TMJ Dislocation: From Etiology to Management

David Y. Cho, DDS
Resident, Department of Oral and Maxillofacial Surgery, Texas A&M College of Dentistry
Resident, Department of Oral and Maxillofacial Surgery, Baylor University Medical Center
Lieutenant, Dental Corps, United States Navy

Andrew Read-Fuller, DDS, MD, MS
Clinical Assistant Professor, Oral and Maxillofacial Surgery, Texas A&M College of Dentistry
Attending Physician, Baylor University Medical Center

INTRODUCTION

The temporomandibular joint (TMJ) is a complex system of parts that, when working in harmony, allows for the movements of the mandible necessary for proper speech and mastication. Improper function of any component of this dynamic system can result in a multitude of pathophysiologic disorders referred to collectively as temporomandibular joint disorders (TMDs), one of which is TMJ dislocation. Dislocation of the TMJ occurs when the normal "ball and socket" anatomic relationship between the mandibular condyle (i.e. the ball) and the glenoid fossa (i.e. the socket) is lost. Anterior dislocation is most common[1-4] representing an estimated 95% of TMJ dislocations,[5] but dislocation from the fossa in other directions is also observed. While it represents only 3% of the joint dislocations in the body,[6-7] is uncommonly encountered in an ED setting,[8] and is found in as few as 1.8% of symptomatic TMJ patients,[9] TMJ dislocation requires proper diagnosis and management by the oral and maxillofacial surgeon to prevent its pathologic progression of disease.[10-11] Unfortunately, current and past literature on this condition lacks consistent terminology, definite guidelines, and generalizable treatment protocols.[10,12-13] The purpose of this article will not only be to review the disease process, but also to explain how its etiology can help guide its management.

BACKGROUND

Anatomy

Normal TMJ function requires a coordinated interplay between several osseous, ligamentous, muscular and dense fibrous structures. While the condyle of the mandible and glenoid fossa of the temporal bone represent the ball and socket components of the TMJ, respectively, proper joint function and stability also relies on the joint capsule, articular disk and retrodiskal tissues, lateral and accessory ligaments, and supra-mandibular muscles of mastication. This complex of tissues forms a ginglymoarthrodial joint capable of the rotational and translational movements necessary for speech and mastication.

The osseous components of the TMJ include the condyle of the mandible and the articular portion of the temporal bone, which is comprised of the glenoid fossa, articular eminence (or articular tubercle), and post-glenoid process. The posterior slope of the articular eminence serves as the anterior, stress-bearing boundary of the fossa during mandibular translation, while the post-glenoid tubercle and squamotympanic fissure act as the posterior border of the fossa, separating it from the osseous external auditory canal (EAC). The glenoid fossa is separated from the middle cranial fossa by a thin layer of compact temporal bone, which, in the absence of trauma or pathologic changes to the joint, is not a major stress-bearing osseous component of the TMJ. The spine of the sphenoid bone and the zygomatic arch lie medial and lateral to the fossa, respectively, and are the osseous origins of two key structures that confer stability and function to the TMJ: the sphenomandibular ligament and masseteric muscle.[7,14-15]

The joint capsule is comprised of collateral (or diskal) ligaments and capsular ligaments. The collateral ligaments are short, paired ligaments that attach the disk (i.e. the meniscus of the TMJ) to the medial and lateral poles of the mandibular condyle (**Figure 1**).

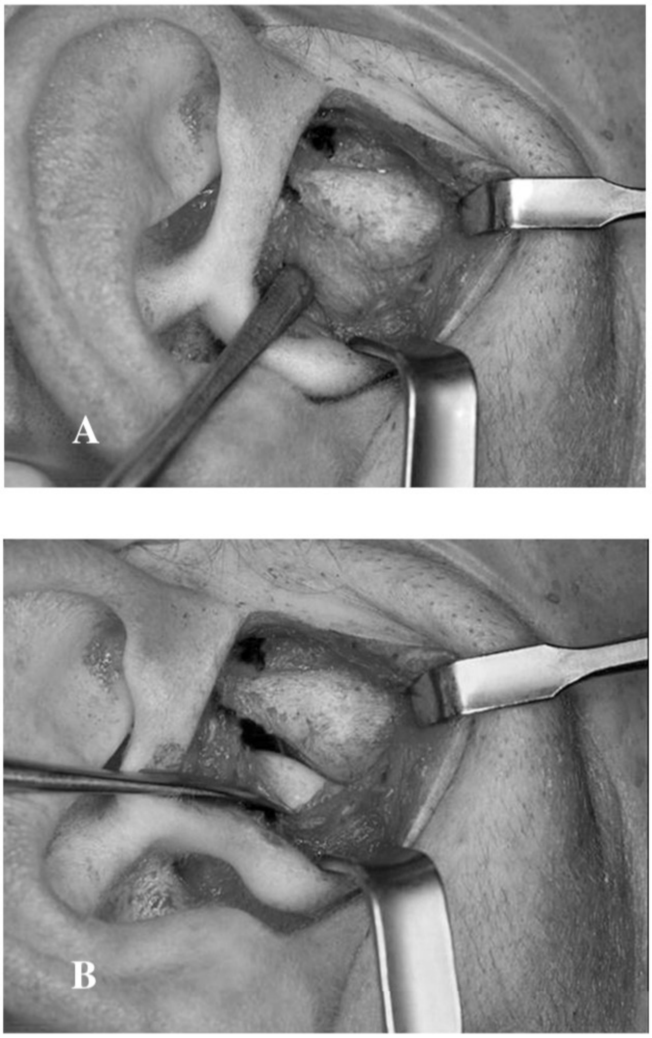

Figure 1:A, View of lateral capsule through a preauricular incision. **B**, Lateral capsule incised and an intact, shiny, white and firm disc is visualized. (From Chung WL, Ochs MW: *Open arthrotomy for the management of internal derangement of the temporomandibular joint*, Selected readings in Oral and Maxillofacial Surgery, Vol 14; 2006)

This attachment to the disk ensures a close spatial relationship between the mobile condyle and the disk. This confers its ability to withstand the compressive forces produced in function – thereby ensuring protection of the osseous structures involved – while also allowing for "smooth, synchronous motion of the disk-condyle complex." [16] Capsular ligaments arise from the neck of the condyle, enveloping the joint circumferentially, and have skull-base condensations which span anteriorly to the pre-glenoid plane and eminence, superiorly and medially to the glenoid fossa, and posteriorly to the squamous portion of the temporal bone. These contiguous ligaments stabilize the joint when subjected to medial, lateral and inferior forces,[5] and it is within this capsule that the articular disk, joint spaces (superior and inferior), and synovial fluid are found.[17-18]

The articular disk, which serves as the meniscus of the TMJ, is comprised of dense, fibrous, and flexible connective tissue which lacks both vascularity and innervation, all of which are properties that allow for it to adapt to, and withstand, repeated compressive and frictional forces without inflammatory changes or relaying of pain signals to the brain. While histologically comprised of the same tissue, it is anatomically divided into three regions based on its macroscopic appearance: the thicker anterior and posterior bands act as anatomic barriers to the thinner, central intermediate zone on which the condyle articulates on normal function. As mentioned previously, the anatomic relationship between the disk and condyle is maintained during function via its attachments to the joint capsule, but also by fibers of the lateral pterygoid, which insert on the medial aspect of the disk.[16-17]

Posteriorly, the disk is contiguous with the bilaminar zone, an area of highly vascular and innervated retrodiskal tissues that are further subdivided into the superior retrodiskal lamina and inferior retrodiskal lamina[19]. While both contribute to the production of synovial fluid – a hyaluronic acid rich, ultrafiltrate of plasma which serves to lubricate, clear debris, and nourish the articular cartilage of the joint – they exhibit some differences in histology. The superior retrodiskal lamina contains elastic fibers which, in extreme translational movements, restrain excessive displacement of the disk through its insertion onto the tympanic plate. In contrast, the inferior retrodiskal lamina contains collagen, rather than elastic fibers, and restrains disk movement away from the condyle through its insertion onto the articular surfaces of the condyle.[17,19]

While the fibrous capsule, articular disk, and retrodiskal tissues confer stability to the joint in function, additional stability is provided by lateral ligament – otherwise known as the temporomandibular ligament (TML) – as well as two accessory ligaments: the sphenomandibular and stylomandibular ligaments. As its name implies, the lateral ligament inserts onto the lateral portion of the fibrous capsule. It is comprised of two distinct portions: the outer oblique and the inner horizontal portion. The outer oblique arises from the lateral aspect of the articular eminence of the temporal bone and descends postero-inferiorly to insert onto the posterior surface of the condylar neck, thereby limiting excessive inferior movement during translational and rotational movements.[19] While the inner horizontal portion also arises from the lateral aspect of the eminence, it travels deep to the outer oblique, in a more horizontal direction, to insert onto both the lateral pole of the condyle as well as the posterior disk. This prevents excessive posterior displacement of the condyle, which in turn protects the highly vascular and innervated retrodiskal tissues which is critical since impingement of these tissues elicits an inflammatory response and relaying of pain signals to the brain. It also plays an essential role in maintaining stability of the ipsilateral condyle during lateral excursive movements, as this condyle is forced posteriorly in its fossa by contraction of the contralateral medial pterygoid.[17] The sphenomandibular and stylomandibular ligaments arise from the spine of the sphenoid and styloid process, respectively. The sphenomandibular ligament inserts onto the medial aspect of the mandible at the lingula while the stylomandibular ligament inserts onto the lower condylar neck and posterior border of the ramus immediately superior to the angle.[16] They both, to a certain extent, serve as a hinge point about which the mandible rotates, with the stylomandibular ligament having the added function of limiting excessive protrusive movements of the mandible. Thesestabilizing forces may be compromised to some degree in individuals with elongated styloid processes due to the resultant change in vector, specifically, the decrease in superior traction it typically provides.[7,17]

The bilateral groups of muscles that insert on the mandible and influence its movements can be classified into two groups: the supra-mandibular muscles and infra-mandibular muscles. The supra-mandibular muscle group – comprised of the temporalis, masseter, medial pterygoid and lateral pterygoid muscles – is commonly referred to as the muscles of mastication for their role in elevating, protruding and retruding the mandible, all of which are necessary for mastication. The infra-mandibular muscle groups – which includes the suprahyoid group (digastrics, geniohyoid, mylohyoid and stylohyoid) and infrahyoid group (sternohyoid, omohyoid, stern thyroid, and thyrohyoid) – serve multiple functions in the neck, including aiding in depression of the mandible.[17]

This intimate network of fibrous and ligamentous components of the TMJ work in concert to resist excessive displacement of the TMJ and mandible upon activation of the supra-mandibular muscles of mastication.[14] Although it is widely understood that the integrity of the capsule and ligaments are most essential in maintaining a stable joint, proper structure and function of all components, including normal dynamics of the neuromuscular mechanism controlling the muscles of mastication, are necessary to maintain a stable joint and prevent dislocation[5,7,14].

Definition and Classification

TMJ dislocation most simply can be defined as a non-self-limiting condition in which the condyle of the mandible is seated outside of its normal anatomic position. (**Figure 2**)

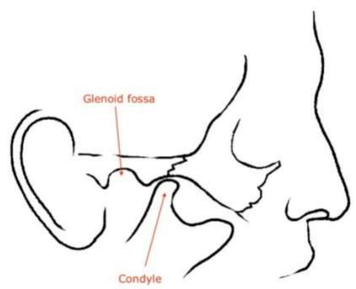

Figure 2: Depiction of the mandibular condyle outside of its normal anatomic position within the glenoid fossa of the temporal bone.

While the term subluxation is sometimes used to describe this state, this is an inappropriate use of the term as subluxation refers to transient and/or partial, and self-limiting, displacement outside of the fossa.[10,14,20] In true TMJ dislocation, the condyle no longer is seated within its normal anatomic and functional position posterior to the articular eminence and within the glenoid fossa. Although the condyle can sometimes be self-reduced by the patient in situations to be described later on, this is inherently different from subluxation, in which the joint reduces back into its normal anatomic position without any overt manipulation.[10,12] TMJ dislocation can be further described and classified in a multitude of ways, including direction of displacement (anterior, medial, posterior, superior, lateral), laterality (unilateral vs. bilateral), duration and/or disease course, and etiology. While it has historically been most commonly classified based on the duration of dislocation and history disease– acute, chronic protracted, and chronic recurrent – as described by Adekeye et. al. and Rowe and Killey,[2,21] utilizing these methods of classification alone is inadequate when determining an appropriate treatment course that can achieve predictable, long-term success.[22] In response, recent literature has advocated basing treatment decisions on etiology,[1] condylar position,[5] history of disease (i.e. number and duration of episodes, previous interventions), as well as medical comorbidities and age[12,24] in order to determine a more case-specific treatment approach that will lend itself to improved, and more predictable treatment outcomes.[5,22,24]

Classification of TMJ dislocation based on duration of episode and history is essential since management of acute dislocations differs from that of chronic protracted and chronic recurrent dislocations. Acute dislocation is most commonly described as an isolated episode of TMJ dislocation. While some argue that this term should be reserved for dislocations that have occurred within 72 hours.[12,25-26] At present, there isn't a universally accepted duration of dislocation that demarcates an acute from a chronic protracted condition.[10,22] Many consider untreated TMJ dislocations that have occurred within 2 weeks to be acute,[5,7] largely based on findings that indicate joints dislocated for longer than 2 weeks require a different treatment approach than those dislocated for a shorter duration of time.[5] For the purposes of this review, chronic protracted dislocation, also referred to as chronic persistent and chronic long-standing dislocation, will refer to anon-reduced TMJ dislocation present for longer than 2 weeks. As an acute dislocation remains untreated, the deranged position of the condyle can result in spasm of the muscles of mastication. This, in combination with fibrotic changes in the muscles and ligaments of the TMJ, impede and complicate reduction, and can contribute to its progression to a chronic protracted state.[1,27] Both the acute and chronic protracted conditions are frequently referred to as "open-lock," as loss of the normal articular relationship between the fossa and condyle-disk complex results in patients presenting with an inability to close their mouth from an open position.[28] (**Figure 3**)

Figure 3: Clinical photograph of patient presenting with mandibular condyle in "open-lock" position (i.e. anterior temporomandibular joint dislocation).

Contrary to the chronic protracted condition, which refers to a single episode, chronic recurrent dislocation, also called chronic habitual dislocation, refers to a disease state in which a patient presents with a history of multiple episodes of dislocation. In these patients, the normal anatomic relationship between the condylar-disk complex and fossa has been lost repeatedly. While acute, chronic protracted, and chronic recurrent dislocations are not mutually exclusive conditions (patients can experience acute or chronic protracted dislocation with a history of recurrent dislocations) changes in the anatomy and neuromuscular dynamics of the TMJ in patients suffering from chronic recurrent dislocations often times facilitate reduction of the condyle, and therefore minimize the chances of an acute or chronic protracted episode.

Clinical Features, Evaluation, and Diagnosis

CLINICAL FEATURES

Acute TMJ dislocation generally presents with a mouth in the open-lock position, palpable muscular spasm and/or tension (particularly in the temporalis region), difficulty with phonation, mastication and clearing of saliva due to both the open-lock position itself as well as an impeded neuromuscular coordination, and – most bothersome to the patient – preauricular pain, discomfort and psychological distress.[6,8,29] Although some studies have found a higher prevalence in females,[30-31] at present, epidemiological prevalence studies fail to show any clear gender predilection.[1] On clinical examination, facial deformityis obvious,[1] particularly in unilateral cases, and a pre-auricular depression due to the empty joint socket may be palpable.[27] In anterior dislocations, which are most frequently encountered,[1,5] the condyle has translated anterior to the articular eminence and now lies in an anterio-superior position in relation to the glenoid fossa. This occurs when posterior traction of the condyle by muscular and ligamentous structures fails to impede translation of the condylar-disk complex beyond the tip of the articular eminence. In normal function, relaxation of the lateral pterygoid muscles allows for postero-superior pull of the mandible by the activation of the temporalis and masseter muscles. However, once beyond the inferior limit and onto the anterior slope of the eminence, continued activation or myospasm of the muscles of mastication without relaxation of the lateral pterygoids can lead to further displacement in an antero-superior direction. Unilateral anterior dislocation presents with a contralateral mandibular midline shift and crossbite malocclusion. Bilateral anterior dislocation, which is exceedingly more common than unilateral, presents with mentalis protrusion and mandibular prognathism with anterior crossbite.[7,32] Superior, or central, dislocations – often the result of a direct blow to a partially opened mouth – may present with concomitant fracture of the glenoid fossa and trauma to other components surrounding and within the temporal bone. This may allow for displacement of the condylar-disk complex into the middle cranial fossa which can cause intracranial hematomas, cerebral contusions, CSF leaks, as well as damage to both the facial and vestibulocochlear cranial nerves.[10,33] Posterior dislocations, also typically a result of trauma to the chin, can present with concomitant EAC injury but infrequently traumatize inner earstructures.[34] Lateral dislocation of the condyle most frequently is seen following a sub condylar fracture, which can present with the condylar head being lateral and superior to the fossa and palpable in the temporal space.[10] Lastly, medial dislocations (**Figure 4**), which are most frequently secondary to unilateral anterior dislocation and subsequent sustained pull and spastic activity of the lateral pterygoid on its condylar insertion, present with contralateral deviation of the mandible.[5,35]

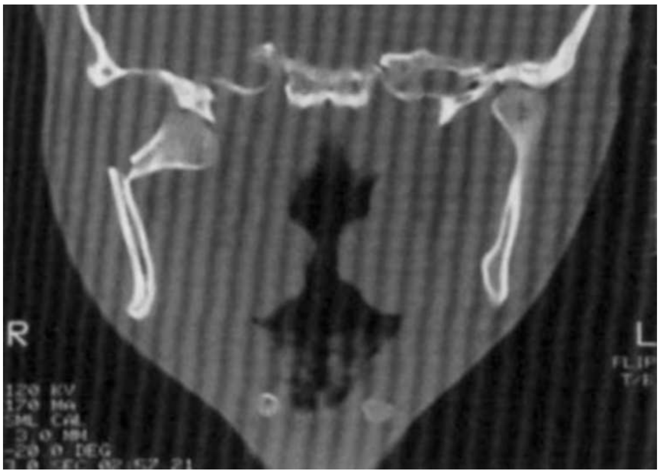

Figure 4: CT image (coronal slice) depicting a medial dislocation of the right mandibular condylar head.

A chronic protracted state often has a similar clinical presentation as acute dislocation with the major difference often times being an inability to simply manually reduce the condyle into its normal anatomic position. Similar to acute dislocations, bilateral anterior dislocation is the most commonly encountered chronic protracted state and presents with the mandible in an often painful, open-lock position.[7,10,12] While a chronic protracted dislocation frequently presents with limitations in any translational or rotational movements, in some cases, a new pseudo-joint is established that allows for limited translational and rotational movement of the condyle. Although some function is re-established, the new position of the condyle results in a change in occlusion which must be addressed. Regardless of whether or not a pseudo-joint is established, as the untreated and non-reduced condyle remains in a dislocated position, spasm of the supramandibular muscles of mastication, particularly the temporalis and lateral pterygoids, further inhibits movement of the condyle back into its native position and increases the difficulty of reduction. Further complicating reduction are progressive fibrotic changes observed in retrodiskal, muscular and ligamentous tissues.[7,14] The result of this pathological progression of disease is maintenance of the condyle in its dislocated position.

As is the case with both acute and chronic protracted dislocation, a chronic recurrent disease state is most commonly seen with anterior dislocation. While chronic recurrent dislocation is commonly reducible by the patient and less frequently presents with pain, it is far from a benign process. Each episode of dislocation is accompanied by hyperactivity of the muscles of mastication as well as occlusal changes, and resultant parafunctional habits can have a destructive effect on the teeth, periodontium, as well as on the components of the TMJ.[36-37] These adverse effects of recurrent dislocation exacerbate any pre-existing congenital or acquired contributing factors, to be described later, and will collectively lead to future episodes and continued pathologic changes within the joint.

EVALUATION AND DIAGNOSIS

At present, there are no standardized methods of evaluating patients who present with concerns of acute TMJ dislocation. Since, for reasons to be discussed later, the failure to treat an acute dislocation expediently can increase the chances of future episodes,[12,27,38] appropriate but not superfluous work-up is essential to prevent any unnecessary delay in reduction of the dislocated condyle. Patients presenting with signs of acute TMJ dislocation without acute facial trauma can be diagnosed after a thorough medical history and physical examination alone if findings are sufficient.[27,38] As previously mentioned, anterior dislocation is the most commonly occurring and presents with the cardinal symptoms of "open-lock," deranged occlusion, and pre-auricular pain and tenderness. Headaches localized to the frontoparietal and occipital regions, and masticatory muscle tenderness may also be observed.[27,38-39] Patients will frequently endorse an inciting event that immediately preceded the dislocation and may report a history of subluxation on excessive opening. On clinical exam, a pre-auricular depression, representing an empty glenoid fossa, may be palpable, but can be masked by the presence of edema, particularly in a trauma setting.[38] When presented with these cardinal symptoms, history and clinical findings in patients without facial trauma, the most appropriate and effective course of treatment is immediate reduction.[1] However, a diagnosis of dislocation may be less apparent in patients who are unable to participate fully in an exam as a result of being sedated or due to cognitive deficits related to dementia.[38-41] In these cases, as well as in the setting of facial trauma, radiographic studies should be employed to assess the position of the TMJ components prior to any intervention.[1,42] In an acute setting, plain film (transcranio-oblique view), orthopantomogram (OPG), computed tomography (CT), and cone-beam computed tomography (CBCT) should be favored over magnetic resonance imaging (MRI) and diagnostic arthroscopy since their shorter study time allows for more rapid intervention when indicated.[38,41]

In patients with a history of recurrent dislocation, and when inflammatory degenerative joint disease is suspected, further studies are warranted and will aid in appropriate treatment planning. MRI and arthroscopic exams allow the surgeon to evaluate the condition and function of the disk and intracapsular components. When used in conjunction with CT findings, particularly the presence of any degenerative changes or anatomic aberrancies, the surgeon can not only diagnose a dislocated joint, but may also be able to identify or rule out the presence of anatomic changes, pathology or fractures that may serve as contributing factors.[26,43]

ETIOPATHOGENESIS

TMJ dislocation is a condition with a complex etiology, the components of which can be categorized as inciting events and contributing factors.[2,14,44] While the inciting event refers to what immediately led to dislocation, contributing factors represent pre-existing anatomic anomalies, pathologies, systemic conditions, and medication-related side-effects that increase the susceptibility of a joint to dislocation.

The inciting event in cases of TMJ dislocation is commonly reported to be trauma to the mandible resulting from a fall, MVC or interpersonal violence.[33,45] However, dislocation also has been found to occur as a result of excessive opening or loading of the joint during everyday activities such as yawning, laughing, and speaking,[38,39,46-47] with Ugboko et.al. reporting yawning as the inciting event in 46% of dislocations seen in their retrospective study of 96 cases in Nigeria.[1,10] Prolonged opening during dental and ENT procedures, as well as after iatrogenic trauma inflicted during intubation and endoscopic procedures, have also been implicated.[27,41,48-49] (**Box 1**)

Box 1: Inciting Events
• **Trauma**: fall, motor vehicle collision, interpersonal violence, etc. • **Everyday activities**: yawning, laughing, speaking, etc. • **Iatrogenic**: prolonged opening during dental and ENT procedures, intubation, and endoscopic procedures.

In a 2011 systemic review published by Akinbami, 425 cases of dislocation from 79 published articles were categorized based on inciting event, direction, chronicity (acute vs chronic) and laterality (unilateral vs bilateral). Greater than 95% (404/425) were anterior dislocations, 73% were chronic recurrent dislocations (311/425), and 99% were bilateral. In this study, the inciting event was found to be trauma-related in 60% of all dislocations, but was higher in posterior, superior and lateral dislocations, which were almost exclusively presented in a trauma setting. Posterior and superior dislocations most commonly occur as a result of direct blows to the chin in a closed position and partially open position, respectively, while medial and lateral dislocations are usually associated with mandible fractures.[5,10,33-34]

Regardless of type of inciting event, dislocation occurs when forces acting on the mandible exceed the stabilizing components of the joint, causing displacement of the condyle out of the glenoid fossa. The likelihood of these events leading to dislocation is not only determined by the amount of force but is also influenced by other contributing factors. As such, it is not only important to describe possible mechanisms, or inciting events, of this disease state, but for reasons to be discussed later, it is quite possibly more important to identify and understand these factors that contribute to a hypermobile joint that is predisposed dislocation. Broadly speaking, these factors include any acquired and congenital conditions that alter normal morphology of osseous, fibrous and ligamentous structures of the TMJ, and its neuromuscular dynamics, all of which are necessary in maintaining a functionally stable joint. (**Box 2**)

Box 2: Contributing Factors
Systemic diseases • Connective tissue disorders (e.g. Ehlers-Danlos syndrome, Marfan syndrome, etc) • Neurologic and neuromuscular diseases (e.g. Huntington's disease, epilepsy, Parkinson's disease, muscular dystrophies such as Duchenne's) **Anatomic abnormalities (congenital or acquired)** • Increased capsular laxity • Weak or atrophic ligaments • Hypoplastic or atrophic mandibular condyles and/or articular eminence • Hypoplastic zygomatic arch and/or narrow/shallow glenoid fossa • Elongated styloid process • Loss of posterior occlusal support (i.e. loss of vertical dimension) **Drug-induced movement disorders (e.g. extrapyramidal symptoms)** • Lithium, psychostimulants (e.g. amphetamines), SSRIs, TCAs (e.g. amitriptyline), MAO inhibitors, anticonvulsants (e.g. valproic acid), and anti-emetic medications (e.g. metoclopramide and prochloperazine)

Congenital or acquired anatomic abnormalities, such as the anatomic changes that are observed in patients with other temporomandibular joint disorders like internal disk derangement, can serve as contributing factors. Anatomic abnormalities that have been found to establish an unstable joint include: increased capsular laxity, weak or atrophic ligaments, hypoplastic or atrophic mandibular condyles or articular eminence, an elongated articular eminence, a hypoplastic zygomatic arch, and a narrow or shallow glenoid fossa.[5,7,50] Capsular and ligamentous laxity reduces the stabilizing forces on a joint, in all directions, while a smaller eminence, zygomatic arch and shallow fossa minimize the prominence of osseous boundaries that typically incarcerate the condyle.[5,7] An elongated styloid process, with its distal tip being in a more caudal position, may also increase susceptibility to dislocation due to a reduced superior vector of traction on the mandible provided by the stylomandibular ligament.[7] In the edentulous population, prolonged lack of posterior occlusal support contributes to pathologic changes to the joint components.[27] Loss of a vertical stop provided by occlusion leads to persistent over-closing of the mandible (i.e. loss of vertical dimension) and may permanently stretch and loosen the TMJ components while also leading bony remodeling of the condyle and fossa.[7,50]

Systemic diseases also can serve as significant contributing factors in establishing a hypermobile joint.[51-52] Connective tissue disorders (e.g. Ehlers-Danlos syndrome, Marfan syndrome) may increase hypermobility due to effects on TMJ-associated ligaments.[1,10,53] In neurologic and neuromuscular diseases (e.g. Huntington's disease, Epilepsy, Parkinson's disease, muscular dystrophies such as Duchenne's) the effect on etiopathogenesis of TMJ dislocation can be two-fold.[1,54-55] Not only can their associated neuromuscular dysfunction (e.g. dystonias) and muscle weakness increase hypermobility through progressive stretching and muscular atrophy, gait disturbance also may contribute to a higher risk of falls in these patients, and therefore can increase the risk of trauma-related dislocation.[11,27] Although not well-documented, it has been suggested that the involuntary and spastic movement of the jaw in oromandibular dystonias, and repeated, excessive opening of the jaw in patients suffering from bulimia, may also stretch and weaken the peri-condylar tissues, thus destabilizing the joint.[14,56-57] Excessive opening related to these conditions also may serve as inciting events to dislocation which serves as an example of how inciting events and contributing factors are not always mutually exclusive. Similarly, medications that can precipitate drug-induced movement disorders (DIMDs), which include extrapyramidal symptoms (EPS), also can serve as both inciting events and contributing factors in TMJ dislocation.[1,44] Although these psychotropic dystonias are most commonly associated with neuroleptics (i.e. typical antipsychotics) – with an estimated 75% of patients on these medications experiencing EPS – DIMDs are also observed, albeit less frequently, with the use of lithium, psychostimulants, selective-serotonin reuptake inhibitors (SSRIs), tricyclic antidepressants (TCAs), monoamine oxidase (MAO) inhibitors, anticonvulsant medication, and anti-emetics metoclopramide and prochloperazine.[1,10,58] (**see Box 2**)

Acute dislocations, when treated expediently and appropriately, are predominantly isolated events without clinically significant long-term sequelae.[14] However, when improperly managed, dislocation itself can become a contributing factor that predisposes a patient to further episodes due to the pathologic changes that occur within the joint when a condyle remains in a dislocated state.[1,7,59] In acute dislocation, displacement of the condyle out of its normal position within the glenoid fossa generates reflex contraction and myospasm of the masseter, temporalis and lateral pterygoids which can exacerbate the dislocated state and impede reduction by non-surgical methods.[2,4] Protracted dislocation, particularly when a joint has been displaced for longer than 2 weeks, can lead to worsening myospasm, ligamentous laxity and capsular weakness, and promote degenerative and fibrotic changes to the disk, retrodiskal tissues, condyle and fossa components due to intra-articular effusions, all of which serve as contributing factors to hypermobility.[1-2,4,7,14] In dislocation with concomitant condylar fracture, fibrous and bony consolidation also may lead to ankylosis and establishment of a new pseudo-joint.[7,60] The extent of these deleterious changes, and the degree to which these changes contribute to the pathological progression of this disease, relies heavily on time to intervention (i.e. reduction) as well as the treatment strategy employed.[1-2,4]

Ultimately, development of TMJ dislocation should be seen as a multifactorial process with inciting events serving as the inflection point between a hypermobile, unstable TMJ that remains in its native position, and a dislocated condyle. The pathophysiology should also be seen as a complex, non-linear process in that many etiological factors can play a dual-role as both inciting events and contributing factors.

TREATMENT AND MANAGEMENT

Appropriate management of all forms of TMJ dislocation relies on achieving two key goals of care: 1) reduction of the joint and re-establishing the normal condylar-disk complex within the glenoid fossa, and 2) prevention of future occurrences. These goals are readily achievable in isolated, non-traumatic acute dislocations with timely intervention using one of many methods of manual reduction and post-reduction patient compliance.[7,25,27] However, in cases of chronic protracted and chronic recurrent dislocations, in which there are several contributory factors leading to progression of the pathologic disease state, other minimally invasive and surgical interventions are indicated for successful treatment. As previously mentioned, anterior dislocation is by far the most frequently encountered direction of dislocation, and as such, its management will be the predominant focus of this review.

Acute Dislocation

Cases of acute anterior dislocation are generally amendable to closed, manual reduction of the condyle back into its normal anatomic position, and infrequently require an open, surgical approach.[7,25,27] In the most commonly employed approach for anterior dislocation, the Hippocratic maneuver,[5,38] the physician positions the patient in a seated position, places his or her thumbs on the patient's mandibular molars, retromolar pad or external oblique ridge bilaterally, first applies caudal traction to the mandible to manipulate condylar head down the preglenoid plane and past the articular eminence, and then provides dorsal and cephalad guidance of the condyle back into the glenoid fossa.[46] (**Figure 5A-C**)

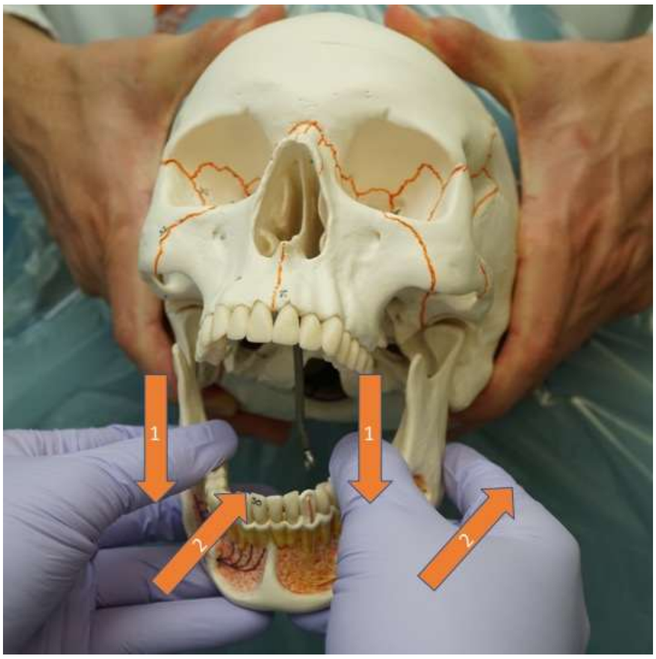

Figure 5A: Hippocratic maneuver displayed on a skull model: first caudal traction is applied to the mandible with thumbs positioned over the posterior mandibular dentition, then dorsal and cephalad guidance is provided as the condyle is manipulated past the inferior-most aspect of the eminence.

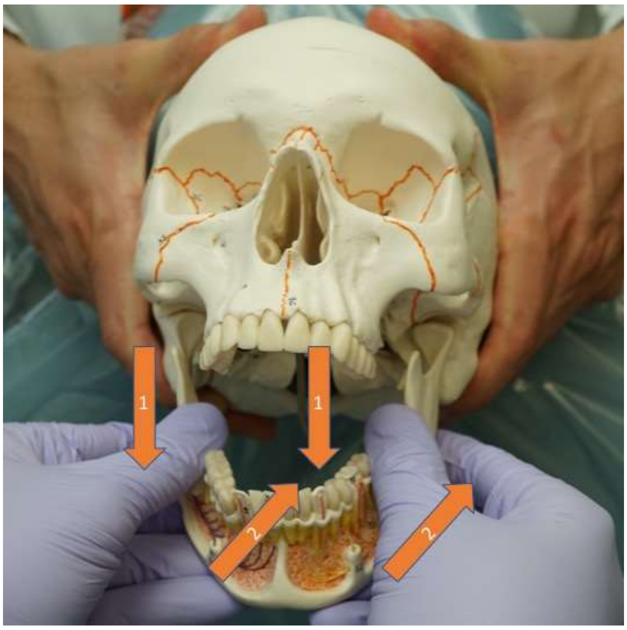

Figure 5B: Modified Hippocratic maneuver displayed on a skull model: thumbs positioned over the retromolar/external oblique.

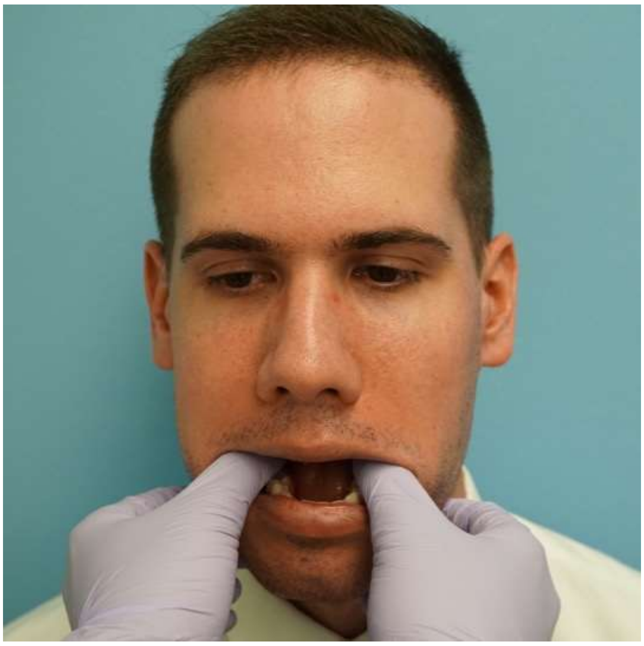

Figure 5C: Hand positioning of the Hippocratic maneuver.

In addition to this bimanual intraoral approach, there have been many other proposed intraoral and extraoral approaches. In the wrist-pivot technique proposed by Lowery et.al., the patient is similarly positioned in front of the physician whose thumbs are then placed on the inferior aspect of the chin extra orally with the remaining digits placed along the occlusal aspect of the patient's mandibular dentition on each side. Utilizing a pivoting motion of the wrist, caudal pressure is applied onto the teeth while superior/cephalad pressure is applied by the thumbs to the chin.[8,27,42,61] (**Figure 6A-B**)

Figure 6A: Wrist-pivot technique displayed on a skull model: caudal traction placed on the mandible with the second and third digits placed over the posterior dentition while cephalad pressure is applied to the chin by the thumbs.

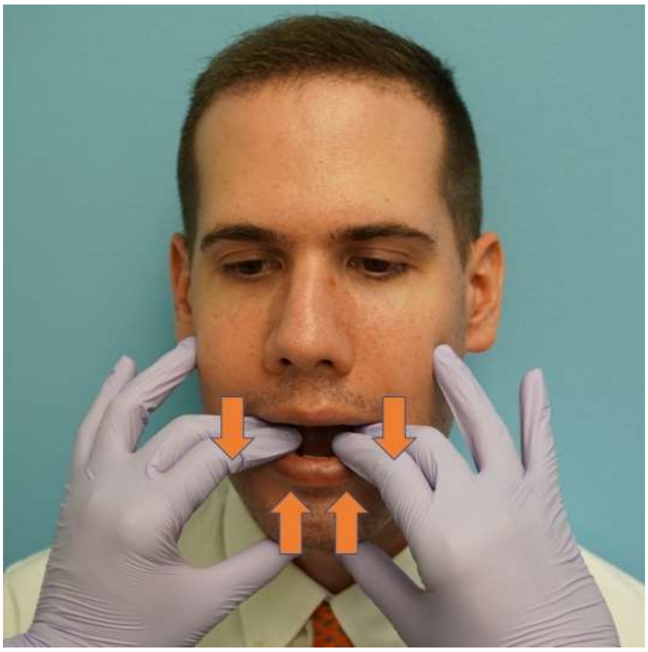

Figure 6B: Hand positioning of the wrist-pivot technique.

As intraoral manipulation with fingers placed between the patient's dentition carries with it an inherent risk to the physician, some alternative approaches have been proposed.[46] In one such technique described by Chen et al., the thumb of one hand is placed on the dislocated coronoid process, with the other fingers placed on the ipsilateral mastoid process.[62] On the opposite side, the other thumb is placed on the malar eminence with the remaining fingers along the angle of the mandible. The condyle is then reduced by applying steady, extraoral pressure to the coronoid in an infero-posterior direction with one hand while the other pulls the ramus anteriorly.[46,62] (**Figure 7A-B**)

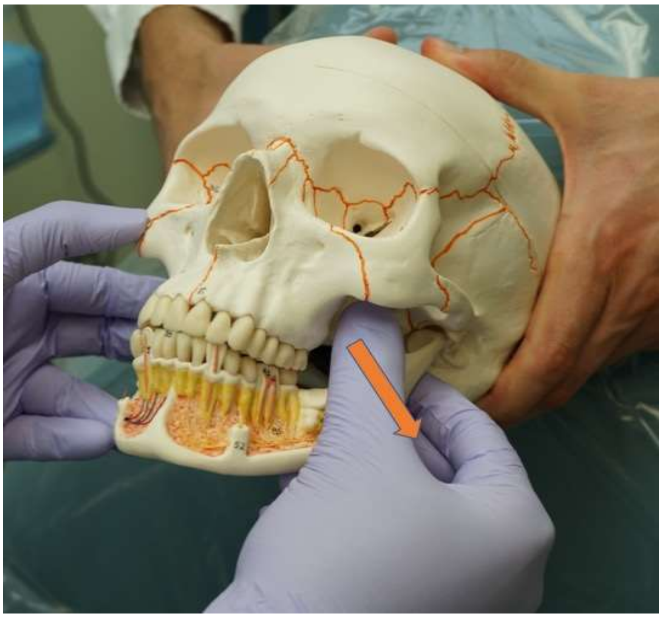

Figure 7A: Unilateral extraoral method of reduction displayed on a skull model: the thumb of one hand is placed on the dislocated coronoid process, with the other fingers placed on the ipsilateral mastoid process.

Figure 7B: On the other side, the other thumb is placed along the malar eminence while the remaining digits pull the mandible anteriorly along the posterior aspect of the ramus.

Another extraoral approach described by Ardehali et.al., allows for bilateral dislocations to be treated at the same time.[42] Thumbs are placed at the bilateral coronoid processes (anterior and inferior to the zygomatic arch) and postero-inferior pressure is applied while the remaining fingers apply anteriorly-directed pressure along the posterior border of the ramus and angle of the mandible.[42] (**Figure 8**)

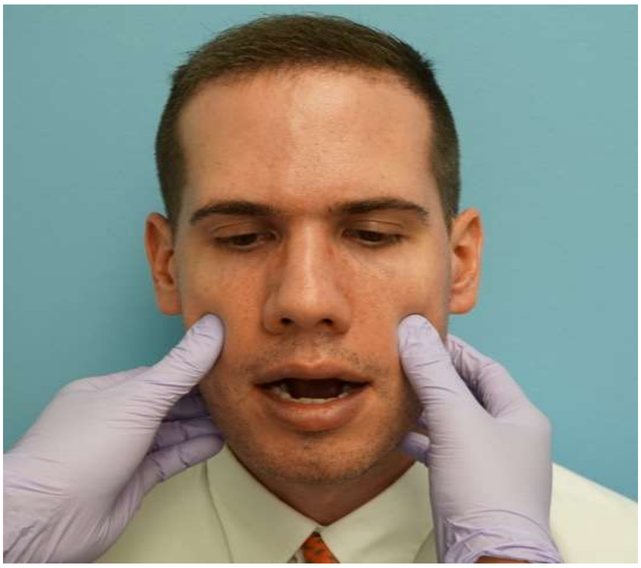

Figure 8: Hand positioning of a bilateral extraoral method of reduction.

Some have described that reduction of the condyles one side at a time via an intraoral, extraoral or combined approach may aid in ease of manual reduction, a technique called the combined ipsilateral staggering technique.[25] In one variation of this, one hand is positioned intraorally, similar to placement for the Hippocratic maneuver, with a downward and anterior traction being placed on the mandible while the other thumb is used to apply posterior pressure to the coronoid process. Alternatively, the hand that remains extraoral can be placed on top of the patient's head for added stability.[27] (**Figure 9A-B**)

Figure 9A: Ipsilateral staggering technique displayed on a skull model: one hand is placed intraorally and traction applied to the mandible in a manner consistent with the Hippocratic maneuver while the other is placed on the patient's head for added stability.

Figure 9B: Hand positioning of the ipsilateral staggering technique.

Certain proposed novel approaches, such as the gag reflex and syringe techniques, avoid manual manipulation by the physician altogether. In a case series of 3 patients, Awang described reduction of an anteriorly dislocated joint by eliciting a gag reflex using a mouth mirror placed along the junction of the hard and soft palate. It is thought that eliciting a gag reflex provokes centrally-mediated activation of depressors and reflex inhibition of elevators which allows reduction of the condyle that is no longer subject to spastic lateral pterygoid contraction.[14,63] The "syringe method, "which involves the use of a 5 or 10 mL syringe placed between the posterior molars, introduced by Gorchynski et al., purports to use the syringe as "a rolling fulcrum" that aids in reduction of the condyle as the patient is asked to roll the syringe back and forth between his/her teeth.[64]

In cases where manual reduction proves difficult due to complicating factors such as reflex contraction and myospasm of muscles, reduction can be facilitated through the administration of anxiolytics, general anesthetics, or muscle relaxants,[8,14,65-66] with much success being reported with Propofol sedation.[43] Pharmacologic assistance also can play a role when treating patients who are unable to tolerate manual reduction due to anxiety, intolerable pain, and cognitive deficits limiting patient cooperation.[3,8] Auriculotemporal, masseteric and deep temporal nerve blocks, as well injection of local anesthetics into preglenoid plane to minimize myospasm, have been shown to aid in reduction and should be considered when the use of sedatives and other medications are contraindicated due to medical comorbidities.[14,65]

Following reduction of an acutely dislocated joint, preventative measures must be taken to reduce the risk of future episodes.[7,46] If not given ample opportunity to heal, the stretched and deformed capsular and ligamentous components can have permanent effects on the stability of the joint. In the compliant patient, instructions for functional limitations for a period of 2 weeks may be sufficient to allow for adequate healing to occur. Specific instructions should include limited opening, soft diet, over-the-counter analgesics (NSAIDs are preferred if not contraindicated), and gradual physiotherapy.[1,27,54] However, maxillomandibular fixation (MMF) with arch bars and wires or elastics, the use of a bandage or dressing such as a Jobst dressing to limit excessive opening, and muscle relaxants are other possible methods of limiting TMJ function in order to facilitate healing.[46] These adjunctive, post-reduction treatments are not typically required in the compliant patient, but should be strongly considered in the setting of dementia, seizure disorder, intellectual disability, oromandibular dystonias related to systemic disease or home medications, or any other factors that may limit post-reductive patient compliance.[27]

Chronic Protracted Dislocation

Chronic protracted TMJ dislocation (also referred to as chronic prolonged or long-standing dislocation) is infrequently manageable with manual reduction alone, even when performed under general anesthesia. Other conservative interventions, such as the use of arch bars or maxillomandibular fixation (MMF) screws with elastic traction and acrylic or impression material spacers, have been found to be similarly unsuccessful in reduction of chronic protracted dislocation.[67-68] Although large sample size studies are limited due to its rare presentation, most agree that dislocations exceeding 3 weeks will require some form of surgical methods of reduction in order to regain function and restore occlusion.[5,12] In general, the longer a joint has been dislocated, the more difficult successful reduction and prevention of recurrence becomes.[12,27,38]

When manual reduction proves to be inadequate, surgical reduction of the dislocated condyle must be performed under general anesthesia in order to produce the forces necessary to counteract the muscular and connective tissue changes that have occurred during a protracted dislocated state. Successful reduction of bilateral anterior dislocation using a bone hook placed into the sigmoid notch was first performed by McGraw in 1899 on a patient that presented with a 5-month history of dislocation.[69] The original maneuver was conducted through an incision beneath the zygomatic arches, with a steel hook modified to have an additional bend that ran parallel to the handle (creating a Z-shape). This then was engaged onto the superior surface of the sigmoid notch and a downward force was applied while an assistant pulled the mandible forward with bimanual, intraoral manipulation.[70] In 1968, Rowe and Killey also employed the use of a bone hook, which they introduced into the sigmoid notch via a submandibular incision (Risdon approach) and through a subperiosteal tunnel. This allowed for downward traction to be placed on the mandible which facilitated successful reduction.[7,71] Alternatively, Lewis described successful reduction of a protracted dislocation with the use of a Bristow's elevator introduced down onto the condyle via an incision similar to the Gillies temporal approach used for reduction of zygomatic arch fractures.[72] Once in contact with the anterior aspect of the condyle, a downward and posterior force was applied until successful reduction of the condyle back into the fossa was achieved.[72] Other methods of applying traction to the mandible involve preparing bur holes at the angle of the mandible through which traction hooks[73] or wires[69] are placed.

Although chronic protracted dislocation most commonly presents with severe limitation in movement of the mandible and deranged occlusion, some patients may present with adequate range of motion owing to development of a pseudo-joint in the new dislocated position.[7,32] In these situations, loss of masticatory function can primarily be attributed to the derangement in occlusion caused by the anteriorly dislocated mandible. In absence of the need for reduction of the condyle back into the glenoid fossa, re-establishment of their occlusion and function can be achieved through osteotomies which allow for the mandible to be set back, while maintaining the pseudo-joint in its new position.[5] While vertical and oblique ramus osteotomies have been successfully employed in these patients,[74] the use of an inverted L-shaped ramus osteotomy or a bilateral sagittal split osteotomy may be favored due to improved stability resulting from increased bone contact between the proximal and distal segments.[2,75] Additionally, vertical and oblique ramus osteotomies may restrict the movement of the new pseudo-joint as setback of the distal segment could result in coronoid impaction on the condyle.[5] Horizontal sub sigmoid osteotomy and midline mandibulotomy have also been proposed as methods to maintain the new joint and restore occlusion.[76]

Successful reduction of a chronic protracted dislocation that has exceeded 6 months in duration typically requires more invasive surgical intervention than those described above.[10,12,14] Proposed methods of treatment include: temporalis myotomy or coronoidectomy,[77] condylectomy, condylotomy, and placement of a TMJ total joint prosthesis.[10,12,14,24] To overcome spasm, shortening, and/or adhesions of the temporalis muscle, which impede reduction of the joint, temporalis myotomy or coronoidectomy can be performed via in intraoral approach.[69,78] In a condylectomy, the inability to reduce a dislocated joint is overcome by osteotomizing and removing the condyle. While this facilitates reduction of the neo-condyle back into its normal anatomic position within the glenoid fossa,[7,10] proper function requires evaluation and treatment of any abnormalities of disk position and anatomy.[79-80] When pathologic changes are extensive, and the disk deemed unsalvageable, repair and repositioning of the disk (i.e. meniscoplasty) is insufficient, and the disk should be removed (i.e. meniscectomy) and replaced to prevent degenerative changes from occurring on the articular surfaces of the neo-condyle and fossa.[80-81] Replacement with autogenous tissues such as temporalis myofascial flap, auricular cartilage graft, and dermal grafts should be favored over alloplastic materials,[80] and have shown clinical success similar to what has been reported when used as interpositional grafts in the treatment of TMJ ankyloses.[80,82] When a condyle is able to be partially reduced, but fibrotic changes in the peri-diskal tissues and disk prevent passive and stable seating of the condyle-disk complex within the glenoid fossa, a disk-sparing condylotomy should be considered.[5] Although first described by Maccaferri in 1951 – who performed a blind, extraoral osteotomy below the level of attachment of the lateral pterygoids as an extra-articular surgery to address internal derangement of the disk – Bouloux proposed the use of an intra-oral vertical ramus osteotomy (IVRO) with post-operative MMF, a procedure he termed the modified condylotomy.[83-84] First, a vertically-oriented osteotomy is made from the sigmoid notch down to the inferior border of the mandible, traveling posterior to the neurovascular bundle. Subsequent inferior displacement of the condyle, which he calls "condylar sag," allows for passive repositioning of the disk and condyle and restoration of a normal anatomic condyle-disk relationship. When anterior movement of the condyle-disk complex is required to achieve completely passive positioning, the proximal segment can be made to sit lateral and anterior to the posterior border of the distal segment. This is achieved by stripping the medial pterygoid attachment and reducing bone along the antero-medial aspect of the proximal segment, which allows for passive seating in an antero-lateral position in relation to the distal segment.[84] The patient is subsequently placed in MMF for a period of 2-4 weeks, with a longer period favored for bilateral cases, followed by 3-4 weeks of light guiding elastics in the absence of malocclusion.[83-84]

These procedures are more commonly described as methods to treat chronic recurrent dislocation and disk displacement, and scarcity of literature to support their use in a chronic protracted dislocation may be attributable to the infrequency with which chronic protracted dislocation is encountered, especially dislocations that exceed 6 months.[27] Despite this lack of literature confirming long-term success, the use of these treatment modalities in this subset of chronic protracted dislocation (greater than 6months) should be considered, particularly when internal derangement of the disk is a contributory factor.[10,12,14]

Chronic Recurrent Dislocation

Management of chronic recurrent dislocation differs from that of acute dislocation and chronic protracted dislocation. This is attributable to the extent of pathologic changes present in these patients' joints. By definition, these patients have experienced multiple episodes of dislocation, which in conjunction with contributing factors, have led to the establishment and progression of a hypermobile joint. Although they are often able to self-reduce and may less frequently present with pain[5,43], persistent concerns about when another episode will occur can have a profound effect on the psyche of these patients, and thereby has a negative effect on their quality-of-life(QOL)[10,22,27]. While early reduction of an acutely dislocated TMJ is adequate in addressing associated oromandibular dysfunction and pain, appropriate treatment of patients with chronic recurrent dislocation hinges on successfully preventing recurrence so that progression of disease is stopped and quality-of-life restored[43]. Approaches to preventing recurrence include both minimally invasive and surgical treatments options, with all treatment modalities falling into one of four approaches: 1) restraining the condyle, 2) creating an obstacle, 3) clearing the condylar path, and 4) total joint reconstruction.

MINIMALLY INVASIVE TREATMENT

Minimally invasive therapies all attempt to prevent recurrence by restraining the condyle. These techniques include administration of local anesthetics in combination with occlusal splints,[11,43] limiting mandibular range-of-motion through adherence to a soft diet and physiotherapy,[1,44,86] and MMF. When unsuccessful, alternative treatments such as chemical capsulorrhaphy, autogenous blood injection (ABI), prolotherapy, botulinum toxin therapy, arthrocentesis, and arthroscopic electrothermal capsulorrhaphy have been proposed.[5,14,87-89] The goal of these therapies is to enhance and strengthen the soft-tissue stabilizing components of the TMJ in order to counteract the intra- and peri-diskal tissue abnormalities that have established a hypermobile joint.

Chemical capsulorrhaphy refers to the use of sclerosing agents (e.g. sodium psyliate, sodium tetradecyl sulfate, alcohol), injected into extra- and intra-capsular tissues, in order to achieve localized fibrosis through induction of an inflammatory response.[5,90] Although this technique was proposed as a non-surgical way to counteract the laxity of ligaments and capsule by inducing fibrosis in these tissues, reports of unacceptably low success rates, facial nerve damage,[91] and exposure causing degenerative changes in the articular cartilage have limited its use today.[3,92] Prolotherapy, otherwise known as proliferation treatment or regenerative injection therapy, employs injection of non-pharmacologic solutions (e.g. 10-50% dextrose, psyllium seed oil, glycerin, phenol) to elicit localized proliferation of fibrous tissue, and has shown promising results in the treatment of recurrent dislocation.[14,93] As is the case with sclerosing agents, it is hypothesized that prolotherapy solutions may achieve this by initiating an inflammatory response in tissue.[14,94] Technique and post-operative instructions vary but typically involve auriculotemporal nerve block which can be administered separately or by mixing lidocaine with a non-pharmacologic solution such as 50% hypertonic dextrose.[95] Administration into the superior joint space, retrodiskal and pericapsular tissues has been shown to decrease laxity of capsule and ligaments, and improve overall stability;[27,92,95] however, a modified method performed on 45 patients by Zhou et.al. in which a mixed prolotherapy solution was injected into the posterior periarticular tissues alone was found to prevent recurrence in 91% of patients during their 6-month follow-up period (41/45 patients).[95] In this study, the needle was inserted at a point roughly 10mm anterior to the tragus along a line drawn from the tragus to the lateral canthus and 10mm inferior, following the posterior border of the condyle. An auriculotemporal nerve block and posterior periarticular injection was accomplished by injecting 0.5mL of a mixed lidocaine and 50% hypertonic dextrose solution on the surface of the condylar neck, injecting another 0.5mL after advancing the needle posterior to the condyle 25mm, and lastly depositing 1.0mL while withdrawing the needle 5mm. Patients were placed on a soft diet and restricted to limited mouth opening for 2 weeks. While 41 of the 45 patients did not have any recurrent episodes of dislocation, 13 patients received a second injection at 4 weeks and 2 patients a third at 3 months due to subjective complaints of hypermobility.[95] Although long-term comparative studies on prolotherapy are lacking, clinical success and prevention of recurrent episodes of dislocation have been reported after a single administration, with some studies finding that weekly injections for up to 6 weeks may be required to achieve their optimal effect.[14,94-95]

Intracapsular autologous blood injections (ABI) into the superior joint space to initiate an inflammatory response was first used by Bracchmann in 1964 as a method of treating recurrent dislocation.[96] Resultant localized fibrosis and adhesion formation within the capsule decreases tissue compliance and therefore decreases the overall mobility of the joint.[96] In a limited sample size, Machon et.al. reported an 80% success rate in preventing recurrent episodes.[97] Coser et. al. reported similar success, with 73% of his patients not having a recurrent episode during a 2-3 year follow-up period.[98] Further studies have shown that recurrence rates decrease when autologous blood is injected both intracapsular (into the superior joint space) and pericapsular, compared to intracapsular or pericapsular ABI alone.[5,97,99] Prior to ABI, two-port arthrocentesis and lavage of the superior joint space is performed with lactated ringers (LR) or normal saline (NS).The outflow needle is then removed, and 2 mL of whole blood drawn from the patient is injected into the superior joint space and 1 mL into pericapsular tissues as the remaining needle is removed.[9] Post-operative regimens vary, but they generally include minimizing jaw function for 2-3 weeks (sometimes through the aid of a facial/jaw bandage), NSAIDs, and close follow-up to allow for supervised physiotherapy.[100-102] ABI can be performed under local anesthesia, sedation or general anesthesia in an operating room setting. Although several protocols have been published, each varying in number of injections (ranging from a single injection to multiple injections per week for several weeks) and duration of MMF, therapeutic outcomes have been found to be similargenerally.[92] Despite reported success, Hasson and Nahlieli, Kato et.al., Machon et. al., and Hegab reported that some patients experienced at least one episode of dislocation after treatment.[97,101,103-104] In the same study, Hegab reported that 6 out of 16 patients treated with a single ABI experienced a recurrence.[104] A second episode of recurrence was prevented by a second ABI in 4 of the 6, with the remaining 2 patients not having any recurrent episodes after a third ABI. The 62.5%, 87.5% and 100% success rates seen after one, two and three ABI treatments, respectively, were compared to a second group of 16 patients who underwent 4 weeks of MMF alone. In this group, 3 out of 16 patients initially failed this therapy and subsequently required an additional 2 weeks of immobilization. Ultimately, Hegab reported that combining a single ABI with 4 weeks of MMF was the most successful in prevention of a recurrent episode of dislocation as all 16 of these study subjects had no episodes of dislocation within their one-year follow-up period.[104]

Injection of botulism toxin to treat various conditions involving hyperactivity of the skeletal muscles is supported by high-quality evidence (i.e. prospective, randomized clinical studies).[105-106] Its successful use in the treatment of temporomandibular joint disorders (TMDs) – a diverse group of pathologic conditions that affect the associated structures of the TMJ as well as its function – is also well documented.[106-108] Of particular importance to the oral and maxillofacial surgeon who is tasked with treating recurrent dislocation is the evidence supporting the use of botulinum toxin in the treatment of TMJ hypermobility and oromandibular dystonias.[109-111] Botulinum toxin causes a dose-related weakness in skeletal muscle via inhibition of pre-synaptic acetylcholine (ACh) release at the neuromuscular junction(NMJ)[14,51,112] and can thereby reduce excessive translational forces exerted on a hypermobile joint when injected into the lateral pterygoid – the muscle principally responsible for protrusion and opening of the mandible.[16,18] Fu et.al. describe an extraoral approach in which 25-50 units of botulinum toxin is injected into the muscle belly of the lateral pterygoid by inserting the needle at two points along an imaginary line drawn between the coronoid notch and the inferior border of the zygomatic arch – the first 1.0 cm below the zygomatic arch, and the second 0.5 – 1.0 cm posterior.[111] (**Figure 10A**) With the patient's mouth in a closed position, the needle is inserted perpendicular to the skin, advanced to the depth of the muscle (measured on CT pre-operatively) (**Figure 10B**), the syringe is aspirated to prevent intravascular injection, and 12.5-25 units are injected into the muscle at each injection site, for a total of 25-50 units/muscle. This is done with the patient's mouth in a closed position.[111] Alternately, a variety of intraoral approaches have been described in which the maxillary tuberosity and the EAC are used as landmarks to direct the needle through the maxillary vestibule towards the condyle with the needle being redirected to the medial aspect of the condyle once encountered.[106]

Figure 10A: Sites of botulinum toxin injection into the lateral pterygoid muscle as proposed by Fu et.al.[111] *Point 1*: 1.0 cm below the zygomatic arch along an imaginary line drawn from the arch to the inferior-most aspect of the coronoid notch; *Point 2*: 0.5-1.0 cm posterior to point 1.

Figure 10B: Depth of advancement determined by measuring skin to muscle belly depth on CT.

Guidance of either an extraoral or intraoral approach by continuous electromyography guidance (EMG) has also been described and advocated for due to the muscle's relatively small size (compared to the other muscles of mastication) and its proximity to the maxillary artery and pterygoid venous plexus.[106,113] Injection into the temporalis, masseter, and medial pterygoid has been shown to be efficacious in the treatment of oromandibular dystonias and should be considered when they are present.[106,110] Incidences of transient velopharyngeal insufficiency, dysarthria and dysphagia related to botulinum toxin administration have been documented,[14] but are infrequent and typically resolve within 2-4 weeks. Its use is contraindicated in pregnant or lactating mothers, and in patients with NMJ disorders such as Myasthenia-Gravis. [5,112]

SURGICAL TREATMENT

In addition to the minimally invasive treatment options, there are surgical treatments focused on each of the four different approaches to increasing TMJ stability mentioned above. Although a variety of surgical approaches provide access to the TMJ, an endaural or preauricular approach with a temporal extension/modification (e.g. Blair's, Popowich and Crane, Al-Kayat and Bramley, etc) (**Figure 11**) is most commonly utilized in open surgical treatments of TMJ dislocations, as they both provide adequate visualization of the joint and articular eminence and have excellent long-term cosmesis. [14,114]

Figure 11: Skin incision marked. Preauricular incision design with temporal extension.

After dissection through skin, subcutaneous tissue, and temporoparietal fascia, an oblique incision is made at the superior aspect of the skin incision. This incision should be parallel to the suspected course of the temporal branch of the facial nerve (i.e. directed antero-superiorly from the EAC) and be no greater than 8mm anterior to the bony EAC as the upper trunk of the facial nerve crosses the zygomatic arch between 8 mm and 35 mm from this landmark.[114] The incision should be carried through the temporalis fascia with subsequent dissection anteriorly performed in a subperiosteal or supraperiosteal plane, depending on the procedure performed. In both situations, anterior retraction of this tissue flap in which the facial nerve is contained, mitigates the risk of iatrogenic damage, with reported rates of transient loss or weakness of frontalis and orbicularis oculi muscles being as low as 9 to 18%.[115]

Restrain the Condyle

Surgical procedures attempting to restrain condylar movement include anchoring of the condyle to the zygomatic arch, lateral pterygoid myotomy, temporalis scarification, condylotomy, arthroscopic and open surgical capsulorrhaphy,[88-89] tethering the disk to the capsule (i.e. meniscorrhaphy), and surgical manipulation of the disk (e.g. meniscectomy, meniscoplasty, discopexy).[14,22,27,80] All of these aforementioned procedures aim to reverse or combat the underlying changes to TMJ components that are contributing to hypermobility.[5,27] In lateral pterygoid myotomy, first described by Bowman in 1949, the goal is to reduce the spastic activity and muscular pull on the condyle by inducing formation of scar tissue within the lateral pterygoid. This is achieved by severing lateral pterygoid attachment to both the condylar and anterior capsule. The condyle can be approached percutaneously(via a preauricular incision)[14] and transorally (via a vertical incision along the anterior border of the ramus).[78,116] Both approaches carry the risk of complications such as bleeding risk, impaired view of the surgical site, and reunion of the muscle during healing.[43] Reunion of the muscle potentiates recurrence and is more commonly seen in myotomy than when excessive muscular pull is addressed with a condylotomy.[5] Temporalis scarification aims to reduce condylar hypermobility by inducing intramuscular scar formation within the temporalis muscle, thereby establishing cicatricial restriction of the temporalis.[14,117-118] Since stretching or anterior displacement of the disk can contribute to hypermobility, surgical capsulorrhaphy aims to reduce capsular laxity by wedge resection and repair, or plication, of the capsule on the side opposite to the direction of dislocation.[119-120] As chronic recurrent dislocation is most commonly seen in the anterior direction, capsulorrhaphy is typically performed on the posterior aspect of the capsule with arthroscopic electrothermal capsulorrhaphy representing the most minimally invasive method available.[88] Repositioning and bolstering of the posterior stabilizing components of the joint is also the goal of meniscus and fibrous capsule tethering to temporalis muscle and fascia in a supero-posterior vector,[7] as well as meniscectomy with replacement, and meniscoplasties. These procedures should be considered when abnormal disk morphology and/or position is a contributing factor in dislocation or when they impede reduction.

Create an Obstacle

Of all the approaches to prevent recurrent dislocation, creation of an obstacle to serve as a barrier to excessive translation has yielded the most innovative methods, albeit with questionable long-term success. In these procedures, implants, grafts, and osteotomies of the eminence or zygomatic arch are used to augment the insufficient eminence with the hopes of preventing dislocation of a hypermobile joint. Although polymer and alloplastic implants, coralline hydroxyapatite (HAP) blocks, and plates and screws have all been employed to augment the eminence, there have been many reported complications and questionable long-term success.[3,24,121] Currently, the most widely used and accepted methods of creating an obstacle include Norman's procedure, in which an interpositional graft is placed between a glenotemporal osteotomy, and Dautrey's procedure, in which an osteotomy is made in the zygomatic arch which is then repositioned inferior and anterior to the eminence.[3,122-123]

The first described use of implants to augment the eminence involved placement of vitallium mesh,[124] mersilene (Dacron) woven strips, and stainless steel pins at the inferior aspect of the inadequate eminence.[125-127] Although early success was published in several small case series, fracture and failure of implants were notable complications that required secondary surgery to remove the hardware.[54] Delayed failures also were observed with the use of silastic (silicone) and polymethyl-methacrylate implants, and were attributed to displacement of the implants, severe immune reactions, and other complications.[24,121] Onlay grafting of the eminence with coralline HAP often resulted in resorption of the underlying bone, a complication also seen in cases where HAP blocks were used as onlay grafts in maxillary and mandibular alveolar reconstruction, and were often displaced under functional loads.[43] Long-term success of these grafts were impeded by these complications and led to abandonment of these treatment modalities.[3,24,121] Similar inadequacies in treatment outcomes have been reported with autogenous bone and cartilage placed as onlay grafts[54,127] at the eminence.[128]

In 1988, Buckley and Terry reported an alternative method to create an anterior obstacle when they placed a bone plate along the lateral aspect of the eminence that extended inferiorly and medially, a technique they had been using since 1981.[129] This was later termed miniplate eminoplasty by Puelacher and Waldhart who employed the use of a T-shaped titanium miniplate.[54,123] Advantages of this technique include ease of procedure compared to other open surgical treatments, avoidance of violation of the joint space, no resorption of the eminence as seen with alloplastic implants, and reversibility.[54,66,115,129-130] Puelacher and Waldhart reported that 7 joints (4 patients) treated with T-shaped titanium and vitallium miniplates had no recurrence of dislocation, fracture or loosening of implants, or impedence in masticatory function during their follow-up period which ranged from 6-months to 2.5-years.[54] Shibata et.al. found similar success and no complications with T-shaped titanium miniplates in a series of 15 joints (9 patients) during a 9- to 54-month follow-up.[66] A retrospective study by Kuttenberger and Hardt in 2003, however, brought into question the long-term success of miniplate eminoplasty.[123] While 13 of 13 (100%) patients that presented with pain pre-operatively reported a reduction in pain score at 1-year post-T-shaped miniplate eminoplasty, they found that 7 out of 20 patients presented with fracture of one or both plates within 2- to 7-years.[123] Another retrospective study that included 8 patients treated with miniplate eminoplasty found recurrence in 1 patient and plate fracture in 2.[115] Delayed failures have also been seen in the use of stainless steel pins or titanium screws placed at inferior aspect of the zygomatic arch or eminence to serve as an obstacle.[126,131] As is the case with miniplate eminoplasty, when these other methods fail, patients present with severe pain and resorption of both the eminence and condylar head in 20% of cases,[5] which can be attributed to fracture and loosening of the implant which necessitates surgical removal.[43,115]

Norman's procedure, or glenotemporal osteotomy with interpositional bone graft, was conceived as an alternative means of enhancing the eminence without the use of an onlay graft.[50,132] While onlay grafts are understood to lead to resorption of the underlying osseous structures, interpositional grafts that maintain intact periosteum on the repositioned segment allow for maintenance of basal bone and consolidation and maturation of the interpositional graft.[54,128] First proposed in 1984, Norman described using a horizontal glenotemporal osteotomy to separate the eminence from the overlying arch, with care taken to keep the periosteum on the distal eminence intact and subsequent placement of an interpositional bone graftto serve as a buttress for the inferiorly repositioned segment.[132] (**Figure 12**)

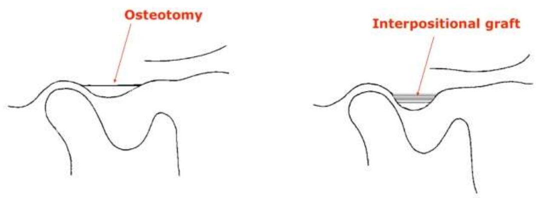

Figure 12: Illustration of a horizontal glenotemporal osteotomy (*left*) with subsequent placement of an interpositional graft to augment the eminence (*right*).

While many slight modifications have been made to the type of graft used, as well as the method of fixation, general tenets of this procedure that remain are 1) adequate medial extension (at least 1.5 cm) to prevent leaving a medial path of escape for the condyle,[50] 2) adequate stability of the graft,[3]) preservation of the periosteum on the osteotomized and inferiorly displaced segment to maintain a vascular pedicle and preserve morphology,[128] 4) avoidance of intracapsular violation when detaching the superior and anterior attachments of the capsule from the posterior aspect of the eminence.[133] Autogenous cancellous bone from the ilium has commonly been utilized by many as the interpositional graft;[3,50,57,133-134] however, others have proposed alternatives due to concerns for extensive resorption and remodeling of cancellous bone graftdlong-term.[119,132] Although the use of autogenous cortical bone from the calvarium[50,135] exhibits less morphological change throughout the consolidation and maturation process than observed in iliac crest autografts, Güven et.al. advocate for the harvesting of a cortical wedge from the chin, citing avoidance of possible complications related to calvarial harvest (e.g. dural tear, arachnoid bleeding and intracranial hematoma),[50,133] cosmesis of incision, and decreased surgical time required.[133] A similar maintenance of original graft volume, and thus amount of vertical augmentation of the eminence, has been reported with the use of coralline HAP blocks[132] and processed bovine cartilage (Chondroplast®), the latter of whichcan attribute its long-term dimensional stability to progressive calcification.[119]

Methods of stabilizing the graft also vary, with some advocating for the use of titanium miniplates or microplates and screws to fixate the augmented eminence.[14,50] Others prefer utilizing wire osteosynthesis[50,130] due to concerns for plate fracture,[14,66] which may be more likely to occur as loading forces on the eminence impart a torsional force on the plate which is fixated along the lateral aspect of the eminence.[123,136-137] Complications related to wire osteosynthesis, although less frequently documented in literature, have also been reported, with impingement of the condyle on the wires potentially causing preauricular pain.[122] Medra & Mahrous reported complete resolution of pain after removal of wires in three patients but failed to report how many of the sixty patients in their prospective study had wire fixation.[50] Despite a dearth of data in regards to specific failure rates of various fixation methods, others have modified Norman's procedure to achieve a stable interpositional graft without the need for wire or plate and screw fixation.[133,135] Guven et.al. describe an oblique, rather than horizontal, osteotomy of the eminence that extends into the arch, keeping the anterior slope of the eminence intact (i.e. creating a greenstick fracture), followed by gently tapping a wedge-shaped autogenous cortical graft into the osteotomy site to serve as a buttress that maintains the inferiorly displaced distal segment.[133] Their 12 patients remained in MMF for 1-week post-operatively and were subsequently placed on a soft-diet to minimize loading forces and damage on the augmented eminence. They had no further episodes of recurrent dislocation within a follow-up period of 2- to 6-years (mean of 4.2).[133] Although more limited in the amount of augmentation obtainable because it requires maintenance of a greenstick fracture, this modification to Norman's procedure eliminates the possibility of hardware-related complications while also utilizing a graft donor site with decreased morbidity compared to iliac crest or calvarial bone autografts. [133,138]

Other methods of creating an obstacle to anterior condylar dislocation focus on manipulation of the zygomatic arch. The Dautrey procedure involves an osteotomy on the posterior aspect of the zygomatic arch, which is then swung inferiorly to act as an augmented extension of the eminence. Mayer in 1933 first described this when he employed a segmental osteotomy of the arch and dislocation in an inferior direction. Ten years later, LeClerc and Girard performed a vertical osteotomy anterior to the eminence and moved the arch medio-inferiorly to form a barrier to translation.[14,139] However, this technique, coined the LeClerc procedure, lost favor in its original form due to the fact that the new position of the osteotomized arch was difficult to maintain.[138] To address problems of insufficient stability of the arch segment, Gosserez and Dautrey further modified the procedure.[14,140] In what has subsequently become known as the Dautrey procedure, an oblique osteotomy is made from the posterior-superior aspect of the arch, just anterior to the eminence, and is completed in an anterior-inferior direction. The arch is then manipulated in an inferior direction in a manner which Dautrey originally described as, "repeated movements gradually and gently increasing in strength," in order to create a greenstick fracture in the anterior archat or near the zygomaticotemporal suture.[139,141] The mobilized segment is first manipulated laterally and inferiorly, and then medially, locking the mobile segment under the eminence, which is made possible by the oblique direction of the osteotomy. Stability of the inferiorly repositioned arch in its new position is conferred by two main factors: 1) preservation of cortical continuity and elasticity of the anterior arch, and 2) the oblique osteotomy allowing for buttressing of the osteotomized end against the eminence (**Figure 13**).

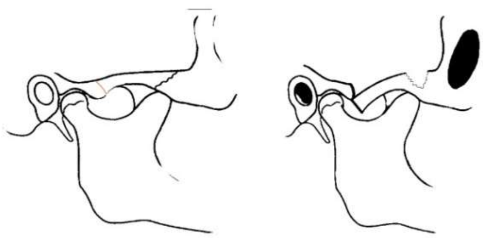

Figure 13: Illustration of the use of an oblique osteotomy in Dautrey's procedure (marked in red) to allow for stable, inferior repositioning of the arch underneath the eminence. Greenstick fracture at the zygomaticomaxillary suture is depicted in the image on the right.

It is therefore imperative to maintain a greenstick fracture anteriorly. Minimizing the risk of complete fracture can be achieved by avoiding excessive forces while repositioning the osteotomized arch,[30,141] maintaining fascial and periosteal attachment to the anterior arch,[139] and avoiding the procedure in patients with brittle bone (e.g. the elderly population).[5,142] Guidelines to successful treatment include: 1) maintaining a purely extra-capsular dissection,[5,14,139] 2) adequate medio-lateral width of the arch to prevent medial escape of the condyleon translation,[133,143] 3) adequate inferior projection (i.e. arch thickness in the cranio-caudal dimension),[138] and 4) stability of the displaced arch. Prevention of an intracapsular inflammatory response, which may lead to degenerative changes, is contingent on careful detachment of the anterior and superior capsule from the eminence[139] while still achieving adequate exposure and visualization of the medial eminence to confirm the absence of an antero-medial path of escape. Confirmation of medial extension is particularly important in the setting of atrophic condyles, which have been implicated as causative factors in recurrent dislocation.[143] In cases where short arch height limits its inferior projection, therefore limiting its efficacy as an obstacle, placement of a wedge-shaped bone graft between the osteotomized segments should be considered.[138-139] Although many have reported success without the use of fixation,[30,144] others have recommended the use of rigid fixation to enhance stability and prevent recurrence.[139,141,145] The necessity of rigid fixation, however, should be evaluated case-by-case based on confirming intraoperatively that there is not a complete fracture at the zygomaticotemporal suture, assessing the degree of stability of the archin its new position (with or without a graft), and a risk-benefit analysis of additional stability provided by fixation versus the risk of hardware-related complications.[115,139] A novel method of achieving enhanced stability and successful treatment without the use of fixation was proposed by Akioka et.al. in 1997, who utilized what they described as a modified LeClerc procedure with oblique osteotomy and V-shaped notch without fixation. In this procedure, a retentive feature meant to straddle the anterior corner of the non-mobile segment was created in the form of a V-shaped notch at the supero-posterior tip of the mobilized arch segment. [138,146] Prevention of recurrence using this method was subsequently reported in three patients during a 7-year follow-up period by Kushida et. al.[138] Despite its small sample size, further studies may be warranted as this procedure could be a viable alternative to wire or plate and screw fixation of the osteotomized arch, and a means to eliminate the risk of hardware-related complications without compromising stability and long-term prevention of recurrence.

Clear the Condylar Path

In a stable TMJ, the articular eminence serves as an obstacle to excessive anterior translation of the condyle. However, in recurrently dislocated joints, the eminence can serve as an impedance to reduction of the condyle from an anteriorly dislocated position back into the glenoid fossa and can therefore lead to periods of open-lock.[43,147] As such, an eminectomy can be used to treat recurrent dislocation and may also prove helpful in cases of protracted dislocation where the condyle is unable to be reduced by the other surgical interventions described above.[12,15] While minor modifications to the approach and surgical methods have been proposed since it was first described by Myrhaug in 1951, such as performing the procedure in combination with other TMJ procedures,[5,148-150] its general success in treatment of chronic recurrent dislocation has been well-documented.[5,151-155]

Once adequate dissection and exposure of the eminence is achieved using the surgical approach described above (**Figure 14A-B**), reduction of the eminence is then performed first by a horizontal osteotomy made with a fissure bur (**Figure 14C**), with care taken to follow the natural curvature of the floor of the middle cranial fossa which dips inferiorly at the medial aspect of the eminence, and subsequently by completing the osteotomy with medio-inferiorly directed osteotome and mallet.[14,147] A large, carbide round bur or rasp can then be used to create a rounded, smooth surface.[153] (**Figure 14D**)

Figure 14A: Preauricular skin incision with temporal extension.

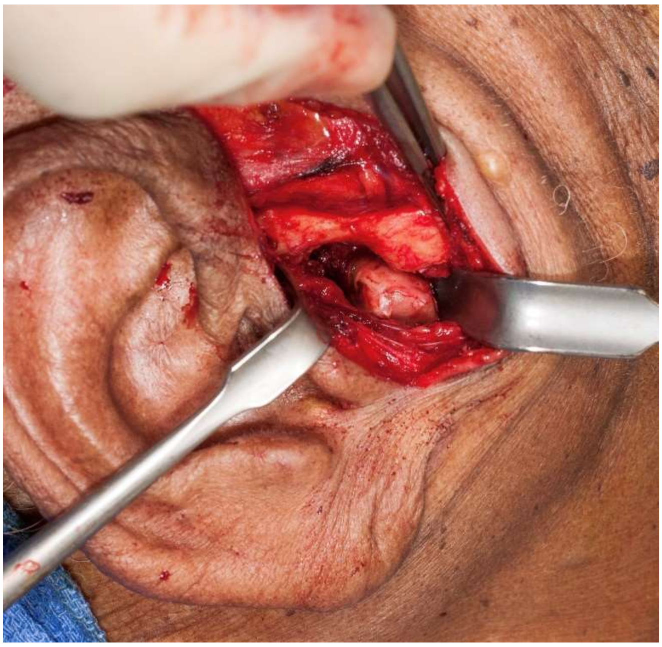

Figure 14B: Exposure of the articular eminence.

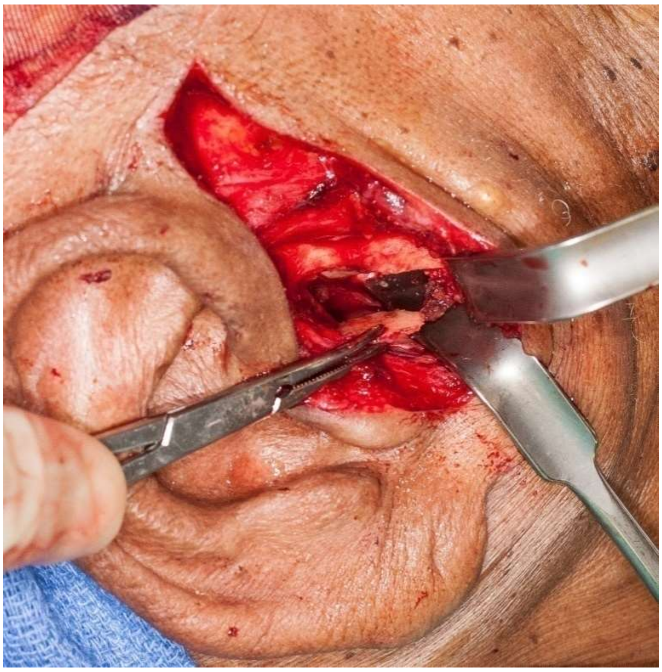

Figure 14C: Removal of the osteotomized segment.

Figure 14D: Use of a rasp to establish a well-contoured surface for unimpeded, smooth translation of the condyle.

Recurrent episodes of dislocation after eminectomy are commonly attributed to incomplete medial extension of the osteotomy. Therefore, adequate inferior retraction of the disk and condyle is imperative to achieve adequate visualization of the medial aspect of the eminence during this procedure.[155] Post-operative recommendations include maintenance of a pressure dressing to the pre-auricular region for 24- to 48-hours, a strict non-chew diet, and joint mobilization (i.e. physiotherapy).[14] Although there are many variations in protocol, diet restrictions are typically prescribed for one- to three-weeks, and early physiotherapy is initiated due to evidence supporting that initiation of aggressive range-of-motion exercises after TMJ surgery, as early as post-op day 1, may prevent long-term functional limitations and aid in clearing of post-operative edema.[82,104]

Advantages of eminectomy compared to other surgical options include that it is less invasive, has a shorter operative time, does not require post-operative MMF, and doesn't involve harvesting of a graft or introduction of a foreign body.[43,147] Eminectomy does, however, pose a risk of damage to adjacent structures, including branches of the facial nerve, trigeminal nerve, and maxillary artery.[156-158] Osteotomy of the eminence also has a potential to result in intracranial violation and therefore warrants pre-operative CT evaluation of the zygomatic arch and articular eminence, as pneumatization of these structures may increase the likelihood of perforation into mastoid air cells, which could lead to intracranial bleeding or infection.[43] Inspired by successful arthroscopic treatment of internal derangement by Ohnishi and Segami et.al. sought to mitigate some of the risks associated with eminectomy by performing the procedure arthroscopically, and in 1999 published a preliminary study on the successful treatment of 11 patients with arthroscopic eminoplasty.[159-160] A 2003 study comparing conventional eminectomy and arthroscopic eminoplasty found comparable success rates in their treatment groups: 72% and 75%, respectively.[59] However, despite being less invasive and potentially as efficacious as eminectomy, arthroscopic eminoplasty has not gained widespread adoption. This is likely due to limited evidence without long-term follow-up, surgeons feeling uncomfortable performing arthroscopic procedures, and concerns that iatrogenic trauma to the articulating surfaces and disk may lead to adhesion formation and hypomobility long-term.[89]

Total Joint Replacement (TJR)

Reconstruction of the TMJ with alloplastic materials was first performed in 1946 when Eggers utilized tantalum foil as an interpositional graft in the treatment of recurrent ankyloses.[161] Over the years, others, including Robinson and Christensen, attempted to reconstruct the glenoid fossa with a variety of metals which were secured to the zygomatic arch. In today's available prostheses, a combination of metal and surgical-grade plastic is used to reconstruct the ball (i.e. condyle) and socket (i.e. glenoid fossa) components of the TMJ, and are available as both stock and custom-made prostheses. (**Figure 15A-C**)

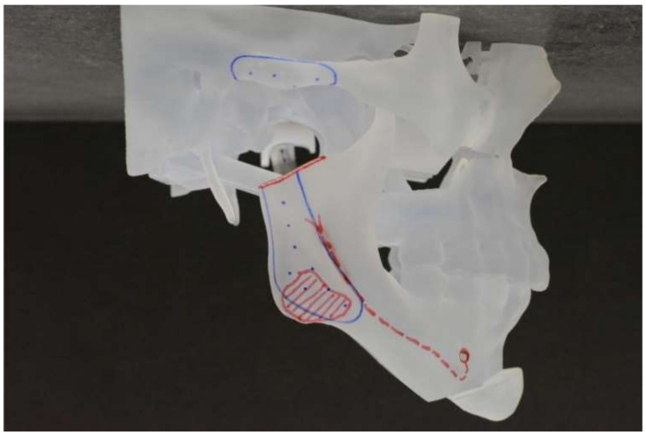

Figure 15A: Model surgery (condylectomy) performed on a sterolithic model in preparation for fabrication of custom total joint prosthesis. (*Courtesy of Dr. Larry Wolford*.)

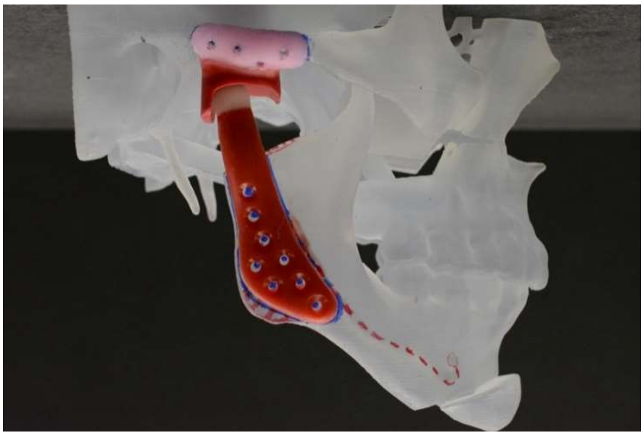

Figure 15B: Wax-up of proposed prosthesis. (*Courtesy of Dr. Larry Wolford*.)

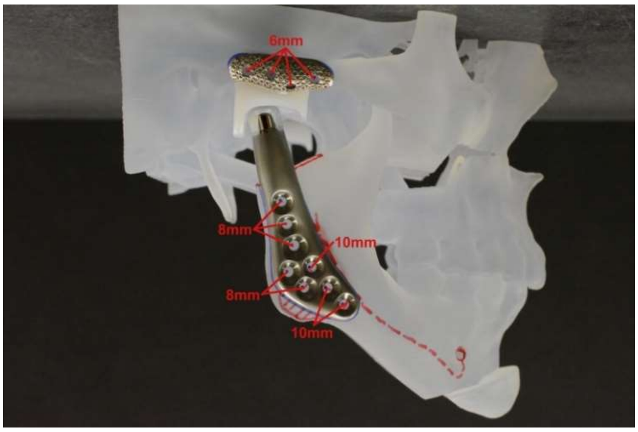

Figure 15C: Custom-made total joint prosthesis. (*Courtesy of Dr. Larry Wolford*.)

Although early use was primarily limited to treatment of recurrent ankyloses,[162] surgeons have since published successful use of alloplastic TJR in the treatment of TMDs which have been refractory to other surgical treatment modalities, including failed reconstruction with other alloplastic or autogenous grafts, advanced degenerative joint disease (Wilkes stage IV or V), and inflammatory arthritis that has been refractory to other surgical treatment modalities. Custom-made prostheses can be utilized to restore proper vertical height and/or occlusal relationship that has been lost as a result of resorption of the condyle or fossa components, trauma, developmental abnormalities or pathologic lesions requiring resection.[163-164]Its use as a salvage procedure in the reconstruction of severely damaged and/or mutilated joints resulting from severe joint disease which have failed conservative and multiple surgical treatments, is well-documented, with many studies reporting significant improvements in pain, function and quality-of-life scores.[165-167]

Total joint replacement also has been proposed as a treatment option for chronic protracted dislocations exceeding 6 months in duration. The use of a custom-made prosthesis, designed to fit a patient's specific anatomic requirements, affords the surgeon the ability to reconstruct deficient functional anatomy.[10,12,14] Properly designed comparative studies testing its efficacy compared to conservative and other surgical treatment modalities in the treatment of chronic protracted and chronic recurrent dislocation are not currently available to support its use as a primary, first-line treatment modality. However, its use as a last-resort salvage procedure and means to restore form and function in the setting of chronic dislocation with concomitant end-stage degenerative TMJ disease should be considered.[5,10]

DISCUSSION

TMJ dislocation in all its forms (i.e. acute vs chronic protracted vs chronic recurrent) should be treated with the following primary goals in mind: 1) reduction of the joint by re-establishing the normal condylar-disk complex with the glenoid fossa, and 2) prevention of future occurrences. In addition, it should be the surgeon's intent to achieve these two goals with the fewest number of surgical interventions possible. While successful use of the plethora of minimally invasive and surgical treatment options have been reported, there is still much controversy regarding which treatment strategy most predictably prevents dislocation long-term.[22] Therefore, critical review of the quality of evidence provided by the available literature is paramount, and the patient's unique set of contributing factors should be considered when determining which is the most appropriate treatment strategy to employ.

Acute TMJ dislocation is predictably manageable with manual reduction, with recent studies suggesting that the Hippocratic maneuver[5,38] and wrist-pivot method are the most commonly utilized today.[8,27,42,61] Although the extraoral approach protects the surgeon performing the procedure, a systematic review by Prechel et. al. found that it has a low success rate (54.5%) in bilateral cases, as well as being more time consuming and painful to the patient.[27,46] A randomized controlled trial (RCT) by Ardehali et. al. comparing the Hippocratic maneuver, wrist-pivot method, and extraoral method also found that the extraoral approach had a lower success rate (66.7%) compared to that of the conventional method (86.7%) and wrist-pivot method (96.7%) in the treatment of acute anterior dislocation in 90 consecutive patients.[46] They did, however, find that the extraoral method had a higher success rate in unilateral dislocations (100%) than in bilateral cases (54.5%).Therefore, the use of an extraoral approach in manual reduction should be limited to patients that pose an increased bite and/or infection risk to the physician and should also be considered when treating unilateral anterior dislocation.[46] In the absence of these, the use of bite blocks, gloves, gauze wraps or plastic splints around fingers, and manipulation of the mandible along the external oblique (modified Hippocratic maneuver)rather than the occlusal surface of the posterior dentition (conventional Hippocratic maneuver), is recommended to minimize the risk of injury to the physician when utilizing an intraoral approach.[27] When initial attempts at manual reduction fail, local anesthetics (e.g. auriculotemporal nerve block, masseteric and deep temporal nerve blocks) and/or with oral muscle relaxants, analgesics, or anxiolytics (e.g. Diazepam)should be administered before further attempts are made.[46,65-66] Manual reduction under IV sedation with general anesthetics should be considered when oral medications prove to be insufficient in aiding manual reduction.[5,8,12,14,44]

Although attempts at manual reduction are also recommended in the treatment of chronic protracted TMJ dislocation, reduction is typically unsuccessful, even with the use of local and general anesthetics, when the duration of dislocation exceeds 3-weeks.[12,67-68] These cases typically require open surgical methods of reduction, such as the use of wire traction or bone hooks, performed under general anesthesia,[5,27,139] with cases that have persisted for 4- to 12-weeks often requiring partial stripping of the periosteum and muscles of mastication from the ramus and coronoid process to facilitate reduction.[12] Literature regarding cases of dislocation that have persisted for 3 to 6 months is limited, but many report that dislocations that have persisted for longer than 6 months are rarely reducible without more invasive surgical intervention. In these extreme cases of chronic protracted dislocation, temporalis myotomy or coronoidectomy,[77] condylectomy, condylotomy, disk procedures (i.e. meniscorrhaphy, meniscoplasty, meniscectomy, discopexy), and TJR with an alloplastic total joint prosthesis[10,12,14,24] may be required to achieve passive repositioning of the dislocated disk-condyle complex while also restoring function and proper, balanced occlusion.[10,12,14,141] Eminectomy also has been reported as a successful “rescue procedure” in these scenarios,[13,115] and may be considered, but limited available literature regarding its use in protracted dislocation should preclude its widespread adoption as a primary treatment modality.

Much of the current literature on the management of chronic recurrent dislocation states that minimally invasive treatments are rarely successful in preventing future recurrence when used as a primary treatment modality,[134] especially in patients with contributory systemic diseases and when poor post-operative compliance is anticipated.[13,67-68] These studies largely attribute reported "successful" treatment of recurrent dislocation with sclerosing agents, autologous blood, prolotherapy, and botulinum toxin alone as a product of inadequate follow-up.[13] In spite of this, attempts at treatment by conservative, minimally invasive means is still commonplace as a first line treatment and should be considered especially when the patient presents with medical comorbidities that preclude them from safely undergoing invasive surgery, or when the patient feels that recurrent episodes are not detrimental enough to their quality-of-life to warrant consenting to surgery.[29] Among the open surgical treatment options, variations of Norman's procedure and Dautrey's procedure continue to be used in treatment of chronic recurrent dislocation that is refractory to conservative interventions. While there is consensus about anatomic considerations that, when present, contraindicate the use of these treatment modalities (e.g. avoidance of Norman's procedure in the setting of a pneumatized eminence and Dautrey's procedure in patients with brittle bone or short and narrow arches), there is less agreement about thenecessity[57] and use of fixation with wires, plates and screws. A case report by Kuttenberger and Hardt published in 2003 found that 35% of patients who underwent eminoplasty with miniplate fixation presented with plate fracture necessitating surgical removal within 2- to 7-years.[123] Other studies have also reported high rates of plate fracture,[14,66,136] and while some recognize this risk and deem it clinically acceptable,[115] exposing patients to this risk should be minimized by preferentially choosing treatment modalities that do not require application of hardware. Guven's modified eminoplasty and Kushida et al.'s modified LeClerc procedure with oblique osteotomy and V-shaped notch both represent promising methods of creating an obstacle in which its stability does not rely on application of hardware that can fail and necessitate additional surgical interventions. [133,138]

When attempts to restrain the condyle or create an obstacle have failed to prevent recurrence, eminectomy has frequently been used as a "rescue procedure."[13,115] Although less invasive than both Norman's and Dautrey's procedure, some elect to not use eminectomy as their first-line surgical treatment for a variety of reasons: 1) it is irreversible (unlike Norman's and Dautrey's procedure), 2) remodeling of the decorticated temporal bone can continue, particularly in young patients,[24] and 3) surgeon's preference for alternative procedures based on anecdotal experience. Still, since its introduction by Myrhaug in 1951, it has consistently been the most widely accepted and utilized treatment for recurrent dislocation, with many considering it the "gold standard" for treatment of this condition due to reported success rates higher than 85%.[6,11,29,98,168] Although typically performed in an OR setting under general anesthesia, an eminectomy can also be performed under IV sedation with Propofol.[15,169-170] This approach may be considered when treating recurrent dislocation in patients for whom undergoing general anesthesia is not an option due to age or medical comorbidities. It is important to note, however, that long-term studies with ample sample size and good study design (e.g. proper criteria, controls, etc.) comparing eminectomy and other treatment options are lacking, and that the use of eminectomy as a rescue procedure, or consideration as a gold standard treatment, is predominantly based on anecdotal evidence, and case series and reports rather than RCTs. [13,115,169]

While eminectomy remains as a commonly used surgical treatment for chronic recurrent dislocation today, some have questioned its use as a singular treatment for a multifactorial disease process involving a variety of contributing factors that differ from patient-to-patient.[5,13-14] Although there is ample evidence that clearing the condylar path can prevent recurrent dislocation,[26,67,149,152,168] a common criticism of the procedure is that eminectomy does not address the underlying pathological changes to TMJ structures that have contributed to its hypermobile state (i.e. Capsular and ligamentous laxity, internal derangement, and uncoordinated or spastic muscle activity), which may contribute to long-term functional impairment if not addressed.[5,132,150] This is particularly concerning in cases of unilateral chronic protracted dislocation. Although the contralateral (i.e. non-dislocated) condyle may not have a history of dislocation, its positional dependence on the contralateral, symptomatic condyle subjects its joint structures to pathologic forces which can lead to symptomology down the road.[171]Excessive muscular pull, a lax capsule, and inadequate strength and elasticity of the posterior attachment of the articular disk often results in anterior disk displacement (i.e. internal derangement of disk),none of which are addressed when eminectomy is performedalone.[50,134] This has led to some surgeons performing additional procedures in conjunction with eminectomy,[43,148-149,172] with procedures performed being dictated by the etiology (i.e. contributing factors) of their individual patient's disease process.[1,150] (**Table 1**).

Table 1: Procedures that Mitigate/Treat Contributing Factors	
Contributing factors	**Procedures**
• Internal derangement (e.g. non-anatomic disk-condyle relationship)	• Meniscectomy and replacement • Meniscoplasty • Disk plication (meniscorrhaphy) • Meniscocondylar imbrication or discopexy with Mitek mini anchors
• Spastic or excessive activity of muscles of mastication	• Lateral pterygoid and temporalis myotomy • Scarification • Botulinum toxin injections
• Lax capsule	• Chemical capsulorrhaphy • Autogenous blood injections • Prolotherapy • Electrothermal capsulorrhaphy (arthroscopic or open) • Plication

In the setting of internal derangement or a non-anatomic disk-condyle relationship, a meniscectomy and replacement, meniscoplasty,[80,132,149,152,173] complete or partial plication of the fibrous disk to posterior ligament (i.e. meniscorrhaphy),[79] or use of

Mitek mini anchors to reposition the disk over the condyle (i.e. meniscocondylar imbrication or discopexy) should be considered.[150,174] Lateral pterygoid and temporalis myotomy,[117-118] scarification, or botulinum toxin injections[111,113] may be used to address spastic or excessive muscle pull.[78,173,133] Lastly, a lax capsule may be addressed by minimally invasive means such as chemical capsulorrhaphy, ABI, prolotherapy, and botox,[97,101-103] or with arthroscopic and open surgical procedures such as electrothermal capsulorrhaphy[88] andplication. Although a comprehensive evaluation of patients with chronic recurrent dislocation through the use of MRI and CT may help to identify contributory factors, intraoperative evaluation of ligament and capsular laxity, disk condition and position, and osseous components after eminectomy should also be performed as it can help guide treatment.[150]

A review of current literature reveals insufficient evidence of adequate strength comparing the efficacy of this multi-modal approach to that of the current mainstays of treatment to warrant widespread adoption. However, given the current understanding of the multifactorial etiopathogenesis of chronic dislocation, further studies are warranted to determine whether combining eminectomy with other procedures intended to address hypermobility can improve long-term success. Keeping in mind the generally held belief that prognosis and outcomes worsen as more open surgical treatments are performed on a TMJ,[163,166] it seems prudent to consider addressing all contributory factors at the same time rather than risk recurrence and having to perform additional surgeries under less favorable conditions.[164]

FUTURE RESEARCH

The lower incidence of chronic protracted dislocation and chronic recurrent dislocation as compared to acute dislocation[27]presents an inherent barrier to producing studies that provide high-quality evidence such as systematic reviews of randomized controlled trials (RCTs) and individual RCTs (Level I evidence as defined by the Oxford Centre for Evidence-based Medicine; (**Figure 16**)[175] from which generalizable recommendations can be drawn.[22,27]

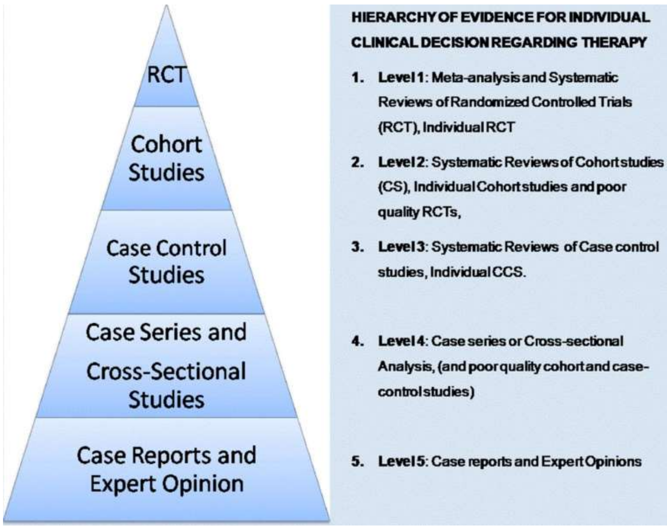

Figure 16: Hierarchy of evidence for clinical decision-making regarding therapy.
(From open-access article Esene IN, Baessa SS, Ammar A. *Evidence-based neurosurgery: Basic concepts for the appraisal and application of scientific information to patient care (Part II).*Neurosciences 2016; 21(3):197-206.)

Further complicating matters are variable and/or inadequate follow-up periods found between studies, some ambiguity in regards to classification of an acute vs chronic dislocation,[10] and a lack of uniformity in the definition of a successful treatment, with some defining “success” to be the absence of recurrence and others examining additional outcome variables such as functional measurements (e.g. maximal incisal opening) and subjective findings (e.g. quality of life, pain scores, etc.).[13,27,29] These differences in study design that limit our ability to compare a large number of studies explain the lack of consensus and absence of a well-defined treatment algorithm as it pertains to the management of chronic recurrent dislocation. Future, prospective, randomized controlled trials that address these shortcomings and compare the use of an etiology-based, multi-modal approach to individual treatments (e.g. eminectomy) for the treatment of chronic dislocation may allow us to more predictably prevent recurrence and halt pathologic progression of disease, and therefore eliminate the need for multiple interventions.

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