**A. Patient-related risk factors for infection include:**
1. Previous revision arthroplasty or infection associated with a prosthetic joint
2. Tobacco abuse
3. Obesity
4. Rheumatoid arthritis
5. Concurrent neoplasm
6. Immunosuppression and diabetes mellitus

**B. Surgical-related risk factors include:**
1. Simultaneous bilateral arthroplasty
2. Operative time longer than 160 min
3. Allogeneic blood transfusion

**C. Postoperative-related risk factors include:**
1. Wound healing complications (e.g., superficial infection, hematoma, delayed healing, wound necrosis, and dehiscence)
2. Atrial fibrillation, myocardial infarction, urinary tract infection
3. Prolonged hospital stay
4. *Staphylococcus aureus* bacteremia

**Fig. 8.3** Patient, surgical, and postoperative related risk factors in orthopedic periprosthetic joint infections

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Patient, surgical, and postoperative related risk factors in orthopedic PJI have been spelled out and must be considered [9–17] (Fig. 8.3).

To date, there is no diagnostic test that produces "absolute" accuracy, and due to this lack of a "gold standard" for the diagnosis of PJI, diverse and sometimes conflicting criteria have been proposed [18].

### *8.2.1 TMJ TJR PJI Diagnosis and Management Algorithm*

Based on the review of the TMJ TJR PJI literature [4, 8, 18–20] and the American Academy of Orthopedic Surgeons' (AAOS) Clinical Practice Guideline for Diagnosis of Periprosthetic Joint Infections [21], practical diagnostic and management algorithms were developed for early and delayed TMJ TJR PJIs (Fig. 8.4).

#### 8.2.1.1 Early TMJ TJR PJI

As with any diagnosis, the clinical history and physical examination are important. A suspected PJI occurring within days or < 3 weeks after TMJ TJR typically manifests as increasing pain, low-grade fever, swelling and erythema at the pre-auricular and/or retromandibular incisions, and drainage from either or both surgical sites [19].

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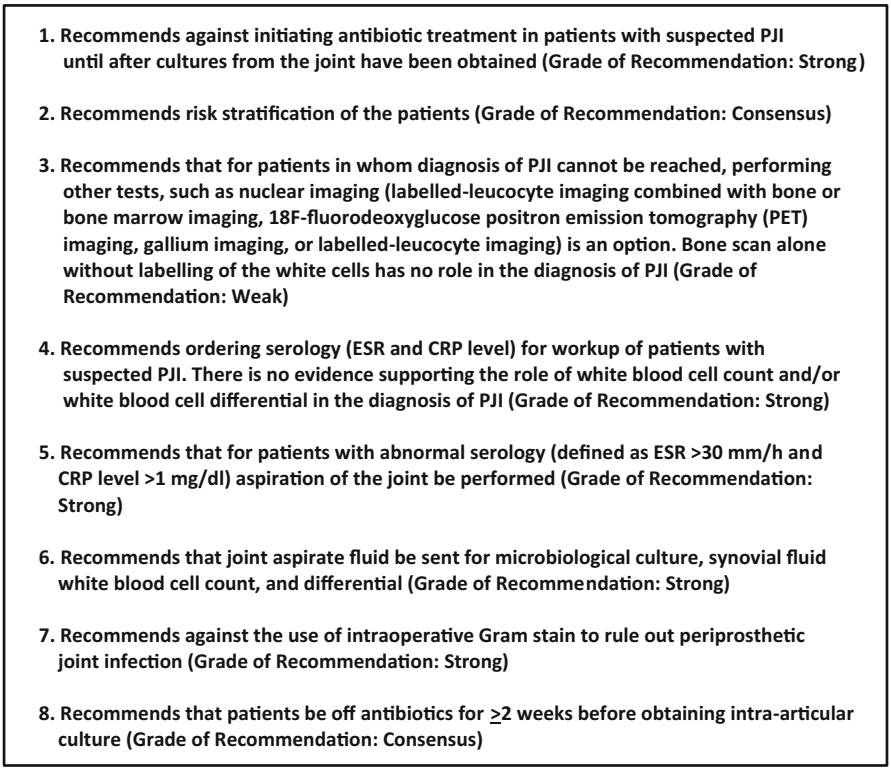


**Fig. 8.4** AAOS clinical practice guideline for the diagnosis of periprosthetic joint infection. Key: AAOS, American Academy of Orthopedic Surgeons; PJI, periprosthetic joint infection; ESR, erythrocyte sedimentation rate; CRP, C-reactive protein

| | Early PJI |
|---|---|
| History | *Days to < 3 weeks* |
| Clinical | *Pain, swelling, redness, drainage* |
| Serology | *ESR and CRP* ↑ |
| Synovial Fluid WBC | *+* |
| Synovial Fluid Culture | *+* |
| Imaging (Plain, CT) | *Stable components* |
| Nuclear Medicine | *+* |
| Management | *Incision and Drainage, debridement, antibiotics#* |

**Fig. 8.5** Diagnosis and management of early periprosthetic TMJ TJR infections. Key: ESR = Erythrocyte Sedimentation Rate (>30 mm/h); CRP = C-Reactive Protein (>10 mg/L); #*Wolford LM*, *Rodrigues DB*, *McPhillips A*: *Management of the Infected Temporomandibular Joint Total Joint Prosthesis. J Oral Maxillofac Surg 2010*:*68*: *2810–2823*

Serology (ESR and CRP) will be elevated as will the peripheral WBC. There is no need to aspirate the joint, but aspiration wound cultures should be taken before antibiotics are employed to assure proper identification of the etiologic organisms.

In early PJI cases, plain film and/or CT imaging should reveal stable component fixation. Should there be any evidence of component or fixation loosening, these issues must be addressed along with the PJI for there to be any resolution. MRI, ultrasound, and nuclear medicine scans are unnecessary in the diagnosis of an early TMJ TJR PJI (Fig. 8.5).

#### 8.2.1.2 Delayed TMJ TJR PJI

Patients presenting >3 weeks or longer after TMJ TJR with complaints of increasing pain and diffuse swelling with no evidence of localized erythema, no fever, and no drainage present a difficult diagnostic dilemma unless there is clinical evidence of a draining skin or auditory canal fistula directly communicating with the device. This sign is pathopneumonic of a TMJ TJR PJI and requires delayed TMJ TJR PJI management [8, 18, 20] (Fig. 8.6).

Intrinsic causes for these signs and symptoms should be ruled out (Fig. 8.7) by imaging (plain film or CT). Since ESR and CRP can be equivocal in Late PJI, their value as diagnostic tests is diminished in a suspected delayed TMJ TJR PJI.

Sterile aspiration of the TMJ TJR articulation to obtain fluid for WBC analysis (>1100–4000 cells/μL; 64–68 % polymorphonucleocytes) and culture is indicated. Labeled-leukocyte imaging (e.g., leukocytes labeled with indium-111) combined with bone marrow imaging with the use of technetium-99 m-labeled sulfur colloid is more accurate than technetium-99 alone, combined bone and gallium-67 imaging, or labeled-leukocyte and bone imaging, when compared head to head, and it is considered the imaging test of choice when imaging is utilized [22].

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| | Late PJI |
|---|---|
| History | *> 3 weeks to years* |
| Clinical | *Pain, swelling, ± fistula* |
| Serology | *ESR and CRP ±* |
| Synovial Fluid WBC | *+* |
| Synovial Fluid Culture | *+* |
| Imaging (Plain, CT) | *Unstable component(s)* |
| Nuclear Medicine | *+* |
| Management | *2-stage removal/replacement** |

**Fig. 8.6** Diagnosis and management of late periprosthetic TMJ TJR infections. Key: ESR = Erythrocyte Sedimentation Rate (>30 mm/h); CRP = C-Reactive Protein (>10 mg/L); **Mercuri LG*: *Avoiding and Managing Temporomandibular Joint Total Joint Replacement Surgical Site Infections. J Oral Maxillofac Surg 2012:70: 2280–2289*

| |
|---|
| Infection |
| Heterotopic bone formation |
| Aseptic component or screw loosening |
| Dislocation |
| Neuroma formation |
| Material sensitivity |
| Synovial entrapment syndrome |
| Component or screw fracture |
| Osteolysis |

**Fig. 8.7** Intrinsic causes of TMJ TJR increasing pain and swelling, and decreasing mandibular function

Because there is no single PJI diagnostic test that offers perfect sensitivity and specificity, this field continues to evolve. New assays will become available in the future that will be incorporated into any diagnostic algorithm as they are developed and subjected to clinical testing.

## 8.3 Preventive Measures

The risk of infection after TMJ TJR can be decreased with appropriate consideration to preoperative patient risk assessment; properly timed antibiotic prophylaxis; and intraoperative, postoperative, and post-discharge attention to detail [20].

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### *8.3.1 Preoperative Patient Assessment*

There are a number of endogenous (patient-related) and exogenous (process-/procedure-related) variables that affect a patient's risk for the development of an infection. Some endogenous factors cannot be changed, such as age, gender, and genetic factors [23]. However, a number of exogenous factors may exist that can be improved to decrease the potential for the development of an infection and enhance TMJ TJR outcomes.

#### 8.3.1.1 Nutrition

TMJ TJR surgical candidates, such as those with ankylosis or other pathologic conditions that prevent them from maintaining a proper diet over an extended period of time, may require nutritional and hematologic evaluation and intervention before TMJ TJR. Serum albumin level (normal range, 3.4–5.4 g/dL) is the surrogate marker most commonly used to classify nutritional status.

#### 8.3.1.2 Systemic Disease Control

As with the implantation of any device into medically compromised patients, it is essential that any risk-related systemic pathology be controlled before surgery. Two common systemic diseases that can negatively affect TMJ TJR, diabetes mellitus and rheumatoid arthritis, illustrate the value and importance of preoperative risk assessment and control.

Candidates for TMJ TJR surgery with a personal or family history or symptoms consistent with diabetes mellitus should be considered for fasting serum glucose (FSG) as well as hemoglobin A1c screening to evaluate the presence of preexisting diabetes.

When TJR infections arise in rheumatoid arthritis patients, two important modifiable risk factors have been identified—recent primary TJR or revision in the previous year, especially if tumor necrosis factor (TNF-α) blockers are not withdrawn, and steroid intake reduced before surgery [24, 25]. Therefore, it may be prudent to consider carrying out TMJ TJR before the introduction of TNF blockers. In patients already taking TNF-α blockers, withdrawal during the perioperative period is advocated. Furthermore, steroid intake in patients taking TNF-α blockers should be reduced as low as possible before TMJ TJR [25]. Dosage and/or medication modifications should be made in consultation with the patient's rheumatologist. The author has found most rheumatologists in agreement with cessation of TNF blockers for 2 weeks preoperatively and 1 week postoperatively [20].

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#### 8.3.1.3 Smoking

Cigarette smoking is associated with inhibited wound healing and decreased circulation to the skin due to microvascular obstruction from platelet aggregation and increased nonfunctioning hemoglobin. In addition, smoking has been found to compromise the immune system and respiratory system [26]. Postoperative healing complications occur more often in smokers than in nonsmokers and in former smokers than in those who never smoked. Perioperative smoking cessation intervention reduces infection but no other healing complications [27].

Smoking should be discontinued 6–8 weeks before surgery. In a randomized study, participation in a preoperative smoking cessation program was found to reduce postoperative complication rates. No wound-related complications occurred in the patients who stopped smoking before surgery [28, 29]. In an experimental study, use of transdermal nicotine patches did not impair wound healing [30].

Cigarette smoking may also be one of the preexisting exogenous factors amenable to intervention, especially with the relatively new smoking cessation supports now available, such as the nicotine patch or bupropion hydrochloride. Patients should also adhere to nutrition and physical status guidelines, including the intake of vitamins such as A, B, C, D, E, and K, as well as supplements of zinc, manganese, magnesium, copper, and iron [31].

### *8.3.2 Preexisting Remote and Local Site Infections*

Infections at a site remote from the TJR have been linked to a three- to fivefold increase in TJR infection rates [32, 33]. The most common sources of blood-borne infection are the skin and urinary and respiratory tracts. Therefore, any remote infections should be identified and managed before TMJ TJR.

It is not uncommon for multiple dental extractions to be required in order for oral infections to be eliminated preoperatively. Although the underlying evidence is weak, it is advisable to perform dental extractions before TMJ TJR [34, 35].

*Propionibacterium acnes* (*P. acnes*) has been increasingly recognized as an important agent in orthopedic shoulder device infections. *P. acnes* is a Gram-positive bacterium that forms part of the normal flora of the skin, oral cavity, large intestine, the conjunctiva, and the external ear canal. Although primarily recognized for its role in acne, *P. acnes* is an opportunistic and difficult to culture pathogen that can cause a range of postoperative and device-related infections [36].

Therefore, any history of severe acne or prior TMJ TJR infection where the pathogen was not clearly identified should be pursued by a dermatology consultation before undertaking TMJ TJR.

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## 8.4 Preoperative Patient Preparation

### *8.4.1 Skin*

The primary source of infection for most TJR infections is the patient's own endogenous microorganisms. All patients are colonized with bacteria, fungi, and viruses—up to 3 million organisms per square centimeter of skin [37]. All surgical wounds will be contaminated with bacteria during surgery, but only a small percentage become infected. This is because most patients' host defenses are capable of controlling and eliminating the offending organisms when the wound inoculum is small, the bacterial contaminants are not overwhelmingly virulent, the wound microenvironment is healthy, and the host defenses are intact.

Despite the intervention, the patient's skin will never be sterile, but a number of strategies can be used to reduce bio-burden. A Cochrane Database Review provided no clear evidence of benefit of preoperative showering or bathing with chlorhexidine over other wash products to reduce SSI. However, the benefit of day-of-surgery showering or bathing in an effort to reduce the incidence of nosocomial infection was demonstrated [38].

Preincision skin preparation is of critical importance, ensuring not only that the antibacterial solution used has broad-spectrum properties but also that the product is properly applied. Additional strategies used to reduce bacterial migration into the surgical incision include the use of antiseptic-impregnated adhesive drapes and/or novel cyanoacrylate-based skin sealants that can be applied over the skin preparation site to immobilize residual skin flora, including those imbedded in hair follicles [39, 40].

### *8.4.2 Preincision Antibiotic Prophylaxis*

Systemic intravenous antibiotic prophylaxis reduces the risk of postoperative infections. Cephalosporins are widely used, based on their good efficacy against staphylococcal species and uropathogens. Vancomycin is indicated in high-risk patients carrying methicillin-resistant *Staphylococcus aureus*. If the patient has an allergy to β-lactam antibiotics, clindamycin or vancomycin can be used [41].

The association between time of administration of the antibiotic and infection rate can be presented as a U-shaped curve, with a higher risk of infection both before and after the optimal time frame of administration [42].

In a prospective study in 364 consecutive total knee replacement patients, Levent et al. examined the significance of 5 variables commonly associated with the potential for infection after TJR: (1) classic risk factors (e.g., diabetes and rheumatoid disease); (2) incomplete preoperative skin preparation; (3) methicillin-resistant S aureus-positive patient; (4) perioperative antibiotic use; and (5) duration of surgery.

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After a 1-year median follow-up, they report a 1.4 % infection rate; of the 5 variables, only perioperative antibiotic use and duration of surgery showed significance [43].

Rosenberg et al. reported that the delivery of antibiotic prophylaxis within 1 h before surgical incision is important in decreasing the incidence of SSI in orthopedic TJR. Therefore, it is recommended that verification of antibiotic administration be part of the pre-incision "time-out" protocol to ensure compliance with appropriate timing of prophylactic antibiotic administration in all TJR cases [44].

### *8.4.3 Anesthesia*

Contamination of the surgical site and/or displacement of the anesthetic naso-endotracheal tube (NET) during TMJ TJR can be avoided by suturing the NET to the nasal septum. The NET, as well as associated tubing and equipment, can then be directed caudad away from the surgical field. This affords better access to the surgical site, as well as easier head movement in bilateral cases, and it decreases the potential for NET contamination of the sterile field and/or displacement [40] (Fig. 5.15).

### *8.4.4 Eyes*

After the patient is anesthetized and the airway secured, the eyes should be lubricated and protected to prevent corneal injury, conjunctivitis from blood/irrigation, or contamination of the surgical field from tearing of the eyes [40] (Fig. 5.16).

### *8.4.5 Hair*

The patient should be directed to thoroughly wash and rinse his or her hair the night before surgery with a mild shampoo and avoid the use of hairspray or styling gels the day of surgery. Hair in the surgical incision area should be carefully arranged and/or parted to facilitate the skin incisions. Hair trimming or removal should be performed with clippers, not razors, immediately before surgery. Care should be taken to avoid cutting or nicking of the skin in the area of the surgical incision so as not to introduce skin bacteria [40].

After shearing the hair above the ear, the remaining hair should be drawn up toward the crown of the head, away from the planned incision sites. Foam tape can be used to wrap the head circumferentially (forehead–above the ear– occiput) so that the hair will be kept out of the surgical field, under the tape, and off the skin over the planned incision sites [40] (Fig. 5.17).

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### 8.4.6 Ear

The auditory canal and tympanic membrane should be inspected with an otoscope to ensure that there is no preoperative infection or pathology and the results documented in the operative report. The external auditory canal should be occluded to prevent wound contamination during surgery from the egress of bacterial flora and/or accumulation of irrigation fluid and/or blood intraoperatively [40].

After careful cleansing of the auditory canal with a gentle bactericidal solution, a cotton pledget moistened with sterile mineral oil provides one among many occlusive options. Care must be taken to avoid pushing whatever occlusive material is chosen too deeply, causing injury to the auditory canal and/or tympanic membrane [40].

### 8.4.7 Oral Cavity

Any intraoral procedures such as application of intermaxillary fixation appliances (arch bars, Ivy loops, etc.) should be completed before skin preparation and final sterile draping. All contaminated intraoral instruments and power equipment must remain separate from the sterile instruments to be used in the surgical field [40] (Fig. 5.18).

## 8.5 Intraoperative Considerations

### 8.5.1 Incisions

Not only is the anatomic placement of the incision for access to the surgical site important, but also the incision must be large enough to expeditiously execute the procedure. Incisions that are small, though potentially less conspicuous, may require more forceful retraction, resulting in excessive pressure on the wound skin edges, resulting in ischemia. This can lead to poor healing, increasing the potential for infection, or result in excessive scarring.

### 8.5.2 Saliva

Parotid gland tissue is typically encountered during the surgery to implant a TMJ TJR device. Care should be observed during dissection, retraction, instrumentation, and the use of power equipment to avoid injury to parotid tissue. Injury to this tissue can result in the contamination of the surrounding host bone, tissue, and device

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components with potentially bacteria-laden saliva, resulting in an infection. Therefore, although there are advocates of the retromandibular/trans-parotid approach to the mandibular ramus in trauma surgery [45], it is recommended that the parotid capsule remain intact during TMJ TJR [40].

### *8.5.3 Device Contamination*

Direct contamination of the device components before implantation as a result of improper handling in the operating room environment or indirect contamination from the skin, ear flora, or saliva during multiple "try ins" of templates and/or the device components themselves can lead to an infection.

Mercuri and Psutka state that it appears prudent to soak the components and/or perform irrigation of the implant components with antibiotic or antibacterial solution intraoperatively [4]. Furthermore, the work of Levent et al. demonstrated that the time required to implant a TMJ TJR device is a statistically significant determinant of TJR infection. Therefore, the experience of the operator and the use of TMJ TJR systems requiring a shorter operating time are considered important variables [43].

### *8.5.4 Hemostasis and Irrigation*

Intraoperatively and before wound closure, the surgeon must ensure that adequate hemostasis has been achieved to prevent hematoma formation. Hematomas have been implicated not only in the development of infections [46] but also in the need for revision surgery after TJR [47].

Copious irrigation with saline or antibiotic solution to remove any clotted blood, soft tissue, and bony fragments before wound closure is extremely important in decreasing the potential for infection. The use of drains can be a potential source of contamination [40].

Irrigation with an antibiotic solution (neomycin and polymyxin B [4, 19] or vancomycin [4]) before and after implantation of the device components may decrease the potential for local contamination, although no definitive studies to prove or disprove this have been published in the TMJ TJR literature to date.

## 8.6 Immediate Postoperative Considerations

### *8.6.1 Auditory Canal*

After precise and careful wound closure, the auditory canal and tympanic membrane should be reinspected with a speculum to ensure that there was no intraoperative accumulation of irrigation fluid or blood, or surgical incursion into the auditory

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canal or tympanic membrane. This inspection should be documented in the operative notes [40].

Blood clots should be removed with gentle, warm irrigation and careful suction. Instillation of antibiotic/steroid otic drops and occlusion of the external auditory canal with a cotton pledget is recommended to decrease the potential for the development of infection and/or inflammation of the auditory canal and/or tympanic membrane [40].

Intraoperative incursion into the auditory canal can occur as a result of pathologic or iatrogenic involvement. Pathology, like ankylosis, can directly involve both the cartilaginous and bony auditory canal. Therefore, care must be taken when performing gap arthroplasty in such cases [40].

Preoperative imaging awareness, as well as intraoperative careful, controlled manipulation of instruments and power equipment, is critical. However, sometimes, because of obscure involvement of the pathology with the auditory canal, perforation or tearing can occur. Should either occur or be discovered, consultation with an otolaryngologist is advised to determine the best management options [40] (Chapter 5).

### *8.6.2 Dressing*

A pressure dressing should be applied for a minimum of 8 to 12 h to aid in minor hemostasis and reduce postoperative edema.

### *8.6.3 Postimplantation Antibiotic Coverage*

There appears to be little consensus on the need for postimplantation antibiotics in orthopedic TJR [48]. Until similar studies are available for TMJ TJR, an antibiotic that covers the spectrum of potential skin, ear, and saliva contaminants (i.e., clindamycin and cephradine) is recommended for 7–10 days postoperatively, especially for the high-risk patient [4].

As for prophylactic antibiotic coverage prior to dental procedures in patients with orthopedic TJR devices, the AAOS and ADA developed three related recommendations: (1) The practitioner might consider discontinuing the practice of routinely prescribing prophylactic antibiotics for patients with hip and knee prosthetic joint implants undergoing dental procedures; (2) The expert panel was unable to recommend for or against the use of topical oral antimicrobials in patients with prosthetic joint implants or other orthopedic implants undergoing dental procedures based on the available data; (3) In the absence of reliable evidence linking poor oral health to prosthetic joint infection, it is the opinion of the work group that patients with prosthetic joint implants or other orthopedic implants maintain appropriate oral hygiene [35].

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However, postoperative antibiotic prophylaxis before invasive dental, urologic, gastrointestinal, and aero-digestive procedures might be important after TMJ TJR because the tips of the condylar component ramus fixation screws lie in the pterygomandibular space and can be contaminated during inferior alveolar nerve anesthesia administration techniques [4].

### *8.6.4 Nosocomial Infections*

Although nosocomial infections are difficult to predict and manage, the duration of hospitalization after TMJ TJR should be minimized to reduce the risk of colonization of the patient's skin with hospital-acquired organisms. Meticulous wound care and personal hygiene (hand washing) by both the surgeon and patient both during hospitalization and after discharge are absolutely essential [40].

### *8.6.5 Discharge Considerations and Information*

The risk of infection continues even after the patient leaves the hospital. Surgeons should educate the patient and relatives regarding proper incision care, personal hygiene, how to recognize early signs of an infection, and the importance of reporting symptoms to their surgeons as soon as they arise. Providing preprinted instructional information and answers to frequently asked questions should be considered [40].

The patients should be directed that when washing their hair postoperatively, they should have someone help them so that their head is tilted backward as in a salon sink so as to avoid soaking the incision sites. The incision sites should then be patted dry and a Neosporin (Johnson & Johnson, New Brunswick, NJ) ointment applied [40].

## 8.7 Heterotopic Bone Formation

The development of heterotopic bone around any TMJ TJR device will limit mandibular function and cause pain. Heterotopic bone formation is the presence of bone in the soft tissue surrounding a TJR where bone normally does not exist, leading to decreased joint mobility and pain. History and imaging are used to distinguish it from other diagnostic possibilities. As management or prophylaxis, either a nonsteroidal anti-inflammatory drug, such as indomethacin, or a diphosphonate, such as ethane-1-hydroxy-1, 1-diphosphate [49], or local radiation therapy [50] has been recommended.

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Surgical removal of the heterotopic bone is used to preserve joint mobility, but heterotopic bone formation is likely to recur and possibly progress. Therefore, it is recommended that an autogenous fat graft be packed around the articulation of TMJ replacement devices to decrease potential recurrence [51, 52].

It is not only essential to thoroughly irrigate the bone debris out of the ostectomy site, but also assure good hemostasis as the presence of a blood clot or large hematoma can result in the development of a re-ankylosis. One of the advantages of the aforementioned autogenous fat graft besides filling the dead space around the device articulating components is that fat has a hemostatic effect on surrounding tissues, therefore decreasing the potential for development of a hematoma or clot [52].

Heterotopic bone can form along the anterior, lateral, posterior, or medial aspect of the articulating components. Confirm with axial and coronal CT imaging the location of the heterotopic bone around the device components. Determine the exact location of that bone in relation to the device components.

Heterotopic bone isolated mainly to the anterior, lateral, or posterior aspect of the device articulation with no medial extension (Fig. 8.8) typically can be addressed and can often be removed through a standard pre-auricular incision. Once isolated, the heterotopic bone can be sectioned away from the components with a rotary instrument utilizing a 701 bur with copious irrigation. Extreme care must be taken not to damage either the bearing surfaces of the TMJ TJR components while doing

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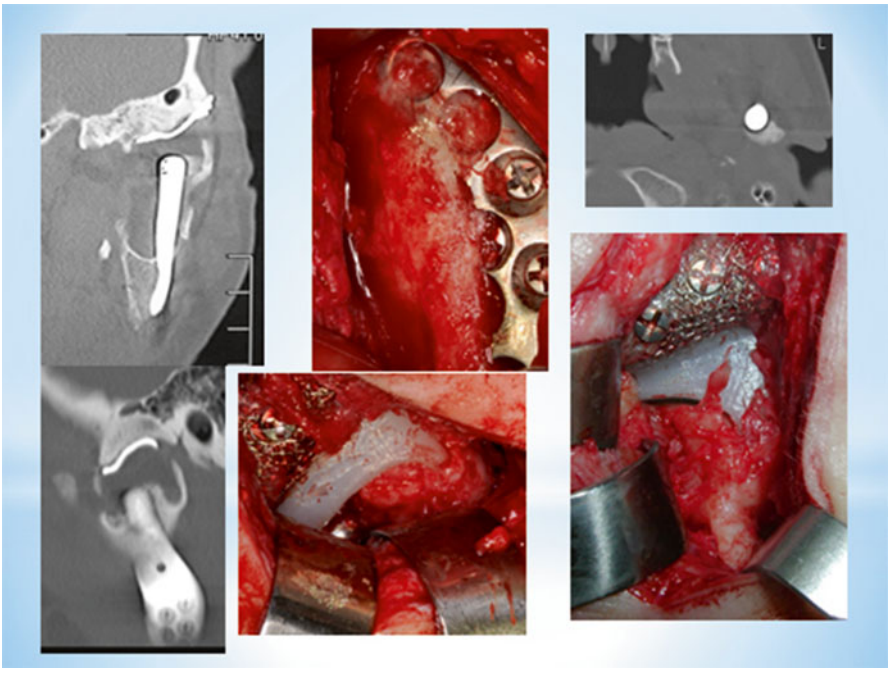

**Fig. 8.8** Heterotopic bone isolated mainly to the anterior, lateral, or posterior aspect of the device articulation with no medial extension

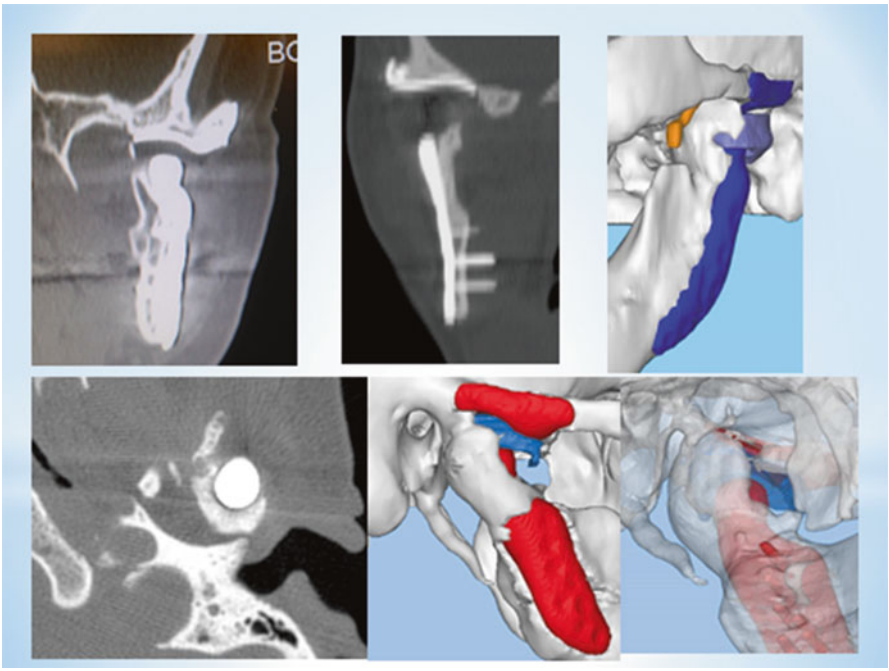

**Fig. 8.9** Heterotopic bone medial to the TMJ TJR bearing surfaces

this! Protecting those components with careful retraction is essential. The sectioning can be accomplished by NOT completing the bony cut, "snapping off" the heterotopic bone with an instrument, and removing it as one would when separating the crown from the roots when sectioning a tooth during impaction surgery. A bone file can be used to remove the remaining un-sectioned attached bone fragment.

If the heterotopic bone lies medial to the TMJ TJR bearing surfaces, but BELOW the medial aspect of the fossa component, access will require both a pre-auricular and a retromandibular approach since the ramal component will have to be removed to gain proper access to the heterotopic bone (Fig. 8.9). When ramus component requires removal, keep an account of the screw lengths for each hole, unless you have the original screw length recommendations from TMJ Concepts. Even after the fixation screws have been removed, the ramus component is attached medially to the lateral mandible. After removing the heterotopic bone, the ramus component can be replaced using the TMJ TJR system "rescue" screws to re-fixate the ramus component. Occasionally, bone will have grown over the ramus component and filled in the screw access sites. A screw removal system with a "back out" function that makes this an easier task should be available for all such cases.

If the heterotopic bone lies medial to the articulation, and ABOVE the medial aspect of the fossa component, this will require both a pre-auricular and a retromandibular approach, since both the fossa and the ramal components will have to be

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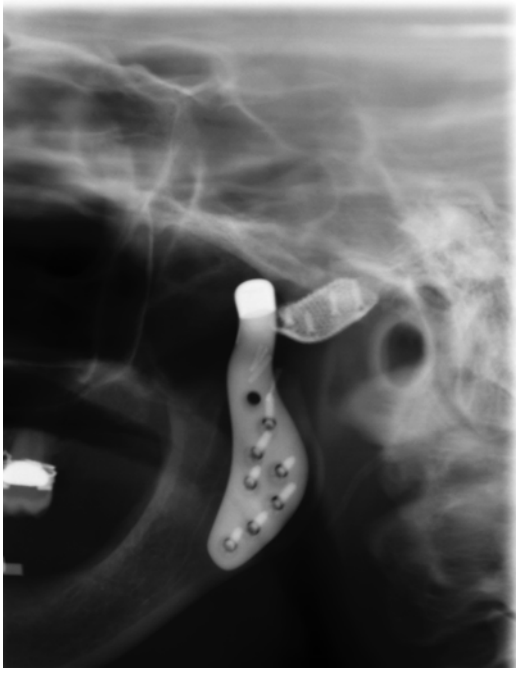

**Fig. 8.10** Left TMJ TJR dislocation on orthopantomogram

removed to gain proper access to the heterotopic bone. When both components of a custom TMJ TJR system must be removed in order to address the heterotopic bone in this scenario, both components of the custom device must be remade. Therefore, placing a spacer (Fig. 5.13) applying MMF, ordering a new protocol CT scan and new TMJ TJR components designed and manufactured for later implantation, are all required.

In all of these techniques, copious irrigation followed by placement of an autogenous fat graft should precede closure. Active physical therapy daily with a jaw exercising device should be maintained for a minimum of 6 months.

## 8.8 Dislocation

Diagnosis of anterior condylar component dislocation is typically clinically evident and can be demonstrated on imaging (Fig. 8.10). The patient most likely to anterior dislocate in the immediate post-TMJ TJR period is the one who has undergone either unilateral or bilateral coronoidectomy. During TMJ TJR surgery, all of the supporting mandibular masticatory musculature, except the medial pterygoid, is either stripped or sacrificed (lateral pterygoid) during the procedure. This allows the

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action of the suprahyoid musculature to depress the mandible unchecked and dislocate especially when coronoidectomies are part of the procedure.

While routine coronoidectomy is not recommended, there are cases where coronoid hyperplasia or coronoid ankylosis requires removal of the coronoid process to obtain maximum mandibular range of motion [53]. In such cases, it is recommended that light intermaxillary elastic traction be placed and maintained for 1 week to allow the development of enough periprosthetic fibrous tissue formation which will prevent spontaneous dislocation.

Immediate post-TMJ TJR dislocation can easily be managed by standard mandibular dislocation manual reduction and placement of light intermaxillary elastic traction or a Barton-type dressing for 1 week. Late dislocation may require sedation and/or general anesthetic in combination with manual reduction, or possible surgical intervention to achieve relocation.

Posterior condylar component dislocation is rare and typically only seen in patients where a stock TMJ TJR device without a posterior stop has been utilized in combination with orthognathic surgery (Fig. 5.14). Resolution of this situation usually involves removal of the stock device and replacement with a custom TMJ TJR device.

## 8.9 Continued or Increasing Post-TMJ TJR Pain

The definition of pain endorsed by the International Association for the Study of Pain is: "Pain is an unpleasant and emotional experience associated with actual or potential tissue damage, or described in terms of such damage." Acute pain almost always originates from nociception in somatic or visceral tissues (intrinsic); however, not every pain sensation originates from nociception (extrinsic) [54]. There are both intrinsic and extrinsic causes for pain in patients after TMJ TJR (Fig. 8.11). The surgeon must rule each out in a systematic manner and manage the cause appropriately.

| *Intrinsic etiology* | *Extrinsic etiology* |
|---|---|
| Infection | Prior misdiagnosis |
| Heterotopic bone formation | Chronic centrally mediated pain |
| Dislocation | Persistent myofascial/muscular pain |
| Material sensitivity | Complex regional pain syndrome I |
| Aseptic component or screw loosening | Neurologic injury (CPRS II) |
| Component or screw fracture | Temporalis tendonitis |
| Osteolysis | Coronoid impingement |
| Neuroma formation | Frey's neuralgia |
| Synovial entrapment syndrome | Integrin formation |

**Fig. 8.11** Intrinsic and extrinsic causes for post TMJ TJR pain

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Persistent or chronic post-TJR pain can be a significant clinical and economic problem. While the estimated mean incidence of post surgical pain is high and varies between 10 and 50 %, in the surgical literature it is most often related to procedure-specific conditions such as thoracotomy, breast, inguinal hernia surgery, and amputations [55].

Liu et al. undertook a multicenter, cross-sectional study of 897 patient electronic medical records to identify preoperative risk factors for acute moderate to severe pain after total hip and knee replacement at rest and with activity. Moderate to severe pain was reported by 20 % at rest and 33 % with activity. Among the significant predictors for postoperative pain at rest were: (1) Female gender; (2) Increased severity of preoperative pain in the hip or knee area; (3) Preoperative use of opioids. Predictors for postoperative pain with activity were: (1) Severity of the preoperative hip and/or knee pain; (2) Preoperative use of anticonvulsants and antidepressants; and (3) Prior previous hip/knee surgery [56].

Judge et al. state that although the majority of patients after total knee replacement (TKR) surgery have symptomatic improvement, up to 30 % reported no improvement or are worse. Therefore, they undertook a prospective study of 1991 TKR patients to identify possible predictors of outcome by administering the Oxford Knee Score questionnaire 6 months postoperatively and applying regression modeling. The strongest predictors of outcome were as follows: (1) Preoperative pain/function—those with less severe preoperative disease obtained the best outcome; (2) Diagnosis—those with rheumatoid arthritis did better than those with osteoarthritis; deprivation—those from poorer areas had worse outcomes; and (3) Anxiety/depression—those with anxiety/depression were associated with poorer pain symptom relief. Older patients and women had poorer outcomes [57].

These results confirm previous reports that patients with better preoperative pain and functional status have better postoperative pain/function outcomes [58–62]. These data are similar to those found in TMJ TJR outcome studies [63–68].

The orthopedic literature also reveals that the greater the number of preoperative comorbidities, the poorer the outcomes [58, 60, 61, 69]. These data are consistent with similar TMJ data that bring to light that the presence of comorbid conditions may explain why 50 % of patients seeking care for TMJ pain, some of whom were multiply operated and/or exposed to failed materials or devices, still report experiencing pain 5 years later. 20 % of chronic pain patients experience long-term disability from their pain [70–72].

## 8.10 Intrinsic Etiologies

The surgeon must first determine if one of the common causes for continued post-TMJ TJR pain and dysfunction infection, heterotopic bone formation, or dislocation is present. The clinical, imaging, and laboratory manifestations, diagnosis, and management options for each have been discussed earlier in this chapter. Material sensitivity is discussed at length in Chapter 9 Failed and failing devices as a result

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of aseptic component or screw loosening as well as component or screw fracture and osteolysis will be discussed later in this chapter.

### *8.10.1 Neuroma Formation*

A traumatic, amputation, or postsurgical neuroma is a non-neoplastic mass of entangled Schwann cells, fibrous scar tissue, and inflammatory cells [73, 74]. The orthopedic, neurosurgical, and plastic surgery literature contains numerous reports, case series, and reviews concerning postsurgical neuroma formation and management options [75–79].

Neuromas are thought to occur more commonly in the presence of scar formation, either from a decreased ability of cytokine signaling diffusion or contracture making nerve migration more difficult [73, 75]. Also, mechanical stimulation or motion can interfere with nerve migration, resulting in neuroma formation [73]. Some patients have been identified who develop chronic refractory pain several months after otherwise successful joint reconstruction.

Neuromas are known to cause chronic pain and it has been shown that the excision of these masses can result in a decrease in pain [74–79].

Diagnosis may be complicated because peripherally mediated neuropathic pain leads to centrally mediated pain, and this combination may be contributing to the patient's pain perception [73].

The initial evaluation should include a thorough description of the pain by the patient. The area involved should be documented and correlated to known nerve distributions if possible. Pain spanning beyond the area of known nerve distribution may suggest a strong psychological component or a myofascial diagnosis. Thermal perception as well as hyperalgesia and allodynia should be checked. It is also important to try and elicit a Tinel's sign (paresthesia or a tingling sensation in the distal distribution of a nerve following percussion). It is suggestive of nerve regeneration. A painful response may indicate neuroma formation [80]. Local anesthetic carefully injected into the joint avoiding scuffing of the bearing surfaces with the needle tip may be of differential diagnostic value.

Granquist et al. evaluated pain scores and maximal incisal opening in patients who after TMJ TJR were found to have postsurgical neuromas and compared these scores with patients who underwent revision arthroplasty without neuromas. On mean 1.9-year follow-up, 3 of 7 patients in the neuroma group had clinically significant pain reduction, three reported lower pain scores, and 1 had no pain change. No patients had increased pain. 1 of 4 patients in the scar revision group had clinically significant pain reduction, 2 had no change, and 1 reported increased pain. They concluded that their small retrospective study suggested that removal of these neuromas may benefit some patients, but that additional studies comparing surgical and medical management should be performed [81] (Fig. 8.12).

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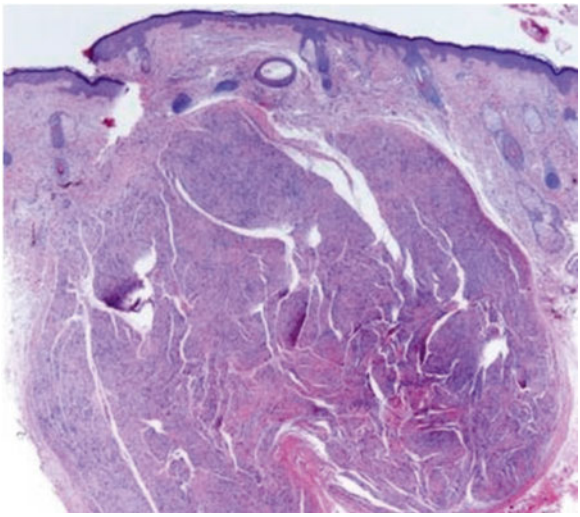

**Fig. 8.12** Neuroma from TMJ TJR (*Granquist EJ, et al.: Post-surgical neuromas in patients with TMJ TJR: A retrospective case series. Int J Oral Maxillofac Surg. 2011. 40:366–71.*)

### *8.10.2 Synovial Entrapment Syndrome*

Synovial metaplasia was first described by Brody and White after their studies on implanted silicone joints in chickens [82]. The first human cases were descriptions of the formation of synovial-like membranes reported by investigators studying the reaction of the peri-articular tissues to joint and tendon prostheses [83, 84]. Later, Gonzalez et al. reported the occurrence of synovial metaplasia occurring in the skin in healed surgical scars [85].

Murray and Drachman suggested that traction or motion provides the biological signal that stimulates differentiation and organization of individual cells into synovial tissue. That is, mechanical forces (movement, shear forces, repeated surgery, trauma, and so on) are necessary for the development of synovium. These authors also named other rarely cited factors necessary for the formation of normal joint spaces: loose areolar tissue that would develop spaces due to the movement and relatively smooth gliding surfaces that would resist penetration by growing fibroblast processes [86].

Edwards et al. provided data supporting this concept. They repeatedly injected air into subcutaneous tissue. Analysis of the lining tissue of this cavity by light and electron microscopy and histochemical techniques showed that after 5–30 days, the lining membrane was indistinguishable from synovial tissue. These studies appear to show that mechanical forces, in association with the natural developmental response of mesenchymal tissues surrounding an implanted foreign body, and the chemical and physical composition of the foreign body are most likely primarily involved with the formation of the synovial metaplasia [87].

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Westermark et al. reported histologic findings in soft tissue samples obtained from around 2 types of TMJ TJR devices (Biomet Microfixation and TMJ Concepts) after up to 8 years of function. All joint capsule samples showed dense, fibrous connective tissue with no inflammatory cells or foreign-body reactions. The joint disc tissues showed even denser fibrous connective tissue, free from inflammatory reactions (Fig. 8.13). Some samples from the junction between capsule and disc showed synovial-like tissue [88] (Fig. 8.14). Monje et al. reported similar findings [89].

In the orthopedic literature, symptomatic synovial plicae have been reported in the knee [90–92] and other extremity joints such as shoulder [93] and elbow [94]. Although the common signs and symptoms in the medial plica impingement of the knee include crepitation, popping, snapping, instability, catching, and pain [90–92], there is significant crossover of symptoms and clinical findings associated with more commonly seen diagnoses [92]. Thus, the specific diagnosis of plica syndrome is still controversial.

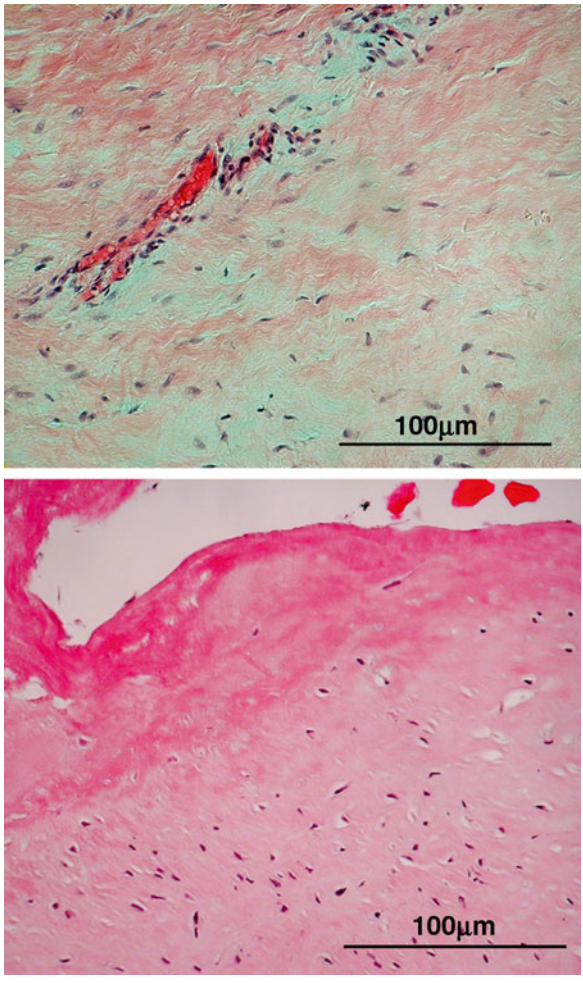


**Fig. 8.13** Sections stained with hematoxylin–eosin from soft tissue between the joint components showing a dense, fibrous connective tissue with only limited focal chronic inflammation. (*Westermark A, Leiggener C, Aagaard E, Lindskog S. Histological findings in softtissuesaround temporomandibular joint prostheses after up to eight years of function. Int J Oral Maxillofac Surg. 2011. 40:18–25*)

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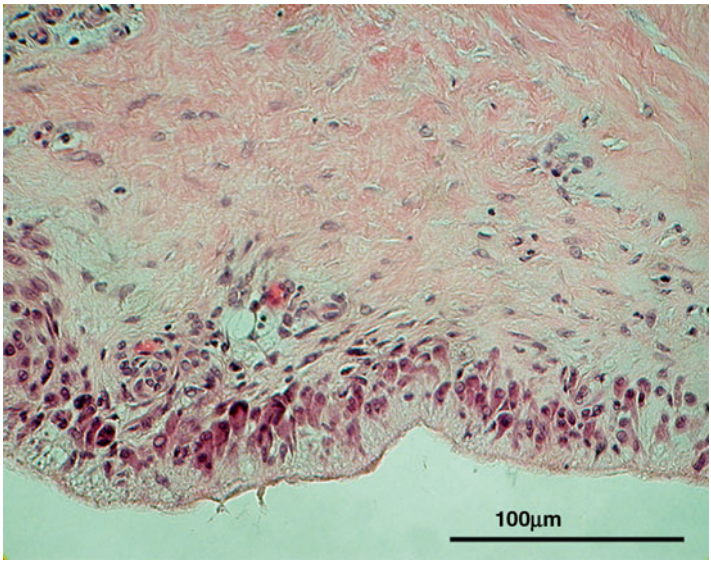


**Fig. 8.14** Section stained with hematoxylin–eosin from tissue adjacent to the condylar component showing a surface resembling a synovial membrane. (*Westermark A*, *Leiggener C*, *Aagaard E*, *Lindskog S. Histological findings in softtissuesaround temporomandibular joint prostheses after up to eight years of function. Int J Oral Maxillofac Surg. 2011. 40:18–25.*)

The anatomical occurrence and distribution of the plicae in orthopedic joints have been demonstrated on MRI; however, MRI has not been able to distinguish between pathologic and non-pathologic plicae [91, 92, 94–96]. MRI findings may be useful to evaluate the thickness and extension of plica and synovitis with concomitant effusion [91, 94–96]. The "gold standard" for plicae diagnosis in orthopedics is arthroscopy followed by excision [90–93, 95, 96].

To date only cadaveric studies demonstrating the presence, appearance, and histology of synovial plicae have been reported in the TMJ literature [97–99]. However, there are clinical reports of synovial plicae-like structures in patients with TMJ disorders [100, 101] (Fig. 8.15).

Kim et al. concluded that an intra-articular synovial fold located only in the inferolateral parapatellar area underwent hypertrophy and fibrosis following chronic irritation and that this caused the impingement symptoms [102].

Since it is clearly evident that a "neo-synovium" develops around TJR devices and TMJ synovial plicae have been identified both in both laboratory [97–99] and clinical [103] studies, development of these plicae with entrapment between the bearing surfaces of TMJ TJR devices can result in inflammation and pain. Diagnostic local anesthetic block and surgical debridement and placement of an autogenous fat graft followed by active jaw exercises to regain and maintain range of mandibular motion may provide relief. However, further studies of this phenomenon in TMJ TJR are recommended.

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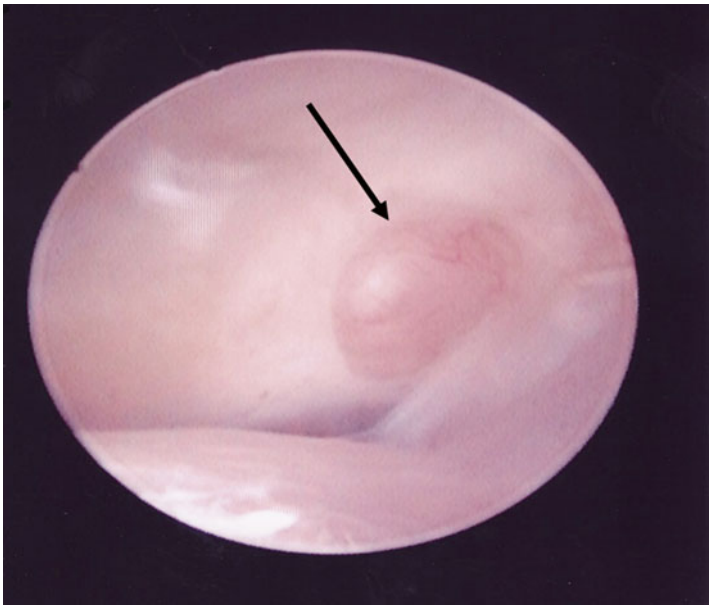

**Fig. 8.15** Synovial plica left TMJ (*Kenichiro Murikami*, *Kyoto*, *Japan*)

### *8.10.3 Extrinsic Etiologies*

After ruling out all possible internal etiologies for a patient's continued or increasing post-TMJ TJR pain, the following extrinsic etiologic possibilities should be examined (Fig. 8.11).

### *8.10.4 Prior Misdiagnosis*

It has been well documented that misdiagnosis plays a role in unsuccessful TMJ surgical outcomes [104–108]. The experience of the past 150 years in the diagnosis and management of chronic orofacial pain conditions has shown that a mechanistic, "tunnel vision" approach is likely to produce iatrogenic harm, e.g., unnecessary equilibrations, extractions, restorations, TMJ surgery, etc. [109].

Such "tunnel vision" can easily lead to misdiagnosis of potentially more serious conditions that mimic either TMD pain distribution or limitation of mouth opening, possibly resulting in unnecessary treatments or more serious consequences for the patient.

How do practitioners develop "tunnel vision"? Mohl and Ohrbach provide some possible insights: (1) These practitioners doggedly continue to rely on their early professional training and experience instead of furthering their education on the subject by reading and/or attending relevant CE programs; (2) They are only famil-

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iar with certain procedures and are unwilling to change or try something "new"; (3) Inertia; (4) Isolation in "private" practice; (5) Insecurity with change; (6) Unfamiliarity with the literature or not willing to make the effort to assess scientific evidence; and (7) Blind belief in "schools of thought" sponsored by charismatic gurus (cults). The last is the most dangerous motivation because it is self-perpetuating and often based essentially on economics [110].

Mercuri reported that after taking comprehensive histories from failed multiple-operated patients (>10 prior unsuccessful TMJ surgeries), signs and symptoms present before any of prior TMJ surgery revealed that in most cases the original diagnosis was not correct. More than half of the patient did not have an intra-articular problem and, therefore, unlikely to have resolution via intra-articular surgery [63].

TMJ TJR is a mechanical solution to a biological problem and should be reserved as a management option for definitive and demonstrable end-stage TMJ disease. Multiple-operated patients must be informed that due to the complex nature of joint-related masticatory muscle functional and anatomical associations, it is unreasonable to expect a surgically modified TMJ will be returned to its "normal" premorbid function. Therefore, the patient and surgeon must both accept that there will always be some functional disability associated with any invasive TMJ procedure, especially TMJ TJR in multiple-operated cases.

The literature supports the fact that TMJ TJR subjective outcomes (pain, jaw function, and diet) are inversely related to the number of prior failed TMJ surgeries [63–68].

### *8.10.5 Chronic Centrally Mediated Pain*

Chronic pain can be the consequence of central sensitization. Central sensitization, increased neuronal responsiveness to repetitive and noxious stimulation, causes hyperalgesia, allodynia, and referred pain leading to chronic pain. Triggers discussed as causes for sensitization are wind-up or temporal summation [111], dysregulation of descending inhibitory pathways [112], and upregulated facilitatory modulation by cognitive emotional sensitization [113].

Certain cognitive styles and personality traits have been associated with amplification of pain and its extension in the absence of tissue damage. These include somatization, catastrophizing, and hypervigilance [114]. Data from this and other studies suggest generalized hyper-excitability of the central nociceptive system in patients with chronic pain [115, 116].

Human genetic studies have demonstrated associations between certain genetic polymorphisms and the development of chronic pain. Coupled with environmental triggers such as depression, anxiety, somatization, catastrophizing, and hypervigilance, genetic factors will contribute to enhanced pain perception, psychological dysfunction, and increased risk and onset for TMJ-related pain and related idiopathic pain disorders [72, 117].

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Chronic pain patients presenting for TMJ TJR with poor or no mandibular function due to iatrogenic end-stage TMJ disease as a result of misdiagnosis or multiple failed prior surgeries should be considered as potential centrally mediated pain patients who could possibly have post-TMJ TJR continued chronic or increased pain.

Management of such cases begins with patient education. Unrealistic outcomes of total pain relief must be dispelled. These patients and the surgeon must understand that the primary goal of TMJ TJR is increased mandibular function. Any pain relief is of only secondary benefit. The TMJ TJR literature demonstrates clearly that as the number of prior TMJ surgeries increases, any significant decrease in pain does not [63–68]. However, these patients report an increase in their quality of life [65–68].

Many chronic TMJ pain patients are under the care of a pain management specialist and taking multiple medications to control their pain. Prior to TMJ TJR, it is advisable for the surgeon to contact the pain specialist to discuss the most appropriate perioperative and postoperative analgesic regimens for control of the surgically related pain in light of any analgesics the patient may be using regularly.

If this type of patient is not already under the care of a pain management specialist, the surgeon should provide the patient with a referral to one before undertaking surgery.

Finally, since families of chronic pain patients are often negatively affected, it is advisable for the surgeon to include significant family in discussion of expected surgical outcomes so that their expectations will also be reasonable.

Low-dose antidepressant medication has also been shown to be effective in modulating chronic pain symptoms [118].

## 8.11 Persistent Myofascial/Muscular Pain

Myofascial pain is a subcategory of temporomandibular disorders and it is accepted that jaw muscle pain and motor function are interrelated [119]. The exact nature of this interrelationship has been the subject of much discussion [120–127].

Three major theories have been proposed to explain pain and its relationship to muscle activity: the Vicious Cycle Theory, the Pain Adaptation Model, and the Integrated Pain Adaptation Model.

The Vicious Cycle Theory proposes that an initiating factor such as abnormal structure, posture, movement, or stress results in pain that leads to reflex muscle hyperactivity. This leads to spasm, fatigue, and further pain and dysfunction in a self-perpetuating cycle [122–126, 128].

The Pain Adaptation Model proposes that pain arises from causes other than muscle hyperactivity and that pain leads to alterations in muscle activity that limits movement, thereby protecting the skeletomotor system from further harm and promoting healing [129–131].

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The Integrated Pain Adaptation Model attempts to explain the motor effects of pain. In normal function, the brain will activate whatever motor units required producing an appropriate movement. However, in the presence of pain, the pain interacts in a unique way with the individual's somatosensory system [119]. There is evidence that there is considerable variability in behavioral response to pain with both genetic and psychosocial factors playing crucial roles.

Mense discussed the transition from acute to chronic muscle pain on the basis of central sensitization. He states that if nociceptive muscle input is strong or ongoing, the functional changes in the spinal cord and brainstem will outlast the peripheral lesion. Neuroplastic changes, such as the opening of synapses, are one of the primary steps in the transition from acute to chronic pain because they persist for a long time. The next step toward chronic muscle pain is the lesion-induced metabolic changes that take place in the sensory spinal neurons. Finally, actual morphological changes occur in the spinal dorsal horn which may last for years or become permanent. Therefore, an important principle in the management of muscle pain is to abolish the nociceptive input from the muscle to the spinal cord as early as possible to prevent lesion-induced CNS alterations resulting in chronic muscle pain [132].

Management of chronic masticatory muscle pain after TMJ TJR is both challenging and frustrating since often an initial misdiagnosis of a muscle-related etiology for TMJ pain disorder is involved. After ruling out an intrinsic or other extrinsic problem, chronic pain management and patient education as to the issue may prove helpful.

## 8.12 Complex Regional Pain Syndrome I and II

Complex regional pain syndrome (CRPS) is a chronic pain condition most often affecting one of the limbs (arms, legs, hands, or feet), usually after an injury or trauma to that limb. CRPS is characterized by prolonged or excessive pain and mild or dramatic changes in skin color, temperature, and/or swelling in the affected area.

There are two similar forms, called CRPS-I and CRPS-II, with the same symptoms and treatments. CRPS-II (previously called causalgia) is the term used for patients with confirmed nerve injuries. Individuals without confirmed nerve injury are classified as having CRPS-I (previously called reflex sympathetic dystrophy syndrome).

CRPS symptoms vary in severity and duration. Studies of the incidence and prevalence of the disease show that most cases are mild and individuals recover gradually with time. In more severe cases, individuals may not recover and may have long-term disability.

The key symptom is prolonged pain that may be constant and, in some people, extremely uncomfortable or severe. The pain may feel like a burning or "pins and needles" sensation, or as if someone is squeezing the affected limb. The pain may spread to include the entire arm or leg, even though the precipitating injury might

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have been only to a finger or toe. Pain can sometimes even travel to the opposite extremity. There is often increased sensitivity in the affected area, such that even light touch or contact is painful.

People with CRPS also experience constant or intermittent changes in temperature, skin color, and swelling of the affected limb. This is due to abnormal microcirculation caused by damage to the nerves controlling blood flow and temperature. An affected arm or leg may feel warmer or cooler compared to the opposite limb. The skin on the affected limb may change color, becoming blotchy, blue, purple, pale, or red.

In more than 90 % of cases, the condition is triggered by a clear history of trauma or injury. The most common triggers are fractures, sprains/strains, soft tissue injury (such as burns, cuts, or bruises), limb immobilization (such as being in a cast), or surgical or medical procedures. CRPS represents an abnormal response that magnifies the effects of the injury.

Peripheral nerve abnormalities found in individuals with CRPS usually involve the small unmyelinated and thinly myelinated axons that carry pain signals to blood vessels. Because small fibers in the nerves communicate with blood vessels, small nerve fiber injuries may trigger the many different symptoms of CRPS. Molecules secreted from the ends of hyperactive injured small nerve fibers are thought to contribute to inflammation and blood vessel abnormalities. These peripheral nerve abnormalities in turn trigger abnormal neurological function in the spinal cord and brain, leading in some cases to complex disorders of higher cortical function.

Another abnormality in CRPS involves the blood vessels in the affected limb, which may dilate (open wider) or leak fluid into the surrounding tissue, causing red, swollen skin. The underlying muscles and deeper tissues can become starved of oxygen and nutrients, causing muscle and joint pain. At times, the blood vessels may over-constrict, causing cold, white, or bluish skin.

CRPS also affects the immune system. High levels of cytokines have been found in the tissues of people with CRPS. These contribute to the redness, swelling, and warmth reported by many patients. CRPS is more common in individuals with other inflammatory and autoimmune conditions.

Currently, there is no single diagnostic test to confirm CRPS. Diagnosis is based on the affected individual's medical history and signs and symptoms that match the definition. Testing may be used to help rule out other conditions, such as arthritis syndromes, Lyme disease, generalized muscle diseases, a clotted vein, or small nerve fiber polyneuropathies. Magnetic resonance imaging or triple-phase bone scans sometimes identify CRPS-characteristic changes in the bone metabolism. CRPS is often associated with excess bone resorption.

The outcome of CRPS varies from person to person. Occasionally, individuals are left with unremitting pain and crippling, irreversible changes despite treatment. Anecdotal evidence suggests early treatment, particularly rehabilitation, is helpful in limiting the disorder, but this benefit has not yet been proven in clinical studies.

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### 8.12.1 Rehabilitation Therapy

An exercise program to keep the painful limb or body part moving can improve blood flow and lessen the circulatory symptoms. Additionally, exercise can help improve the affected limb's flexibility, strength, and function. Rehabilitating the affected limb also can help to prevent or reverse the secondary brain changes that are associated with chronic pain. Occupational therapy can help the individual learn new ways to work and perform daily tasks.

### 8.12.2 Psychotherapy

CRPS and other painful and disabling conditions often are associated with profound psychological symptoms for affected individuals and their families. People with CRPS may develop depression, anxiety, or posttraumatic stress disorder, all of which heighten the perception of pain and make rehabilitation efforts more difficult. Treating these secondary conditions is important for helping people cope and recover from CRPS.

### 8.12.3 Medications

Several different classes of medication have been shown to be effective for CRPS, particularly when used early in the course of the disease. No drug is approved by the U.S. Food and Drug Administration specifically for CRPS. No single drug or combination of drugs is guaranteed to be effective in every person (Fig. 8.16).

1. **Non-steroidal anti-inflammatory drugs to treat moderate pain, including over-the-counter aspirin, ibuprofen, and naproxin**
2. **Corticosteroids that treat inflammation/swelling and edema, such as prednisolone and methylprednisolone (used mostly in the early stages of CRPS)**
3. **Gabapentin, pregabalin, amitriptyline, nortriptyline, and duloxetine, botulinum toxin**
4. **Opioids such as oxycontin, morphine, hydrocodone, fentanyl, and vicodin**
5. **N-methyl-D-aspartate (NMDA) receptor antagonists such as dextromethorphan and ketamine nasal calcitonin, especially for deep bone pain**
6. **Topical local anesthetic creams and patches such as lidocaine.**

**Fig. 8.16** Drugs to treat CRPS

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### 8.12.4 Sympathetic Nerve Block

Some individuals report temporary pain relief from sympathetic nerve blocks, but there is no published evidence of long-term benefit. Sympathetic blocks involve injecting an anesthetic next to the spine to directly block the activity of sympathetic nerves and improve blood flow.

### 8.12.5 Surgical Sympathectomy

The use of this operation that destroys some of the nerves is controversial. Some experts think it is unwarranted and makes CRPS worse; others report a favorable outcome. Sympathectomy should be used only in individuals whose pain is dramatically relieved (although temporarily) by sympathetic nerve blocks. It also can reduce excess sweating.

### 8.12.6 Spinal Cord Stimulation

Placing stimulating electrodes through a needle into the spine near the spinal cord provides a tingling sensation in the painful area. Typically, the electrode is placed temporarily for a few days to assess whether stimulation will be helpful. Minor surgery is required to implant all the parts under the skin on the torso. Once implanted, the stimulator can be turned on and off, and adjusted using an external controller. Data show that about one-fourth of individuals develop equipment problems that may require additional surgeries.

### 8.12.7 Other Types of Neural Stimulation

Neurostimulation can be delivered at other locations along the pain pathway, not only at the spinal cord. These include near injured nerves (peripheral nerve stimulators), outside the membranes of the brain (motor cortex stimulation with dural electrodes), and within the parts of the brain that control pain (deep brain stimulation). A recent option involves the use of magnetic currents applied externally to the brain (called repetitive Transcranial Magnetic Stimulation, or rTMS). The advantage is that no surgery is required; the disadvantage is need for repeated treatment sessions.

Intrathecal drug pumps. These devices pump pain-relieving medications directly into the fluid that bathes the spinal cord, typically opioids and local anesthetic agents such as clonidine and baclofen. The advantage is that pain-signaling targets in the spinal cord can be reached using doses far lower than those required for oral

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administration, which decreases side effects and increases drug effectiveness. There are no studies that show benefit specifically for CRPS.

Several alternative therapies have been used to treat other painful conditions. Options include behavior modification, acupuncture, relaxation techniques (such as biofeedback, progressive muscle relaxation, and guided motion therapy), and chiropractic treatment [133].

Melis et al. reviewed the features of complex regional pain syndrome (CRPS), including its pathophysiology, diagnosis, and treatment with a focus on the literature reporting cases in which the face, head, and neck were affected. Very few cases were found that met the International Association for the Study of Pain criteria for the disease. The clinical characteristics were similar to those of CRPS elsewhere in the body, with the main features being burning pain, hyperalgesia, and hyperesthesia starting after trauma to the craniofacial region. Physical signs were reported less frequently. The treatment of choice was a series of stellate ganglion anesthetic blocks, which resulted in a good outcome in all the cases reviewed [133].

## 8.13 Temporalis Tendonitis

Temporal tendinitis is described as a disorder of the fibrous insertion of the temporalis muscle tendons on the coronoid process of the mandible characterized by both inflammation and degeneration [134].

Ernest et al. describe a patient with the classic signs and symptoms of temporal tendonitis who underwent excision of the temporal tendon and associated mandibular coronoid process. Histopathologic examination revealed focal atrophy and tissue necrosis which the authors' statement attests to the focal nature of the painful condition of temporal tendonitis [135].

Physical examination and diagnostic local anesthetic infiltration can provide evidence for this diagnosis. If intra-oral digital palpation along the anterior border of the ramus at the insertion of the temporalis tendon elicits the pain and medial and lateral infiltration of local anesthetic relieves the pain, stripping the temporalis tendon with a v-notch sagittal split osteotomy instrument will typically deal with this postimplantation pain issue.

## 8.14 Coronoid Impingement

It is important for the surgeon to understand the relationship of the coronoid process of the mandible to the zygoma and the zygomatic arch preoperatively in all TMJ TJR cases. Careful examination of preoperative CT imaging and/or the virtual or actual SL model is essential to determine the coronoid processes involvement in the pathology so that a determination can be made as to whether a coronoidectomy should be part of the surgical plan (Fig. 8.17).

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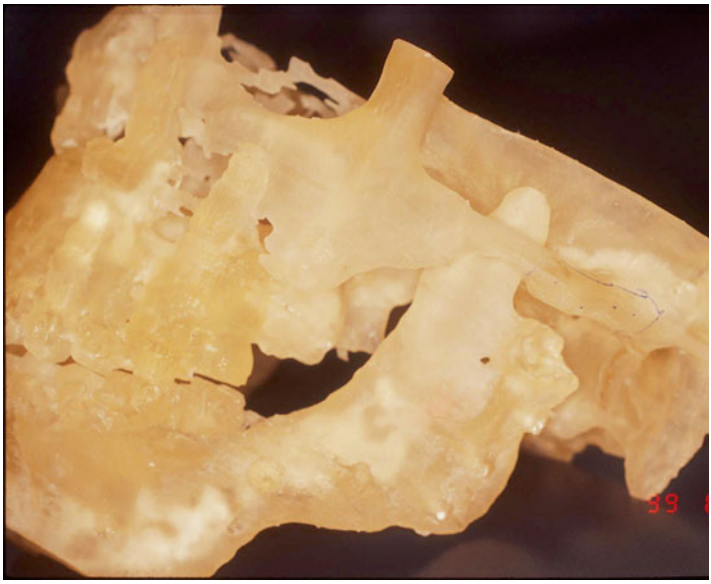

**Fig. 8.17** Stereolithic model demonstrating coronoid hyperplasia secondary to lost posterior mandibular height

In cases where there has been long-term posterior mandibular height loss (e.g., arthritic disease [53], ICR, or PCR [136]), there will be compensatory increase in the height of the coronoid process (hypertrophy) due to the influence of the temporalis muscle on the coronoid process bone as the posterior mandible height decreases. Using TMJ TJR to reposition the mandible into the appropriate posterior vertical relationship can result in potential impingement of the hypertrophied coronoid against the posterior zygoma resulting in postoperative pain and decreased mandibular range of motion.

Typically, this is evident in the preoperative planning when it is recognized and coronoidectomy becomes part of the procedure. However, there are cases where the coronoid process redevelops at some time postoperatively leading to functional pain and decreased mandibular opening. If CT imaging will reveals this, coronoidectomy will resolve it.

## 8.15 Frey's Syndrome and Frey's Neuralgia

Frey syndrome (auriculotemporal syndrome, gustatory sweating) is characterized by episodes of warmth, flushing, and sweating of the face in the pre-auricular region initiated by gustatory stimulus [137] (Fig. 8.18). Frey syndrome is a common

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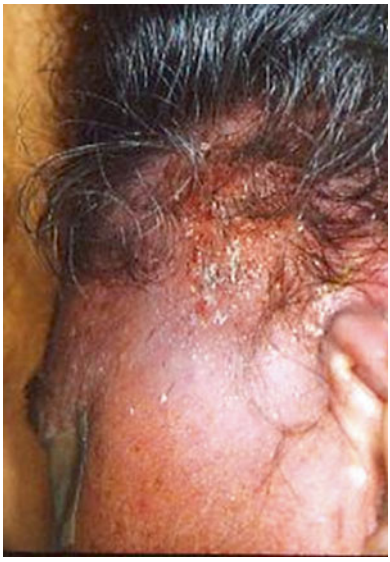

**Fig. 8.18** Frey's Syndrome after left TMJ surgery

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complication after operations on the parotid gland and the temporomandibular joint [138, 139]. The most common hypothesis is that regenerating parasympathetic fibers to salivary glands connect in error with the sweat glands and subcutaneous blood vessels of the skin. The onset has usually been 12–18 months after surgery. The most effective treatment has been subcutaneous infiltration of botulinum toxin into the affected area [140].

De Benedittis reported two cases of Frey's syndrome presenting as trigeminal tic douloureux. This extremely rare condition is characterized by gustatory sweating and facial hyperemia, and a tormenting gustatory pain occurred in excruciating brief paroxysms [141]. Consultation with a neurosurgeon for management is indicated should it occur.

## 8.16 Integrin Formation

Integrins are heterodimeric (i.e., alpha beta heterodimers) cell surface receptors, which enable adhesion, proliferation, and migration of cells by recognizing binding motifs in extracellular matrix (ECM) proteins. As transmembrane linkers between the cytoskeleton and the ECM, they are able to recruit a huge variety of proteins and to influence signaling pathways bidirectionally, thereby regulating gene expression and cell survival. Hence, integrins play a key role in mechanoreception and various physiological as well as pathological processes [142].

Milam et al. demonstrated that trigeminal ganglion neurons supplying the rat TMJ expressed integrin subunits. These subunits identify subfamilies of integrins

that may be involved in TMJ mechanoreception and proprioception [143]. This finding along with neuroplasticity may account for the phenomenon of chronic pain with function after TMJ TJR despite the removal of diseased joint components. Management options involve the assistance of pain specialists. Reoperation does not appear to be a viable option in such cases.

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# Chapter 9
# Material Hypersensitivity

**Nadim Hallab**

## 9.1 Material Sensitivity

Excessive reactivity to metal implant debris or hypersensitivity to implant debris is relatively rare, where it is estimated that only 1–3 % of aseptic failures are due to hypersensitivity responses among traditional metal-on-polymer type total joint replacement (TJR) hip and knee designs. The percentage of aseptic failures due to biomaterial hypersensitivity in alloplastic total temporomandibular joint TMJ-TJR is not known. Other more prevalent reasons for long-term aseptic implant failure in TJR are bone fracture, infection, implant failure, and aseptic osteolysis due to particle-induced subtle inflammatory responses (Fig. 9.1) [1–6]. It is important to understand that implant surfaces are NOT the cause of hypersensitivity reactions or indeed almost all unwanted immune reactivity to orthopedic implants. It is implant debris (particles and ions) emanating from implant surfaces that are capable of activating interactions with immune cells and are thus able to elicit an immune response. This distinction is important in order to combat these elevated hypersensitivity immune responses, i.e., when implant debris is minimized, metal hypersensitivity is also minimized [7].

Hypersensitivity is characterized by cell-mediated adaptive immune responses where conditioned lymphocytes respond to specific stimuli, as opposed to the more typical and less specific response of macrophages to implant debris. Although undocumented in TMJ-TJR, the typical manner of long-term failure is induced by a slow progressive debris-induced osteolysis or "particle disease." This refers to the normal process of peri-implant osteolysis which takes place over the long term (>7 years), where implant loosening and inflammation are due to implant particulate debris nonspecifically interacting

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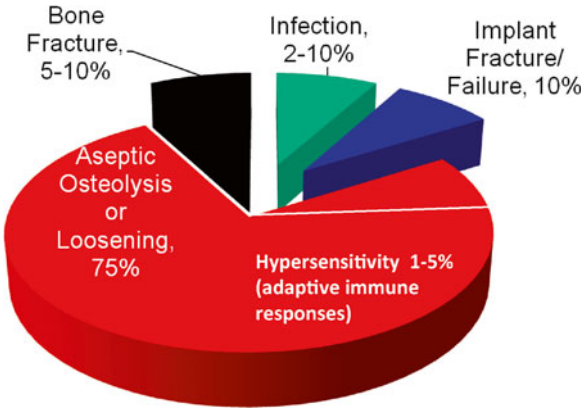


**Fig. 9.1** A compilation of investigations show the averaged percentages of metal sensitivity among the general population for nickel, cobalt, and chromium, among patients after receiving a metal-containing implant, and among patient populations with failed implants. All subjects were tested by means of a patch test, metal-LTT (lymphocyte transformation test), or histological diagnosis

with innate immune system cells (i.e., tissue macrophages termed histiocytes). In contrast, "metal sensitivity" or hypersensitivity is a more specific immune response which takes place over a shorter time frame. From its onset it is a more severe lymphocyte-based immune response, i.e., delayed-type hypersensitivity (DTH). These responses have been associated with the failures of certain types of metal-on-metal bearing hip arthroplasty implants but remain unreported in the area of TMJ-TJR.

To a large extent, implant materials and metals currently in use have evolved over time to the more successful devices that resist wear and corrosion. However, they are not the strongest metals or plastics, and despite the low corrosion and wear potential of modern TJR implants, metal particle and ion release are inevitable so metal sensitivity remains reported in both case and group studies [8–10]. All implant metals degrade by both corrosion and wear in vivo [11, 12], and the released debris (particles and ions) immediately are coated (opsonized) or complexed with plasma proteins and it is these organometallic complexes that interact biologically both locally and systemically. Released metal ions become antigenic "allergens," haptens which activate the immune system not by themselves, but by forming complexes with native serum proteins and altering their natural conformational structure [13–16]. Metal-altered-self-protein complexes are engulfed by antigen-presenting cells (APCs) and recognized as foreign by lymphocytes triggering a hypersensitivity response.

In its broadest definition, "metal hypersensitivity" to TJR implants is any aseptic (non-bacterial) material-driven "excessive" immune response that causes peri-implant pathology, such as bone loss caused by local T-cells, B-cells, and/or macrophages. It remain unclear just what constitutes an "excessive" immune response. However, when an implant fails prematurely (<7 years) due to an exuberant cell-mediated immune response to metal implant debris, while an equivalent amount of implant debris is typically well tolerated, that response can be categorized as "metal allergy," "implant allergy," "implant sensitivity," or "hypersensitivity." The allergy/sensitivity/hypersensitivity terms are liberally used interchangeably in immunology and orthopedics despite specific

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nuanced differences between them that imply different immune-based reactivity. For simplicity, any nuanced differences between them will not be discussed here.

Skin or dermal sensitivity to metals affects approximately 10–15 % of the population [8, 9, 16–19] and has been reported to cause skin hives, eczema, redness, and itching (Fig. 9.1). Hypersensitivity to nickel is the most common (approximately 14 %) [8, 17–20], followed by cobalt and chromium [8, 16, 20]. Other metals that are reported to cause sensitivity responses include beryllium and to a lesser degree tantalum [21], titanium [22, 23], and vanadium [21]. How these metals elicit sensitivity responses will be discussed in the following sections.

### *9.1.1 Metal Sensitivity Mechanism*

In general, hypersensitivity responses can take one of two main forms: (1) a humoral type of response that occurs fast (within minutes), initiated by circulating antibodies to antigen complexes, classified as types I, II, and III reactions, or (2) a cell-mediated delayed type of response that occurs hours to days after "recall" challenge (2nd challenge) [24, 25]. Metal hypersensitivity reactions are almost exclusively delayed-type responses mediated by antigen-activated lymphocytes and are classically categorized as type IV delayed-type hypersensitivity (DTH) responses.

A cell-mediated delayed type of hypersensitivity response is characterized by T-helper lymphocytes of the $T_H1$ subset that have been sensitized to the specific antigen. Once activated they release inflammatory cytokines, thus recruiting more antigen-presenting cells such as macrophages which in turn amplifies the $T_H1$ response which continues in a vicious cycle if not stopped by regulatory immune cells. These $T_H1$ cells release inflammatory cytokines, including interferon-γ (IFN-γ), tumor necrosis factor-α (TNF-α), interleukin-1 (IL-1), and interleukin-2 (IL-2). Without these $T_H1$ cells, we are vulnerable to pathogens such as *Staphylococcus pneumonia* (and other organisms that occur in HIV infections where $T_H$ are lost). However, when $T_H1$ cells are erroneously activated, they can result in autoimmune diseases.

In this fashion, metal-sensitized and activated T-cells, in conjunction with antigen-presenting cells (APCs), will secrete a variety of pro-inflammatory cytokines that recruit and activate other innate immune cells, e.g., macrophages, monocytes, and neutrophils. Signature cytokines of this response include IFN-γ and TNF-β which, among the many pro-inflammatory destructive effects they exert on local cells (e.g., endothelial cells), induce chemokines such as migration inhibitory factor (MIF), which prevents the migration of recruited macrophages away from the site of the metal-DTH reaction. The hallmarks of a DTH response are infiltration, activation, and eventual migration inhibition of innate antigen-presenting immune cells (e.g., macrophages) that are largely controlled by adaptive immune cells, i.e., T-cells. These recruited and activated macrophages have an increased ability to phagocytose, process, and then present pieces of the phagocytosed metal-protein complexes on their surface for T-cell recognition. These "immune epitopes" are nestled in "class II MHC complexes" surface receptors on antigen-presenting cells for interaction with T-cell receptors, TCRs. The release of cytokines from the recruited antigen-presenting cells

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(such as IL-1) can trigger the recruitment/activation of more T-cells, which in turn activates more macrophages in a vicious cycle. Under certain circumstances, such as in some autoimmune diseases, there is an inability to turn off this DTH self-perpetuating vicious cycle response which can result in extensive tissue damage. Thus, current strategies to mitigate these types of responses are geared toward immunosuppressive therapies to temporarily stop this vicious cycle, thereby facilitating normal noninflammatory homeostasis.

However, antigen-specific targeted therapy has yet to be developed as there is still much to learn about metal sensitivity, including the following: (1) how to address the fact that different specific lymphocyte populations are involved in this reaction in different individuals [26], (2) the specific cellular mechanisms of recognition and activation remain unknown, and (3) why serum metal-protein complexes become antigenic in only some people.

Dermal sensitivity is relatively easily studied and to some extent characterized. Skin is a primary immune barrier. Antigen-presenting cells (APCs) of the skin, called Langerhans cells, are exquisitely good at gathering and presenting antigen. Each dendritic Langerhans cell is responsible for the immunosurveillance of 53 epidermal cells, which is surprisingly consistent from person to person [27]. However, these cells differ in several ways from APCs in TJR peri-implant tissues, where APCs are primarily composed of macrophages, endothelial cells, lymphocytes, dendritic cells, and parenchymal tissue cells.

Tissue macrophages (histiocytes) are considered primary APCs around TJR implants and are primarily responsible for implant debris-induced phagocytosis. T-cell receptors (TCR) that recognize the metal-protein complex presented by APCs have been widely acknowledged as central to metal sensitivity response and can be mitigated by blocking these receptors [28–30]. To complicate matters, metals such as nickel have also been shown to activate T-cells in both this classical and other nonclassical ways. One nonclassical way is to simply cross-link TCRs and costimulatory receptors on T-cells (e.g., VB17 of CDR1 T-cell receptor) to create what is termed a "superantigen" activation of T-cells receptor [29, 31]. Despite the possibility of nonclassical activation of T-cells by released metals complexed with serum proteins, identification of ways that non-typical metal-induced lymphocyte activation can occur is not known. The traditional DTH response remains the dominant mechanism associated with implant-related hypersensitivity responses [32–34].

### *9.1.2 Testing for Metal Sensitivity*

Currently approved methods for human diagnostic testing for metal allergy include both skin testing (patch testing) and in vitro blood testing using lymphocyte transformation testing (LTT testing). There are commercially available assays for physicians that contain some of the metals contained in orthopedic implants [24, 35].

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#### 9.1.2.1 Dermal Testing

Clinical patch testing kits used to diagnose dermal metal-DTH responses do exist for a variety of common metals [24, 35]. However, there are serious questions regarding the applicability of skin testing to diagnose in vivo immune responses to orthopedic implant debris—there may be more harm than good. There are a number of questions regarding skin challenge verses metals mixed with serum proteins to accurately form metal-protein complexes to mimic metal challenge agents in vivo [13–15, 36]. Is the allergenic (hapentic) potential of metals in a dermal environment (in which dermal Langerhans cells are the primary effector cells) the same as that of an in vivo closed peri-implant environment? Not likely [25, 37]. Unique antigen-processing/endosomal-recycling organelles, called Birbeck granules, are present in Langerhans cells of the skin but are not found in the dominant peri-implant APCs such as macrophages [38, 39]. Other important drawbacks of dermal testing for implant-related metal sensitivity include the following: (1) The biggest risk associated with patch testing is the possible development of metal sensitivity in a previously nonsensitive individual [40]. (2) The non-quantitative and subjective nature of grading a dermal reaction as a 0 to +3. This precludes the use of patch testing to discern more subtle, but statistically significant, group differences between potential study cohorts (e.g., patients with different kinds of implants) and incorporates the widely different opinions of clinicians on what constitutes a +1,+2, or +3 response. (3) Dermal testing may be affected by location-specific immunological tolerance (i.e., suppressed skin reactivity to implants but not peri-TMJ areas or vice versa) [35, 41]. (4) There may be person-dependent impaired host immune responses that are genetic, or environmental, e.g., concurrent medications affecting dermal reactivity [42, 43]. (5) The conditions of immune challenge during patch testing are also highly variable (i.e., non-standardized), where the environment of a patch test placed on a hairless area of the skin (typically the upper back) for 48–72 h is highly inconsistent from patient to patient and can be uncomfortable, where such aspects as cleanliness of the area and home environment are not standardized. (6) Finally, there are no well-established challenge concentrations/doses and methods for several orthopedic metals available in commercially available/approved patch test kits (e.g., Al, Mo, V, and Zr; see Table 9.1).

#### 9.1.2.2 Lymphocyte Transformation Testing

Metal lymphocyte transformation testing, LTT or metal-LTT, measures the proliferative responses of blood-drawn lymphocytes after they are exposed to specific metal antigens for 3–6 days, the time required for a delayed sensitivity response by the lymphocytes. These lymphocytes are obtained from a heparinized blood draw where the mononuclear cell fraction is isolated after centrifuging on a layer of Ficoll (density gradient separation). Proliferation of lymphocytes (both basal levels and those responding to antigen) are measured using a radioactive marker (i.e., [$H^3$]-thymidine) that is added to cultured lymphocytes. The incorporation of the

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**Table 9.1** The percentages of metals in different orthopedic alloys

| Alloy | Ni | N | Co | Cr | Ti | Mo | Al | Fe | Mn | Cu | W | C | Si | V |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Stainless steel (ASTM F138) | 10–15.5 | <0.5 | * | 17–19 | * | 2–4 | * | 61–68 | * | <0.5 | <2.0 | <0.06 | <1.0 | * |
| CoCrMo Alloys (ASTM F75) | <2.0 | * | 61–66 | 27–30 | * | 4.5–7.0 | * | <1.5 | <1.0 | * | * | <0.35 | <1.0 | * |
| (ASTM F90) | 9–11 | * | 46–51 | 19–20 | * | * | * | <3.0 | <2.5 | * | 14–16 | <0.15 | <1.0 | * |
| (ASTM F562) | 33–37 | * | 35 | 19–21 | <1 | 9.0–11 | * | <1 | <0.15 | * | * | * | <0.15 | * |
| Ti alloys | | | | | | | | | | | | | | |
| CPTi (ASTM F67) | * | * | * | * | 99 | * | * | 0.2–0.5 | * | * | * | <0.1 | * | * |
| Ti-6Al-4 V (ASTM F136) | * | * | * | * | 89–91 | * | 5.5–6.5 | * | * | * | * | <0.08 | * | 3.5–4.5 |
| 45TiNi | 55 | * | * | * | 45 | * | * | * | * | * | * | * | * | * |
| Zr alloy (95 % Zr, 5 % Nb) | * | * | * | * | * | * | * | * | * | * | * | * | * | * |

*Note*: Alloy compositions are standardized by the American Society for Testing and Materials (ASTM, vol. 13.01)

*Indicates < 0.05 %

radioactive marker into cellular DNA upon mitosis facilitates accurate quantification of proliferation responses through the measurement of incorporated radioactivity after 5–6 days of challenge (with 0.001–0.1 mM $Al^{+3}$, $Co^{+2}$, $Cr^{+3}$, $Mo^{+5}$, $Ni^{+2}$, $V^{+3}$, and $Zr^{+4}$ chloride solutions). The amount of radioactivity incorporated into dividing cells' DNA is measured after "harvesting" (collecting) cells onto a paper membrane using liquid scintillation measurement of radiation counts per minute (cpm). This is a very precise way to measure DTH proliferation responses because a small subset of antigen-activated cells that are proliferating can be easily discerned against a background of many non-activated (nonproliferating but viable) cells. All proliferation is compared to non-treated control cells from the same individual. A normalized proliferation or stimulation index is calculated:

$$\text{Proliferation index}\left(\text{factor}\right) = \left(\text{mean cpm with treatment}\right) / \left(\text{mean cpm without treatment}\right).$$

The use of LTT testing in the assessment of orthopedic implant-related metal sensitivity is growing. It is well established for testing hypersensitivity in a variety of clinical settings [44–49]. Some reports seem to indicate that LTT testing may be equal to, or better suited for, the testing of implant-related sensitivity than dermal patch testing [45] given that metal sensitivity can be more readily detected by LTT [50–57]. This is particularly important with orthopedic implants because high sensitivity (minimized false negatives) may be more critical than specificity (minimized false positives), thus erring on the side of caution. This is because there are equally efficacious commercially available orthopedic TJR implants made from different metals, and these different implants have similar reports of clinical success. Thus, it is more important to be able to detect more people who likely have metal sensitivity (at the expense of some false positives) since the risk of choosing a different better suited implant material carries little to no risk. In comparison, the sacrifice of method sensitivity for better method specificity (minimized false positives) carries with it the risk of missing the diagnosis of metal sensitivity and thus early TJR failure and the need for revision surgery.

Another advantage of metal-LTT is that soluble metal chloride challenge agents are complexed with serum proteins from the individual undergoing the LTT testing using autologous serum drawn at the same time [58–60]. These in vitro metal-protein challenge agents have been shown to be chemically similar to those produced in vivo [61–63]. However, the metal-protein complexes that are formed during patch testing produced by placing petroleum jelly with metal salts on the dermis remain unknown. Another advantage of metal-LTT testing is the highly quantitative results that are not physician/technician/operator dependent to produce or interpret (unlike patch testing). In LTT testing, a highly quantitative stimulation index is produced from multiwell replicates that enable calculation of an average and standard deviation for each metal challenge agent at multiple concentrations. This facilitates detection of dose-dependent responses (e.g., 0.001–1 mM of metal). Most immune responses are dose dependent where too little or too much will not induce a response. Testing at different concentrations provides a means of assessing those people who are sensitive at lower

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than normal (e.g., 0.01 mM) or higher than normal (e.g., 1 mM) concentrations of a metal challenge. This scenario is illustrated in Fig. 9.2 where LTT results of a metal-sensitive individual demonstrate dose-dependent increased reactivity to nickel. LTT testing is becoming more popular and is even more relevant than ever, due to the increasing numbers of TJR devices implanted.

However, there are some limitations to contemporary LTT testing. Metal solutions complexed with proteins may only approximate the kinds of products generated by corrosion and wear (see Chap. 10) during metal implant degradation [60, 62, 63]. It

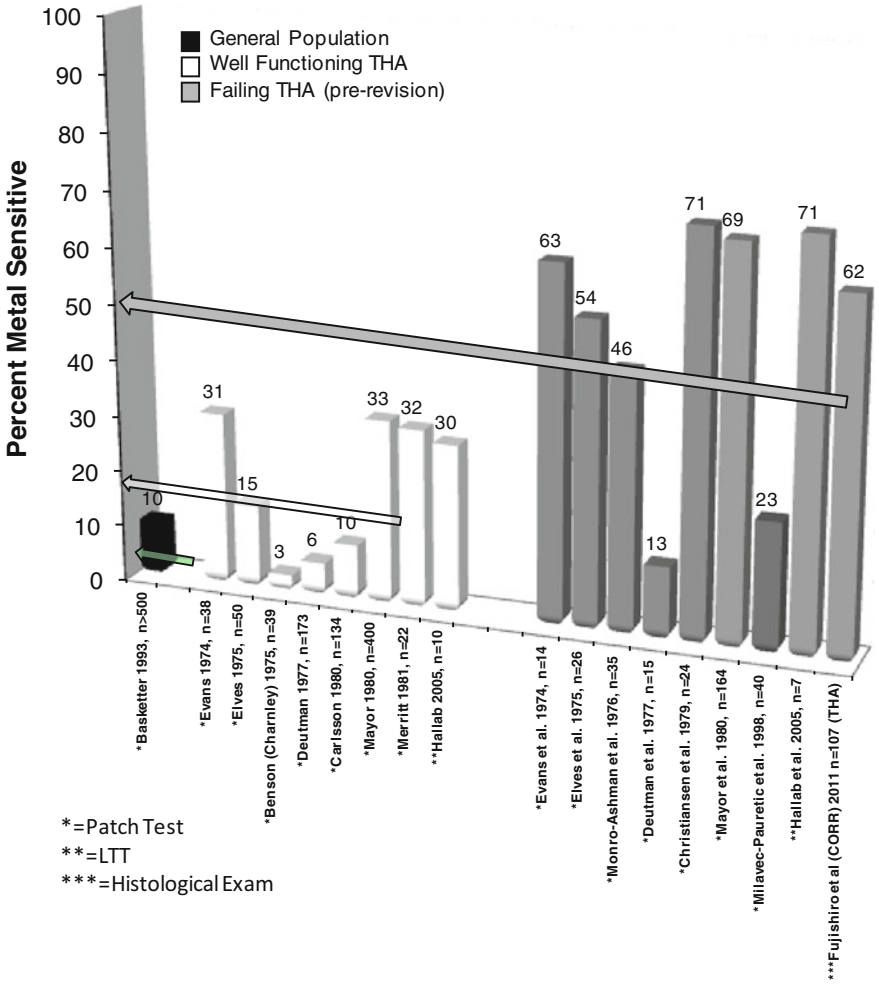


**Fig. 9.2** A compilation of investigations show the averaged percentages of metal sensitivity among the general population for nickel, cobalt, and chromium, among patients after receiving a metal-containing implant, and among patient populations with failed implants. All subjects were tested by means of a patch test, metal-LTT (lymphocyte transformation test), or histological diagnosis

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remains unclear what stimulation index number (i.e., threshold) best indicates a clinically relevant (or irrelevant) hypersensitivity response (i.e., an SI index of > 2 or > 3). In the past a stimulation index threshold of SI>2 ($p<0.05$) has been used to indicate mild drug sensitivity and metal hypersensitivity, and SI>8 was used to indicate severe metal reactivity, consistent with drug allergy literature over the last half century [28, 46, 52, 64, 65]. However, it remains unclear whether this criterion is too strict or too permissive.

Prospective, longitudinal clinical studies, such as the metal-on-metal study discussed in the following section, epitomize why both LTT and patch testing have clinical utility. Generally TJR implants with greater propensity to release metals in vivo are more prone to induce metal sensitivity. For example, failures of total hip prostheses with metal-on-metal bearing surfaces have been associated with metal sensitivity when compared to similar designs with metal-on-ultrahigh molecular weight polyethylene bearing surfaces [41, 66]. Many case and group studies indicate the clinical utility of metal sensitivity testing; some of these studies are summarized in the following.

## 9.2 Case Studies in Metal Implant-Related Metal Sensitivity

Many reports over the past 40 years have indicated metal allergy or sensitivity-type responses temporally connected to adverse clinical responses such as dermatitis, urticaria, vasculitis [67–72], and/or nonspecific immune suppression [42, 73–76].

One of the first studies of these pathological dermal metal reactions to the poor performance of a metallic orthopedic implant was made in the mid-1960s [77] where a nickel-containing implant was reported to be accompanied by dermal reactions characteristic of hypersensitivity. Since then there have been many case reports that link immune responses with metal implant-induced sensitivity responses in the cardiovascular [71, 78, 79], orthopedic [9, 67, 69, 70, 72, 80], plastic surgery [81], and dental [82–88] literature. In many of these reports, excessive early immunological reactions (aseptic inflammation) necessitated device removal, after which the associated immune reactions dissipated [67–72]. Clinically, severe skin reactions [68, 70, 71, 78–80, 89, 90] were seen accompanied by aseptic inflammation, surgical descriptions of metallosis (dark metallic staining of tissue due to excessive implant debris), periprosthetic fibrosis, and in some rare cases adjacent muscle necrosis [72, 91, 92].

In one of the earliest cases of metal implant sensitivity [69], a 20-year-old female developed a rash on her chest and back approximately 5 months after stainless steel screws were used to treat chronic patellar dislocation. While topical steroids managed this condition for 1 year, it eventually worsened to more generalized dermal eczema. When the stainless steel screws were removed, her dermal rash completely disappeared within 72 h [69]. The actual words of the report state, “the orthopedist still doubted that the steel screws could be the cause of her dermatitis and applied a stainless steel screw to the skin of her back. In a period of 4 h,

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generalized puritis and erythema developed." [69] The only diagnostic technology of the time (dermal patch testing) showed aggressive sensitivity reactions to nickel and the steel screw itself. Thus, early on the phenomena of metal hypersensitivity was demonstrated to be real in that it satisfied Koch's postulates, a key test for causality in medicine. According to this postulate, an agent can be considered as causative if when it is removed, the symptoms abate and when the patient is rechallenged, the symptoms return. Thus, metal sensitivity complications associated with implant materials was conclusively demonstrated nearly 40 years ago, albeit only in a case study. A large number of case studies followed demonstrating similar temporal and physical evidence of delayed-type hypersensitivity response reactivity to implant metals [9, 16, 67, 70, 72, 81].

There are generally more cases of stainless steel and cobalt alloy metal sensitivity and less to titanium alloy implants [9, 16, 67, 68, 70, 79, 80, 90, 93, 94]. An early case report of cobalt metal sensitivity indicated periprosthetic fibrosis, patchy muscular necrosis, and chronic peripheral inflammatory changes occurring several years after the initial implantation of cobalt alloy plates and screws during fracture fixation of a 45-year-old female's left radius and ulna [43]. This case demonstrated the extent of time it took to develop this kind of response. As is generally NOT the situation with such case reports, after the implant was removed and the symptoms (swelling) disappeared, the patient remained reactive to cobalt as indicated by patch testing [43].

## 9.3 Cohort Studies of Implant-Related Metal Sensitivity

The clinical utility of metal sensitivity testing can be largely attributed to the many retrospective studies that indicate a strong correlation between the performance of a metal-containing implant and metal sensitivity [9, 35, 41, 95–103]. These studies showed that the incidence of metal sensitivity among patients with elevated metal exposure with well-functioning implants is approximately 25 %, roughly twice as high as that of the general population (Fig. 9.3) [35, 41, 66, 94, 96, 98, 99, 102, 104]. This dramatically increases to 60 % in patients with a painful or poorly functioning implant [66, 94, 96, 98, 104]. Thus, the incidence of metal sensitivity in people with painful/failing implants is about sixfold that of the general population and approximately twofold greater than that of people with pain-free well-performing implants.

Specific types of implants that release more metal are more likely to induce metal sensitivity. Metal-on-metal total hip prostheses designs result in metal sensitivity to a greater extent than similar designs with metal-on-ultrahigh molecular weight polyethylene bearing surfaces [41, 66, 105]. New generations of metal-on-metal (MoM) total hip replacements generally have the advantage of lower overall wear than metal-on-polymer implants, but because of the release of more metal,

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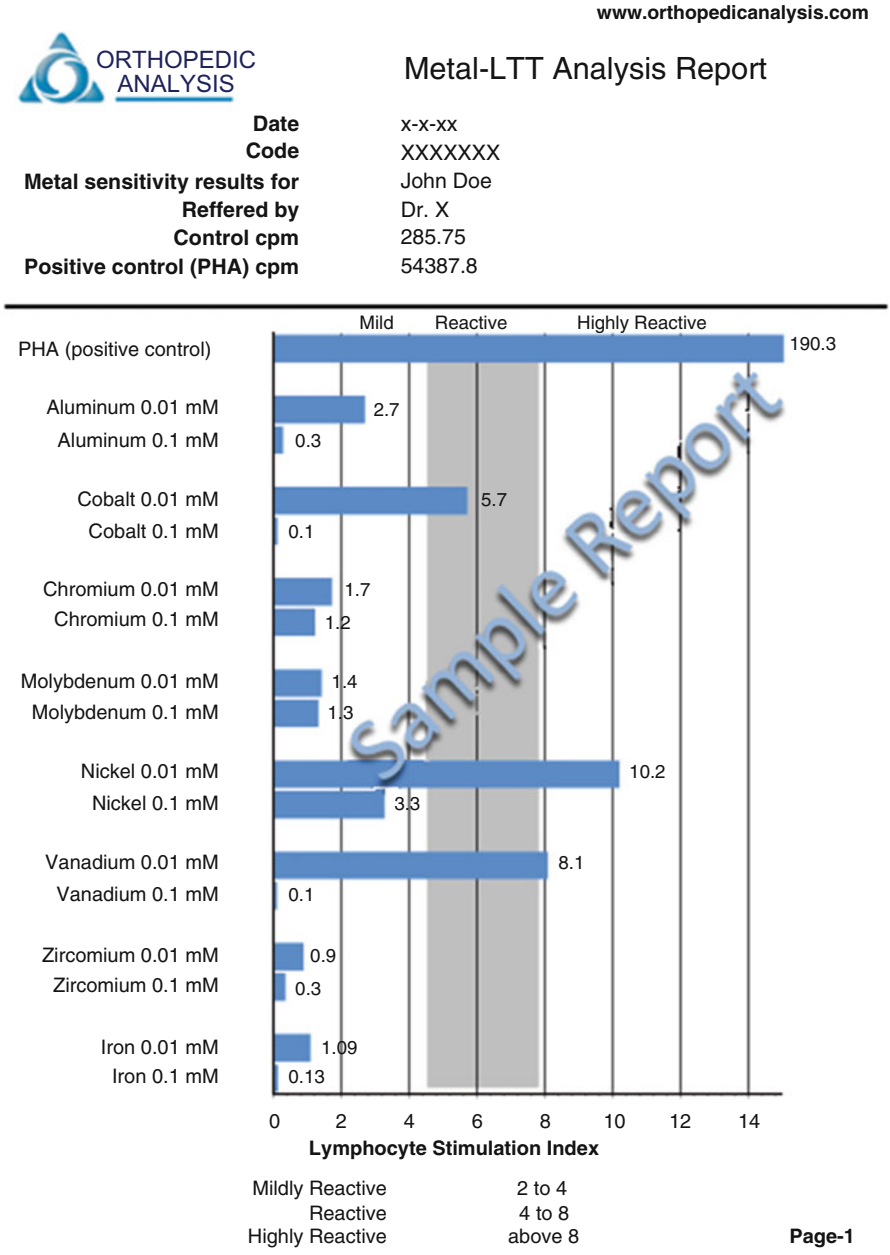


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**Fig. 9.3** Sample results of a metal-LTT (lymphocyte transformation test) indicate high reactivity to nickel at all 3 concentrations tested. Metals are generally used at 3 different concentrations of 0.001, 0.01, and 0.1 mM (courtesy of Orthopedic Analysis LLC)

there are more reports of short-term failures attributable to excessive inflammatory reactions with these devices and they have been the subject of significant litigation against implant companies that produced them. Hypersensitivity-like reactions have been reported to be as high as 76–100 % of the people with failing metal-on-metal total hip devices [106, 107].

Currently there is no hypersensitivity data on TMJ-TJR failures due to this type of bearing couple or any other. These metal-on-metal hip replacement device failure sensitivity responses include evidence of histological adaptive immune response inflammation, i.e., extensive lymphocyte infiltrates [106, 107]. Patients at early time points (<4 years) and some people with MoM implants developed metal sensitivity responses without evidence of implant pain and inflammation, adding further evidence that this condition is likely causal or contributory to the eventual high rates of failure of MoM THA implants [105]. One study reported a significant increase in metal sensitivity from 5 % pre-op to 56 % at 1–4 years post-op in people with well-performing (asymptomatic) MoM surface replacement hip arthroplasties. The same investigation of cohorts with asymptomatic MoM implants in place for longer than the prospective study group (i.e., >7 years on average) had an even higher average incidence of metal sensitivity at 76 %. This incidence of sensitivity, while high, is actually less than those previously reported for painful/symptomatic MoM patients (i.e., 81 % in failing MoM [108]). Thus, there is strong evidence that there is a causal or contributing relationship between local adaptive immune responses and the pathogenesis of MoM failure. Regardless of the role of the immune response in implant failure (which may not be generalized to individual patients), the overall findings support the use of sensitivity testing for assessing implant performance. In people with MoM THA implants, lymphocyte sensitivity responses to Co and Cr are not apparent at 3 months postoperatively (when serum levels of metal were already high), but seem to develop over time when systemic exposure levels are high (i.e., after 1–4 years, Fig. 9.4). However, this "gradual" increase in immune reactivity contrasts with relatively fast elevations in serum metal-ion levels (e.g., Co and Cr at 3 months postoperatively). This suggests that metal sensitivity responses to this MoM THA implants may develop over time and are related to metal ion exposure levels. Additionally, patch testing has been reported to not correlate at any time point with in vivo metal ion levels or other measures of metal-induced immune responses such as metal-LTT and flow cytometry or cytokine analysis [105]. Adding evidence to suggest that patch testing may not accurately reflect adaptive immune responses in the local implant environment.

Elevated levels of circulating metal ions corresponds to metal sensitivity responses: reports have shown that some MoM hip implants with radiographically identifiable large soft acellular fibrous tissue growths (termed pseudotumors) had a nearly twofold increase (80 % vs. 45 %) in the incidence of metal reactivity to Ni (LTT, SI>2) and had >5 fold increases in both Co and Cr serum ion levels, when compared to people with MoM implants without pseudotumors [109].

Pain levels associated with aseptic implants also correlate with metal sensitivity reactivity. One study has shown that the percentage of people with metal sensitivity (metal-LTT with SI>2) was significantly higher for people with more painful implants vs. non-painful (Fig. 9.5) [110]. Furthermore, when broken down into

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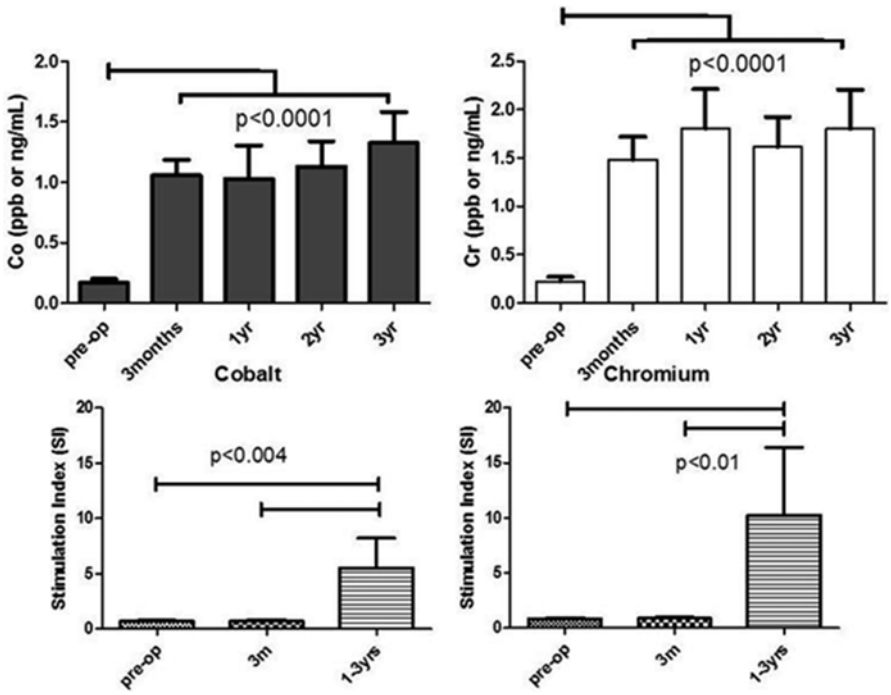


**Fig. 9.4** Metal ion levels of cobalt and chromium are shown increased as early as 3 months in serum in people with metal-on-metal hip arthroplasty implants. However, increases in metal reactivity as measured by lymphocyte proliferations (SI) were only increased after 1–3 years of metal exposure in the same people with metal-on-metal hip arthroplasties. All people with metal implants used in this study were asymptomatic ($n=21$) ($p<0.04$, Mann Whitney) (Adapted from Hallab et al. [105])

categories of metal-induced reactivity (i.e., mild ($2<SI<4$), moderate ($4<SI<8$), or high ($SI>8$) sensitivity categories) and compared with self-reported mild, moderate, and high pain levels, there were significantly different pain levels between people with moderate vs. high sensitivity levels (LTT) [110]. Conversely, people with TJA and no pain or low pain levels demonstrated a relatively low incidence of metal sensitivity (not significantly different, Fig. 9.5), indicating that pain level may be connected to lymphocyte-associated immune reactivity to metal implant degradation products.

## 9.4 Clinically Relevance

The clinical relevance of avoiding chronic inflammation associated with an adaptive immune response to metal implant products (metal sensitivity) is self evident, from both the point of pain and bone/joint homeostasis. However, other complications have been associated with chronic inflammation such as increased risk of cancer

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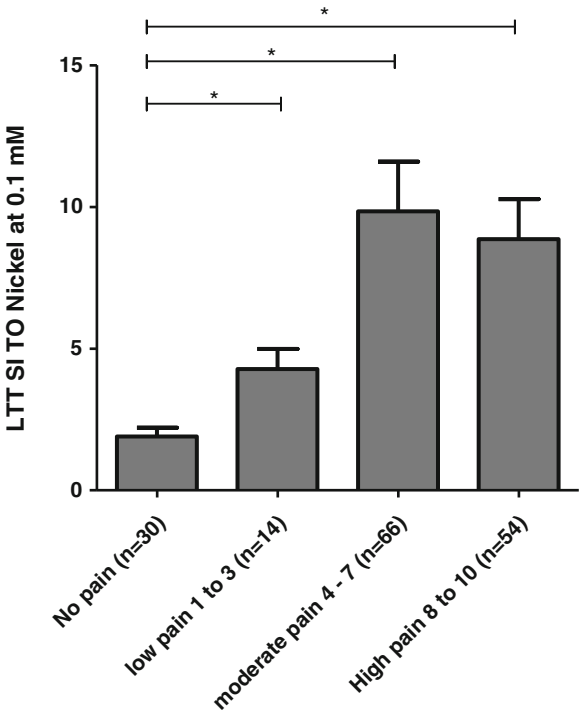


**Fig. 9.5** Incidence of nickel reactive subjects (LTT) according to self-reported pain levels in patients with no history of any allergy at a challenge concentration of 0.01 mM. Nickel reactivity in TJA subjects was based on their lymphocyte SI and was categorized as follows. Pain levels were denoted as follows in a scale of 1–10: no pain (0), mild pain (1–3), moderate pain (4–7), high pain (8–10). To obtain the incidence of metal reactivity, the percentage of subjects nonreactive, mildly reactive, reactive, and highly reactive to nickel at 0.01 mM concentration were calculated within their respective pain level group. No pain ($n=30$), mild pain ($n=14$), moderate pain ($n=66$), high pain ($n=54$) (courtesy of Orthopedic Analysis LLC)

[111]. In one study increased cancer risk was shown in animal models of dermal metal sensitivity (allergic contact dermatitis, a metal-DTH response) [111]. However, the degree to which this risk manifests itself with DTH metal responses to implants is unknown. A pervasive problem in orthopedics due to the pain and poor implant performance associated with such metal-DTH allergy responses where the resultant revision surgical intervention limits the length of chronic inflammation/exposure is not likely.

The clinical utility of sensitivity testing two categories of patients seems clear: (1) people with a known history of metal sensitivity and (2) people with a painful implant where infection has been ruled out. Metal sensitivity testing is a direct measure of immune cell reactivity to metal-serum complexes. It is mechanistically linkable to implant pathology and is not merely the correlation of a biomarker with elevated implant metal reactivity or failure. Immune reactivity to metal is both correlated and mechanistically linkable with implant performance, and thus, diagnostic

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assay measurement of metal-associated immune function if detectable, reproducible, and quantifiable represents a useful clinical tool available to physicians. Metal sensitivity testing is a direct test of an individual's immune response to metal-protein challenge, and the results indicate levels of immune reactivity that have been used for the past half century to diagnose delayed-type hypersensitivity responses drugs (such as antibiotics) and the persistence/effectiveness of vaccines such as tetanus toxin. Thus, once a sensitivity response to an implant metal is initiated (either before or during aseptic implant failure), that response directly contributes to inflammation and most likely dominates the cycle of further implant failure. Thus, the question of whether preexisting or developed metal sensitivity initiates the pain, loosening, etc., is moot once sensitivity-type immune response is established and a vicious cycle feedback loop is formed, where a loose implant causes more metal to be released which causes greater inflammation. It is well established that metal-stimulated lymphocytes can participate in the pathogenesis of aseptic osteolysis through the release of powerful cytokines such as IL-2, IFN-γ, and RANKL (receptor activated NF-KB ligand), which can both directly and indirectly increase bone resorption and inhibit bone deposition Fig. 9.6 [112].

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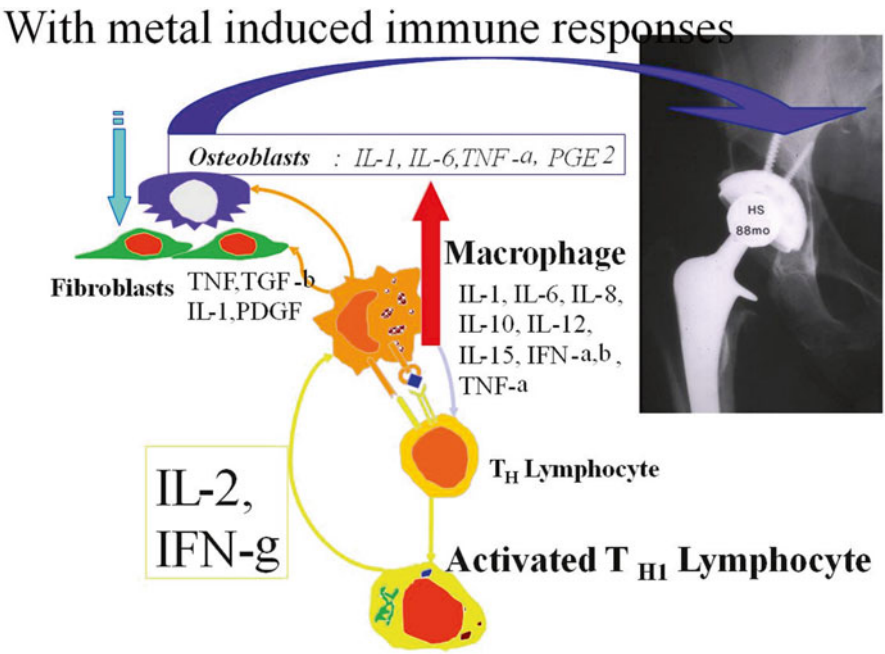


**Fig. 9.6** Metal-induced immune responses can be due to both innate immune (e.g., macrophage) or adaptive (e.g., lymphocyte) immunity. Adaptive immune responses (i.e., hypersensitivity) can negatively affect bone homeostasis both directly and indirectly leading to osteolysis (courtesy of Orthopedic Analysis LLC)

## 9.5 Conclusions

The effect of implant debris on the immune system generally has one of three possible outcomes: (1) metal degradation products are immunogenic [28, 33, 113, 114], (2) metal degradation products are immunosuppressive [115–117], or (3) metal degradation products are immuno-neutral (i.e., non-bioreactive) [118, 119]. All three possibilities have been shown to occur in case and group studies. The type of reaction that will occur in any individual is dependent on both genetic and epigenetic factors.

The key immune cells in a metal sensitivity response are CD4+ lymphocytes, which traffic locally through the periprosthetic space. After ingestion and processing of metal-protein complexes by antigen-presenting cells (such as macrophages), the relevant lymphocytes proliferate and activate, which can dominate the cascade of inflammatory events leading to osteolysis and aseptic loosening. Potent pro-inflammatory cytokines are released in this scenario such as IL-2, IFN-gamma, and RANKL that can activate osteoclasts directly (increasing bone resorption) and inhibit osteoblasts (decreasing bone production). Thus, as the number of patients receiving joint replacement implants including TMJ-TJR implants continues to grow and the clinical specialties expected to evaluate this phenomena widen, metal sensitivity testing offers a relatively risk-free additional tool in the armamentarium of physicians seeking to optimize implant success.

Not all people who test positive to metal sensitivity will proceed to early implant failure if their implant has the offending metal as a constituent. In fact, it remains unclear to what extent positive results to sensitivity responses to metallic biomaterials affect orthopedic implant performance [120, 121], but new evidence continues to demonstrate that concrete relationship and benefits of sensitivity testing improve success rates and implant performance [16, 24, 122].

It is clear that some people experience excessive immune reactions to the metals released from implanted metallic materials [9, 67, 69, 70, 72, 80]. Metal sensitivity testing is currently the only form of testing to discern those individuals that are highly susceptible to excessive metal-induced immune responses (lower estimates of this are about 1–5 % of general joint replacement recipients) [36]. Metal- LTT likely provides greater sensitivity relative to patch testing, but the clinical outcome studies needed to validate the sensitivity and specificity of patch or LTT testing (i.e., a clinically identifiable pathology) are still in progress [105, 108, 109]. Because metal sensitivity testing is a highly complex immune test, is it very important that testing facilities are both experts with this type of immune testing and have expertise in orthopedic/biomaterials in order to adequately advise attending physicians. They should also be able to fully disclose all testing parameters/methods/protocols to physicians, researchers, and the general public. Physicians ordering this testing should be familiar with criteria such as (1) test conditions, including challenge agents (soluble metal ions and metal particulate), culture medium, time of incubation, etc.; (2) method of proliferation detection; (3) whether autologous serum is used for culturing or if human AB pooled serum is used to supplement human cell cultures; (4) if there is statistical assessment or an acceptable level of redundancy, e.g., triplicate, duplicate, etc. in the assays; (5) the pharmacologic profile of the patient at the time

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of testing; and (6) if there is strict adherence to all patient privacy and Health Insurance Portability and Accountability Act regulations, required by law.

Given that <1 % of the over one million people receiving total joint replacement implants in the USA annually are metal sensitivity tested pre-op or at revision, it is likely that implant-related metal sensitivity has been underreported and remains underestimated. However, the slow and continuing improvements in sensitivity testing technology and availability will likely continue to provide accumulative clinical evidence into the utility of metal sensitivity testing along with more basic understanding into how and when metal sensitivity develops.

There are reports that patients receiving implants who are diagnosed preoperatively by metal sensitivity testing have better outcomes than those where sensitivity testing results are not accommodated by altered surgical procedure [122]. It is clear that more studies are needed to build a consensus and confirm the clinical utility of pre-op and/or post-op testing. However, as more reports build a scientific foundation, there will be increasing attention paid to the phenomenon of metal sensitivity. Many surgeons now take this testing into account when deciding what type of implant is optimal for each patient. Optimizing implant and material selection tailored to individual immune reactivity profiles is important given that >1 in 4 older Americans will eventually require a joint replacement implant and the increasing need for TMJ-TJR [123–125]. With the specter of early poor performance and revision surgery mortality risk of >10 % when over the age of 75 [126, 127] for THA, appropriate preoperative testing can extend implant performance and save lives.

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# Chapter 10
# Tribocorrosion and TMJ TJR Devices

**Mathew Mathew, Shelley Kerwell, Maria Alfaro, Dmitry Royman, Valentim Barao, and Sukotjo Cortino**

## 10.1 Introduction

This chapter discusses the role of tribocorrosion alloplastic total temporomandibular joint replacement (TMJ TJR). Tribocorrosion is a relatively new field of physical science research in which two degradation processes, mechanical wear and electrochemical responses to that wear, are studied. Understanding these processes is essential in preventing joint replacement device complications and failures. The fundamentals of tribocorrosion, general testing methodologies and testing protocols, and results from a TMJ TJR retrieval study will be included as evidence of tribocorrosion in TMJ TJR devices.

## 10.2 Tribocorrosion: Definition and Fundamentals

Tribocorrosion deals with two separate scientific domains: "tribology" [1, 2] and "corrosion" [3–6]. As an established branch of mechanical engineering, tribology is the science of two contacting surfaces in relative motion and the consequences

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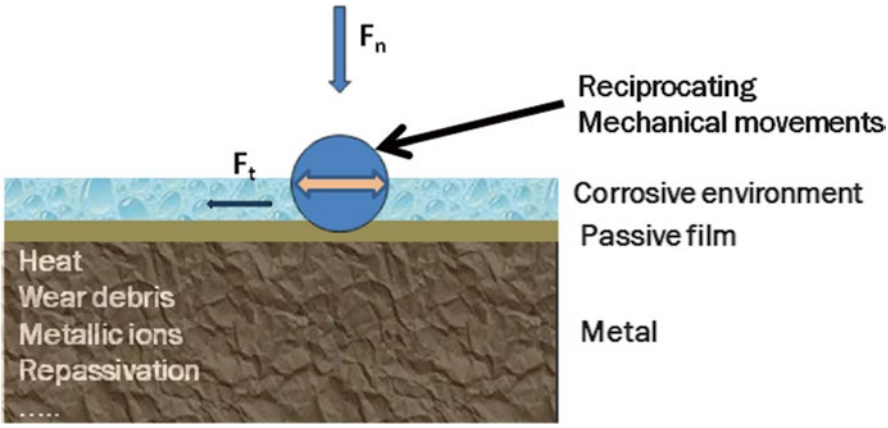


**Fig. 10.1** Principle of tribocorrosion

of wear, friction, and lubrication occurring at this interface (Fig. 10.1) [2, 7–9]. The prefix "tribo" originated from the Greek word "tribos," which means rubbing. Otherwise known as an electrochemical process, *corrosion* is the degradation of metals into their constituent atoms due to chemical reactions occurring within their surroundings. This leads to the development of a simple definition of *tribocorrosion*, which could be stated as: "A science related to the surface degradation mechanisms and processes when mechanical wear and chemical/electrochemical reactions interact with each other [10]." Further, bio-tribocorrosion is directly related to the application of tribocorrosion within a biological environment, particularly implants used in orthopedics and dentistry (Fig. 10.2).

One of the important features of tribocorrosion is its interdisciplinary nature. The investigation of tribocorrosion includes disciplines of tribology, corrosion science, material science, and clinical science. This provides a common platform for experts from the aforementioned disciplines to explore mechanisms of material degradation.

Although "tribocorrosion" is a relatively new subject, it has a long history. In the eighteenth century, Faraday observed potential changes of surfaces under applied friction, while the material surface was exposed to mechanical sliding. In 1960, reports from Germany researchers mentioned the possible effect of tribocorrosion in material degradation terming this phenomenon as: tribo-oxidations, mechanical-oxidations, and tribo-electrochemistry. In 1992, Celis et al. [11, 12] reported that sliding and corrosion influenced the thin film coating on the surface of materials which brought this area of research to the forefront. They called it tribo-electrochemistry. In the biomedical field, it was termed "mechanically assisted corrosion" (MAC), particularly in orthopedic hip retrieval studies [13, 14]. Over the last 20 years, this discipline has developed and has been lauded for its industrial, biomedical and commercial applications [7, 15–17].

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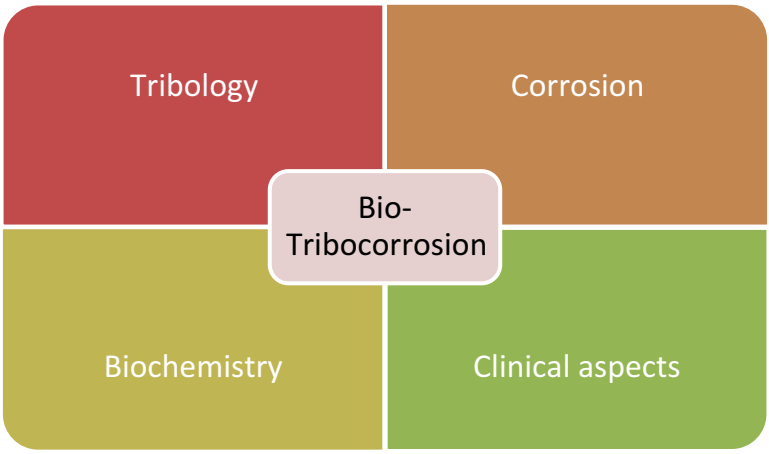


**Fig. 10.2** Bio-tribocorrosion definition

## 10.3 Tribocorrosion: Basic Principles

A tribosystem is concerned with the electrochemical environment, as shown in Fig. 10.3. Tribology, when coupled with corrosion, is able to induce solid particle erosion, abrasion, cavitation erosion, and fretting of material surfaces. Tribocorrosion is encountered when two or more materials undergo mechanical motion, such as rubbing, or sliding motion. The rate of material surface corrosion and wear is not yet fully understood; however, key factors are known to influence material and surface corrosion such as the properties of the material and local environment. In a tribocorrosion system, the surface of the material is able to counter the effects of the environment. Strong corrosion-resistant surfaces are able to form thin oxide surface films to create a barrier that can prolong electrochemical charge transfer between the environment and the material. However, as the material undergoes mechanical motion, the thin oxide film begins to erode and diminish allowing environmental factors to come directly into contact with the bulk material. As a result of this loss of the thin oxide layer, an electrochemical charge transfer between the material and the surrounding environment causes material degradation and corrosion to occur. Evidence of corrosion includes, but is not limited to, surface pitting with variable material grain size and location, indentations, and bidirectional boundary lines.

## 10.4 Tribocorrosion Testing in the Laboratory

The details of a laboratory tribocorrosion apparatus are shown in Fig. 10.3. A custom-built tribocorrosion cell is used to contain the electrolytic media (i.e., simulated joint solution) and hold the electrodes in place. Generally, four types of corrosion tests are recommended in tribocorrosion testing [9, 12, 16, 17].

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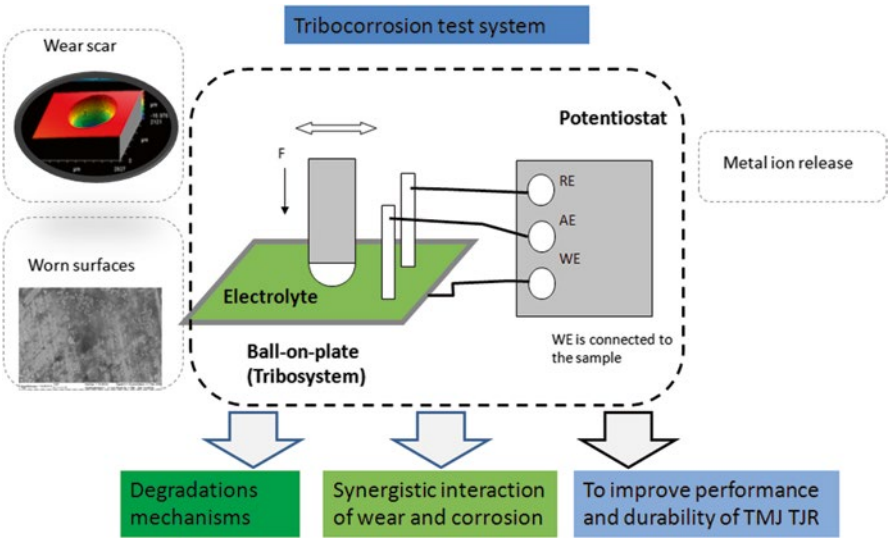


**Fig. 10.3** Schematic diagram of the basic tribocorrosion setup and basic output variables and main objective of the study

(1) Cathodic condition: A cathodic potential is applied and the current is monitored. (2) Free potential: The evolution of potential is measured and monitored, (3) Potentiostatic test: A specific potential is applied (typically anodic, E-passive, $E_{corr}$ (corrosion potential), or any selected potential while current is monitored, (4) Potentiodynamic test: Is a shorter test, in which a potential scan is made during tribo-tests.

During testing, it is possible to apply a potential and monitor the current generated as a function of time [18]. The selection of a potential is based on the Potentiodynamic curves generated during the basic corrosion tests (with tribological events). Before and after the testing motion, electrochemical impedance spectroscopy (EIS) measurements are performed in frequency ranges from 100 kHz to 10 mHz, with an AC sine wave amplitude of ± 10 mV applied to the electrode at its corrosion potential [7]. During tribocorrosion testing, evolution of current or potential is monitored as shown in Fig. 10.4.

Volume loss can be estimated based on profilometry measurements of the wear scar using a laser scanner (Smartscope, OPG Inc.). To determine the amount of metal released into the electrolyte media, solution samples (minimum of 1.5 mL) are taken at two discrete time points. The determination of metal ion content is determined by atomic absorption (GF-AAS) and mass spectrometry (high-resolution ICP-MS).

Examinations of the corroded and worn surfaces play a vital role in understanding wear mechanisms and material degradation processes. An optical microscope and scanning electron microscope (SEM) are used to establish primary surface characterization. Energy dispersive spectrometer (EDS) and X-ray photoelectron spectroscopy

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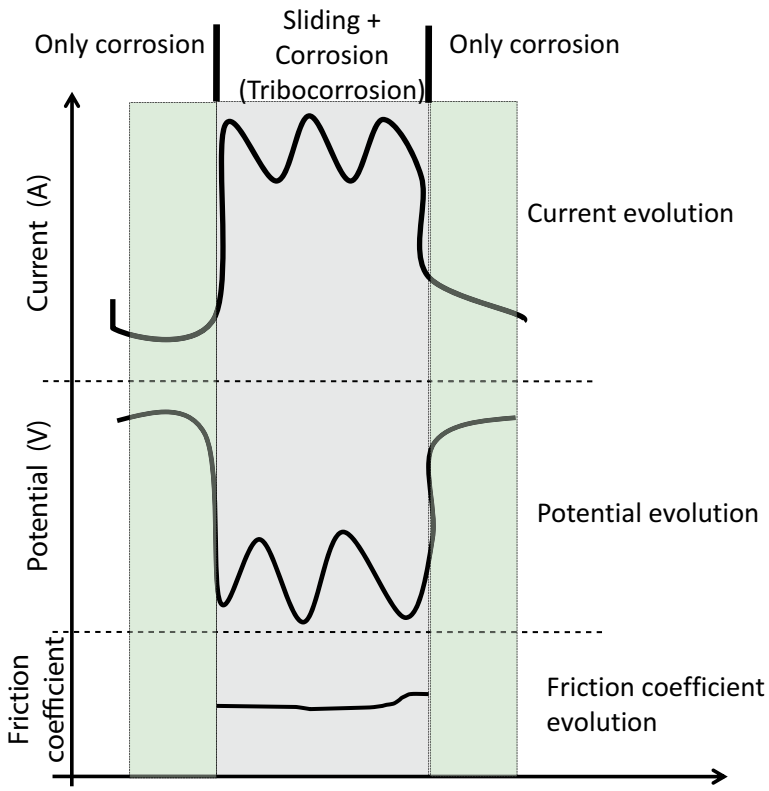


**Fig. 10.4** Typical data collection from tribocorrosion test (Current, Potential, Friction coefficient)

(XPS) techniques are employed to evaluate oxide film formation and composition. Finally, atomic force microscopy (AFM) and white light interferometry (WLI) microscope are employed to capture three-dimensional images of the corroded surface, particularly from surfaces with pits.

## 10.5 Tribocorrosion: Synergism or antagonism

The main challenge of studying tribocorrosion is understanding the synergistic interaction of wear and corrosion, which generates wear debris and metal ions as shown in Fig. 10.5. Quantifying the effect of corrosion on wear, the effect of wear on corrosion, or both scenarios, could lead to either beneficial or detrimental effects on the degradation process. The testing results are used to construct the tribocorrosion maps/models.

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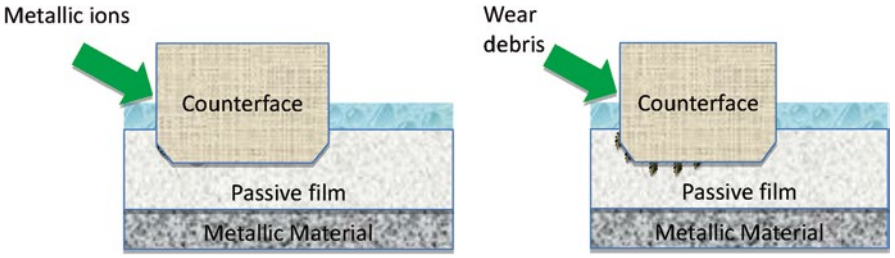


**Fig. 10.5** Synergistic interactions in the tribocorrosion process, generation of metal ions and wear debris

The following terms describe the synergistic effects in tribocorrosion interactions [5, 7–9, 19].

The total wear volume loss, $K_{wc} = K_w + K_c$, can be split into two components:

$$K_{wc} = \left(K_{wo} + \Delta K_w\right) + \left(K_{co} + \Delta K_c\right)$$

$K_{wo}$ = Wear rate in the absence of corrosion
$\Delta K_w$ = Change in the wear rate due to corrosion
$K_{co}$ = Corrosion rate in the absence of wear
$\Delta K_c$ = Change in the corrosion rate due to wear

These terms assist in quantifying the synergistic interactions of corrosion and wear. Therefore, synergistic analysis is the first step used to understanding the mechanisms involved in any degradation process.

## 10.6 Tribocorrosion: Current Status

Since tribocorrosion is a relatively new field of study, many experiments are under way to understand the mechanism of corrosion and mechanical wear. Works conducted by Landolt et al. in 2001 indicated the importance of electrochemical methods to tribocorrosion. His group controlled mechanical parameters and the contact geometry of the materials. The findings indicated how mechanical parameters and contact geometry affect the electrochemical response of the system when a known electrode potential is applied.

Additionally, the function of thin oxide layer films and coatings on material surfaces under tribocorrosion testing is currently being explored. Wood et al. [20] reviewed the tribocorrosion integrity of such coatings and their degradation under wear–corrosion. Some of the major factors influencing coating performance include microstructure, defect level, cohesion, adhesion, and substrate properties.

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Four tribocorrosion symposiums have taken place, under the leadership of Prof. Margaret Stack (University of Strathclyde, Glasgow UK). She also leads a tribocorrosion network, which is an international network of scientists who are actively involved in tribocorrosion research.

Due to clinical concerns regarding total hip replacement, tribocorrosion research expanded into the orthopedic community. This area is specifically called biotribocorrosion [2]. Simulated in vitro tribocorrosion hip simulators and experimental protocols have been developed at Leeds University (UK) [8, 16, 17, 21] and Rush University (Chicago, USA) [8, 16].

Similarly, wear and corrosion influence the early failure of dental implants, and several researchers are involved in the investigation of in vitro simulated oral environment studies [22, 23].

### *10.6.1 Evidence of Tribocorrosion from TMJ TJR Retrieval Studies: Preliminary Observation*

In the TMJ TJR community, clinical concerns related to metal ion release and its relation to failure of these devices have increased. Hence, research activities have been initiated as a collaborative effort between Rush University (Chicago, USA) and University of Illinois at Chicago—College of Dentistry (Chicago, USA) [24].

Thirty-one TMJ TJR samples were collected from two independent sources: (1) a group of international TMJ surgeons and (2) the retrieval collection of the TMJ Implant Registry and Repository located at the University of Minnesota. The inventory is comprised of one group of non-implanted devices (Control) and three groups of failed retrieved TMJ TJR devices: Group 1—comprised of 3 never implanted control metal (CoCrMo) fossa-on-metal (CoCrMo) condyle TMJ TJR devices (MoM Control), Group 2—consisted of 19 failed retrieved metal (CoCrMo) fossa-on-metal (CoCrMo) condyle devices (MoM Retrieved), Group 3—consisted of a total of 7 failed retrieved Metal (CoCrMo) fossa-on-polymethylmethacrylate (PMMA) condyle devices (MoP Retrieved), and Group 4—consisted of 2 titanium nitride coated fossa-on-condyle devices (TiN Coated Retrieved). The implant inventory (Table 10.1) also provides the number of years each implant was placed

**Table 10.1** Characteristics of TMJ prosthesis devices

| | Manufactures | | |
|---|---|---|---|
| Components | Nexus CMF | TMJ Concepts | Biomet Microfixation |
| Fossa | CoCrMo | Ti (UHMWPE surface) | UHMWPE |
| Condyle | CoCrMo | CoCrMo | CoCrMo (Ti surface) |

*CoCrMo* Cobalt–Chromium–Molybdenum alloy, *UHMWPE* Ultrahigh-molecular weight polyethylene, *Ti* Titanium

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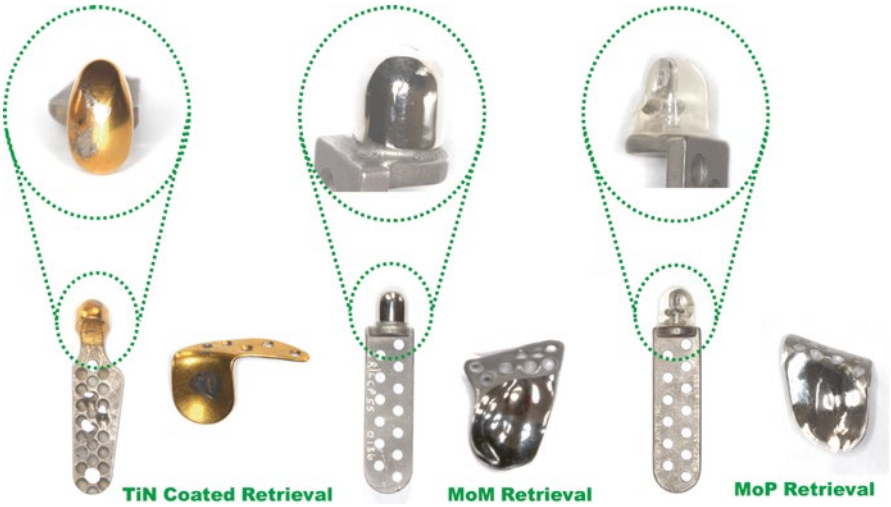


**Fig. 10.6** Evidence of tribocorrosion from TMJ TJR implant surfaces

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in vivo before removal. The type and total number of TMJ TJR retrieved implants are displayed in Fig. 10.6.

In order to evaluate the areas of contact between the condyle and the fossa and peripheral areas of the bearing surfaces, a standardized orthopedic retrieved TRJ protocol was employed. For each device, clinical history, type of material, years of implant service, and origin implant were collected. SmartScope optical images done at 93.5× magnification of the condylar heads of each implant type are shown in Fig. 10.7a–d. These images reveal that the MoM Control condyle demonstrated bidirectional scratches (Fig. 10.7a). This may be due to the polishing protocol used by the specific manufacturer. The MoM Retrieved displays significantly more scratching than the MoM Control (Fig. 10.7b). This indicates that these bearing surfaces sustained damage due to wear when the device was under functional loading in vivo. The TiN Coated condyle displays signs of surface delamination due to surface fatigue (Fig. 10.7c). Lastly, the MoP Retrieved also indication signs of surface scratching and wear, similar to the MoM Retrieved (Fig. 10.7d).

White light interferometry (WLI) images of a corresponding MoM Control condyle and fossa are shown in Fig. 10.8a and b Although this is a Control, visible signs of grooves and scratches can be seen as indicated by blue and green-colored areas. Hence, it is evident that the bearing surfaces of TMJ TJR devices are affected by in vivo wear and corrosion processes during joint function.

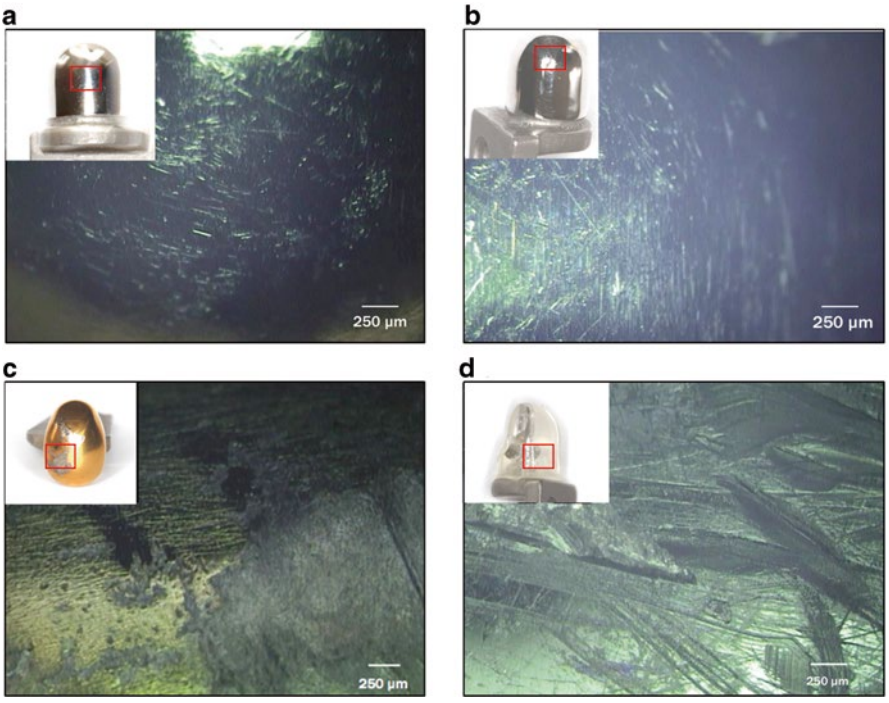


**Fig. 10.7** Smartscope images. (**a**) MoM control condyle. (**b**) MoM retrieved condyle. (**c**) TiN coated retrieved condyle. (**d**) MoP retrieved condyle

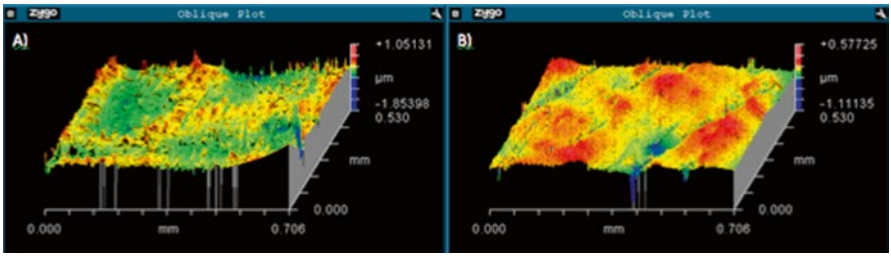


**Fig. 10.8** White light interferometry of MoM Control condyle and fossa. The image shows the scratches and pits due to the corrosion and wear, generated from the TMJ TJR in vivo functional motion

## 10.7 TMJ TJR and Hip Replacement in Orthopedics: Tribocorrosion Research

Unlike the orthopedic community, tribocorrosion research in the TMJ TJR system is still relatively new. The materials utilized for manufacturing these implants (titanium, cobalt–chromium–molybdenum (CoCrMo), and ultrahigh-molecular weight

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polyethylene (UHMWPE)) were selected to minimize wear and fragmentation and to reduce the potential for the development of foreign body reactions [20–22].

TMJ TJR devices consist of a fossa and condyle, which are similar to the acetabulum and femoral components in a total hip replacement (THR) system. In a TMJ TJR device, the ball is smaller in diameter (10 mm) compared to the 28–52 mm diameter found in standard THR systems. These two joints also exhibit differences in functional performance, kinematics, and loading conditions [25, 26]. Currently, there are no available methods to accurately measure TMJ forces in human subjects. Given the difficulties in direct measurement of TMJ forces in animals, it is clear that direct measurements in humans may not be feasable [25]. It has been estimated that the natural masticatory loads range between 250 to 450 N. Investigators have estimated the loads transmitted to the TMJ to be roughly half of masticatory loads (125 to 300 N) [27].

The maximum bite force for an average male is normally 300 N, while the maximum bite force for the average female is normally 210 N. Therefore, the average expected load on TMJ implant is 100–150 N. In contrast, the forces in THRs can be up to 2500 N [25, 26, 28]. Due to the anatomy of the TMJ, it is considered to be unconstrained joint more like the knee than the hip [23, 24].

## 10.8 Tribocorrosion: Future Perspective

Tribocorrosion is a relatively new approach in TMJ TJR research. Therefore, studies should start with retrieval analysis of failed TMJ TJR devices, simulated in vitro wear, and corrosion testing under loads determined by finite element analysis (FEA) models (Fig. 10.9). Further investigation into the response of surrounding tissues to wear generated metal ions and debris, in addition to understanding the synergistic interactions of wear–corrosion on the longevity of these devices [29–32] (Fig. 10.5).

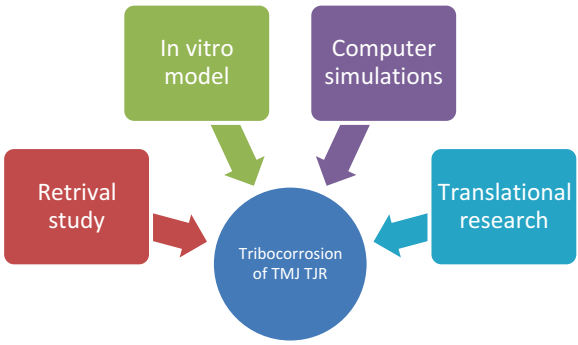


**Fig. 10.9** Future scope of TMJ tribocorrosion research

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Many studies have been reported on tribocorrosion in the THR [14–17, 19]. Hence, the possibilities of translational research between orthopedics and TMJ TJR should be pursued, encorporating the knowledge from orthopedic device research to TMJ TJR research.

Tribocorrosion can occur in a variety of ways, such as the repeated rubbing of metal components against each other, in turn affecting the protective passive oxide layer formed on the surface of the metallic portion of the implant. Corrosion can also be caused by the spontaneous breakdown of the passive film on the exposed area of an implant without any mechanical stimulation. Metal ions resulting from the tribocorrosion phenomenon have also been shown to decrease DNA synthesis, mitochondrial dehydrogenase activity, mineralization, and mRNA expression of alkaline phosphate. There have also been traces of metal ions found in the liver, lungs, and lymph nodes [32, 33]. Additionally, the debris caused by corrosion and material wear can promote peri-implant tissue reactions, jeopardizing both the mechanical stability of the device components and the longevity of the implant [34–36].

In 2013, the Food and Drug Administration (FDA) ordered the three manufacturers of TMJ TJR devices to conduct post-market surveillance studies to determine the length of time before their implants are removed or replaced due to pain or failure [37–40]. Some studies that examined the peri-implant tissue of retrieved TMJ implants found wear debris from the breakdown of the implant due to wear and corrosion processes [24, 41–44]. As this chapter shows, there is evidence of early device failure associated with corrosion and wear of the implant materials. Hence, systematic wear–corrosion studies (tribocorrosion) are required to understand the degradation mechanism of TMJ TJR devices.

## 10.9 Summary

This chapter provides a review of tribocorrosion and its prospective applications in the study of TMJ TJR device interactions with peri-implant tissues. The progress made in THR studies is extensive; hence, the already acquired knowledge should be translated to improve TMJ TJR. However, the biomechanics of the TMJ must be considered in tribocorrosion studies. Further tribocorrosion studies of the TMJ TJR devices may provide answers to questions related to the peri-implant tissue reaction and their relationship to periarticular infections, bone formation, cellular damage, and material hypersensitivity.

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# Chapter 11
# Management of Failing and Failed TMJ TJR Devices

**Louis G. Mercuri**

## 11.1 Introduction

It is unlikely that an alloplastic joint with an infinite life span can be developed. All alloplastic joint prostheses will develop wear under functional loading. This wear can be decreased, thus prolonging the life of these devices by using the appropriate materials especially at the articulating interfaces, proper implant design and articulating geometry, development of osseointegration of the components with stable fixation from the time of implantation, and eliminating parafunctional loading. Despite attempting to control wear-promoting factors, eventually wear and its sequelae will result in the revision of all alloplastic total joint replacement (TJR) devices [1].

Since the vast majority of TMJ replacement patients are reported to be younger than their orthopedic counterparts, planning for future revisions of these devices is appropriate [2–14]. Using the orthopedic experience with revision arthroplasty, this chapter presents a paradigm for the revision of failed and failing total alloplastic TMJ replacement (TMJ TJR) devices.

## 11.2 Classification of Failures

To be able to diagnose a failing or failed device, surgeons must understand the reason a device is failing or has failed so that the cause of that device failure is not repeated with its revision or replacement.

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Schmalzried and Brown classified orthopedic alloplastic joint device failures as a result of one or more of the following categories: (1) failure of the concept; (2) failure of embodiment; (3) failure of the implantation technique; or (4) limitations of technology [15]. Defining and applying each of these to TMJ TJR devices is both appropriate and instructive.

### *11.2.1 Failure of the Concept*

To be considered a conceptual failure, the failure must be independent of the device design and/or materials. It results because the device cannot satisfy the fundamental principles of anatomy, physiology, immunobiology, and/or mechanics.

Conceptualizations that include cantilevers, multiple moving parts, or muscle and/or bone attachments that violate the mechanical and immunobiological principle of this joint would fall into this category.

Hemiarthroplasty, a metallic bearing surface articulating with normal articular cartilage, is frequently utilized in orthopedic surgery for fractures of the hip and shoulder in geriatric patients. The surgery can be quite successful in such cases where functional demands are low; however, over time the metallic component against the articular cartilage causes cartilage wear and may cause pain, requiring total joint replacement. For this reason, hemiarthroplasty is generally not performed in young patients or in patients with preexisting degenerative joint disease [16, 17] (Fig. 3.6).

### *11.2.2 Failure of Embodiment*

Embodiment is the specific application of a concept. It includes the variables of implant design, materials from which the component parts are made, manufacturing tolerances and surface finishes, and the type but not the quality of the fixation. Failure of a single feature of an embodiment can result in failure of the reconstruction even though the other variables were satisfactory. The classic example of this category is polytetrafluoroethylene (PTFE or Teflon). It was introduced in 1958 by Charnley because it was thought to be a good candidate in joint reconstruction because of its low friction characteristics and inertness, but it failed because of its unfavorable wear properties and unacceptable tissue reaction resulting in wear products [18].

Polytetrafluoroethylene (PTFE or Proplast PTFE with added carbon fibers) is the most inert of plastic materials. Although possessing an extremely low coefficient of friction, PTFE has poor wear properties. PTFE represents a classic example of how tissue responses can differ when the body is exposed to the bulk form versus the particulate matter of the same material. Although the tissue reaction to the solid PTFE was quite minimal and possibly better than any other material, the wear debris particles provoked a serious histolytic and giant-cell response that resulted in loosening and failure of the prosthesis [19] (Figs. 3.3 and 3.4).

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Polymethylmethacrylate (PMMA) is another example. The first prosthesis to utilize this polymer was the Judet hip implant [20]. Unfortunately, surface abrasion and stem breakage led to disastrous failures due to massive osteolysis from wear particles [21, 22]. PMMA is a combination of pre-polymerized grains and un-polymerized monomer that is mixed and caused to polymerize into a peri-implant grout by the use of catalysts, in addition to the heat generated during polymerization [23] (Fig. 3.5).

It is known that failure of total polymerization will result in residual unfused particles that will be available at the surface for phagocytosis when the body recognizes them as foreign particles. In addition, the various irregularities of the cement as it flows into the bone will produce a variety of fragments and protruding masses of varying sizes. Some of these cement protrusions can be sites of mechanical disruption during cyclical loading and fractures can occur resulting in mechanical abrasion and the formation of particles. When particles of polymers are available to the tissue for phagocytosis, it appears that the number and size of the particles are as important as the presence of the material itself. Large particles that cannot be phagocytized even by multinucleated hystiocytic giant cells tend to remain in situ and be encased by giant cells. In effect, there is a stalemate between the cells and the particles in such cases, leading to a relatively localized and often inert tissue response. However, when the particles are small enough to be phagocytized a relatively brisk cellular sequence of events results [24].

Extracellular particles are known to become coated with tissue proteins. This makes them more easily identifiable to phagocytic cells for ingestion. Once these particles are in the phagosomes within the cells, the cells undertake to convert the phagosomes to a lysosome by the release and activation of enzyme systems. The most blatant responses are those of proteolytic enzymes. The cell is unable to dissolve or digest the majority of these materials; the result is that the particle is extruded from the cell together with all of the enzymes that the cells had attempted to use on the foreign material. As a result, there is a chemotactic attraction for more phagocytic cells. There will be dissolution of the intercellular matrix by the enzymes, leading to loosening of the tissue with separation of collagen fibrils. The protein polysaccharide moieties that hold them together are disrupted by enzymatic digestion.

As a result, it is now easier for cells to travel within the matrix and the vicious cycle of phagocytosis, enzymatic activation, exocytosis, chemotactic effect, and repeat phagocytosis by additional cells is set in motion. As the tissue loosening occurs, support for the implant will weaken and there is enhancement of micromotion. This leads to further micro-injury to the surrounding tissue and eventually catastrophic collapse and clinical failure [24].

Polydimethylsiloxane (silicone rubber) also falls into the embodiment failure category. The first silicone rubber compounded specifically for medical purposes was patented in 1948 by Dow Corning (Midland, MI) [25]. Since then, this elastomer material has found many applications in the field of reconstructive surgery of non-weight bearing areas of the body because of its excellent biocompatibility [26–28]. When used in hand surgery, although dramatic relief of pain and restoration of motion ensued, problems related to implant fracture were not uncommon [29–36] (Figs. 3.1 and 3.2).

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Despite the development of a high-performance silicone rubber, with long-term follow-up of these implants, the issue of host tolerance of the silicone material emerged as a clinical concern. Several reports surfaced of implant wear and cold flow associated with erosive cystic changes of adjacent bones resulting in a severe synovitis [37–39]. Histologic studies of synovium, cyst content, cartilage, and bone have shown a marked inflammatory reaction to particulate silicone debris with numerous foreign body granulomas being found. This aggressive giant-cell response to the silicone implant particulate matter is known as silicone synovitis and has been reported with an incidence between 51 and 84 % of long-term silicone carpal implants [40, 41].

It is well known that the hystiocytic response to particulate debris plays a major role in osteoclast activation and bone resorption in tissues surrounding implants. This may well be the final factor leading to progressive loosening of the support for the implant and its eventual clinical failure. The use of these materials in TMJ reconstruction and the resulting scenarios has been well documented [42].

Metal-on-metal total hip prostheses made from cast cobalt-chrome (Co-Cr) alloy were used in total hip replacement in the 1960s and early 1970s, but by the mid-1970s they were replaced by metal-on-high molecular weight polyethylene. The main factors that led to the abandonment of metal-on-metal articulations were the success of the Charnley prosthesis [43] and implant component loosening due to fretting, galling, and seizing experienced with the metal-on-metal prostheses [44–46].

Historical failures of metal-on-metal articulations have been attributed to one or more of the following factors: (1) poor control of sphericity and radial clearances greater than 200 μm (high wear); (2) poor implant design and/or implantation technique; (3) inadequate radial clearance via matched head-cup pairs (seizing and high friction); and (4) galling and fretting resulting in wear. This means that the only type of embodiment using metal-on-metal design is a highly constrained joint such as the hip because only in that situation can the optimum tolerances be possible to prevent abnormal wear [47].

Attempts at using metal-on-metal geometry in relatively non-constrained joints like the knee have resulted in abnormal wear due to fretting and galling with the development of particulate debris leading to the formation of foreign body granulomas, osteolysis and catastrophic device failure [48] (Fig. 5.9A).

### *11.2.3 Failure of Implantation Technique*

The variables to be considered in this category include the specifics of the joint surgical procedure as related to component position, joint biomechanics and initial fixation. Surgeons may be nominally doing the same procedure, but variations in general technique or in a specific case may be substantial. Schmalzreid and Brown stated that this category is in general the greatest source of unrecognized variability [15].

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Ravi et al. reported the after primary total hip and knee replacements, the risks for dislocation and early revision in patients whose surgeons had carried out less than 35 procedures were 48 and 44 % higher respectively than In patients whose surgeons had carried out greater than 35 [49].

### *11.2.4 Limitations of the Technology*

In some cases, the requirements for a specific case may exceed the capabilities of the technology. For example, attempts to span large anatomical defects with standard, non-specialized, stock implants may result in early failure due to inadequate bone support.

All alloplastic total TMJ reconstruction devices undergo in vitro testing utilizing theoretical load and motion data. The key element of this statement is 'theoretical load'. Normal functional loading forces on the TMJ have been only postulated from mathematical, finite element computer analysis and crude anatomical modeling. To date, most TMJ devices have been bench tested over time at these postulated loads. But it has yet to be determined what these loads actually are under both normal and compromised anatomical and biophysiologic states [50].

The TMJ is not normally exposed to the same functional loads as is the hip, especially in complex, multiple-operated patients. The surgical procedure to implant a TMJ TJR device involves the elimination of the functional forces of the lateral pterygoid muscle and the temporalis muscles on the mandible if a coronoidectomy is indicated. Therefore, the subsequent functional loads delivered to the bearing surfaces of these devices are reduced [51].

In cases where the functional loading demands are beyond what is technically possible for the TMJ TJR device being used, it can lead to failure. In the case of a chronic heavy clencher/bruxer who delivers functional loads to the fixation and/or components that exceed the ability of the materials to tolerate, there can be excessive wear and/or fracture of the components with failure of the reconstruction. The use of a full-coverage oral orthotic should be considered for daily use in such cases to reduce bearing surface overloading in such cases.

Stock TMJ TJR devices should be avoided in cases where the host bone anatomy has been so architecturally modified by the disease process or prior surgery that it makes providing a stable fitting of the components difficult.

The fossa components of stock devices are designed without a posterior stop to prevent the TMJ TJR device condyle from displacing posteriorly. If the stock condyle is not perfectly aligned in the center of the stock fossa medio-laterally and/or antero-posteriorly, the stock condyle can displace posteriorly and impinge on the tympanic plate and/or the auditory canal. This can result in pain and mandibular dysfunction and facial deformity. There is also the potential for infection should there be a pressure-related perforation of the auditory canal. This is of special concern when using a stock TMJ TJR in combination with orthognathic surgical procedures involving counterclockwise mandibular rotation [52]. The custom TMJ TJR fossa has a posterior stop, alleviating this concern [53] (Fig. 5.14).

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## 11.3 Indications for Revision

Based on Bourne's work [54], the following are the indications for revision or replacement of a failing or failed TMJ TJR:

1. Failed component/components
2. Breakage of a component or components and/or fixation screws
3. Aseptic loosening
4. Sub-acute or chronic infection
5. Osteolysis
6. Peri-prosthetic bone fracture
7. Ankylosis.

## 11.4 Important Considerations Before Revision/Replacement

The following should be considered before a patient undergoes revision of a failed or failing TMJ TJR device.

### *11.4.1 Medical*

The revision/replacement patient will be older and may have had significant changes in their medical history. It is important that the revising surgeon takes a new and complete medical history and consults with the patient's primary physician as he/she assesses the patient's surgical and anesthetic risks.

### *11.4.2 Anesthetic*

Ankylosis may be the result of a failed TMJ TJR device. Careful consideration to anesthetic induction and postoperative care may indicate tracheostomy especially in patients with recurring ankylosis or heterotopic bone formation where limitation of mouth opening provides a challenge to conventional intubation techniques.

### *11.4.3 Local Skin Disorders*

Any skin conditions affecting the incision sites should be evaluated and managed by a dermatologist before revision/replacement.

*Propionibacterium acnes* (P. *acnes*) has been increasingly recognized as an important agent in orthopedic shoulder device infections. *P. acnes* is a Gram-positive bacterium that forms part of the normal flora of the skin, oral cavity, large

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intestine, the conjunctiva, and the external ear canal. Although primarily recognized for its role in acne, *P. acnes* is an opportunistic and difficult to culture pathogen that can cause a range of postoperative and device-related infections [55].

Therefore, any history of severe acne or prior TMJ TJR infection where the pathogen was not clearly identified should be pursued by a dermatology consultation before undertaking revision/replacement.

### *11.4.4 Uncooperative or Drug Dependent Patient*

Psychotic, drug-dependent and patients with clearly inappropriate expectations should be evaluated carefully before surgery.

In the case of drug dependence, consultation with a pain management specialist is essential to develop both surgical and postoperative pain management regimens. It is advisable that both the patient and their pain management professional understand before revision/replacement that the surgeon will provide, based on consultation with the pain management professional, analgesics to control the surgical pain, but long-term pain management must be provided by the pain management physician, not the surgeon.

### *11.4.5 Local Resources*

Revision/replacement surgery can often be difficult; therefore, the surgeon who performs an occasional TMJ procedure must examine his/her experience, operating room personnel, assistants, and the TMJ TJR implant system availability to determine whether the patient would be better served by being sent to a more experienced TMJ revision/replacement surgeon.

Also, since post-revision/replacement physical therapy is essential, there must be therapists available who are familiar with the rehabilitation of these patients.

## 11.5 Evaluation of the Patient

### *11.5.1 TMJ History*

When evaluating patients with complaints of a failing or failed TMJ TJR device, an accurate and complete TMJ history is essential. All prior problems, including initial complaints prior to any surgical treatment, should be documented. Prior noninvasive and invasive management modalities should be documented as well as rate of recovery and outcomes.

Mandibular function and pain should be assessed based on the patient's perceived mouth opening over time (increased or decreased, relation to symptoms), diet consistency (normal diet or liquids only), pain, and swelling (with or without function).

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The patient's primary and secondary outcomes expectations after revision/replacement must be discussed. The primary outcome expectation for most patients in this situation is pain relief. Typically mandibular function and aesthetics are secondary. It is essential that the revision/replacement surgeon make it completely clear that any relief of pain is an unrealistic expectation of revision surgery. The patient must understand that primary goal is the replacement of the joint is improvement of mandibular function [56, 57].

### *11.5.2 Physical Examination*

A thorough head and neck examination is important. Extra-oral facial symmetry, prior incision site, cranial nerve, otoscopic, facial, masticatory, and cervical muscle examination should be included and documented. Since many of these patients have Facial Nerve weakness from prior surgery, this should be documented preoperatively using one of the classic Facial Nerve grading systems such as House Brackmann [58].

Intra-oral examination should include documentation of dental and mucosal lesions, occlusion, and mandibular range of motion (MIO, lateral and protrusive movements). Pre-revision/replacement extra-oral and intra-oral photographs provide good documentation of the presenting clinical situation.

### *11.5.3 Imaging*

When more sophisticated imaging is required, CT, CBCT, and MRI studies provide useful information despite the fact that most of the components of TMJ TJR devices are metallic. Plain radiographs still play an important role for initial imaging of TMJ TJR devices. The orthopantomogram and anterior–posterior (AP) cephalometric or AP skull films provide good screening images.

As is the case with any radiographic examination, the quality of the study is important. When examining TMJ TJR device imaging, one should first evaluate the quality of the film, not only to decide on the necessity to repeat the radiograph but also to ensure that future radiographs will be of good quality. A good TMJ TJR device image should demonstrate both fossa and ramus components and the screw fixation. The best plane image is the AP cephalometric or skull. These radiographs provide excellent and reproducible images for initial evaluation and serial comparison of component position and screw integration at postoperative and follow-up visits.

Since most of the components of TMJ TJR devices are metallic, CT, CBCT, and MRI can yield images affected by metallic scatter and can be difficult to interpret; although recent advances in this equipment have improved image quality. However, PA and lateral tomography should be considered as an alternative if more sophisticated imaging is deemed necessary.

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In cases where there is a question of inflammation or hypersensitivity, radionuclide bone scanning with Indium-111 may be helpful in the diagnostic evaluation.

Based on the work of Ghelman [59], the following points should be examined when observing any TMJ TJR device radiograph:

1. The position of the components, not only to each other, but also in relationship to the adjacent bone.
2. The position of the fixation screws. The fixation screws should be bicortical and of the proper length with the medial aspect not penetrating into underlying soft tissue. This can cause irritation of that tissue with pain and swelling during later function [53].
3. The presence of metal fractures, either major components or fixation screws. Look to see if the head of the fixation screw appears to be out of its component recessed position indicating fracture.
4. Areas of ectopic bone formation. Heterotopic bone is most commonly seen between the medial aspect of the ramus and fossa, or lateral and posterior to the fossa component.
5. Metal fragments in and/or around the joint. This is an indication of wear fretting and/or galling in metal-on-metal TMJ TJR devices.
6. Host bone osteolysis around major components and/or fixation screws. This is an indication of loosening and impending implant failure.
7. Fracture of the host bone. Loosened major components and/or screw fixation add stress to the underlying bone, and under function, fractures can result.
8. Infection. Manifestations of osteomyelitis (periosteal reaction, sequestrum, involucrum, etc.)
9. Other abnormalities, such as primary or metastatic neoplasm, bone cysts, odontogenic lesions developing around or close to these devices.

## 11.6 Guiding Principles of Revision/Replacement of Failed or Failing TMJ TJR Devices

Based on the information gained in the history, physical, and radiographic examination, the following should be considered as principles for the revision/replacement of TMJ TJR devices:

### *11.6.1 Establish Failure Mechanism*

The most basic TMJ TJR revision/replacement principle is the establishment of why the prior device failed. The classification of failures discussed above should be reviewed and applied. If the failure mechanism is not identified, the revision/replacement surgeon may repeat the same error. Further, lack of understanding by both the surgeon and the patient for the cause of the pain and/or functional limitation often leads to failure of the revision.

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### 11.6.2 Rule Out Sepsis

Failure to know or understand the role of sepsis in the failure of a case to be revised will doom the revision procedure to failure. Fortunately, the incidence of deep infection following TMJ TJR is rare [60]. This is at least partially attributable to the abundant blood supply to the head and neck area. This will be discussed in detail in the complications Chapter 8.

### 11.6.3 Perform Adequate Preoperative Planning

It is essential that in revision/replacement planning the proper posterior and anterior mandibular vertical mandibular height and proper maxillomandibular skeletal and dental relationships be established.

### 11.6.4 Utilize the Appropriate Revision System

Often, the failure of TMJ TJR components leads to alteration of host bone architecture. The use of custom TMJ TJR devices is recommended for such cases especially if orthognathic surgical procedures are combined with the TMJ TJR replacement. Custom TMJ TJR components can be designed and manufactured to replace lost host bone and can be made to develop the most functionally stable articulation as well as salvage anatomic distortions [53].

### 11.6.5 Minimize Complications

Good preoperative planning, use of the appropriate TMJ TJR devices, and careful surgical technique will lead to less complications and better results.

### 11.6.6 Optimize Rehabilitation

One of the major advantages of a well-fixated and stabilized TMJ TJR device is that physical therapy can begin immediately. Salter demonstrated that early surgical joint mobilization results in increased long-term range of motion.[61] This is very important in patients who have had failed TMJ TJR devices where masticatory muscle range of motion has been definitely compromised and needs improvement.

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# Part V
# Future Considerations

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# Chapter 12
# Bioengineered Tissue TMJ TJR

**E. Weston Santee, Sharon Aronovich, and Stephen E. Feinberg**

## 12.1 Introduction

One of the major obstacles that have plagued the reconstruction of the temporomandibular joint (TMJ) has been the adverse reactions seen with the use of alloplastic, non-biologic materials. These inert and passive materials, by themselves, do not respond to normal biochemical or biomechanical signals, which are present in situ within the TMJ. The patient, because of the biologic inertness of these materials, must adapt to the material or mechanical device that has been used. This may result in related complications or compromised functional outcome [1].

The use of viable autogenous tissue offers an exciting alternative to alloplastic or non-biologic materials. Autogenous tissue has the advantage of being able to respond to biologic cues; however, it also has the disadvantages of donor site morbidity, limited quantity and quality, and less than perfect match to the tissue being replaced or reconstructed. Some alloplastic TMJ devices have been unsuccessful in the past because patients must adapt to the implanted synthetic materials they contained [2, 3]. In contrast, the use of autogenous tissue for total TMJ repair or replacement allows biological remodeling to occur by the functional forces placed on the implant by the patient.

The successful use of autogenous costochrondral (CCRGs) has been attributed to the presence of a cartilaginous cap atop of the bony strut of the rib [4, 5]. Studies have shown that the articulating condylar fibrocartilage enables the TMJ to withstand compression and loading, which assists in the morphological adaptive

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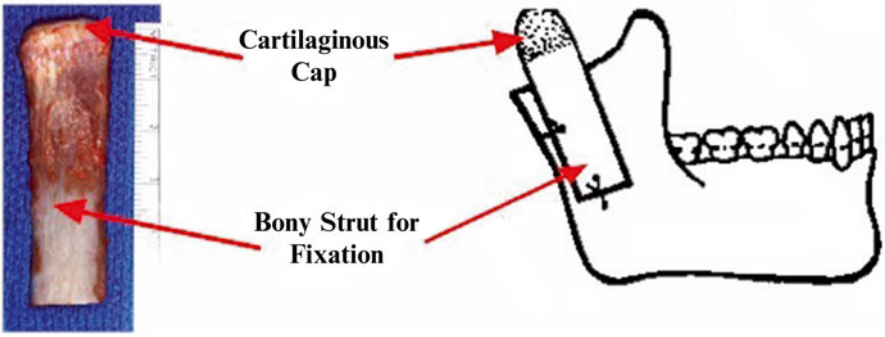


**Fig. 12.1** CCRG and lateral placement on ramus

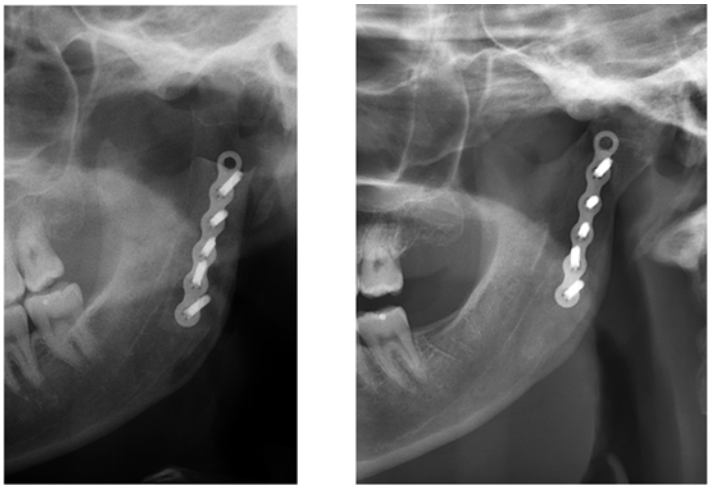

**Fig. 12.2** *Left*: radiograph placement of CCRG. *Right*: radiograph 5 years later showing marked changes in rib that now looks like a normal condyle. In fact if one removed the titanium plate, you would not know that the person had a CCRG placed

responses to biomechanical stress [6]. It has been demonstrated that mechanical stimuli elicited by joint function can determine the ultimate growth and shape of the condyle [7] (Figs. 12.1 and 12.2).

The development and use of biologically responsive materials will allow grafted or reconstructed tissue anatomic and functional adjustments in situ so that it provides for the unique needs and demands of the specific anatomic site and/or functional load. A tissue engineering/regenerative medicine approach may allow for the development of a new TMJ prosthesis that could eliminate or minimize the disadvantages of the use of autogenous tissues such as donor site morbidity and the poor anatomical shape of the CCRG when used in TMJ reconstruction.

Within the last decade, the newly emerging field of tissue engineering/regenerative medicine has developed to a level of sophistication that it now may offer alternatives

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to traditional TMJ reconstruction. Tissue engineering is defined as the application of principles of biomimetics for the restoration, repair, replacement, and assembly of functional tissue and organs [8]. Biomimetics is defined as an interdisciplinary field that combines information from the study of biological structures and their function with physics, mathematics, chemistry, and engineering in the development of principles that are important for the generation of novel synthetic materials and organs.

In the past, TMJ reconstructive joint surgery focused on the designing of replacement parts. With the debut of tissue engineering/regenerative medicine (TE/RM), the focus has shifted to reconstruction using functional biological components of tissues. The ex vivo construction of a TMJ replacement, using a TE/RM approach, will be determined by several components: stem cells or progenitor cell populations, regulatory signals such as growth factors or biophysical cues such as mechanotransductional forces and electric fields, scaffold architecture (composed of the extracellular matrix and/or the suprastructure of the reconstructive region), and restoration of a vascular component to the area to be reconstructed.

These components must follow the cardinal rules of tissue engineering. First, optimal regulatory signals must be present. Second, the cells must be able to respond to these regulatory signals. Third, one must have an instructive and interactive scaffold whose geometry and surface coating can influence cells that migrate in and/or attach to it. And lastly, an active perfusion system must exist that allows for the restoration of vasculogenesis, which is critical to maintain cell viability and function.

One vision of a tissue-engineered TMJ prosthesis utilizes a 3D designed and manufactured biodegradable scaffold shaped similar to a condylar head and neck, i.e., a condyle-ramus unit (CRU). The fabricated CRU scaffold would be constructed such that it would impart biologic cues to implanted cells placed within its interstices. These biologic cues should influence scaffold-implanted mesenchymal stem cells (MSCs) or bone marrow stromal cells (BMSCs) to form a fibrocartilaginous joint surface, or cap, on top of a bony strut, similar to a costochondral rib graft (CCRG), which could then be fixated to the mandibular ramus. The disc would form from an extracellular matrix that would be able to modulate its shape and form in response to a functional joint. Presently, TMJ ligaments pose a problem, since the technology to accomplish this does not currently exist. This new approach to tissue engineering a TMJ would be advantageous because of its site-specific anatomical configuration as well as its potential ability to adapt to the functional forces placed on it during function.

In this chapter we will discuss the various components and some possibilities and difficulties in tissue engineering a complete temporomandibular joint.

## 12.2 Scaffolds

A scaffold can be defined as a transitional physiochemical framework within which cells populating it create a replacement tissue as the prosthesis disappears or is incorporated into the surrounding tissues. The ideal properties for fabrication of scaffolds from biomaterials have been outlined by Bell [9]. The ideal scaffold

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should be biodegradable and nontoxic, should have degradation products that are nontoxic, should allow cell attachment, and should be able to be remodeled by the cells within or surrounding the scaffold. The physical properties of the scaffold should be similar to those of the tissue it is replacing with respect to strength, compliance, and density. The scaffold should be a good substrate for the extracellular matrix (ECM) enzymes to modify as necessary. It should also allow cell motility and ingress of angiogenic elements while having a low level of immunogenicity and thrombogenicity. It should be capable of being fixated, if necessary, with screws or sutures. Finally, the scaffold must be interactive with its surrounding environment.

The different types of scaffolds that are available fall into three basic categories: (1) non-bioabsorbable (man-made materials with little or no information content for cells, i.e., Dacron, nylon, polytetrafluoroethylene [PTFE]); (2) bioabsorbable (man-made with low information content for cells, i.e., polylactic acid [PLA], polyglycolic acid [PGA], combinations of PLA/PGA, calcium phosphate ceramics [hydroxyapatite (HA), tricalcium phosphates (TCP)], and polycaprolactone (PCL); and (3) naturally occurring materials with high information content for cells (secreted biomatrix, animal and human tissues [dermis, dura, ligaments, heart valves], or animal polymers [collagens, elastin, laminins, fibronectin, glycosaminogylcans]).

## 12.3 Role of Interactive Scaffolds

Scaffolds should be enriched with instructive materials that could be interactive and influence host and/or seeded cell production of the ECM. It is only through this approach that functional tissue replacement parts can be accurately fabricated [9]. This could be accomplished by making the scaffolds responsive to site-specific needs of the area to be reconstructed, i.e., TMJ. One way to accomplish this is by controlling the degradation rate of the scaffold such that it is replaced seamlessly by natural tissue. Another alternative approach is to control the surface properties of the scaffold so that it could influence or determine the types of cells that attach, their behavior, growth, differentiation, and/or migration through the scaffold. One could also control the three-dimensional internal architectural structure, such as pore size, channel direction, and trabecular orientation, as well as surface chemistry or local surface texture of the scaffold as seen on dental implant surfaces.

Another interesting approach would be the use of conducting polymers to create electric fields to manipulate and direct behavior of either cells placed within the scaffold or cells that migrate into the scaffold from the peripheral tissue bed. In addition, through the process of mechanotransduction, forces transmitted through the scaffold influence the behavior of the resident cells within or migrating into the scaffolds.

What is most important about this approach for TMJ reconstructive surgery is the ability to create a dynamic functional interaction between the cells, ECM, and

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the scaffold. This is seen when cells synthesize and secrete multiple molecules into the surrounding immediate environment to form an ECM. The secreted ECM can impart important signals and properties to the scaffold. Collagen fibrils secreted within the ECM can enhance tensile strength. The release of proteoglycans, or ground substances, that are incorporated into the ECM can bind fluid to improve the compressive properties, as is seen in cartilage. The secretion of cytokines or growth factors into the surrounding environment can influence cell growth and behavior (BMP, PDGF, bFGF, TGF-β). Specific cell surface receptors can interact with the ECM and scaffold affecting cell behavior, attachment, migration, and/or differentiation.

The signal transmission between cells and the ECM can be accomplished through mechanotransduction. This is germane to reconstruction of a TMJ since this joint is loaded during function. The signal could be either a solid deformation resulting directly from muscle and bite forces or a fluid flow effect secondary to deformation. The importance of an interactive ECM is illustrated in a study in which hepatocytes are placed into two different environments [9]. One is a non-physiologic environment in which the ECM or collagen is in contact with the cells only on one side (not naturally seen in vivo). The second is a more natural environment in which the hepatocytes are placed in contact with collagen (ECM) on both sides (a situation more typical in the liver). If one compares cell function of the hepatocytes, as measured by their secretion of bile salts, protein, and urea, the cells in the more natural environment (collagen on both sides) will secrete exponentially more of these products [9].

## 12.4 Importance of Intra-architectural Scaffold Geometry

A scaffold for a TMJ prosthesis should provide interactive and/or functional biologic cues or signals to guide incremental matrix production by cells that are invading or those already implanted [9]. The architectural design of the scaffold/matrix should be instrumental in influencing biological activity (cell infiltration, attachment, differentiation, and function) and mechanical integrity (ability to withstand or distribute mechanical forces) [10].

Several studies have shown that scaffold pore size can influence formation of bone or cartilage regeneration. Coralline HA scaffolds, with a pore size of 500 μm, allowed extensive cellular and vascular invasion and new bone formation after 12 weeks in vivo, while no bone formation or cellular invasion was found within 200 μm scaffolds [11]. Gauthier and others, utilizing macroporous biphasic calcium phosphate ceramics, showed a 563 μm pore size provided more new bone formation, both in peripheral and deep pores, than a 300 μm pore size [12]. Tsuruga et al. noted that a HA structure with pore sizes of 300–400 μm, complexed with rhBMP-2, was optimal for attachment, differentiation, and growth of osteoblasts and for vascular ingrowth [13]. Kuboki et al. showed that rhBMP induced only osteogenesis when porous particles of HA were used as a carrier, whereas only chondrogenesis

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occurred within a carrier of fibrous glass membrane [14]. They concluded that vasculature was the crucial factor that determined osteogenesis or chondrogenesis. Tsuruga and Kuboki's findings showed that scaffold geometry, which restricted vascular invasion, would produce cartilage, while other geometries that could accommodate a haversian system would produce bone. Ripamonti and Reddi showed that pore sizes of 150 μm could not support neovascularization [15]. These studies demonstrate the significant effect scaffold pore size can have on bone or cartilage regeneration and vascular ingrowth.

## 12.5 Cells

There are four basic strategies for using autogenous bone marrow stem cells (BMSCs) in cell-based bone tissue engineering: (1) local targeting of BMSCs where new tissue is needed, (2) transplanting autogenous BMSCs to augment the local population, (3) transplanting cultured-expanded or modified BMSCs, and (4) transplanting fully formed tissue [16]. These strategies involve the use of progenitor/stem cells, cytokines (BMP, PDGF, etc.), or proteins attached to scaffolds (protein sequences), alteration of cells via genetic manipulation, and/or the development of carriers or scaffolds for their delivery to the regenerative site. To date, none of these approaches alone or in combination have effectively transferred into the clinical arena for a variety of reasons.

The use of pluripotent cells on hydroxyapatite (HA) ceramic scaffolds is a promising approach for the engineering of a hard tissue construct. Either mesenchymal stem cells (MSCs) or bone marrow stem cells (BMSCs) can regenerate bone and given the appropriate microenvironment may also regenerate cartilage. Bruder and Dennis noted bone formation in a rat femoral defect model when a porous ceramic carrier was loaded with MSCs [17, 18]. Negligible bone formation was noted in carriers without MSCs. Kadiyala et al. saw both bone and cartilage formation in porous ceramic carriers loaded with canine MSCs implanted subcutaneously into dogs and mice [19]. Krebsbach et al. found significant bone formation in mice with HA/TCP carriers loaded with BMSCs [20, 21]. These results indicate that cells from marrow stroma have the capacity to form significant amounts of bone and/or cartilage when placed in defects on either HA or HA/TCP ceramic scaffolds.

In summary, stromal cells have the potential to regenerate bone and/or cartilage when placed on bioceramic scaffolds. The geometry or pore size of a biomaterial scaffold carrier can play an important role in the type and amount of tissue regenerated. The recent advances in three-dimensional (3D) printing and image-based design methods allow us to study the effect of scaffold architecture (pore size) on bone and cartilage regeneration of bioceramic scaffolds loaded with marrow stromal cells [20–23]. Unfortunately, bioceramic scaffolds are brittle under structural force and are not an ideal material to use. A much better material would be the biodegradable and FDA-approved polymer, polycaprolactone [24]. The superior rheological and viscoelastic properties over many of its aliphatic polyester

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counterparts render PCL easy to manufacture and manipulate into a large range of implants and devices. Coupled with relatively inexpensive production routes and FDA approval, this provides a promising platform for the design and fabrication of longer term degradable implants which may be manipulated physically, chemically, and biologically to possess tailorable degradation kinetics to suit a specific anatomic site.

Another approach is to inject cells parenterally and take advantage of the process of cell homing. Cell homing is a technique that relies on induction and chemotaxis of undifferentiated host progenitor MSCs to migrate into the scaffold, making the scaffold their new home, and differentiate into specialized matrix forming cells.

In 2010, Lee et al. demonstrated successful regeneration of a rabbit humeral condyle after having resected the original structures and implanting a custom-fitted composite polycaprolactone and hydroxyapatite (PCL-HA) scaffold [25]. A group containing a TGFβ3-adsorbed collagen gel was compared to a TGFβ3-free collagen hydrogel group and to a group that underwent resection without scaffold implantation. It was hypothesized that the TGFβ3-infused scaffold would recruit and stimulate chondrogenic endogenous cell homing, which was the mechanism responsible for recruitment of host progenitor cells from sources such as synovium, bone marrow, and adipose tissue (TGFβ3 provided the chemotactic cue for cell homing). Together with a favorable local tissue response and functional stimulation, only the TGFβ3-adsorbed constructs produced a histologically sound articulating osteochondral joint unit with stratified avascular cartilage and vascularized subchondral bone at 4 months after implantation. In addition, functional recovery, evidenced by locomotion and weight bearing, was achieved 3–4 weeks after surgery. The PCL-HA scaffold was not only designed to provide the mechanical strength needed for weight bearing, but incorporated a design with interconnected microchannels (200–400 μm) that served as conduits for cell homing and diffusion, and to encourage angiogenesis. This study suggests that complete articular tissue regeneration is possible by cell homing instead of cell delivery. How does this apply to the clinical scenario? Aside from known differences between tissue regeneration in rabbits and humans, Lee et al. inserted their bioscaffold into an intramedullary tunnel within the proximal humerus immediately after the proximal articulating head was osteotomized [25]. In addition, we must keep in mind that this was carried out within a healthy joint where all soft tissue attachments and the joint capsule were preserved. It remains to be seen how successful this cell-free bioscaffold would be in disease states such as ankylosis or osteoarthritis with chronic degenerative joint changes.

Alhadlaq and Mao also demonstrated that while MSCs in inductive culture media can differentiate and synthesize the specific chondrogenic and osteogenic matrix, a high cell density (on the order of $20 \times 10$ [6] cells/mL) is necessary to improve tissue maturation and grow a well-integrated osteochondral construct [26, 27]. At lower densities (such as $5 \times 10$ [6] cells/mL), the osteochondral junction lacked the mutual infiltration of osseous and cartilaginous tissue seen in mature condyles. Thus, it was found that cell density matters, with cells preferring close contact and interaction with adjacent cells for functional tissue matrix synthesis and maturation.

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The advantages of cell homing are:

1. It would overcome key scientific, technical, commercialization, and regulatory issues associated with cell transplantation.
2. Bioactive cues (cytokines) for cell homing are readily packaged as off-the-shelf products and delivered in a single procedure from the injected cells.
3. Ease of clinical delivery of packaged and stored molecular delivery products.
4. Maximizes the body's own regenerative capacity.

## 12.6 Biophysical Manipulation of Cells

Mechanotransduction can influence cell and scaffold interactions through stretch-activated stress channels through ECM connections with the cytoskeleton via integrins such that mechanical loading could influence matrix synthesis assembly and degradation [20]. This is illustrated by the work of Guldberg and others in which they placed pneumatically loaded bone chambers into the tibial plateau of canines [28]. They then varied the force that was generated within the chambers and assessed bone formation. The chambers that were mechanically loaded showed a higher density of bone formation than the unloaded chambers.

The introduction of biocompatible electroactive (conductive) materials into a biological system has the potential to not only provide a physical substrate for cell growth and tissue repair but also to allow the local delivery of an electrical stimulus to a specific site to foster cell growth and repair damaged tissue. The delivery of an electrical stimulus may also promote the in vitro development of tissue for implantation. The former studies concluded that heat-conducting polymers such as polyaniline and polypyrrole in powder or film form are biocompatible materials, showing cell and tissue compatibility in vivo and in vitro.

Covalent grafting of bioactive molecules is one of the effective strategies used for conducting polymer film surface modification, having as benefits the avoidance of biomolecular denaturation, the leaching of the entrapped biomolecules, and decreasing of the conductivity by orders of magnitude as observed in the doping or entrapment techniques. The covalent links of the biomolecules on the surfaces are based on the presence of the COOH or $NH_2$ functional groups on the top of the films and provide both electrical and biological stimulation, which represent major advantages. Being reversibly switchable between different oxidation states, conducting polymers allow control over polymer characteristics including surface energy, conductivity, morphology, and Young's modulus; all these characteristics may be modulated to enhance or control the behavior of responsive cells. Various composite materials and copolymers of conducting polymers with biodegradable, biocompatible counterparts can be obtained and studied as alternative materials for tissue engineering scaffolds. Different polymerization techniques can be employed for the synthesis of polymer nanostructures such as soft and hard template polymerization, emulsion polymerization, admicellar polymerization, and also modern

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processing techniques that can be applied for 2D and 3D scaffold formation. These composite materials can be processed as porous membranes, nanofibers, nanotubes, or nanofilaments. These nanostructures can be used to generate scaffolds with large surface areas, desirable topography (e.g., 3D porosity, nanometer-scale size, and alignment), high porosities, ease of construction into different shapes, and surface functionalization (e.g., surface immobilization of bioactive molecules or functional groups).

The work of Carl Brighton from the University of Pennsylvania illustrates the importance of utilization of various strengths of electric fields to influence cell, chondrocyte, and osteocyte behavior both in cartilage and bone formation, respectively [29–31]. Normal human tissue cells have well-known electrical properties that have been found to play a role in embryogenesis and tissue repair. A well-known example during development is the regulation of cellular phenotype by voltage-gated ion channel expression [32]. Can the application of electric fields enhance a tissue-engineered condylar construct composed of bone and cartilage in an osteochondral graft? Alternating current (AC) devices to aid healing of bone fractures and nonunions with electric field stimulation are already in clinical use (commercially available). Electric fields change cell membrane potential and alter ionic currents in the extracellular space upregulating gene transcription and translation via the activation of cellular signaling cascades. Stimulating osteoblasts with a pulse electromagnetic field increased calcium influx which in turn upregulates PGE2, insulin receptor substrate-1, and TGF-beta. This effect on voltage-gated calcium channels may explain how electric fields aid in bony healing. Thus, researchers using biocompatible devices that are capable of generating electric fields to stimulate 2D or 3D engineered tissue constructs have been exploring the potential to influence cell proliferation, adhesion, differentiation, migration, and function. Direct current (DC) field stimulation may have an important role in endogenous cell homing by impacting cell migration, known as galvanotaxis, into an implanted scaffold. Moreover, the exposure of hMSCs to an intermittent DC electric field can lead to osteogenic differentiation, increased alkaline phosphatase, and mineralization. There are also several examples showing upregulation of mRNA expression and protein levels of TGF-beta, BMPs, FGF-2, osteocalcin, and ALP, with a similar increase in osseous matrix deposition (osteocalcin, osteopontin, type 1 collagen) under the influence of capacitively coupled electric fields. Similarly, when articular chondrocytes undergo electrical stimulation, there is a fourfold increase in aggrecan and type II collagen mRNA which is inhibited completely when calcium channel blockers are added. Compared to unexposed cell-scaffold constructs, significantly greater proliferation of osteoblasts and hMSCs is achieved when cell-seeded scaffolds are exposed to electromagnetic fields [33].

In summary, in order to obtain an optimal reconstructive outcome, one would need to control all of the variables: cells, growth factors, scaffold makeup, and design, as well as the interactions among all of the above. The ability to control or direct these variables would enable successful site-specific anatomical reconstruction.

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## 12.7 The Fabrication of a TMJ Disc

The disc has a biconcave structure and is composed of dense, fibrous connective tissue. It is non-vascularized and non-innervated. When the disc becomes irreparably damaged, patients may complain of pain during mastication and functional limitations, both of which may progress to the point of lowering the patient's quality of life. The surgical removal of the disc is a widely performed procedure for a patient with the aforementioned injury and symptoms. Discectomy without a replacement has been shown to be effective at resolving pain and mobility limitations even at 5-year follow-up [34]. MRI studies have shown that when a disc replacement is not used, there appears to be thick tissue that develops, which also may prevent the two articulating surfaces from coming into contact [35]. Other studies show degenerative changes in the articular surfaces, regardless of whether a substitute was used [36]. There still remains concern about long-term damage to the articular surfaces and joint adhesion after meniscectomy without replacement. Autologous disc substitutes such as the temporalis flap have been better tolerated than alloplastic substitutes [37]. However, a suitable and reliable disc replacement with minimal morbidity has been elusive.

Recently, there have been studies demonstrating the potential for a xenogeneic disc replacement that can act as an inductive substrate for TMJ disc tissue. Badylak and colleagues have used porcine bladder to create a urinary bladder matrix-extracellular matrix (UBM-ECM) disc substitute. The UBM-ECM substitute is composed of an outer layer of acellular, treated porcine bladder with particulate porcine bladder extracellular matrix packed inside. Using a canine TMJ model, it was shown that the UBM-ECM disc substitute acted as a scaffold that remodeled over time to closely resemble the shape and size of the native disc. In addition, the scaffold remodeled into a collagenous tissue with fibers that also resembled fibers found in the native disc. Another very promising finding is that at the periphery of the UBM-ECM substitute integration with the adjacent musculature occurred. No evidence suggestive of pathology was seen in the articulating surfaces of the glenoid fossa or the head of the condyle. These promising results and the fact that UBM-ECM products already exist and are FDA approved suggest that a UBM-ECM disc substitute may be a beneficial and efficient means to replace the TMJ disc [38].

The challenge is how to incorporate the replacement of the disc into the overall strategy to create a tissue-engineered TMJ. Currently, there are no studies investigating the use of a CRU with an attached disc substitute overlying the head of the condylar portion. One approach would be to create ligamentous attachments, which allow for appropriate disc mobility. Creating or guiding the ligamentous attachments between the CRU and the disc may prove difficult, but such a schematic may one day be feasible with advancements in tissue engineering. Another approach that is perhaps less complicated is to plan for two surgeries, the first being removal of the pathologic tissue and insertion of a tissue-engineered CRU (as well as the articulating fossa if necessary) and the second being the insertion of a UBM-ECM disc substitute after the tissue-engineered CRU has had sufficient time to mature and

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begin to remodel. There may be an issue with the timing of the surgeries, though, as the tissue-engineered CRU will be expected to remodel according to the load and function under which it will be operating. Knowing the appropriate time for the second stage of a two-stage surgery will be very important for successful integration and remodeling that allows for appropriate tissue adaptation during the patient's physiotherapy.

## 12.8 Vision of the Process of Tissue Engineering a TMJ

The patient would present with either ankylosis or advanced degenerative joint disease with various limitations of joint movement. The following would be the stages of treatment to include in fabrication of a total TMJ:

1. **Imaging** of the unaffected side if unilateral or, if bilateral, use a computer-generated rendition of an appropriately fitting condylar-ramus construct or scaffold (Fig. 12.3).
2. **Design** of interior scaffold architecture via computer modeling. This step would take into consideration the structural integrity of the scaffold that is necessary to withstand functional loads. One would also minimize the amount of polymer that is utilized in concert with maximizing volume void such that the biodegradable scaffold will in time be replaced by mineralized tissue without compromising the structural integrity of the condylar-ramus construct/scaffold. The target properties would closely approximate those of bone and cartilage at the articulating surface (Figs. 12.4 and 12.5).

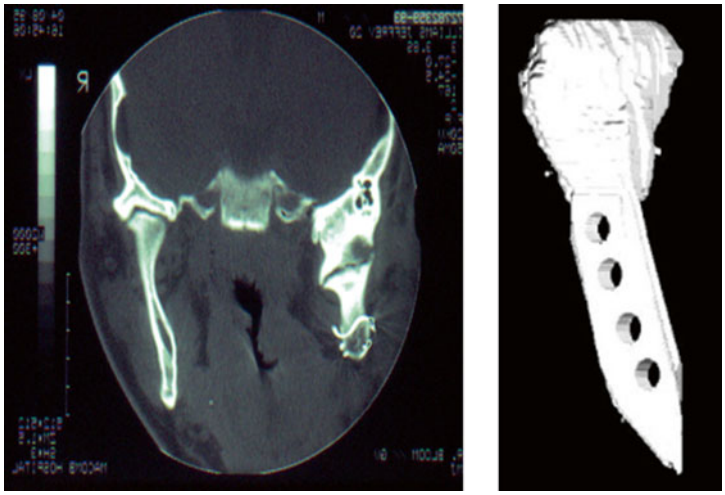

**Fig. 12.3** CT used to perform computer-generated rendition of unaffected side

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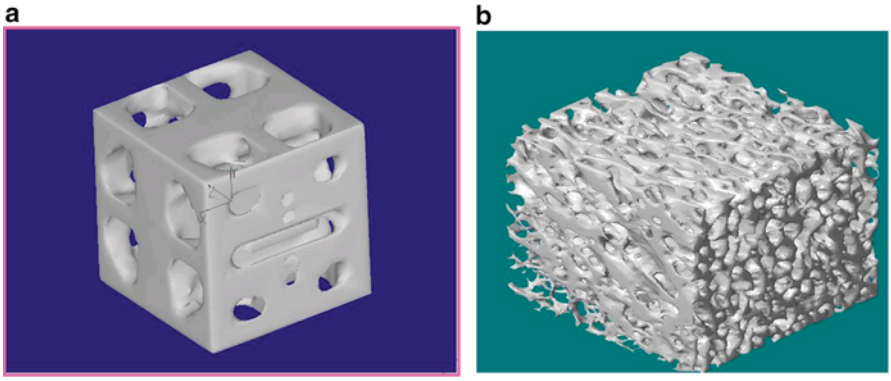


**Fig. 12.4** Designing the internal architecture of the scaffold: (**a**) micropores and/or (**b**) similar to bone

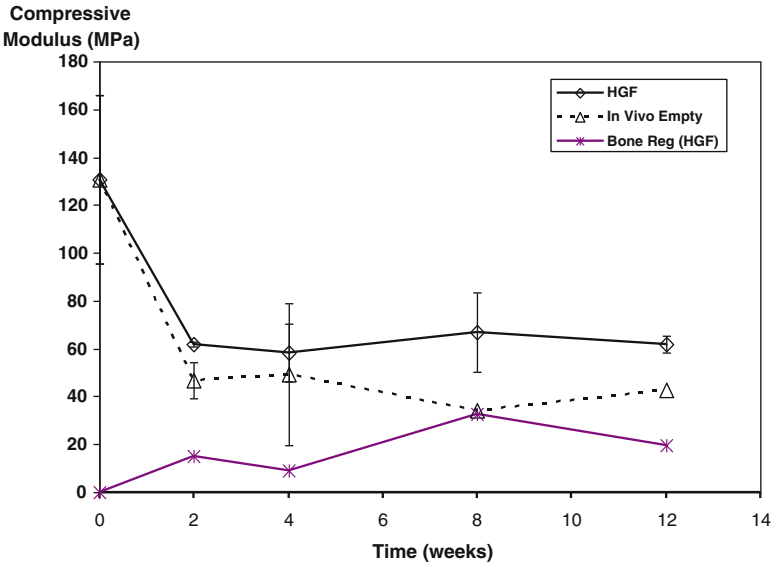


**Fig. 12.5** Bone stiffness increases with time; scaffold stiffness decreases; overall stiffness remains constant >2 weeks [39]

3. **Combining steps 2 and 3** via Boolean operations on a computer to finalize the external and internal architecture to optimize the condylar-ramus scaffold (Fig. 12.6).

   The design pattern would also take into consideration fixation of the scaffold to the remaining ramus of the mandible using either a standard lateral approach seen with costochondral rib grafts (above) or a more innovative design using a "U"-shaped scaffold to fit onto the posterior border of the ramus (Fig. 12.7).

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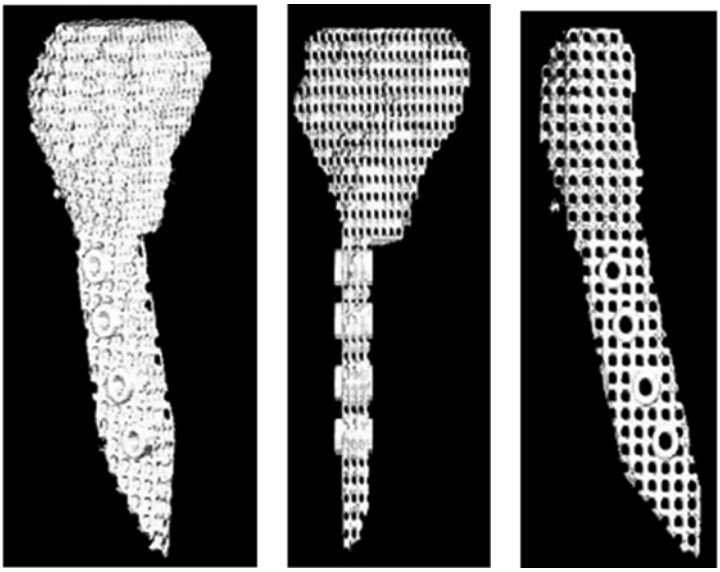

**Fig. 12.6** Internal and external architecture modified using computer software

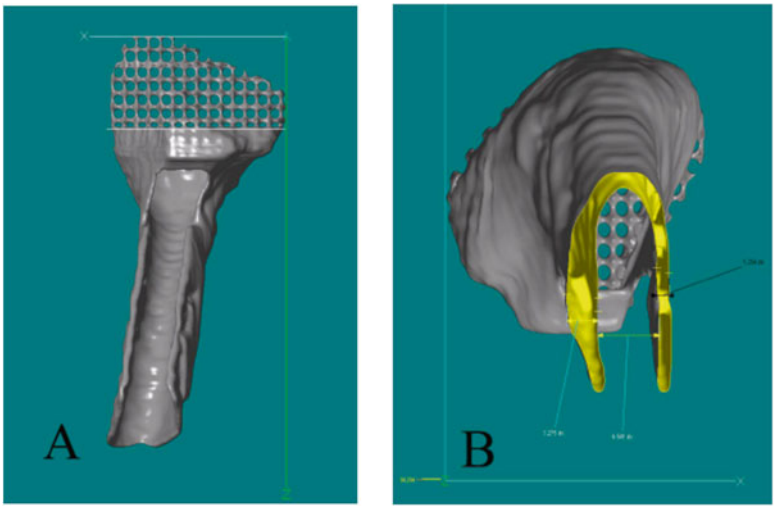


**Fig. 12.7** Computer-designed "U"-shaped scaffold

4. **Manufacturing** of the patient-specific designed condylar-ramus scaffold using a solid free-form fabrication technique, i.e., a 3D printer. This could be selective laser sintering, fused deposition modeling, etc. This has been accomplished in the past using polycaprolactone (PCL) by our research group. To make the scaffold more interactive, we could consider doing several of the following:

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(a) Create either an osteoconductive or osteoinductive coating prior to implant placement. Murphy and others have created an osteoconductive coating that has already been tested in vitro and in vivo and achieved FDA approval with a PCL scaffold [40]. Liu et al. are working on a fluoride-based osteoinductive coating of PCL that to date has only been tested in vitro [41–43]. Murphy et al. have shown that these scaffolds also have the capability to bind osteobiologics, such as BMP, and control their kinetic release over time.

(b) The scaffolds could be made out of a composite of PCL and a conducting polymer such as polyaniline. The electric fields created could be optimized for bone or cartilage on the same condylar-ramus construct/scaffold [44, 45]. This would encourage bone to form on the ramus portion and cartilage to form on the articulating surface. This approach could be combined with one of the coatings mentioned above.

(c) Loading of scaffolds with a cell type of choice such as MSCs or adipocytes on either of the above types of scaffolds. The most efficacious way to do this would be in the operating room for several reasons. Once cells are removed from the operating room, the complexity and cost in both time and labor increase exponentially. There are numerous FDA regulatory guidelines that need to be followed prior to placement of the cells back into the same or a different individual. It would be more cost-effective to either perform a hip aspirate or use a machine in the operating theater that could isolate adipocyte stem cells from a liposuction aspirate. The scaffolds could then be loaded within the operating theater prior to or at the time of implantation of the condylar-ramus scaffold. Still another approach would be using the cell homing method mentioned previously in which stem cell homing from bone marrow via bloodstream or from the tissue niche can occur [46, 47].

5. **Implantation** of the condylar-ramus scaffold

   The condylar-ramus scaffold would be secured with biodegradable PCL screws with the patient in maxillo-mandibular fixation to assure proper positioning of the tissue-engineered prosthesis (Figs. 12.8 and 12.9).

If the glenoid fossa needs reconstruction, it should be done at the same time as placement of the condylar-ramus construct/scaffold and be manufactured with the same composite material, coating, and cells such that like material is articulating with like material.

The issue of if and when to place a disc presents a quandary. The articular disc assists in distribution of loads and absorbs "shocks" to the TMJ during function. Would it be necessary to replace the disc with the tissue-engineered condylar-ramus scaffold with or without prosthesis of the glenoid fossa? If so, when should it be done? At the time of the implantation of the prosthesis or in a second staged surgery? It would seem that if a disc was placed, one would utilize, at this moment in time, the unique FDA-approved UBM-ECM scaffold. How this would remodel in a TMJ with biodegradable scaffolds for the condyle alone or also with a fossa scaffold will have to be determined by future studies.

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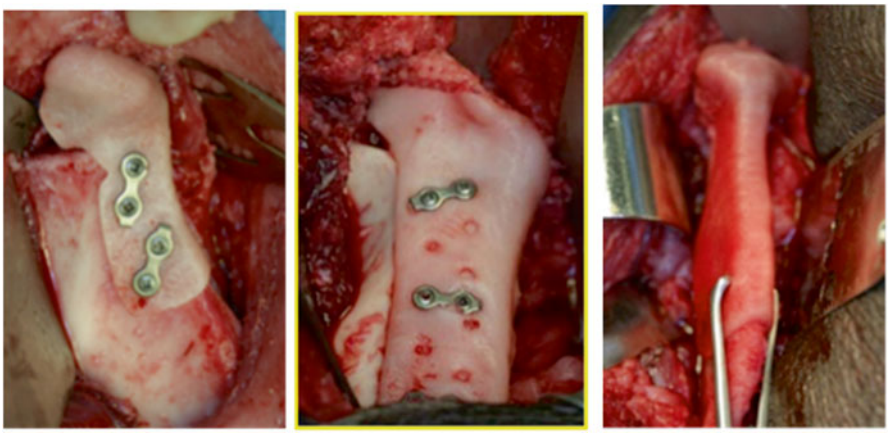

**Fig. 12.8** Illustrate placement of such a "U"-shaped scaffold with titanium plates and screws in a Yucatan minipig. Once secured in place, the patient would immediately be placed into function to activate the cells and initiate functional remodeling

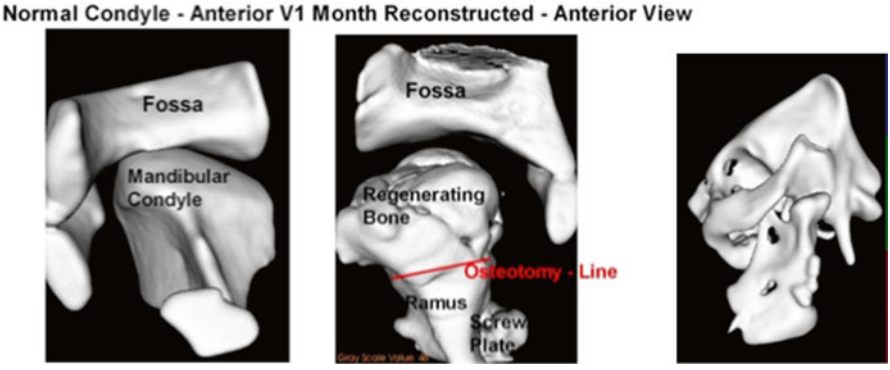


**Fig. 12.9** Remodeling of scaffold after 3 months in a Yucatan minipig placed into function immediately after placement

The other area of controversy is placement of the capsular ligament. To date, no FDA-approved ligaments have been approved, but other ligaments, such as the anterior cruciate ligament, is under investigation and awaiting clinical trials [48].

Future issues that will also have to be addressed through further studies would include:

1. Function: range of motion of prosthesis and joint
2. Fixation: means of stabilization of prosthesis with joint movement especially in early physical therapy
3. Wear resistance

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4. Problem: absence, in most cases, of an articular disc which assists in distribution of loads and absorbs "shocks" to the TMJ
5. Since this is a biologic replacement, subject to systemic disease (high inflammatory arthritic diseases such as RA) and local functional loading forces (concomitant orthognathic surgery where posterior mandibular vertical dimension is increased and the mandible is rotated counterclockwise), can such a device provide long-term stability?
6. Can a biologic TMJ device be used to reconstruct an ankylosis, failed prior autogenous or alloplastic device, or the multiply operated failed TMJ case?

In summary, the advantages of a tissue engineering TMJ prosthesis would be:

1. No secondary donor site
2. Decreased surgical time and hospital stay
3. A construct that mimics the anatomic contours of structures being replaced (or a patient-specific anatomical restoration of missing structures)
4. More easily adapted to surgical site
5. Can be used in growing children
6. Remodels to functional forces "Wolff's law" (theory of mechanotransduction)

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