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Guidelines for the Management of Patients With Orofacial Pain and Temporomandibular Disorders

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The management of patients with orofacial pain and temporomandibular disorders is challenging. Recognizing the opportunity to advance patient care, in 2019 the National Academies of Science, Engineering, and Medicine (NASEM) convened representatives from the Food and Drug Administration, Medical Device Epidemiology Network (MDEpiNet), patient advocacy groups, oral and maxillofacial surgeons, and orofacial pain experts to identify specific steps to improve care for patients. In response, the American Association of Oral and Maxillofacial Surgeons (AAOMS) created the Special Committee on Temporomandibular Joint Care (SCTMJC) whose assignmentwas to developcontemporary evidence-based guidelines for the management of patients with orofacial pain and temporomandibular disorders. These guidelines represent the findings and recommendations of that committee.

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Chapter 1. Glossary of Terms

Internal Derangement (ID)

This term has historically been used to describe a constellation of symptoms, signs, radiological and histological findings as it pertains to the disc position within the Temporomandibular joint (TMJ). ID has most commonly been classified using Wilkes stages I-V. The historical reliance on ID to explain intraarticular pain and dysfunction (IPD) has been superseded by a contemporary understanding that focuses on biological and inflammatory processes. Although the Wilkes stages of ID are a useful tool to describe the relationship between the condyle, disc and glenoid fossa; it offers little ability to identify the cause of IPD or the most ideal treatment.

Intraarticular Pain and Dysfunction (IPD)

This term refers to a range of intraarticular pathology including synovitis, chondromalacia, capsular impingement, fibrous adhesions, pseudo walls, disc morphology and disc position. It is an all-inclusive term that better reflects the multiple causes of IPD. It will be used throughout these guidelines in lieu of ID.

Symptomatic Disc Displacement (SDD)

Disc displacement often occurs in the general population without any symptoms. Disc displacement in any direction, either with or without reduction, that results in pain, limited opening or a reduced quality of life is considered SDD. The presence of pain, limited function or reduced quality of life does not correlate with disc displacement in the majority of patients.

Inflammatory Arthropathy

The presence of degradation of joints, articular cartilage and subchondral bone secondary to a high inflammatory state that may result from one or more autoimmune conditions including rheumatoid arthritis, systemic lupus erythematosus, psoriasis, ankylosing spondylitis and reactive arthritis.

Failure

Failure can be defined as an outcome, either nonsurgical or surgical, where the patient considers the post treatment state (pain, range of motion and/or QoL) is unsatisfactory. It should be considered in light of ensuring that the patient and surgeon expectations are both realistic and concordant.

Arthroplasty

An open surgical procedure of the TMJ that may involve repositioning of the articular disc with sutures/anchors, disc removal without replacement or disc removal with replacement including temporalis muscle/fascia, fat or dermis. Reshaping of the mandibular condyle, glenoid fossa or articular eminence may also be performed.

Total Joint Replacement

An open surgical procedure involving the replacement both the mandibular condyle and glenoid fossa with an alloplastic material.

Osteoarthritis

Osteoarthritis is a degenerative joint disease that involves the degradation of joints, articular cartilage and subchondral bone from mechanical stress. The inflammatory state is low and may be conspicuous by its absence or limited presence.

Chapter 2: Differential Diagnosis of Diseases/Disorders Affecting the Head, Neck and Temporomandibular Joint Structures

Objectives:

- Appreciate the many disease processes and disorders that can affect the head and neck region resulting in pain and loss of function
- Allow the Oral and Maxillofacial Surgeon to develop a broad differential diagnosis in patients presenting with oro-facial pain.

The differential diagnosis of oro-facial pain must remain broad in order to ensure that all potential sources of pain and dysfunction are considered.[1]

INTRACRANIAL PATHOLOGY

1 Ischemic cerebrovascular disease or Transient Ischemic Attack
- Acute onset of headache in association with neurological deficit such as aphasia, ataxia, facial weakness, diplopia, extremity weakness or altered sensation.
- Requires emergent CT with contrast to evaluate for hemorrhagic process versus ischemic stroke. The latter requires the administration of tissue plasminogen activator (tPA) within 3 hours of onset which is followed by anticoagulation with heparin, warfarin, antiplatelet drugs, Factor X inhibitors, Direct Thrombin inhibitors or aspirin.

2 Intracranial hemorrhage
- Acute or subacute onset of headache may suggest epidural or subdural hemorrhage.
- Epidural hemorrhage may occur following trauma resulting in pain and a lucid interval leading to progressive unconsciousness. Requires emergent CT with contrast followed by craniotomy and decompression.
- Subdural hematoma is less likely to produce pain and may result in more subtle neurological deficits and cognitive impairment. Risk is highest in patients on anticoagulation. May require judicious observation or craniotomy and decompression.

3 Vascular malformation
- Vascular malformations may result in nonspecific headaches although most are asymptomatic until they rupture. This typically results in the ''worst headache imaginable'' and altered mental status.
- Magnetic resonance angiography (MRA) and computed tomographic angiography (CTA) remain the diagnostic imaging modalities of choice.
- Craniotomy and surgical repair are indicated following rupture or when they are identified and pose a significant risk of rupture.

4 Giant Cell Arteritis
- Giant cell arteritis (GCA) is an inflammatory vasculitis that affects the temporal artery as well as other branches of the maxillary artery including the ophthalmic artery.
- It can result in headache, jaw claudication and vision loss. Furthermore, it may be associated with generalized muscle pain (polymyalgia rheumatica).
- It is diagnosed with a temporal artery biopsy and is often associated with non-specific inflammatory markers such as C Reactive Protein (CRP) and Erythrocyte Sedimentation Rate (ESR).
- Treatment includes steroids and immunosuppression

5 Cervical Carotid Dissection or Vertebral artery dissection
- Dissection may follow trauma or may be spontaneous. Pain typically accompanies the dissection which may also result in Transient ischemic attacks (TIA) or cerebrovascular accidents (CVA). Focal neurological deficits are common.
- The diagnosis can be made with ultrasound, CTA or MRA.
- Treatment is surgical although the endovascular approach has gained popularity

6 Increased Intracranial pressure
- Increased intracranial pressure may result from an obstruction to the outflow of Cerebrospinal fluid (CSF) or may be idiopathic.
- It typically results in headache, papilledema, diplopia, and focal neurological deficits. Idiopathic intracranial hypertension (IIH) is the most common cause and is more common in obese females.
- The diagnosis requires an increased opening pressure during lumbar puncture and a normal brain MRI.
- Treatment includes weight loss, diuretics and on occasion surgery.

7 Neoplasia
- Tumors of the brain may be malignant or benign. They often result in headaches and focal neurological deficits.
- The diagnosis requires CT or MRI. Treatment depends on the type and nature of the neoplasm (malignant vs benign).

8 Arnold Chiari malformation
- Herniation of the cerebellar tonsils through the foramen magnum can result in headaches. Depending on the severity and level of herniation, patients may also suffer focal neurological deficits, nystagmus, dysphagia, and coughing.
- Severe cases require surgical decompression.

9 Hypertensive Emergency
- Hypertension that is poorly controlled may result in a hypertensive emergency when associated with end organ damage. This includes hypertensive encephalopathy, CVA, subarachnoid hemorrhage or intracranial hemorrhage. The presence of elevated blood pressure, headache and focal neurological deficits is suggestive.
- Treatment involves intravenous antihypertensive medication.

10 Acute Glaucoma
- Acute angle-closure glaucoma may result in headache as well as eye pain, visual disturbance, eye redness and visual halos.
- The diagnosis is made by demonstrating raised intra-ocular pressure (IOP) measured with tonometry. Normal IOP is 12-22 mm Hg. Failure to treat raised IOP may lead to blindness that begins in the peripheral visual field.
- Treatment includes topical beta blockers and carbonic anhydrase inhibitors. Surgery may also be needed when topical agents are ineffective.

HEADACHE DISORDERS

1 Tension-Type Headaches
- The most common type of headache with characteristics of a dull pain, non-pulsating, bilateral headache of mild to moderate intensity and with a pressure-type of pain presentation in a band-like distribution ‘‘as in a hat that is too tight ’. There is no nausea, vomiting or agitation with physical activity noted with these headaches and they are usually related to stress, anxiety, depression, or impaired sleep. These usually occur in the forehead region.
- Tension-Type headaches can be infrequent (less than 1 per month and less than 12 days per year), frequent (more than 1 day per month and less than 15 days per month for at least 3 months) or chronic (more often than 15 days per month for at least 3 months). The headache duration can be from 30 minutes to over 7 days.
- Most are treated with over-the-counter medications (NSAIDs, aspirin and acetaminophen). Amitriptyline, mirtazapine, venlafaxine, and clomipramine are occasionally used to treat headaches. Botulinum toxin may also be used to treat chronic daily headaches.

2 Migraines
- The next most common headache is a migraine. They can occur with or without aura and are considered the most disabling of all the headaches. The headache usually presents as a pulsating moderate/severe pain which is unilateral.
- Migraines can be recurrent and last 4-72 hours. The headache of a migraine can be broken down into 4 separate phases: Prodrome phase, Aura phase, Headache phase and Postdrome phase. The Prodrome phase, which is predictive of an attack, occurs approximately 2 -24 hours prior to such attack, is seen in approximately 60% of all migraine patients. Prodrome phase can consist of a variety of symptoms such as depression, tenderness, yawning, sensory hypersensitivity, food craving, irritability, euphoria, fatigue, and polyuria. The second phase is the Aura phase experienced by over 33% of patients and includes visual and sensory changes with scintillations (flashes or twinkle of light), scotomas (an area of partial alteration in the field of vision, a blind spot) and can be associated with nausea, vomiting, photophobia and phonophobia, language disorders and agitation with physical activity, and then a clearing state. The third phase is the Headache phase and consists of a unilateral moderate to severe pain as described above. The headache of migraine usually occurs around 60 minutes after resolution of the neurologic symptoms. The fourth phase involved in the migraine is the Postdrome phase after the headache has subsided or following medical intervention. This usually involves impaired concentration, fatigue, and a feeling of being washed out.
- Migraine treatment includes preventive and abortive medications including Triptans, Selective serotonin reuptake inhibitors, NSAIDS, acetaminophen, aspirin, beta-blockers, topiramate, Ona botulinum toxin A (Botox) and Calcitonin Gene Related Peptide blockers (eptinezumab-jjmr, ubrogepant).

3 Cluster headache
- A rare headache characterized by very severe sharp and throbbing unilateral pain lasting from 15-180 minutes in duration and can be episodic with ‘‘cluster periods’’ lasting from 2

weeks to a few months. These Cluster headaches can have migraine features of nausea, photophobia, and phonophobia in about 50% of cases.

- These headaches are extremely severe and sometimes referred to as ''suicide headaches''. The frequency of attacks ranges from 1-8 per day. The male to female ratio is greater than 3:1.
- Alcohol is a powerful stimulus to provoke a Cluster headache.
- This type of headache is responsive to supplemental oxygen or sumatriptan.

4 Paroxysmal Hemicrania Headache

- A rare but severe throbbing unilateral (orbital-frontal area) pain similar to Cluster headaches but the duration of pain is for shorter periods (usually minutes). Attacks can average 11+ per day. These paroxysmal headaches can have migraine features of nausea, photophobia and phonophobia in about 50% of cases.
- These headaches are more common in females and respond well to indomethacin.

5 Short-lasting Unilateral Neuralgiform headache with conjunctival injection and tearing (SUNCT)

- A rare very severe sharp and throbbing unilateral (orbital) pain. Attacks last 5 seconds to 4 minutes in duration. Pain is noted to be burning, stabbing or electric in nature and there can be 3 - 200 attacks per day.
- Autonomic features of this headache are conjunctival injection and tearing.
- SUNCT headaches can have migraine features of nausea, photophobia and phonophobia in about 25% of cases.
- Treatment includes carbamazepine, lamotrigine, gabapentin, topiramate and intravenous lidocaine.

6 Short-lasting Unilateral Neuralgiform headache with Cranial Autonomic Symptoms (SUNA)

- a rare unilateral sharp but very brief headache similar to SUNCT with attacks lasting 5 seconds to 4 minutes. Less common than SUNCT. Autonomic features include agitation and restlessness.
- Treatment is similar to SUNCT.

7 Hemicrania Continua headache

- A rare and persistent severe unilateral headache that fluctuates in intensity but never truly remits. Hemicrania Continua headaches can have Migraine features of nausea, photophobia, and phonophobia in about 50% of cases. Autonomic features include restlessness and agitation which are found in about 66% of all cases.
- A positive response to indomethacin is diagnostic. Treatment also includes topiramate, greater occipital nerve block and greater occipital nerve stimulation.

8 Cervicogenic headaches

- This is a very common cause of headaches. This is attributed to a disorder of the cervical spine including muscles, soft tissues, intervertebral disc or vertebrae. This headache presents as a moderate unilateral non-throbbing pain that radiates (starts) from the neck. The headache is usually ipsilateral. The male: female ratio is 4:1.
- There is a decreased range of motion (ROM) in the cervical area. If radiculopathy is present, pain may be referred to the upper extremity with concomitant upper extremity weakness.
- Treatment includes NSAIDs, physical therapy, epidural steroids/local anesthetics, or surgery.

NEUROPATHIC PAIN

1 Trigeminal neuralgia

- Pain is felt in the cranial nerve V distribution, most often a single division. It may be spontaneous or triggered by non-noxious stimuli. The pain is typically unilateral and severe, lasting from seconds to up to several minutes. Most common in V2 and V3 distributions.
- About 1/3 of TN is accompanied by a background of dull pain in the same area (termed TN with concomitant persistent facial pain).
- Treatment includes anti-seizure medication (carbamazepine, oxcarbazepine, phenytoin, gabapentin, pregabalin), baclofen, radiofrequency rhizotomy, gamma knife and microvascular decompression.

2 Glossopharyngeal neuralgia

- Pain felt in cranial nerve IX distribution including the ear, base of tongue, oropharynx, submandibular triangle. Typically unilateral, but rarely may have asynchronous bilateral pain. The pain is triggered by chewing, swallowing, yawning, and talking.
- Treatment is similar to trigeminal neuralgia.

3 Painful post-traumatic trigeminal neuropathies (PPTTN)

- Pain develops, sometimes gradually, following injury/trauma to the affected division. It is accompanied by a decrease in sensation as well as pain in one or more divisions of trigeminal nerve.
- Allodynia or hyperalgesia are often present.
- Classic ''phantom tooth pain'' is thought to represent a form of PPTTN and deafferentation.

There is often a history of one or more endodontic treatments or extractions and persistent pain at that site for >4 months.
- Treatment is unfortunately very challenging with limited success.

4 Central neuropathic pain
- This is most often caused by either Multiple Sclerosis or CVA. There are often other non-neuralgia neurologic findings and multiple dermatome involvement is common.
- Treatment is challenging.

5 Complex regional pain syndrome
- There are two types that are distinguished by the presence or absence of known nerve damage preceding the pain. The pain is disproportionate to the inciting event.
- The diagnosis requires a constellation of findings including hyperesthesia and/or allodynia; temperature asymmetry and/or skin color changes and/or asymmetry; edema and/or sweating changes and/or sweating asymmetry and decreased range of motion and/or motor dysfunction (weakness, tremor, dystonia) and/or trophic changes (hair, nail, skin).
- Treatment includes physical therapy, anti-seizure medications, muscle relaxants, sympathetic nerve blocks, ketamine infusions, opioids and rarely surgery (sympathectomy).

6 Persistent idiopathic facial pain (previously atypical facial pain)
- Facial and/or oral pain which recurs daily for >2 hours/day for >3 months, non-anatomical in distribution and dull, aching or nagging in quality.
- Neurological examination is normal and persistent idiopathic facial pain remains a diagnosis of exclusion.
- Treatment is generally unsatisfactory.

INTRA-ORAL PAIN DISORDERS

1 Pulpal pain
- Deep, dull, aching, pain associated with reversible pulpitis, irreversible pulpitis, or pulpal necrosis as result of infectious, traumatic, or iatrogenic injury to the dental pulp. Pain is often worse with biting or with ingestion of hot and cold foods/liquids.
- Treatment may include removal of caries and restoration, endodontic therapy, or dental extraction.

2 Periodontal pain
- Localized dull aching pain or severe unrelenting throbbing pain associated with gingival abscess, periodontal abscess, or periapical abscess. Pain is the result of inflammatory infiltrate in the periodontium. Percussion of the associated tooth often results in pain.
- Treatment of the gingival, periodontal, and periapical abscess involves establishment of drainage of the abscess and removing the source of infection including but not limited to dental extractions or endodontic therapy of the offending tooth or teeth.

3 Pericoronitis pain
- Periodontal pain due to inflammation or infection of the soft tissue surrounding an impacted or partially erupted tooth, most commonly lower 3rd molars. The erythematous and swollen gingival tissue surrounding the tooth may result in radiating pain to the ear, floor of the mouth and the throat.
- Initial treatment of acute pericoronitis include irrigation and lavage under the gingiva with sterile saline, hydrogen peroxide or chlorhexidine. Removal of the tooth is indicated after the acute episode resolves. If an abscess is present surgical drainage should be established with administration of appropriate antibiotics.

4 Aphthous Stomatitis
- Mucosal pain associated with a detectable mucosal lesion due to immune dysfunction. Aphthae generally occur in the non-keratinizing mucosa of the mouth. Aphthae may present in three clinical forms: minor, major and herpetiform.
- The three clinical forms differ in the number of aphthae present, the size of the lesions and the duration of the lesions
- Current treatments such as topical steroid, tetracycline, intralesional steroid or systemic steroid are effective in reducing symptoms and healing time but not in decreasing the recurrence rate.

5 Viral infections
- Viral infections from the Herpes family viruses such as Herpes Simplex (HSV), Herpes Zoster (VZV), Cytomegalovirus (CMV), Epstein Barr Virus (EBV) may manifest as oral blistering lesions that could cause pain during primary exposure or reactivation.
- Once the active infection resolves, postherpetic neuralgia can develop and lead to chronic neuropathic pain of the head and neck region.
- Treatment of primary viral infections focus on supportive care to prevent dehydration. Systemic analgesics may be required for pain management. Systemic antiviral medications such

as acyclovir or valacyclovir may be utilized. For recurrent infections, topical or systemic antiviral medication may be used to either prevent development of lesions or shorten the time to resolution.

6 Fungal infections
- Fungal infections of the oral cavity could present as orofacial pain. Patients may complain of oral tenderness, mucosal burning, taste changes and odynophagia. In pseudomembranous candidiasis, lesions may occur on the palate, buccal mucosa, mucobuccal folds, lateral and dorsal aspects of the tongue and oropharynx.
- Erythematous candidiasis is more commonly seen on the mid dorsal tongue and denture bearing area of the palate. Chronic candida infections could also lead to hyperplastic tissue response.
- Angular cheilitis with local erythema and/or ulceration at the commissures of the lips may also be painful. Fungal infections of the oral cavity can be treated with topical polyenes (nystatin) and azoles (clotrimazole, miconazole). Systemic antifungals such as fluconazole may also be utilized.

7 Malignant tumors
- Primary oral and oropharyngeal malignancies, oral involvement by hematologic cancers, and orofacial spread of metastatic cancers can cause oral pain.
- Pain from cancer may be due to ulceration, pressure on nerves, invasion of nerves, vessels, muscle and periosteum or secondary infections. Pain may occur at the primary site and/or referred to another site. Treatment of head and neck cancer may include surgery, radiation therapy, chemotherapy, or immunotherapy.
- Treatments of malignancies may cause acute pain due to surgical trauma, or mucositis. Management of cancer related pain depends on its cause. Tumor induced pain generally regresses with anticancer treatment. Therapy induced mucositis requires systemic or topical analgesics.

8 Osteomyelitis
- Deep localized bone pain accompanied with swelling, fever, chills, and erythema next to the affected bone indicate acute osteomyelitis.
- Imaging includes panoramic x-rays, CT with contrast and MRI with or without gadolinium. The diagnosis of osteomyelitis requires bone biopsy and bacterial culture. Chronic osteomyelitis occurs after an acute episode of osteomyelitis that has not been completely treated and is often associated with a draining sinus tract.
- Treatment of osteomyelitis often requires a combination of surgical debridement/resection and appropriate oral or parenteral antibiotic therapy.

SALIVARY GLAND DISORDERS

1 Sialadenitis
- Relatively rapid onset of pain in affected gland area associated with swelling, and erythema in overlying skin
- Reduced salivary flow from affected gland is observed and on occasion frank pus from salivary ducts. Fever may be present.
- CT with contrast will identify sialadenitis.
- Treatment includes hydration, sialagogues, antibiotics, stone removal, or gland excision.

2 Sialadenitis and Sialolithiasis
- Pain in the gland area, often evoked or worsened by salivary stimulation (food smell, tart candy, etc.).
- Clinically appreciable or radiographically demonstrable stone in hilus of gland or duct.
- CT is much more sensitive than panoramic film.
- Treatment is the same as for sialadenitis.

EXTERNAL/MIDDLE EAR PATHOLOGY

1 Otitis externa
- Ear pain associated with acute infection of the skin of the ear canal and/or the auricle. Other symptoms may include discharge or hearing loss if the ear canal is swollen shut. Manipulation of the auricle often elicits pain. Diagnosis is based on inspection.
- Treatment includes local debridement and a combination of topical antibiotic, corticosteroids, or acetic acid.

2 Otitis media
- Ear pain presenting with upper respiratory infections is often due to bacterial or viral infection of the middle ear. Patients often have fever, nausea, vomiting, and diarrhea. Diagnosis is based on symptoms in conjunction with otoscopy. Bulging tympanic membrane and presence of middle ear effusions are signs of acute otitis media.
- Treatment includes analgesics, antibiotics and rarely myringotomy.

3 Cholesteatoma
- Feeling of fullness or pressure in the ear and pain may be due to cholesteatoma or a collection of keratinized squamous epithelium in the middle ear. However, the most common presentation for cholesteatoma is painless otorrhea.
- Cholesteatoma may erode and destroy local structures as it grows and may be congenital or due to chronic ear infections or eustachian tube dysfunction. Dizziness may be observed if it invades inner ear structures. Diagnosis is based on clinical presentation, history and radiographic CT findings.
- Excision of cholesteatoma is the treatment of choice.

4 Eustachian tube dysfunction
- Feeling of fullness in the ear, pain, muffled hearing, tinnitus, reduced hearing, and rarely balancing problems are results of eustachian tube dysfunction. Eustachian tube dysfunction occurs when the mucosal lining of the tube becomes swollen or if the tube cannot open or close properly.
- Chronic eustachian tube dysfunction often leads to middle ear effusions, chronic otitis media and middle ear atelectasis. Diagnosis is based on medical history and clinical examination to identify possible underlying causes of the eustachian tube dysfunction.
- Eustachian tube dysfunction generally resolves within a few days without treatment however if chronic both surgical and non-surgical options exist for management.

5 External Auditory Meatus/TMJ perforation
- Ear pain and otorrhea are common symptoms when there is an external auditory meatus perforation with or without temporomandibular joint herniation. This could be a result of trauma, tumor, infection, or inflammatory processes.
- Medical history, clinical examination, and radiographic imaging such as CT scan confirm the diagnosis.
- Management of the external auditory meatus perforation depends on the severity of the perforation and may include surgical intervention or placement of stents or packing or topical otic drops.

6 Mastoiditis
- Pain behind the ear or ear pain associated with redness, swelling, tenderness and fluctuation in the mastoid area and displacement of the pinna due to bacterial infection of the mastoid air cells secondary to acute otitis media.
- Treatment included antibiotic therapy and mastoidectomy if the non-surgical therapy is not effective alone.

SYSTEMIC ARTHROPATHY

1 Rheumatoid arthritis
- Destructive autoimmune disease that causes pain, inflammation, and stiffness of the joints. It is more common in women, usually polyarticular but primarily affecting the wrists and hands and frequently bilateral. When the TMJ is affected, it can cause pain, condylar erosion and intra articular inflammation leading to malocclusion and decreased range of motion.
- Constitutional symptoms include fatigue and malaise.
- May also involve other organs including, the skin, eyes, kidneys.
- Diagnosis is based on clinical and laboratory findings including Rheumatoid factor (RF) and anti-citrullinated protein antibodies (anti-CCP antibodies). Imaging in early stages may not demonstrate osseous findings or the x-ray may show osteopenia near the joint, soft tissue swelling, and a smaller than normal joint space. As the disease advances, there may be bony erosions and subluxation.
- Treatment includes physical therapy, nonsteroidal anti-inflammatory drugs (NSAIDs), Disease modifying anti-rheumatic drugs (DMARDS), steroids, immunosuppressive drugs, biologics, arthrocentesis, arthroplasty, and total joint replacement.

2 Systemic lupus erythematosus (SLE)
- SLE is a chronic, autoimmune disease that affects the connective tissue of many organs, causing inflammation and vasculopathies. Symptoms include painful and inflamed joints (including the TMJ), fever, facial rash, hair loss, mouth ulcers, swollen lymph nodes and malaise. Many organs may be involved including the kidneys, heart, lungs, and brain. The diagnosis requires that at least 4 out of 17 specific criteria be met.
- The diagnosis can be made with serologic diagnostic tests such as Antinuclear antibody (ANA), anti-extractable nuclear antigen (anti-ENA) and anti-double-stranded DNA (anti-dsDNA) blood tests or tissue biopsy.
- Treatment includes physical therapy, nonsteroidal anti-inflammatory drugs (NSAIDs), steroids, immunosuppressive drugs, biologics,

arthrocentesis, arthroplasty, and total joint replacement.

3 Psoriatic arthritis
- Chronic inflammatory autoimmune erosive arthritis that occurs in about one-third of the patients affected by Psoriasis. Commonly oligoarticular affecting two to four joints but it can present itself in multiple joints as well. The joints of the hand that are involved in psoriasis are the proximal and distal interphalangeal as well as the metacarpophalangeal and the wrist. It is associated with psoriasis and affects the nails (thickening and pitting), and the skin producing scaly erythematous lesions.
- It is classified as a type of seronegative spondyloarthropathy although approximately 40–50% of individuals with psoriatic arthritis have the HLA-B27 genotype.
- Diagnosis is supported by a family history of psoriasis or psoriatic arthritis; RF negative, involvement of the distal interphalangeal joints, ridging or pitting of fingernails or toenails (onycholysis) and radiologic images demonstrating degenerative joint changes.
- Treatment includes physical therapy, nonsteroidal anti-inflammatory drugs (NSAIDs), steroids, immunosuppressive drugs, biologics, arthrocentesis, arthroplasty, and total joint replacement.

4 Ankylosing spondylitis
- This type of arthritis affects the vertebral column, usually causing ossification of the spinal ligament, affecting the spine and sacroiliac joint. Occasionally it can affect the hips and shoulders. Often results in kyphosis and bony ankylosis of the TMJ.
- The majority of the patients are HLA-B27 positive.
- Treatment includes physical therapy, nonsteroidal anti-inflammatory drugs (NSAIDs), steroids, immunosuppressive drugs, arthroplasty, and total joint replacement.

INFLAMMATORY/INFECTIOUS CONDITIONS

1 Gout
- This is a metabolic disease caused by the deposition of uric acid crystals in the joints, tendons and tissues resulting in swelling and pain. It is due to elevated levels of uric acid and is a disorder of purine metabolism. It occurs when uric acid crystallizes in the form of monosodium urate crystals triggering a local immune mediated inflammatory reaction initiated by the NLRP3 complex.
- Gout is often diagnosed in someone with hyperuricemia and involvement of the great toe (podagra). Joint aspiration also reveals needle shaped crystals that are positively birefringent.
- Treatment involves drugs to reduce purine turnover (allopurinol), increase urinary excretion of uric acid (probenecid) as well as colchicine, NSAIDS and steroids.

2 Pseudogout
- This is a rheumatological condition that most commonly affects the knee, wrists and hips secondary to abnormal accumulation of calcium pyrophosphate dihydrate crystals within the joint soft tissues. It is also called chondrocalcinosis or pyrophosphate arthropathy and it causes pain and swelling of the affected joint. It mimics the symptoms of gout therefore its name.
- The symptoms can be monoarticular (involving a single joint) or polyarticular (involving several joints) and they usually last for days to weeks, and often recur.
- X-ray, CT, or other imaging usually shows accumulation of calcium within the joint cartilage.
- Treatment includes colchicine, NSAIDS, steroids and arthrocentesis.

3 Reactive Arthritis
- This is a form of inflammatory arthritis that occurs in response to an infection usually in another part of the body. By the time the joint is affected the primary infection may be resolved making the diagnosis difficult.
- It occurs in any joint but typically the knees and ankles are affected and is characterized by pain, swelling, and stiffness accompanied by inflammation of the eyelids, skin, or urinary tract, and typically lasts 1 year or less but may become chronic.
- Reactive arthritis is an RF-seronegative, HLA-B27-linked arthritis often precipitated by genitourinary or gastrointestinal infections. The most common triggers are intestinal infections including Salmonella, Shigella or Campylobacter; sexually transmitted infections including Chlamydia trachomatis; or group A streptococcal infections.
- Diagnosis is very challenging although serology for specific organisms listed above is suggestive.
- Treatment involves the use of long-term antibiotics.

4 Lyme disease
- Lyme borreliosis is an infectious disease caused by tick-borne spirochete Borrelia burgdorferi.

The most common sign of infection is an expanding red rash, known as erythema migrans, that appears at the site of the tick bite about a week after it occurred.

- The systemic infection primarily involves three organ systems: the heart, causing a conduction block; the joints, causing arthralgia; and the nervous system, typically as cranial neuropathy, painful radiculopathy, or lymphocytic meningitis.
- Serological tests using the sensitive ELISA test are performed initially and if positive is followed by a confirmatory Western blot test.
- Antibiotics are the primary treatment. Oral administration of doxycycline is widely recommended as the first choice, as it is effective against not only Borrelia bacteria but also a variety of other illnesses carried by tick. With treatment the prognosis is good.

OTHER

1 Sinusitis

- Inflammation and/or infection of the paranasal sinuses are relatively common. Inflammation is often secondary to an allergy while infection may be due to viral, bacterial, or fungal infection. It can be acute, subacute, or chronic.
- Complicated sinusitis is typically a bacterial infection that extends to involve the orbit or intracranial structures.
- The inflammation or infection results in the accumulation of mucous, sinus lining thickening and loss or sinus patency and drainage, postnasal drip, discolored nasal discharge, nasal stuffiness or congestion, tenderness of the face (particularly under the eyes or at the bridge of the nose), frontal headaches, pain in the maxillary teeth and fever.
- Examination may reveal pain to pressure over the frontal and maxillary sinuses together with rhinitis. The diagnosis can be confirmed with x-rays (Waters' view) although CT imaging is more sensitive. Although many microorganisms may cause sinusitis, Hemophilus influenzae, Moraxella catarrhalis and Streptococcus pneumoniae are often responsible for acute sinusitis.
- An additional consideration relates to chronic sinusitis which tends to involve more anaerobic organisms including Prevotella, Fusobacterium and Peptostreptococcus species as well as Staphylococcus aureus.
- Sinusitis in an insulin dependent diabetic should also raise the suspicion of fungal involvement and mucormycosis with the potential for rhinocerebral involvement.
- Treatment involves nasal lavage (normal saline or neti pot), decongestants, and antibiotics. Amoxicillin and amoxicillin/clavulanic acid are good choices for uncomplicated sinusitis for non-penicillin allergic patients. Third generation cephalosporins such as ceftazidime, cefotaxime and cefdinir are alternate choices if the patient can tolerate them. Doxycycline remains a good choice when penicillin/cephalosporin allergy is severe.
- The management of complicated sinusitis requires hospital admission, broad spectrum intravenous antibiotics and often endoscopic surgical intervention.

2 Bruxism

- Bruxism is typically defined as grinding or clenching of the teeth that results from excessive activity of the masticatory muscles. It can occur during sleep, resulting in nocturnal bruxism, or during the day, resulting in awake bruxism.
- Symptoms of nocturnal bruxism include headache, masticatory muscle or temporomandibular joint pain that is worse upon awakening, jaw tightness and limited opening. Signs may include excessive tooth wear, fractured teeth and lateral tongue crenations.
- Treatment remains challenging with little evidence to support one modality over another. Flat plane occlusal splints and medications remain the most popular treatments.
- The most frequently used medications are muscle relaxants, low dose tricyclic antidepressants and benzodiazepines. If present, trigger point injections with or without local anesthesia can be beneficial.
- The beneficial effect of Botulinum toxin on sleep related bruxism has been reported. It appears to reduce the intensity of muscle activity within the masseter and temporalis muscles with a reduction in self-reported pain and jaw stiffness. This remains an off-label use of Botulinum toxin.
- Daytime bruxism may be treated with physical therapy including massage, dry needling, Transcutaneous Electrical Nerve Stimulation (TENS),

Contingent Electrical Stimulation (CES), Biofeedback and Cognitive based therapy.

3 Dystonia and dyskinesia
   - Dystonia is a tonic contraction of muscle that can affect multiple sites and result in pain and dysfunction. Oromandibular dystonia results in contraction of the masticatory muscles, intrinsic tongue muscles and/or the suprahyoid muscles. This can result in severe tooth grinding and grimacing.
   - If accompanied by blepharospasm, it is referred to as Meige syndrome.
   - Dystonia may also present as hemifacial spasm, torticollis, isolated blepharospasm and oculogyric crisis.
   - Tardive dyskinesia is typically a continuous involuntary muscle movement that develops following the administration of antipsychotic or antiemetic medication. Oro buccal lingual tardive dyskinesia is relatively common resulting in lip and tongue smacking, pursing of the lips and various masticatory movements.
   - The etiology of dystonia/dyskinesia is unclear although antipsychotic, antiemetic, antidepressant, anticonvulsant and anti-vertigo medications have been implicated. The diagnosis is based on the clinical presentation although electromyography (EMG) can be useful.
   - Acute dystonias can be managed by administration of an anticholinergic medication such as benztropine, biperiden or deutetrabenazine. Dopamine agonist medications may also help some patients. The use of botulinum toxin has greatly improved quality of life in this patient population.
   - It remains important to identify other causes of dyskinesia such as cerebral palsy, Huntington's disease, Parkinson's disease, Tourette's syndrome and cerebrovascular accident (CVA).

4 Multiple sclerosis
   - Multiple sclerosis (MS) is a demyelinating disease that affects the central nervous system including the brain and spinal cord. This results in motor and sensory deficits such as diplopia, weakness, paresthesia, loss of coordination, constipation, bladder dysfunction and fatigue.
   - It may also result in central neuropathic pain when there is involvement of the dorsal horn or spinal nucleus of the trigeminal nerve. However, MS is more likely to result in trigeminal neuralgia than central neuropathic pain.
   - The diagnosis of MS relies on an appropriate neurological examination with deficits, MRI of brain and spinal cord (plaques) and lumbar puncture (antibody to myelin). Disease may be classified as clinically isolated syndrome (CIS), primary-progressive, relapse-remitting and secondary progressive MS.
   - Treatment includes various modalities to reduce the immune response, cognitive and emotional changes, depression, including the use of corticosteroids, plasmapheresis, interferon, monoclonal antibodies and other Disease Modifying Treatment (DMT).

5 Fibromyalgia
   - Fibromyalgia is a chronic neurological pain condition that is associated with muscle and joint tenderness/pain, fatigue, altered sleep hygiene and cognitive impairment. Patients may also experience anxiety, depression, headache, irritable bowel syndrome, gastroesophageal reflux, interstitial cystitis, pelvic pain and temporomandibular disorders.
   - This constellation of symptoms is consistent with the diagnosis of chronic overlapping pain condition (COPC).
   - The diagnosis of fibromyalgia relies on a history and physical examination. Fatigue, sleep disturbance and tender points on a physical examination are common. Although there is no longer any requirement for a specific number of tender points, these areas typically involve the neck, chest, elbow, knee, suboccipital triangle, hip and buttocks.
   - Treatment includes non-steroidal anti-inflammatory drugs (NSAIDs), pregabalin and certain Serotonin Norepinephrine Reuptake Inhibitors (SNRI).

6 Stylohyoid ligament inflammation (Eagle syndrome) and Stylomandibular ligament inflammation (Ernest syndrome)
   - Pain in oropharynx, neck, or lower face, may include more general headache.
   - Usually unilateral, evoked by turning head.
   - Radiographs, including panoramic films or CT demonstrate calcified stylohyoid ligament.
   - Treatment is surgical resection or steroid injections

Chapter 3: History and Physical Examination

Objectives:
   - To develop a comprehensive and standardized approach to history taking and physical examination

- To appreciate the sensitivity and specificity of making the correct diagnosis when utilizing a standardized approach to history taking and physical examination.
- To appreciate the importance of a broad differential diagnosis
- To identify patients that are potentially good candidates for surgical intervention

INTRODUCTION

Obtaining a history and performing a physical examination of the new patient with a potential temporomandibular disorder (TMD) complaint creates the foundation for accurately diagnosing and, in turn, properly treating the patient's condition. The process follows the well-established outline for History and Physical Examination (H&P), with particular attention to details pertinent in TMDs. TMD is an ambiguous term which refers to both myogenous and arthrogenous sources of pain and dysfunction. It is critical to separate the myogenous and arthrogenous sources which can be achieved with a structured history and physical examination. As always, accurate, detailed and careful collection of relevant data is essential to developing a rational plan of action. That plan may include immediate treatment for more common, straightforward diagnoses, or in more complex cases, arranging additional tests or imaging to narrow the differential diagnosis. TMDs range in complexity from mild, minimal disease to complex, chronic, multifactorial conditions. However, the clinician must be aware of all facets of a detailed and comprehensive history and physical examination which requires an understanding of all of the following:

- Fundamentals of medical history taking and physical examination.
- Etiology and pathophysiology of disorders and diseases affecting the temporomandibular joint and related structures.
- Acute and chronic pain; peripheral and central mediated pain.
- Molecular basis of inflammation, oxidative stress, and disease course progression.
- Biopsychosocial model.
- Comorbid conditions and how they may be are associated with the current problem/condition.
- Pertinent laboratory and imaging techniques.
- Principles of Making the correct diagnosis and rendering evidence-based treatment.

The key elements to a comprehensive history taking and examination as it applies to temporomandibular disorders are best delineated in the Executive Summary of the Diagnostic Criteria/Temporomandibular Disorders (DC/TMD) (used with permission: Appendix I)

HISTORY

Chief Complaint

- Usually in the patient's own words, the main reason for the visit.

History of Present Illness

A narrative history of the patient's complaint is essential, and includes information on the nature of the problem, onset, precipitating events if any, progression, response to treatment efforts, and its consequences for activities of daily life. The patient should be interviewed with open ended questions eliciting the following information at a minimum:

- There may be additional complaints other than the chief complaint; these should be addressed like the chief complaint but noted as secondary.
- Detailed description of the onset and nature of the problem
- Detailed pain history
  - Onset, location, quality, intensity, radiation, associated symptoms, alleviating and aggravating factors, temporal variation, resolution. Pain should be recorded on a verbal or visual analogue scale.
- Previous treatments and their results
  - Occlusal orthotics, including details on design and use
  - Physical therapy modalities
  - Medications (especially NSAIDS, muscle relaxants)
  - Surgical treatments
- Joint sounds
- Functional limitations
- Other general issues: 'Anything else that I should know about?''

To supplement the patient's narrative, numerous well-validated instruments have been published which standardize data collection related to jaw dysfunction and significant comorbidities including anxiety, depression, functional limitations, pain and quality of life. These greatly enhance diagnostic and prognostic efficiency. It is strongly recommended that one or more of these standard instruments be routinely used as a part of initial patient evaluation:

- TMD Screener and DC-TMD Symptom Questionnaire
- Generalized Anxiety Disorder 7 (GAD 7)
- Patient Health Questionnaire (PHQ-4, 9 or 15)
- Jaw Function Limitation Scale (JFLS-8 or JFLS-20)
- Oral Health Impact Profile for TMDs (OHIP-TMD)
- Quality of life TJR (QOL-TJR)
- TMJ Quality of Life (TMJQoL)

It remains critical to appreciate the biopsychosocial considerations and Axis II Diagnoses that will adversely affect responses to treatment. Surgical outcomes in patients with psychosocial comorbid conditions are generally poor and should be avoided when at all possible (Appendix II).

Psychosocial issues may play a significant role in determining the prognosis for any givenpatient. It remains critical to identify subjects who are likely to do poorly, particularly when surgery will be contemplated. The Brief Symptom Inventory −18 (BSI-18) is an excellent instrument that when combined with Pressure Pain Thresholds (PPT) can allow all patients to be categorized as Adaptive, Pain sensitive or Global symptoms. The latter group are poor surgical candidates and every effort to avoid surgery or at least utilize minimally invasive surgical approaches should be adopted in this patient population.

Past Medical History

In most practices, a medical history is obtained by reviewing a form on which the patient indicates presence or absence of common, relevant medical conditions. Positive findings may be incorporated here by reference with additional details as necessary. Special attention should be paid to a past history of arthritis, psychiatric illness, or generalized pain conditions.

Past Surgical History

- Previous operations, anesthetics, and any unexpected outcomes.

Medications

- Generally obtained as a list provided by the patient.

Allergies

- Drug or environmental allergens and the response they elicit.

Social History

- Smoking/tobacco, alcohol, street drug use.
- Life situation: job, household.

Family History

- Arthritis or similar complaints in 1st degree relatives.

Review of Systems

- Constitutional: weight loss, fevers, sweats, chronic pain, fatigue, dyspnea, malaise and general overall pain, snoring or sleep problems.
- Head, eyes, ears, nose, throat (HEENT): headache, decreased hearing, ringing ears, earache, ear drainage, blurry or double vision, eye pain, dry eyes, nasal stuffiness or discharge, nosebleeds, dental problems, sore tongue or gums, dry mouth, sore throat.
- Musculoskeletal: muscle or joint pain, back pain, joint swelling or stiffness, trauma, weakness.
- Neurologic: dizziness, weakness, fainting, tingling/numbness, tremor, seizures.
- Psychiatric: depression, anxiety/nervousness, memory loss, stress.

PHYSICAL EXAMINATION (WITH EMPHASIS ON HEAD AND NECK FOR TMD)

Vital Signs

- Blood Pressure, heart rate, respiratory rate, weight, height, BMI

General

- Orientation, mood and affect (eye contact, attention to grooming).
- Development, nutrition, body habitus, deformities.
- Head and facial symmetry (or asymmetry), neck and shoulder posture, swellings, thyromegaly.

Eyes, Ears, Nose

- Ocular motility, primary gaze alignment, pupillary function.
- Gross hearing sensitivity.
- Otoscopic examination of external auditory canals and tympanic membranes. Pneumo otoscopy with notation of tympanic membrane mobility can assist with the recognition of otitis media and effusions.
- external inspection of nose and face for scars, lesions, masses.
- Inspection of nasal mucosa, septum and turbinates using a nasal speculum or otoscope.

Neck

- Adenopathy or masses.
- Tenderness and trigger points for the sternocleidomastoid, supraspinal, trapezius and suboccipital triangle muscles.

Masticatory Muscles and TMJ

- Temporalis and Masseter muscle palpation: origin, body, insertion of left and right sides.
  - Note headache, pain at site if pain is familiar, referred pain.
  - Note muscle swelling, hypertrophy, fasciculation, spasticity.
- TMJ capsule, lateral pole and endaural palpation: left and right sides
  - Note pain at site, if pain is familiar, referred pain.
- Intraoral palpation of temporalis tendon, masseter attachment.
  - Note headache, pain at site if pain is familiar, referred pain.

Mandibular Movements

- Pain-free opening
- Maximum unassisted opening
  - Location of pain if pain is familiar.
  - Intermittent locking or catching.
  - Lateral deviation and whether corrected or uncorrected.
- Maximum assisted opening
  - Location of pain if pain is familiar.
- Lateral jaw movement to left and right
  - Location of pain if pain is familiar.
- Protrusive movement
  - Location of pain if pain is familiar.
- Joint sounds during opening and closing, left and right
  - Note palpable or audible click, crepitus.

Intraoral and Dental

- Angle Occlusal Class.
- Occlusion
- General dental health.
- Oral and pharyngeal mucosal appearance.
- Gross salivary flow.

IMAGING

See Imaging Chapter 3.

Assessment and Plan

The new TMD patient is often best approached from a more medical than surgical standpoint. An operation is often not under consideration at the initial visit, and the diagnosis and treatment plan may be uncertain and more complex than routine oral and maxillofacial surgery care. When appropriate, this should be reflected in an assessment which may recognize uncertainty, contributions of comorbid conditions, the need for additional data collection and/or observation of responses to initial treatment. The narrative should include:

- Summary of pertinent findings and related conditions.
- Differential diagnosis.
- Stepwise plan to gather additional information or imaging to identify the correct diagnosis.
- Discussion of next steps.
- Follow-up visit plans.

Chapter 4: Radiological Imaging of the Temporomandibular Joint

Objectives:

- Evaluate the osseous and soft tissue components of the TMJ including the condyle, glenoid fossa and articular eminence
- Evaluate the morphology of the articular disk and its attachments, and tissues within the joint
- Evaluate joint dynamics with respect to joint rotation and translation
- Evaluate the maxillofacial region to identify coexisting diseases and conditions that may contribute to the patient's symptoms
- Monitor treatment outcomes, disease progression or recurrence

Imaging findings support clinical impressions and help confirm the clinical diagnosis. Particularly, imaging may detect pathologic changes that were not clinically detectable and provide information that influences the treatment plan. Therefore, appropriate application of imaging is an essential element in diagnosis and treatment planning of the patient with a TMJ disorder.[2]

Although not appropriate for evaluation of the temporomandibular joint, intraoral imaging may be

	Odontogenic disease	TMJ arthropathy	Developmental Disorders	Synovitis SDD	Trauma	Cysts/benign pathology	Malignant pathology
Panoramic	++	+	+		+	+	+
CBCT	++	++	++		++	++	
MDCT		++	++		++	++	++
MRI		++		++	+	+	+

FIGURE 1. Imaging modalities to be considered with the diagnosis of TMD.
CBCT (Cone beam computed tomography), MDCT (Multidetector computed tomography), MRI magnetic resonance imaging

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indicated when odontogenic sources of pain may exist. Traditional transcranial, transpharyngeal, and transorbital imaging are not currently recommended for TMJ evaluation. While cephalometric imaging should not serve as the primary examination to evaluate the TMJ, frontal and lateral cephalometric imaging may provide additional information on the consequences of a TMJ disorder such as facial asymmetry, growth disturbances of the condyles and treatment-associated changes with orthotic, orthodontic, and orthopedic appliances.

Imaging of the temporomandibular joint will often require different modalities depending on the differential diagnosis.[3] (Fig 1)

PANORAMIC IMAGING

Panoramic radiography provides a two-dimensional overview of the osseous TMJ and mandibular bony anatomy and symmetry. An optimally exposed panoramic radiograph can depict condylar morphology and identify anatomic variants of relevance. Panoramic radiographs can serve as the initial examination for patients with TMJ related symptoms. Serial imaging to assess changes in osseous structures over time may also be beneficial.

LIMITATIONS OF PANORAMIC IMAGING

- The anterior surface of the condyle, a common location for osteophytes, is not adequately visualized
- The shape of the condyle is often distorted.
- The zygomatic process of the temporal bone and the articular eminence are frequently superimposed over the condyle, obscuring osseous changes.
- Panoramic imaging should not be used to assess joint space or cartilaginous structures.

MULTIDETECTOR COMPUTED TOMOGRAPHY (MDCT)

MDCT is a form of computed tomography technology with a two-dimensional (2-D) detector that produces multiple, thinner slices in a single rotation and a shorter period of time allowing for true sagittal oblique cross-sectional images of the TMJ that allows for accurate representation of the relationship of the condyle and the fossa in maximum intercuspation and in open positions in more detail with additional view capabilities.

- Also provides coronal oblique images
- Images may be difficult to interpret, and it is technique sensitive
- Provides excellent hard tissue resolution and better contrast of the soft tissues and allows the assessment of muscles and tissue spaces.
- The articular disk is not well depicted on MDCT.

CONE BEAM COMPUTED TOMOGRAPHY (CBCT)

CBCT utilizes a volumetric scanning machine and generates 3D data of a region of interest at a lower radiation dose and cost along with higher spatial resolution compared with multidetector computed tomography (MDCT).

- Allows for true 3D imaging of maxillary and mandibular osseous structures including the TMJ.
- Superior detail compared to panoramic or tomographic imaging especially for subtle changes in osseous morphology.
- Cannot be used to assess the intra-articular disc or soft tissue details.

COMPUTED TOMOGRAPHIC ANGIOGRAPHY (CTA)

Combines a CT scan with an injection of a radiopaque dye to produce images of blood vessels and tissues to plan medical, surgical or radiation treatment.

- Ideal for TMJ bony ankylosis to identify arterial and venous structures.
- Ideal initial evaluation for vascular pathology including AV malformation and hemangioma.

MAGNETIC RESONANCE IMAGING (MRI)

MRI is the standard imaging modality for evaluating the articular disc and soft tissue structures of the TMJ as well as the presence of intracapsular fluid effusion. Osseous structure details are poor on MRI.

The imaging techniques include sagittal oblique and coronal plane thin slices of 3 mm or less. T1 and proton density weighted or T2 weighted sequences with fat suppression in both closed mouth and open mouth views are preferred for evaluation of the TMJ. Gadolinium based contrast agents may be used to further evaluate for presence of an inflamed synovium or arthropathy.

- Assess intra-articular soft tissue including articular disc position and disc morphology
- Static and dynamic imaging to better characterize TMJ under function
- With gadolinium to enhance images for inflammatory arthropathy and osteomyelitis.
- Allows for evaluation of disc displacement, joint effusions, synovial chondromatosis, and osteoarthritis.

BONE SCINTIGRAPHY

Bone scintigraphy or bone scanning is a nuclear medicine imaging technique. It can help diagnose a number of bone conditions, including primary or metastatic neoplasia, location of bone inflammation and fractures, and infection.

Single-Photon Emission Computed Tomography (SPECT)

SPECT images are taken after an injection of a nuclear medicine. The injected medication 'sticks' to specific areas in the body, depending on what radiopharmaceutical is used and the type of scan being conducted. A SPECT-CT scan is a type of nuclear medicine scan where the images or pictures from two diverse types of scans are combined together. The combined scan can establish reference values of the uptake difference between condyles and the ratio with respect to the clivus.[4]

Technetium 99 MDP/HDP

- 3 phase scintigraphy with SPECT.
- Tracer is incorporated in hydroxyapatite crystals during mineralization to assess overall bone turnover.
- Ideal to assess condylar growth, condylar asymmetry, osteoarthritis and long standing periprosthetic infection.

Technetium 99 HMPAO (Hexamethylpropyleneamine Oxime) WBC Scan

- Ideal for periprosthetic joint infection and osteomyelitis.

Indium-111 Labelled WBC scan.

- Ideal for periprosthetic joint infection and osteomyelitis.[5]

Technetium 99 Antigranulocyte Scan (IgG/Fab)

- Satisfactory for periprosthetic joint infection

Positron Emission Tomography (PET) Computed Tomography

Positron emission tomography is a functional imaging technique that uses radiotracers to visualize and measure changes in metabolic processes, and in other physiological activities including blood flow, regional chemical composition, and absorption.[6]

- Assess metabolically active tissue particularly primary malignancy and metastatic disease

Ultrasound

- Assess articular disc position and joint effusion

Chapter 5: Diagnosis and Management of Temporomandibular Joint and Related Pathology

Objectives:

- To understand the variety of pathology and disease processes that can affect the temporomandibular joint and masticatory muscles
- To appreciate how the history, physical examination findings and imaging are often specific for each disease process.
- To appreciate the variety of treatment options that may exist for each disease process

1. INTRA-ARTICULAR PAIN AND DYSFUNCTION (IPD)

I Synovitis
II Impingement, fibrous adhesions and pseudo walls
III Disc displacement with reduction (DDwR)
II Disc displacement with reduction (DDwR) and intermittent locking

III Disc displacement without reduction (DDwoR) with limited opening
IV Disc displacement without reduction (DDwoR) without limited opening
V Stuck disc
VI Osteoarthritis

History

- Pain with chewing or jaw opening
- History of joint sounds including clicking and crepitus
- Limiting diet to softer foods
- Limited jaw translation and opening

Examination

- Endaural or lateral capsular pain
- Pain, clicking, crepitus, deviation or limitation with protrusion
- Pain, clicking, crepitus or deviation or limitation with lateral excursions
- Pain, clicking, crepitus or deviation or limitation with jaw opening
- Limited MIO without and with pain
- Positive Mahan test
- Altered occlusion

Imaging

- Reduced joint space or osteoarthritis (OA) may be seen with panoramic or CBCT imaging
- Presence of disc displacement with or without reduction, effusion and OA on MRI (Fig 2A-H)
- Presence of OA with Computed tomography (CT)

The original Wilkes classification can be useful to help appreciate disc position, disc morphology and osteoarthritis as well as the impact on function. (Table 1).

Treatment Choices

- Non-surgical management with soft diet, jaw rest and heat
- Medical management with NSAIDs or steroids
- Physical Therapy
- Occlusal Orthotic
- Arthrocentesis
- Arthroscopy
- Arthroplasty
- Total Joint Replacement

2. MUSCULAR

I Myalgia
II Local Myalgia
III Myofascial pain
IV Myofascial pain with referral

History

- Pain with the subsequent bites and prolonged chewing
- Jaw fatigue with prolonged chewing
- Limiting diet to softer foods

Examination

- Pain with palpation of the temporalis and/or masseter muscles, tendons
- Pain with jaw opening
- Limited MIO without and with pain

Imaging

- None

Treatment Choices

- Non-surgical management with soft diet, jaw rest, cold and heat
- Medical management with NSAIDs and/or muscle relaxants
- Physical therapy
- Occlusal Orthotic
- Botulinum toxin

3. ANKYLOSIS

a) Osseous
b) Fibrous

History

- Progressive reduction in MIO
- Very limited or absence of any jaw opening suggests osseous ankylosis
- Pain is usually conspicuously absent
- Prior history of trauma, septic arthritis or previous TMJ surgery

Examination

- Severely limited MIO
- Minimal or absent lateral excursions and protrusion
- Scars from previous TMJ surgery

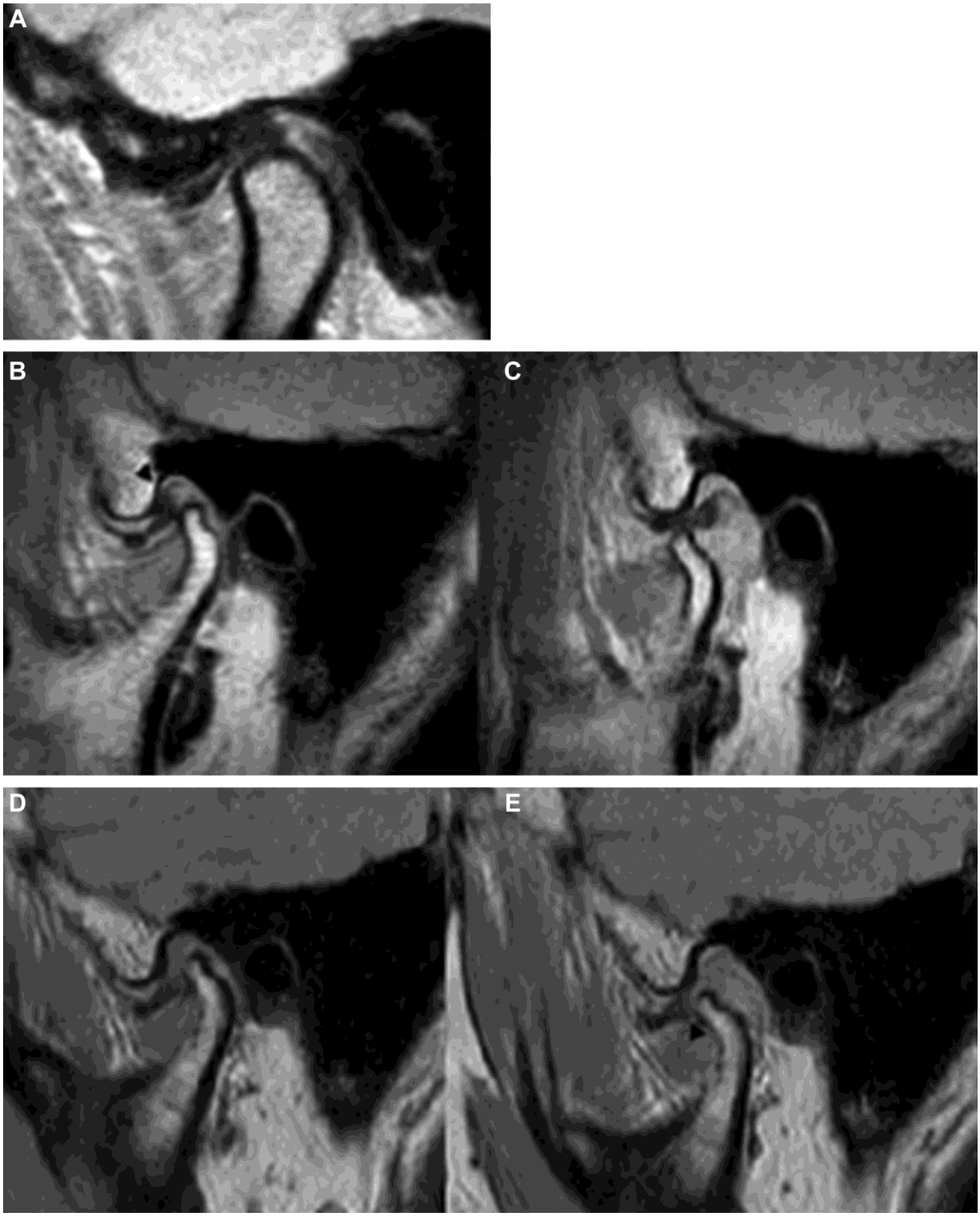

FIGURE 2. *A*, Normal Disc position. *B*, and *C*, Disc displacement with reduction (DDWR). *D*, and *E*, Disc displacement without reduction (DDWOR). (Fig 2 continued on next page.)

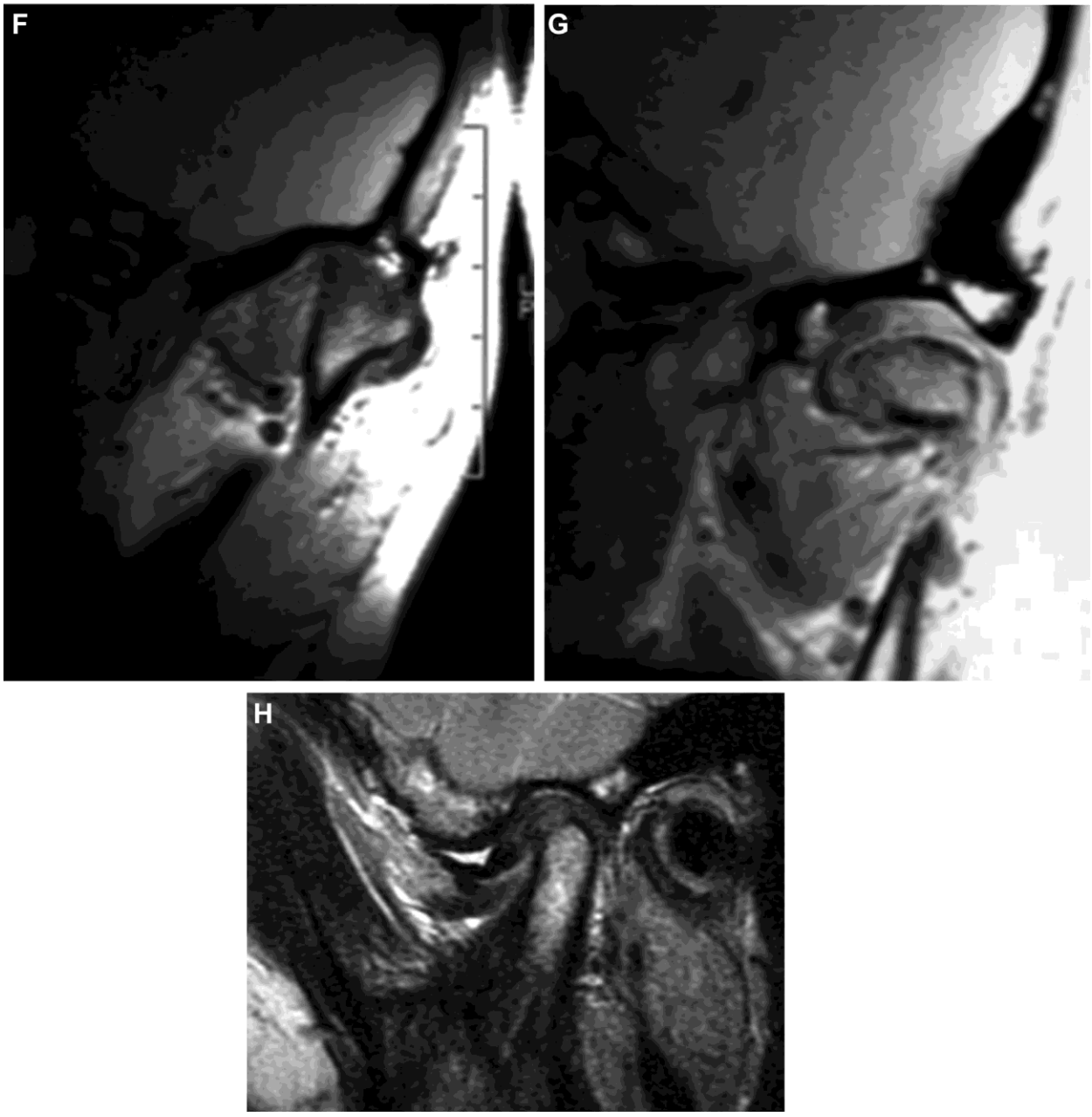

FIGURE 2 (cont'd). *F*, and *G*, Lateral and medial disc displacement. *H*, Joint effusion and osteoarthritis.

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Table 1. WILKES CLASSIFICATION

Stage	Pain	Clicking	Locking	Crepitus and Degenerative Joint Disease
I	None	None	None	Absent
II	Intermittent	Intermittent	Intermittent	Absent
III	Frequent	None	Frequent	Absent
IV	Frequent	None	None	Present
V	Intermittent	None	None	Present

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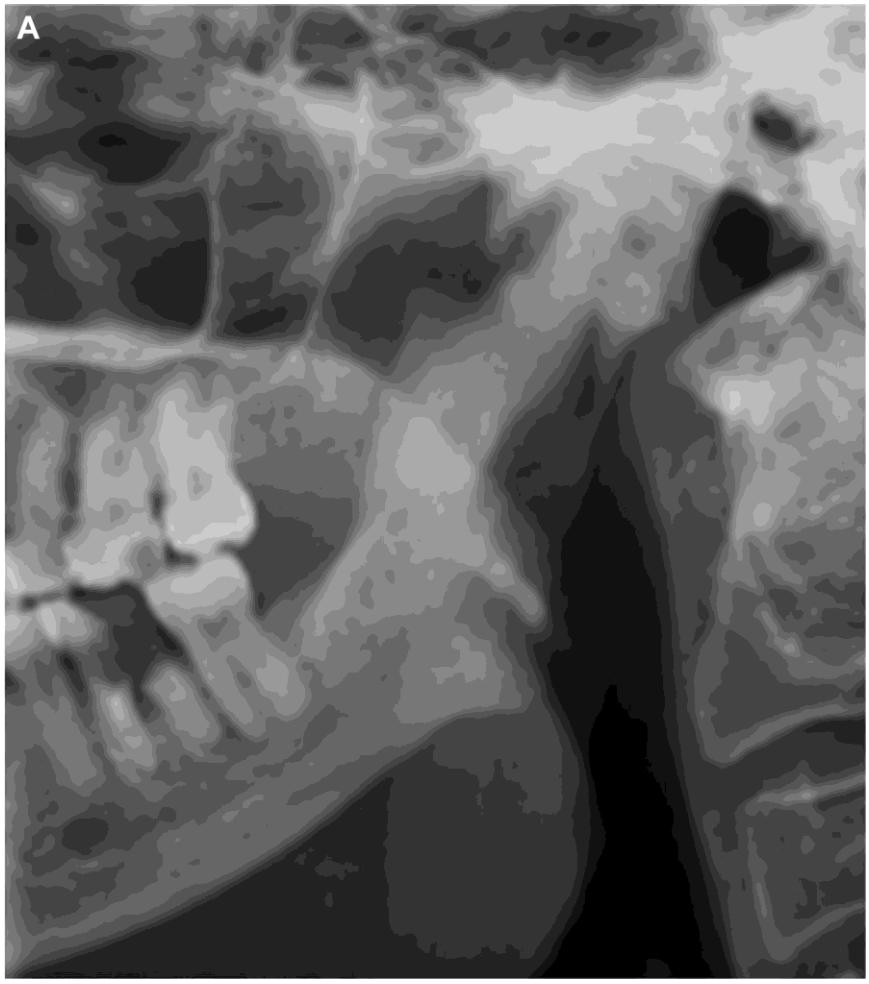

FIGURE 3. *A*, Bony ankylosis. (Fig 3 continued on next page.)

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Imaging

- Screen for loss of joint space and heterotopic bone on Panorex or CBCT (Fig 3A)
- Define extent of ankylosis/heterotopic bone with CT or CTA depending on concern for vascular structures adjacent to or within ankylotic mass. (Fig 3B and C)

Treatment Choices

- Gap arthroplasty with stock or custom TJR
- Arthroplasty with interpositional material

4. HYPERMOBILITY

- Subluxation
- Dislocation
- Ehlers Danlos Syndrome

History

- Acute episodes of open locking that are able to be self-reduced (subluxation) or require closed reduction (dislocation)
- Apertognathia and malocclusion during episodes
- May be isolated, recurrent or chronic
- Pain may be present, particularly with dislocation
- Medical history of Ehlers Danlos based on genetic testing or history of hyperflexible joints, multiple joint dislocations, loose skin, poly articular osteoarthritis, valvular heart disease or aortic dissection.

Examination

- Anterior open bite and pseudo class III
- Pre-tragal hollowing

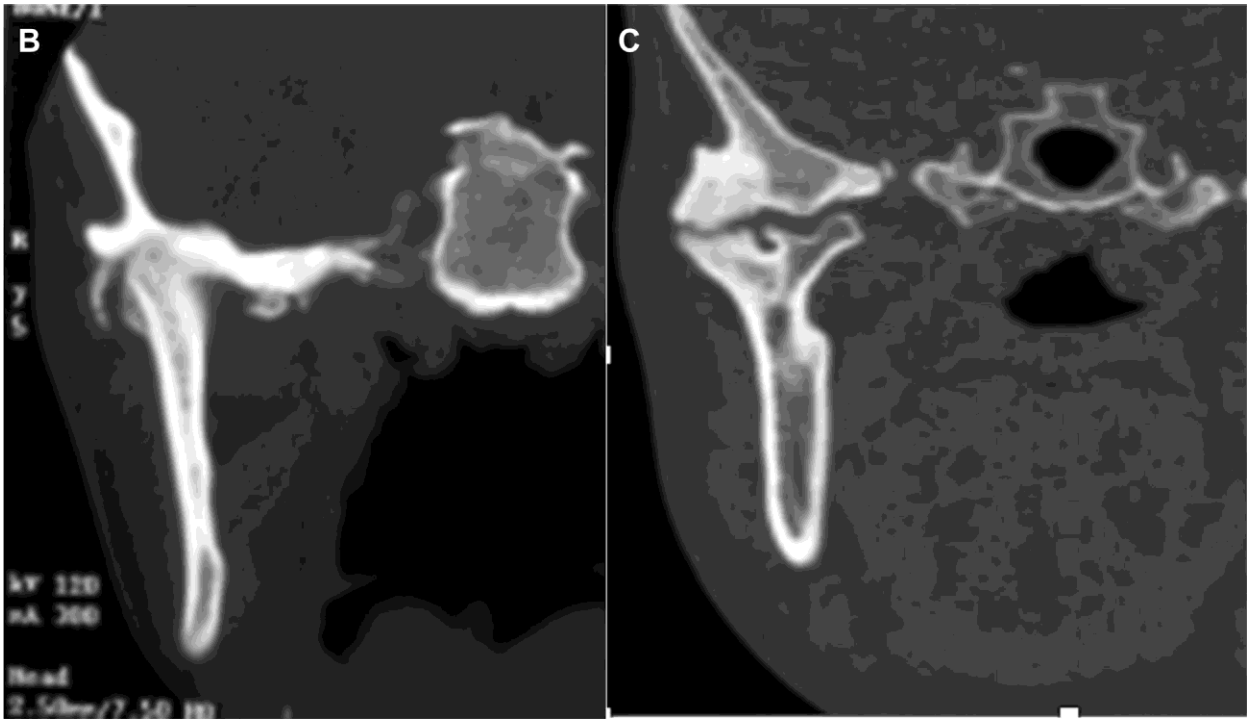

FIGURE 3 (cont'd). *B*, and *C*, Bony ankylosis.

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- Minimal or absent lateral excursions and protrusion

Imaging

- Dislocation can be easily identified with panoramic, CBCT or CT scan imaging (Fig 4A and B).
- Subluxation is best appreciated with MRI which can demonstrate the excessive condyle and disc translation (Fig 5)

Treatment Choices

- Acute dislocation requires closed reduction
- Chronic dislocation considerations
  - I Open reduction (± eminectomy) and class III elastic MMF to correct occlusion and allow the condyle(s) to move posteriorly over time to obtain centric relation
  - II Condylectomy and TJR
- Recurrent dislocation
  - I Intra-articular autologous blood injection and Botox injection to Lateral Pterygoid muscle
  - II Prolotherapy and Botox injection to Lateral Pterygoid muscle
  - III Le Clerc (Dautrey) procedure
  - IV Eminectomy
  - V Custom Total Joint Replacement with large anterior and posterior fossa flanges
  - VI Condylar suspension to post-glenoid tubercle (anchor or MMF screws)

5. INFLAMMATORY ARTHROPATHY

- Rheumatoid arthritis (RhA)
- Systemic Lupus Erythematosus (SLE)
- Psoriatic Arthritis (PA)
- Ankylosing Spondylitis (AS)

History

- History of RhA, SLE, PA or AS
- Other joints may also exhibit swelling, pain and stiffness
- Fatigue, malaise, anemia, rash
- May also report dry eyes, dry mouth, chest pain, hair loss and swollen glands
- Pain with jaw opening, biting and chewing
- Morning symptoms are typically worse
- Decreased MIO or no jaw opening

Examination

- Swollen and tender meta-carpo-phalangeal and proximal interphalangeal joints
- Cutaneous and facial rash
- Subcutaneous nodules

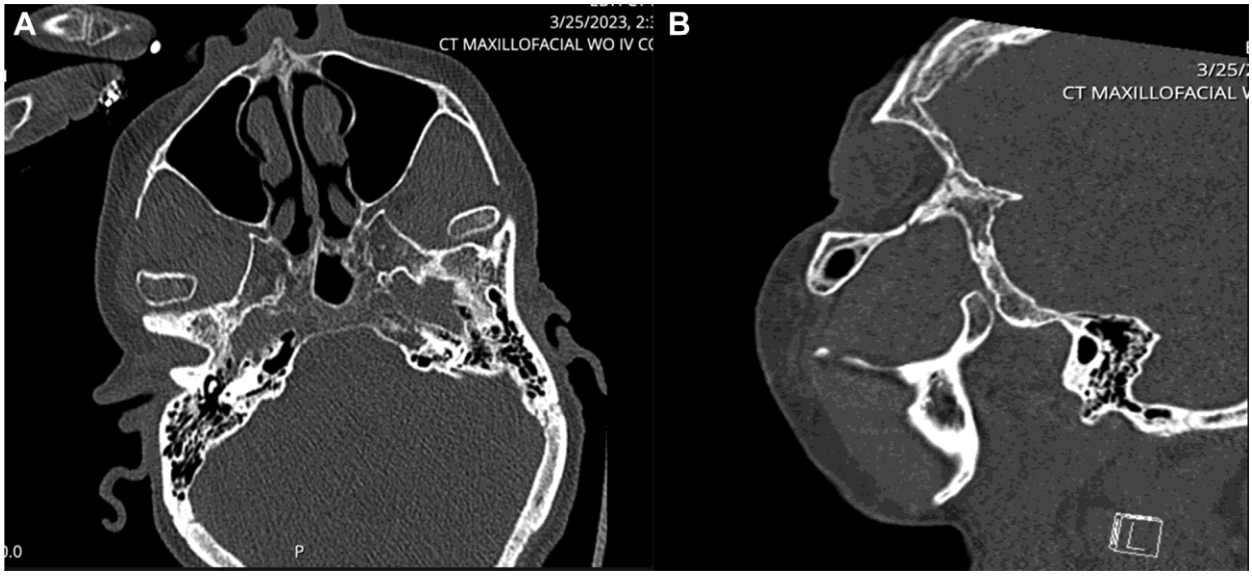

FIGURE 4. *A*, and *B*, Dislocation.

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- Photosensitivity
- Conjunctival injection, xerophthalmia and xerostomia
- Oral ulceration or erythroplakia
- Lymphadenopathy
- Raynaud's phenomenon
- Kyphosis
- Endaural or lateral capsular pain
- Pain, clicking, crepitus, deviation or limitation with protrusion, lateral excursions or opening

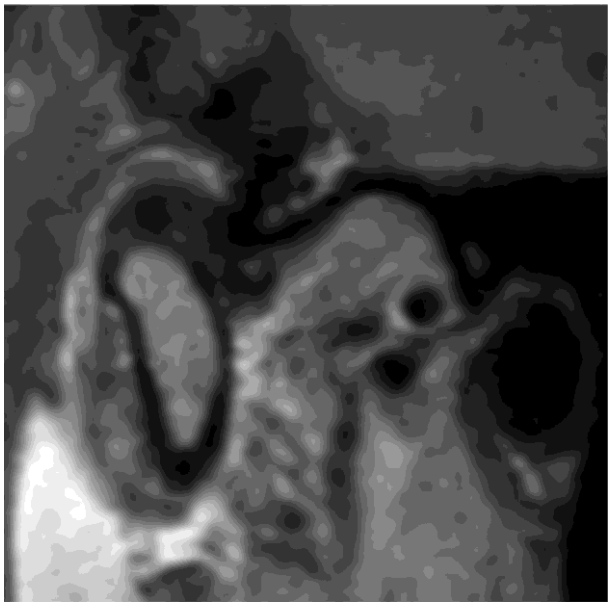

FIGURE 5. Subluxation.

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- Limited MIO without and with pain
- Positive Mahan test
- Altered occlusion
- Minimal or absent lateral excursions and protrusion

Imaging

- CBCT imaging is an excellent screening modality to assess for altered joint space or osteoarthritis. Panoramic imaging is not reliable for assessing joint space.
- MRI remains the best modality to identify internal derangement, pannus formation, effusion and osteoarthritis (Fig 6).
- CT imaging is most suited to identify osteoarthritis and the potential for bony ankylosis seen in AS.

Serology

- C Reactive Protein (CRP), Elevated Sedimentation Rate (ESR), Rheumatoid factor, anti-cyclic citrullinated peptide (anti-CCP) for RA
- CRP, ESR, Anti-nuclear antibody (ANA), Anti Double stranded DNA for SLE
- CRP, ESR and HLA-B27 gene for AS

Treatment Choices

- Non-surgical management with soft diet, jaw rest and heat

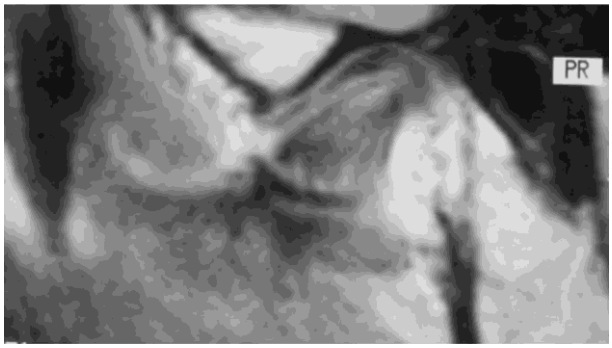

FIGURE 6. Inflammatory pannus.

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- Medical management with NSAIDs, systemic or intra-articular steroids
- Disease Modifying Anti-Rheumatic Drugs (DMARDS) such as methotrexate, leflunomide, sulfasalazine and apremilast
- Biologic drugs targeting TNF-alpha inhibitors, Interleukin 12, 17 or 23 inhibitors and T cell inhibitors
- Arthrocentesis
- Arthroscopy
- Arthroplasty
- Gap arthroplasty
- Total Joint Replacement

6. METABOLIC/INFLAMMATORY/INFECTIOUS

- Gout
- Pseudogout
- Reactive arthritis
- Lyme disease
- Chlamydia
- Gonococcal
- Osteomyelitis
- SAPHO (synovitis, acne, pustules, hyperostosis and osteitis)

History

- Prior diagnosis of one of the diseases
- Swelling and pain in other joints
- Discomfort during urination and sore eyes
- Muscle pain
- Fatigue, malaise and a rash
- Recent facial droop or cardiac arrhythmia
- Pelvic pain
- Vaginal or urethral discharge
- Recent onset of acne
- Pain with jaw opening, biting and chewing
- Jaw Pain

Examination

- Swollen and tender joints including the TMJ
- Cutaneous Rash
- Conjunctivitis
- Facial palsy and/or cardiac arrhythmia
- Urethral or vaginal discharge
- Acne and cutaneous pustules
- Oro-Cutaneous Fistula
- Cutaneous Fistula
- Exposed intraoral bone and purulence
- Tooth mobility
- Endaural or lateral capsular pain
- Pain, clicking, crepitus, deviation or limitation with protrusion
- Pain, clicking, crepitus or deviation or limitation with lateral excursions
- Pain, clicking, crepitus or deviation or limitation with jaw opening
- Limited MIO without and with pain
- Positive Mahan test

Imaging

- Panoramic, CBCT or CT imaging are ideal modalities to identify a reduced joint space, osteoarthritis, osteolysis, punctate calcification or sclerosis associated with most conditions. (Fig 7A).
- MRI remains a more robust choice when concern exists for effusion, limited translation, or altered marrow signal intensity.
- CT with contrast imaging is ideal for identifying the mixed lytic and sclerotic bone changes, sequestra, laminated periosteal new bone formation and soft tissue involvement formation associated with osteomyelitis. MRI with or without gadolinium is also a very sensitive modality that can identify marrow changes that precede the development of the traditional mixed lytic/sclerotic picture. MRI is also helpful when there is soft tissue involvement. On occasion both CT and MRI are needed to define the extent of the lesion (Fig 7B and C). A Tc-99m MDP bone scan is usually able to detect early lesions before radiographic evidence is seen although this modality is not specific. It is particularly useful if multifocal disease is suspected such as SAPHO (synovitis, acne, pustules, hyperostosis and osteitis).

Lab Tests

- CRP, ESR, Uric acid for Gout
- CRP, ESR, calcium, phosphate for pseudogout

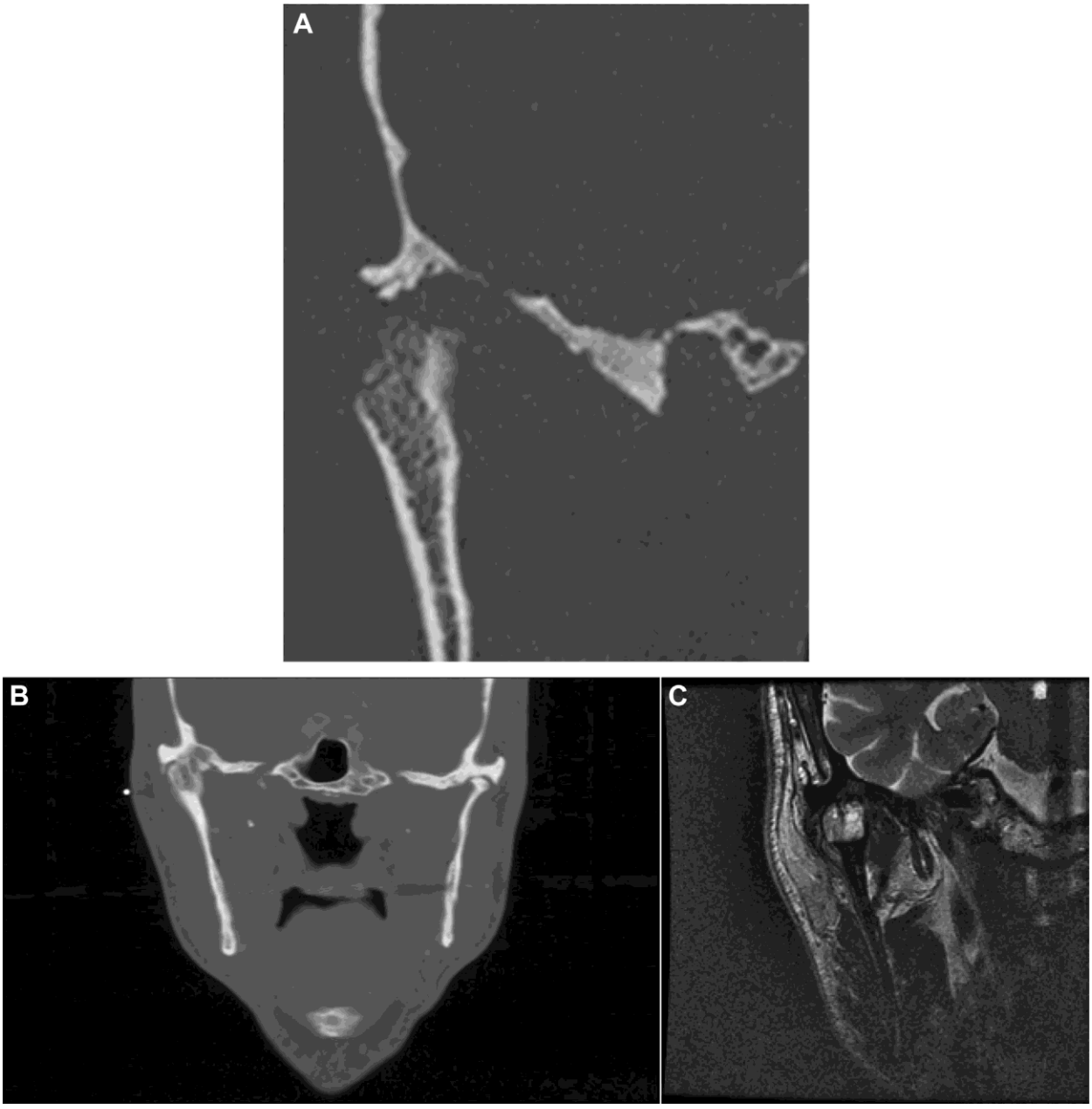

FIGURE 7. *A*, Osteomyelitis, *B*, and *C*, Pseudogout.

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- Urethral or vaginal swab, or mid-stream urine for chlamydia and gonorrhea
- Lyme disease antibody test

Treatment Choices

- Gout: colchicine, NSAIDs, allopurinol, probenecid, steroids
- Pseudogout: colchicine, NSAIDs, steroids and arthrocentesis
- Reactive arthritis Chlamydia, Salmonella, Shigella, Yersinia, Campylobacter): NSAIDs, DMARDs, steroids and antibiotics (doxycycline, azithromycin for Chlamydia)
- Lyme disease: antibiotics (doxycycline, amoxicillin or cefuroxime)
- Osteomyelitis

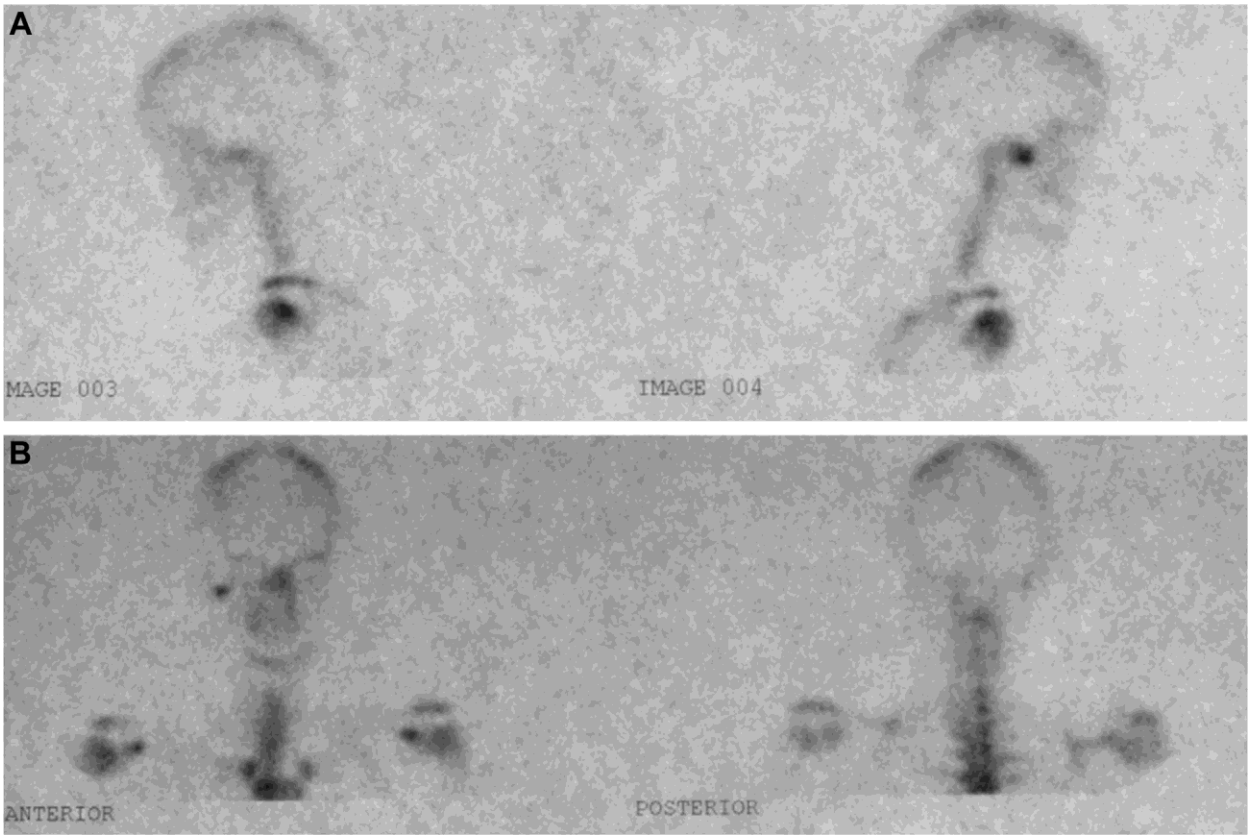

FIGURE 8. *A*, and B, Condylar hyperplasia.

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1 Antibiotics (Oral, IV and PICC)
2 Debridement or Marginal Resection
3 Segmental resection and reconstruction with titanium plate ± vascularized bone/soft tissue graft and/or pedicled flap

- SAPHO
  1 NSAIDs, colchicine, steroids
  2 DMARDs
  3 Antibiotics (clindamycin)
  4 Bisphosphonates
  5 Biologics

7. DISORDERS OF CONDYLAR DEVELOPMENT/ GROWTH/RESORPTION

- Goldenhar syndrome
- Hemifacial microsomia
- Treacher Collins syndrome
- Condylar hyperplasia
- Idiopathic condylar resorption

History

- Congenital defect of the ear, orbit, mandible, soft tissue and cranial nerves
- Progressive loss of facial symmetry and malocclusion
- Pain with biting, chewing and jaw opening
- Development of class II facial profile, steep mandibular plane angle and open bite

Examination

- Scoliosis, epibulbar dermoids, microtia, atresia of the EAM, macrosomia, orbital dystopia, deafness, skin tags.
- Internal organ abnormalities of the heart, lungs or kidneys.
- Asymmetry of the mandible and chin with ipsilateral open bite and contralateral crossbite
- Mandibular retrusion with apertognathia, steep mandibular plane angle and class II dental relationship
- Endaural or lateral capsular pain
- Pain, clicking, crepitus, deviation or limitation with protrusion
- Pain, clicking, crepitus or deviation or limitation with lateral excursions

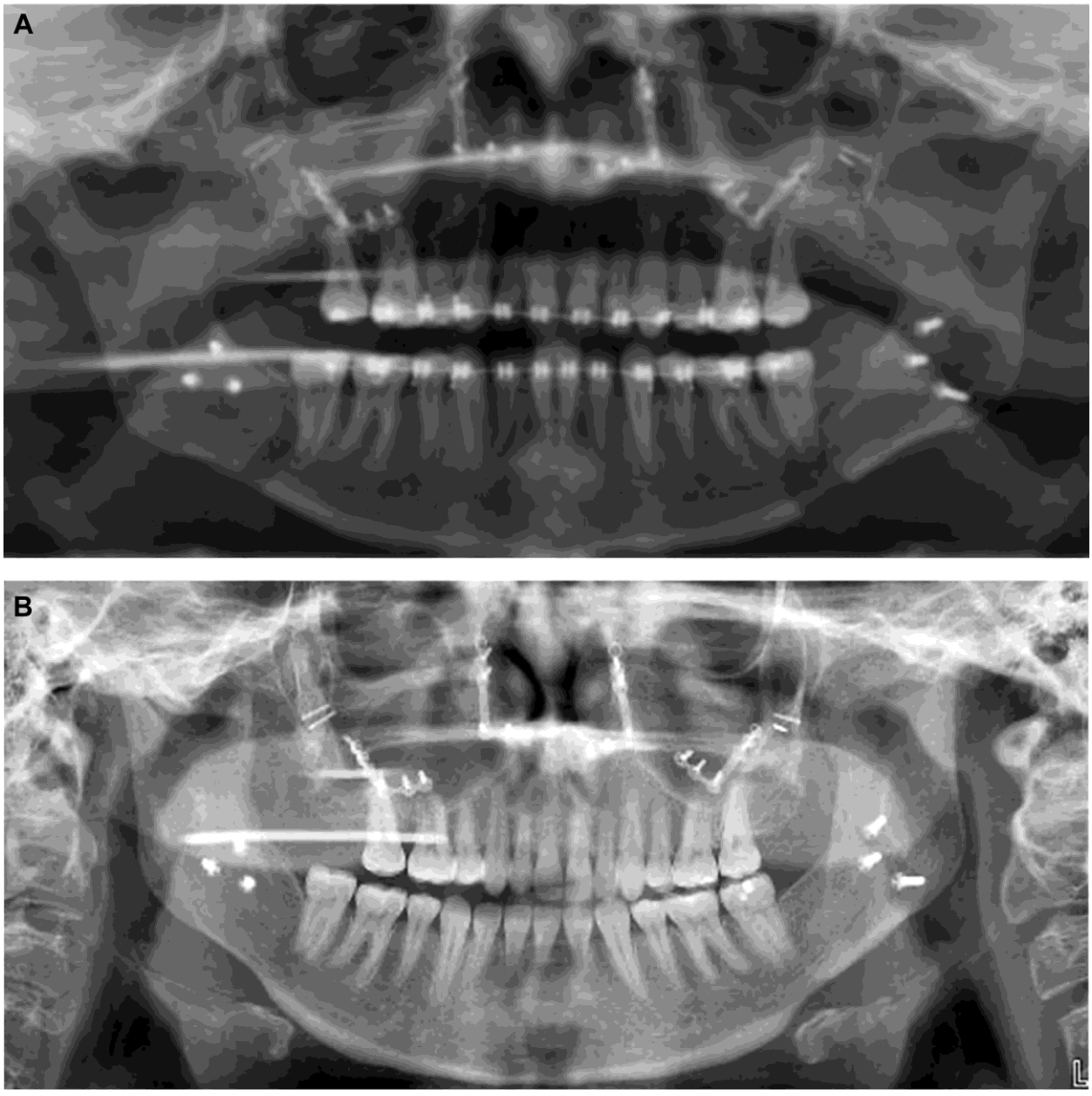

FIGURE 9. *A*, Normal condyle appearance after BSSO. *B*, ICR after BSSO.

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- Pain, clicking, crepitus or deviation or limitation with jaw opening
- Limited MIO without and with pain

Imaging

Goldenhar Syndrome (Hemifacial Microsomia or Treacher Collins Syndrome)

- Panoramic, CBCT and CT are ideal modalities to identify bony anatomy although CT imaging provides the opportunity to appreciate soft tissue deficiencies

Condylar Hyperplasia

- Panoramic, lateral and PA cephalometric, CBCT and CT imaging can identify asymmetry between condyles. These imaging modalities often require serial scans to assess for active condylar growth.
- A Tc-99 MDP/HDP SPECT bone scan remains the most sensitive modality and can recognize asymmetric uptake in tracer that suggests active asymmetric growth (Fig 8A and B). Non growing condyles with appreciable differences in 3D volume may result in a false positive scan.

Idiopathic Condylar Resorption (ICR)

- Panoramic, lateral cephalometric, CBCT and CT can identify condylar resorption and asymmetry (Fig 9A and B).
- MRI is helpful in identifying internal derangement and concomitant degenerative joint disease
- Tc 99 MDP/HDP can also identify condylar resorption, but the specificity is relatively low.

Treatment Choices

- Goldenhar Syndrome, Hemifacial Microsomia or Treacher Collins syndrome:
  1 TMJ: Autogenous Costochondral graft/joint reconstruction or Custom TJR
  2 Mandibular ± maxillary asymmetry: Isolated Orthognathic surgery or in combination with TMJ reconstruction
  3 Ear: Otoplasty with cartilage graft or implant retained auricular prosthesis

- Condylar Hyperplasia:
  1 Isolated orthognathic surgery if growth has ceased on serial examination, CT imaging or Technetium 99 scintigraphy
  2 High proportional condylar shave and simultaneous or delayed orthognathic surgery
  3 Condylectomy and TJR ± orthognathic surgery
- Condylar resorption
  1 Isolated orthognathic surgery if degeneration has ceased on serial examination, CT imaging or Technetium 99 MDP scintigraphy
  2 Condylectomy and TJR ± orthognathic surgery

8. SYNOVIAL DISORDERS

- Synovitis
- Synovial chondromatosis
- Pigmented Villonodular synovitis
- Tophaceous Pseudogout

History

- Pain with the first bite, subsequent bites and jaw opening
- History of Joint sounds including clicking and crepitus
- Limiting diet to softer foods
- History of jaw locking
- Limited jaw opening

Examination

- Endaural or lateral capsular pain on palpation
- Pain, clicking, crepitus, deviation or limitation with protrusion

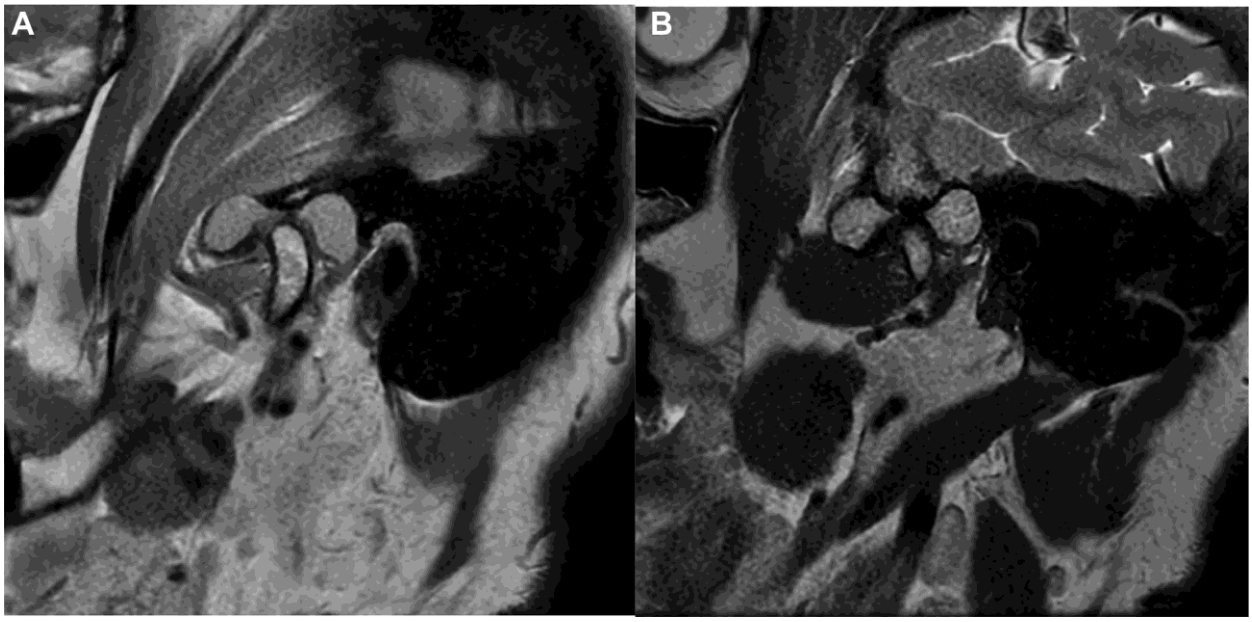

FIGURE 10. *A*, and *B*, Synovial chondromatosis.

- Pain, clicking, crepitus or deviation or limitation with lateral excursions
- Pain, clicking, crepitus or deviation or limitation with jaw opening
- Limited MIO without and with pain
- Positive Mahan test
- Altered occlusion

Imaging

- MRI ± gadolinium is the most ideal modality to assess for the presence of synovitis or synovial chondromatosis. The latter is characterized by an effusion with filling defects that correspond to the cartilage deposits (Fig 10A and B).
- Pigmented villonodular synovitis is best identified with MRI (Fig 11A and B) although the destructive nature of the disease mandates a CT scan to evaluate bony destruction and extension through the ear canal or middle cranial fossa (Fig 11C and D).

Treatment Choices

- Synovitis

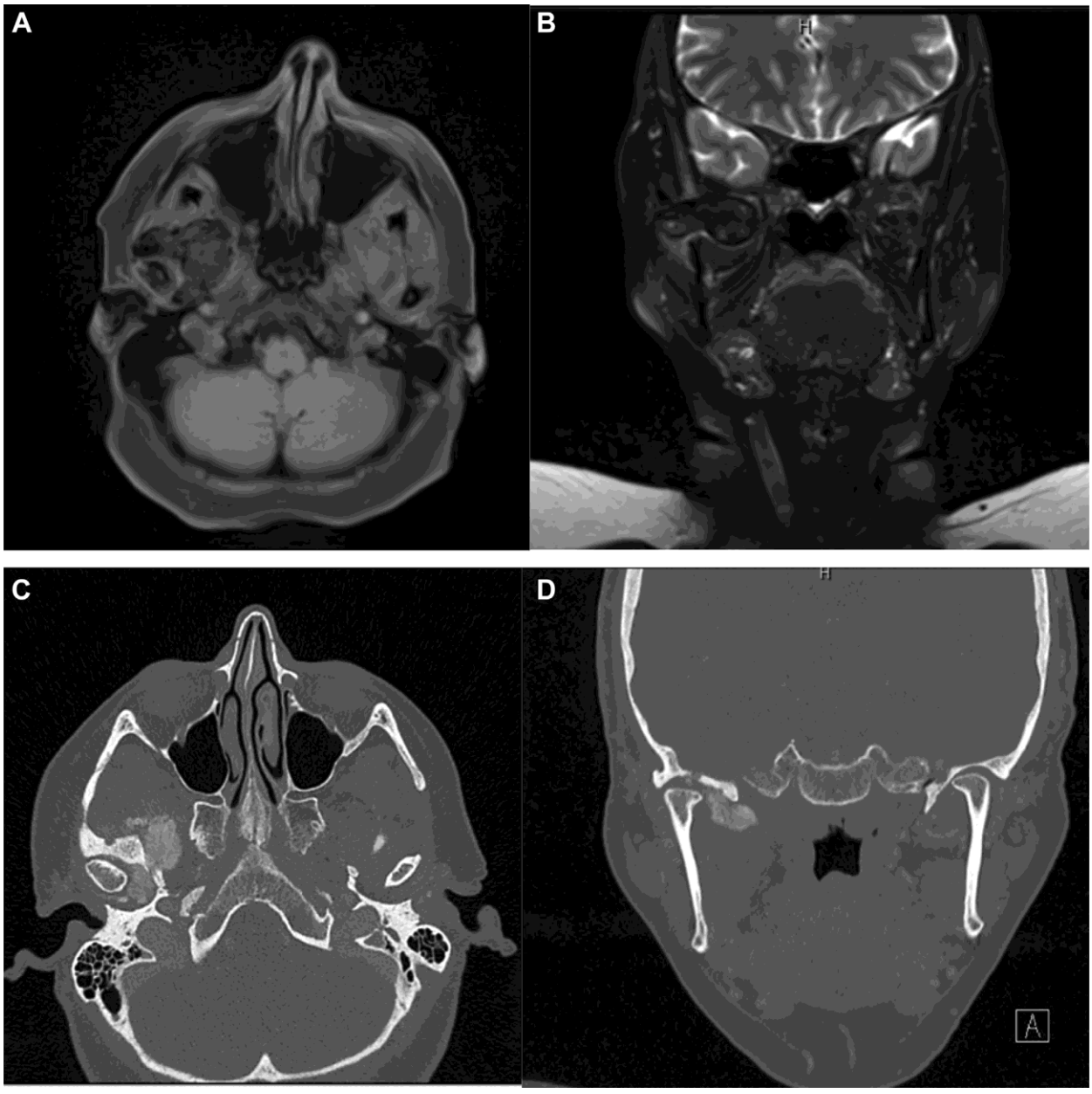

FIGURE 11. *A*, and *B*, Pigmented villonodular synovitis (MRI). *C*, and *D*, Pigmented villonodular synovitis (CT).

1 Non-Steroidal Anti-Inflammatory Drugs
2 Systemic steroids (oral, IM. IV)
3 Intra Articular steroid injection
4 Subsynovial steroid injection
5 Disease Modifying Anti Rheumatic Drugs
6 Biologic Drugs
7 Arthrocentesis
8 Arthroscopy

- Synovial Chondromatosis or Tophaceous Pseudogout
  1 Arthroscopy and limited synovectomy
  2 Arthrotomy and limited synovectomy
- Pigmented Villonodular Synovitis
  1 Arthrotomy with enucleation of the PVNS
  2 Arthroplasty with autogenous reconstruction
  3 Arthrotomy, condylectomy and TJR
- Synovial Plica and pseudo walls
  1 Arthrocentesis
  2 Arthroscopy

9. CYSTS AND TUMORS

a) Synovial cyst
b) Ganglion cyst
c) Other cysts
d) Tumors

Benign

i Osteochondroma
ii Chondroma
iii Chondroblastoma
iv Osteoma
v Osteoid osteoma
vi Osteoblastoma
vii Giant cell tumor
viii Hemangioma
ix Lipoma
x Ossifying fibroma
xi Juxta articular myxoma

Malignant

xii Chondrosarcoma
xiii Osteosarcoma
xiv Fibrosarcoma
xv Ewing sarcoma
xvi Metastatic

History

- Pain with the first bite, subsequent bites and jaw opening
- History of Joint sounds including clicking and crepitus
- Limiting diet to softer foods
- History of jaw locking
- Limited jaw opening
- Preauricular swelling
- Progressive jaw deviation and/or asymmetry
- Malocclusion

Examination

- Preauricular swelling
- Mandibular asymmetry
- Malocclusion
- Endaural or lateral capsular pain
- Pain, clicking, crepitus, deviation or limitation with protrusion
- Pain, clicking, crepitus or deviation or limitation with lateral excursions
- Pain, clicking, crepitus or deviation or limitation with jaw opening
- Limited MIO without and with pain
- Positive Mahan test

Imaging

- Panoramic and CBCT imaging are reasonable screening tools.
- CT scan without contrast remains the most ideal imaging modality for most conditions (Fig 12A and B).
- Vascular lesions are best initially imaged with CT angiography. Angiography ± embolization may be necessary particularly for arterial, high flow and large vascular malformations
- 18^{F} Fluorodeoxyglucose PET/CT scan is ideal for malignant tumors to identify locoregional spread and metastases.

Treatment Choices

- Cysts and Tumors (benign)
  1 Core biopsy or Arthrotomy with biopsy
  2 Arthrotomy with enucleation
  2 Arthrotomy with resection
  3 Arthrotomy with resection and autogenous reconstruction or TJR
- Tumors (malignant)
  1 Core biopsy or arthrotomy with biopsy
  2 Chemotherapy
  2 Arthrotomy with resection and autogenous reconstruction or TJR

10. FRACTURES

History

- Pain with the first bite, subsequent bites and jaw opening

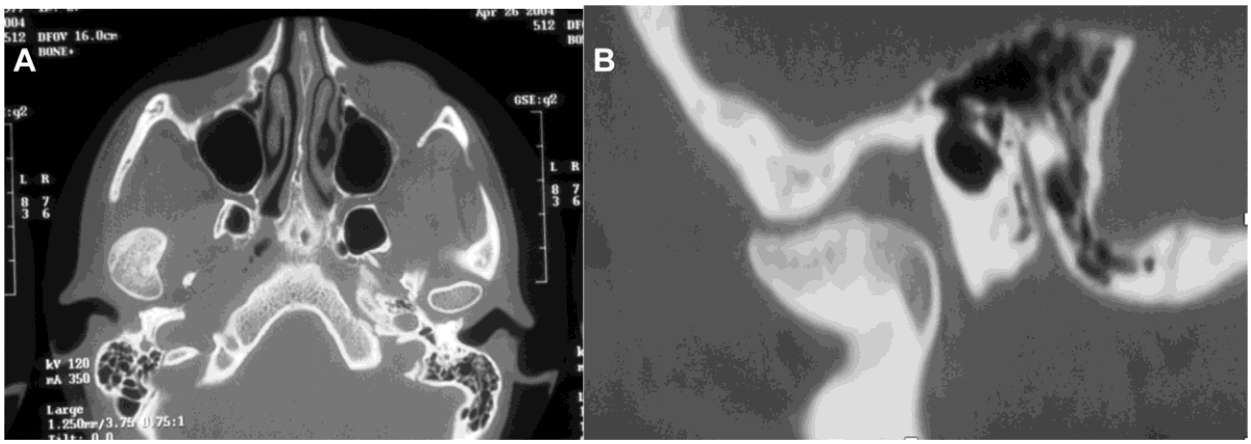

FIGURE 12. *A*, and *B*, Osteochondroma.

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- Limited jaw opening
- Preauricular swelling
- Jaw deviation and/or asymmetry
- Malocclusion

Examination

- Potential lacerations of the external auditory canal
- Preauricular swelling
- Mandibular asymmetry
- Malocclusion
- Endaural or lateral capsular pain
- Limited MIO without and with pain

Imaging

- Panoramic imaging provides a reasonable screening tool to identify condylar fractures (Fig 13A and B)
- CBCT or CT imaging is the modality of choice to identify all fractures, including intracapsular fractures. These modalities also provide additional information about the vector and degree of displacement particularly when contemplating open reduction and internal fixation (Fig 14A-D).

Treatment Choices

The decision to proceed with non-surgical, Closed Reduction (CR) or Open Reduction and Internal Fixation (ORIF) is based on many factors including the type of fracture, unilateral versus bilateral, concomitant facial fractures, patient age, patient medical history and surgeons' ability. General comments that can be made

- Pediatric fractures are often able to undergo restitutional remodeling and do not require ORIF
- Minimally displaced fractures in adults (condyle remains within the glenoid fossa) may be able to be treated with nonsurgical methods or CR.
- Displaced fractures in adults (condyle is not within the glenoid fossa) may often benefit from ORIF
- ORIF is more likely to result in reduced long term TMJ pain when compared to CR

The following treatment strategies should be considered.

a) Non-surgical treatment if the occlusion is unchanged, stable and reproducible.

b) Closed Reduction (CR) with elastic or wire MMF to correct the occlusion.

The choice between elastic and wire MMF will be influenced by the presence of other fractures, the location and displacement of the fracture (Intraarticular/intracapsular vs extracapsular), presence of bilateral condylar fractures, ability to maintain occlusion, the patient's age and medical history.

c) Open Reduction with Internal Fixation (ORIF) with or without elastic or wire MMF.

Consider ORIF when the condyle is displaced into the EAM or MCF, there is loss of posterior facial height (condyle displaced from the glenoid fossa), satisfactory occlusion cannot be established with CR, bilateral displaced condyle fractures are present, or the patient cannot undergo MMF due to medical reasons.

- Fractures in pediatric patients (< age 10 years) should be treated either non-surgically or with CR due to the potential for restitutional remodeling of the fractured condyle.
- Fractures in adolescent and teenage patients (10-19) are more controversial and the ideal treatment less clear. Advancing age decreases the likelihood of restitutional remodeling such that ORIF should be considered for those patients in the mid and late teens.

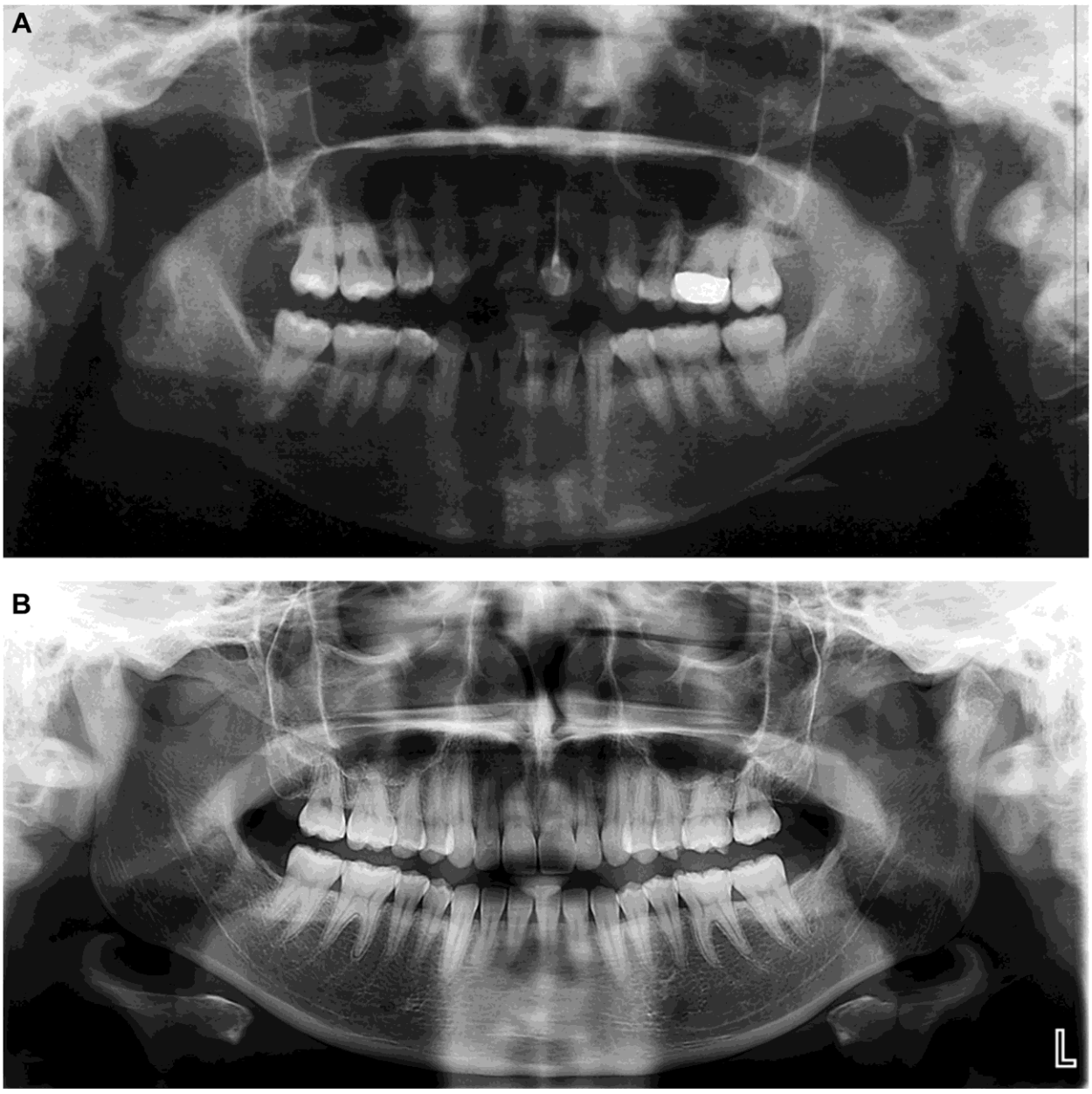

FIGURE 13. *A*, and *B*, Condyle fracture.

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Chapter 6: Non-surgical Management Strategies for TMJ disorders

Objectives:

- To appreciate the various non-surgical approaches to treating cervical pain, IPD and myofascial pain
- To appreciate the role of self-care, physical therapy, orthotics, medications and botulinum toxin

GENERAL CONSIDERATIONS

Temporomandibular disorders (TMD) are defined as a group of musculoskeletal and neuromuscular conditions that involve the TMJs, the masticatory muscles, and all associated tissues.[1] Commonly, but mistakenly, practitioners refer to TMD as a singular disorder despite the fact that patients often present with various and multiple sub-diagnoses which may or may not be painful.[1,7,8] TMD is a relatively prevalent disorder most commonly observed in individuals

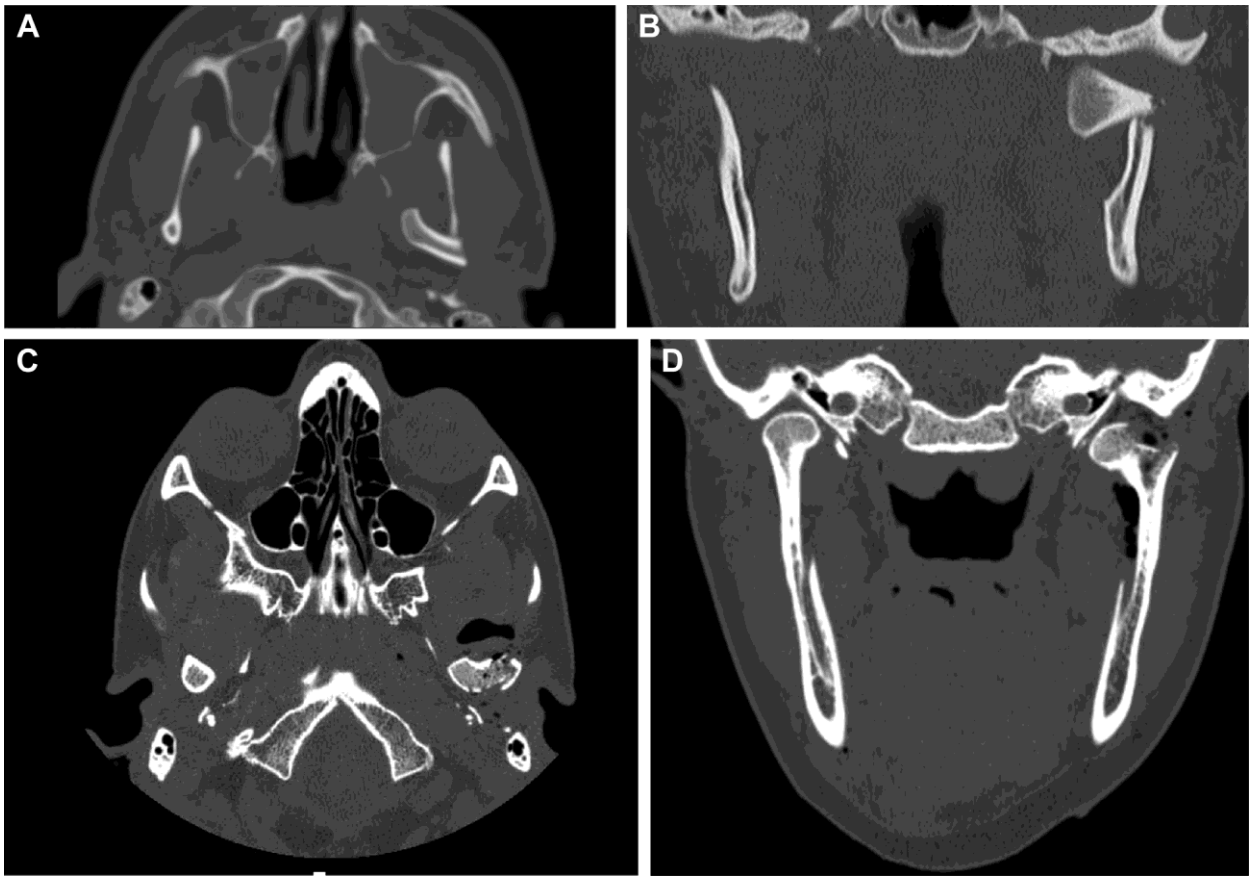

FIGURE 14. *A*, and *B*, Extracapsular left condyle fracture. *C*, and *D*, Intracapsular left condyle fracture.

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between the ages of 20 and 40 years. Approximately 33% of the population has at least one TMD symptom and 3.6 to 7% of the population has TMD with sufficient severity to cause them to seek treatment.[9] It is important to remember that temporomandibular joint disorders (TMJD) are a component of TMD, hence what relates to TMD may impact upon TMJD as well. Furthermore, not all TMJD are painful or need any intervention. Also noteworthy is the bidirectional impact of contraction (bite) forces associated with the masticatory muscles and temporomandibular joint (TMJ) loading forces. TMJD are categorized similar to other musculoskeletal conditions and follow orthopedic principles common to all other joints in the human body. Based upon a systematic review of patient populations using the Diagnostic Criteria for TMD, it was reported that 41% of this population had a disc displacement disorder and 30% were determined to have joint pain disorders.[10] Currently, TMD is considered as a multi-etiological condition hence, interventional strategies, especially in recent decades, have leaned toward multi-modal as well as multi-disciplinary management, in line with that of other chronic musculoskeletal conditions.[11,12] Such strategies often suggest the use of conservative, non-invasive and reversible interventions, which follow supportive and at times, definitive therapy principles. These various therapies often applied singularly or in combination are mainly represented firstly by educating and reassuring the patient and often followed by application of physical self-regulation (PSR) and/or self-management (SM) approaches. Additionally, involvement of additional measures such as psychotherapy, biofeedback, cognitive and behavioral therapies; physical therapy (utilizing exercises, mobilization and various electro-medical therapies); and complementary and alternative medicine therapies (chiropractic, osteopathy, massage, relaxation therapy, acupuncture and others) are often a consideration.

SUPPORTIVE THERAPY

Supportive therapy can be considered any form of treatment provided to a patient to prevent, control, or relieve complications and side effects and to

improve the patient's comfort and quality of life. The intent of this therapy is to relieve symptoms or help the patient live with them rather than attempt changes in character structure. Therapy is aimed toward altering the patient's symptoms rather than having a direct effect on the cause of the disorder. The first step in establishing supportive therapy is education/reassurance followed by PSR and/or SM and if needed, adjunctive measures.

a. Education/reassurance

Education pertains to an evidence-based discussion and explanation of diagnosis and generally favorable prognosis ('optimistic counseling'), when appropriate, and it includes reassurance that TMD is typically a benign group of conditions and self-limiting in the vast majority of cases, but with the potential for fluctuation in symptomatology. This education should also include information related to the patient's role in their treatment as well as the availability of various interventional options and a conversation related to the patient's expectations. An essential component of patient education is an understanding of the concept of a biopsychosocial model toward pain rather than a purely biomedical model and the importance of behavior modification related to the elimination or control of harmful oral behaviors.[13] It is important to realize that harmful oral behaviors may predispose, precipitate or perpetuate painful intra-articular conditions. Changing behavior is not a simple process as it relies not only on patient education but also involves the willingness and cooperation of the patient. Hence, a passive approach of simply providing the patient with informational sheets of instructions may be less than efficacious. The clinician may have to invest some time to educate or re-educate their patients on the importance of these changes or identify obstacles that may interfere with their ability to change behavior and then provide suggestions regarding various strategies for resolution.

Other areas of education where basic information should be included are as follows: sleep practices; sensible and time-limited use of analgesia; avoidance of over the counter/online purchased oral appliances (OA) without consultation involving their dentist; caffeine and or high energy drink consumption; 'doctor shopping'; and an understanding of normal anatomy and function of the TMJ complex and associated musculature. For example, making the patient aware of the masticatory anatomy and why it is painful; a discussion of the presence of intracapsular sounds and why it may or may not be painful while advising patients to avoid becoming hypervigilant or playing with their mandible to constantly check for or reproduce the various intracapsular sounds and often annoying noises.

b. Physical self-regulation

Basically, PSR engages the patient by having them learn how to better manage their personal and environment stressors and/or in the learning of coping skills or methods to enhance control of physical functioning in order to reduce dysfunction and/or dysregulation with the return to homeostasis. There are reports that depression, fatigue, and anxiety characterize the psychologic domain of persons with TMD.[14] Physiologically, it has been shown these individuals over-respond to environmental stimuli with excessive cardiovascular activity and altered breathing rates.[15,16] It has also been demonstrated that persons with TMD report significantly more life stressors than do individuals who are pain-free.[17,18] Taken together, these findings suggest that factors associated with the level of emotional and physical activation may contribute to the chronicity of TMD. While increased autonomic activation is a normal adaptive mechanism for managing life stressors, heightened emotional and physical responsivity is also characteristic of a chronic defense reaction in the presence of relentless stressors. Prolonged stimulation from nociception, for example, is known to be one of the most significant activators of the sympathetic nervous system and can be viewed as an important endogenous stressor itself. Even in non-painful situations, anxiety-induced autonomic activity that alters carbon dioxide levels may cause ectopic impulses to be generated from dense receptive fields within the trigeminal region.[19] Under conditions promoting central sensitization, sympathetic activity from a variety of stimuli may have significant effects on nociceptive transmission or subsequent pain reports. Therefore, management of sympathetic activity can be regarded as an important treatment goal for persons with pain disorders, even though it may not be clear as to whether it is a causative factor or a consequence of the pain experience.

Procedures involved in deactivating an upregulated sympathetic nervous system may be as simple as identifying areas where the patient is performing activities detrimental to the recovery of the stomatognathic system or as complex in the reestablishment of nasal (as opposed to oral) breathing and entrainment of diaphragmatic as compared to chest breathing.

c. Self-management

SM, as the name implies, is what the patient can do for themselves to improve their situation. Since TMD involves multidimensional effects, especially as pain becomes chronic, SM training about behaviors, strategies, and activities may assist in the control of the destructive effects of pain on quality of life. Although SM programs can differ as to interventions, shared features usually include employing strategies to reduce pain by altering individuals' behavioral, cognitive and/or emotional responses to pain and enhancing their self-efficacy for managing pain.[20] SM clinical protocols are commonly used as initial treatment for patients with TMD, after adequate diagnosis, as a conservative and non-invasive approach.[21] They should be simple enough to empower the patient to exert control over the execution of the therapies while enabling healing and/or prevention of further injuries to the musculoskeletal system. The main objectives of any treatment for chronic pain consist of reducing pain, restoring function and improving quality of life. Ideally, the approach should be tailored made to the patient's needs and abilities. In certain circumstances, SM may be all that is required to achieve restoration especially in those individuals who are adaptive copers and/or self-motivated or it may form part of a more complex multi-modal treatment plan.

SM should be a core part of TMD management, and it involves the following principles:

- SM is provided to the patient as a first essential step after diagnosis, but which can be built upon or modified as necessary over time.
- SM is part of a continuous management strategy with the intent the patient will use it as needed throughout their lifetime.
- SM should be delivered verbally and supported with written information/instructions and should be appropriate for any clinical setting. When doing so, the patient's comprehension level must be considered with all materials provided in an easy to follow and understandable format while being language appropriate.
- The decision over who delivers SM resides with the presiding clinician and the particular characteristics of the clinical facility where the patient is being managed. As there is a therapeutic nature to the doctor/patient relationship (therapeutic alliance), there are different models of provision. SM does not necessarily need to be delivered by the clinician but may be delivered by an appropriately trained team member.
- SM contains context-dependent education, including explanation, advice and reassurance (eg, optimistic counseling). Such education can be provided, at least in part, even if a definitive diagnosis is not reached, and can include generic information on the nature of persistent pain and pain related TMD, subject to no 'red flags' being present, such as sensory or motor function changes.
- SM can be reinforced or reviewed to ensure comprehension and adherence. Follow-up could be in person or accomplished through electronic media. Further research is required to determine the optimum mode and time for review.

The most common groups of SM activities are as follows: self-exercise therapy, thermal modalities, self-massage therapy, diet and nutrition, control of parafunctional behavior and jaw muscle exercises with posture training.

1. Self-exercise therapy

One of the key components of SM is related to self-applied exercises by the patient as compared to manual therapies performed by other healthcare professionals. Exercises can be implemented by the clinician (if they have the appropriate education and training to do so) or by referral to allied healthcare professions such as adequately trained physical or occupational therapists who understand the nuances of TMD.

2. Thermal Modalities

The use of heat and/or ice to areas of pain may be beneficial. Often, depending on the circumstance, alternating their use may be of assistance.

3. Self-Massage therapy

Massage is limited to the anatomic location of the painful or tense affected masticatory muscles (most easily accessible to palpation are the masseter muscle and temporalis muscle) as well as the preauricular region to involve the TMJ.

4. Diet and Nutrition

Specific nutritional advice is available in the Temporomandibular Joint Association's (TMJA) guide to nutrition freely available online.[22] Another aspect of this pertains to chewing restriction, which is to be phrased as a 'pain-free diet', as opposed to a 'soft diet', with the recommendation that it be implemented for a 2-week period, followed by a review which will determine whether the individual advances, as tolerated, to a firmer and tougher consistency of foods.

5. Parafunctional Behavior

This component should orient the patient towards awareness, identification, monitoring, and avoidance of any parafunctional behavior that exacerbates the pain. The therapeutic boundaries of this component in terms of self-identification of behaviors and control via avoidance compared to more formalized cognitive behavioral therapy (CBT) are yet to be determined.

6. Jaw Muscle Relaxation and posture training

The therapeutic self-exercise consists in the prescription of muscular contractions and body movements in order to improve general function and help satisfy the demands of daily life. Relaxation exercises involve movements of opening, laterality, protrusion and closing resistance (with the fist as support, for example). Relaxation exercises emphasizing diaphragmatic (abdomen) breathing by exhaling completely with one hand resting on the chest and the other on the abdomen, training to become aware of the mechanism of deep breathing only by the position and hand movement. Diaphragmatic breathing should be performed for 5 minutes every day, every 2 hours. Notwithstanding, use of this coordinated breathing should be followed as frequently as possible throughout the day, if possible. Stretching exercises, utilizing the activity of pain free jaw opening, may be accomplished by opening the mouth slowly only until there is an initial sensation of pain. Opening slightly further with slight pressure by positioning the thumb on the maxillary arch, approximating the region of premolars, and the index finger on the mandibular arch, also in the region of the premolars. The technique should be performed every 2 hours throughout the day, keeping the elongated jaw in position for one minute, six times consecutively. Coordination exercises consist of repeated opening, laterality, protrusion and mandibular retrusion movements. They must be performed slowly and coordinated, often in front of a mirror for self-monitoring, 3 times per day. Finally, mobilization exercises may be performed by attempting mandibular laterality movements to recapture the disc, if possible. This is followed by gentle protrusion and retrusion movements by holding and sliding on some small object positioned between the teeth.

Posture training has been reported to positively affect TMD.[23,24] The goal of this training involves the prevention of dysfunctional muscle activity related to the head, cervical and shoulder regions in addition to the masticatory and tongue muscles. The aim is to maintain orthostatic posture to prevent increased cervical and shoulder muscle activity and possible protrusion of the mandible. It should be noted that the more anteriorly the head is positioned in relation to the spinal column the greater is its weight thereby placing a greater demand on the supporting structures.

ORTHOTIC APPLIANCES

According to the Glossary of Prosthodontic Terms [GPT-9], an occlusal device (also known as: splint, stent, bite guard) is defined as ''any removable artificial occlusal surface affecting the relationship of the mandible to the maxilla used for diagnosis or therapy; uses of this device may include, but are not limited to, occlusal stabilization for treatment of temporomandibular disorders, diagnostic overlay prior to extensive intervention, radiation therapy, occlusal positioning, and prevention of wear of the dentition or damage to brittle restorative materials such as dental porcelain.'[25] The term ''splint', while often appearing in the dental literature as being synonymous with OA, will not be used, because it has several other definitions in dentistry that are unrelated to the management of TMD.

Essentially, OA are fabricated from basically two different materials (based upon consistency) and/or a hybrid of the two. First, there are hard acrylic resin OA that are either chemically cured, or heat/pressure processed, resulting in hard and rigid tooth-borne and occlusal surfaces. Alternatively, there are soft or resilient OA manufactured from plastics or polymers, producing an appliance which has a somewhat flexible and pliable tooth-borne and occlusal surface. The hybrid version is known as dual laminated, because it consists of hard acrylic resin on the occlusal surface and a soft material on the inner aspect (tooth-borne surface). This produces an OA with the positive qualities of a soft material (fitting well and providing comfort for the supporting teeth), with the versatility of a hard acrylic resin adjustable occlusal surface. There are advantages and disadvantages associated with each type of material and costs vary.[26]

There are many different designs of OA with each one having different claims based upon philosophical beliefs rather than robust scientific evidence as to the reason for their effectiveness. The various appliance designs include flat plane stabilization, traditional anterior bite plane, mini-anterior, anterior repositioning, neuromuscular, posterior bite plane, pivot and hydrostatic.

1. Flat plane stabilization appliance

The flat plane stabilization appliance (also known as the Michigan splint, muscle relaxation appliance, or gnathologic splint) is generally fabricated for the maxillary arch but one fabricated for the mandibular arch may also be considered. Ideally, when a stabilization type of appliance is placed intraorally, there is minimal change to the maxillomandibular relationship other than that produced by the thickness of the material. This is the most commonly used type of intraoral appliance, and when properly fabricated it has the least potential for adverse effects to the oral structures.

2. Traditional anterior bite plane appliance

In general, they are designed as a palatal-coverage horseshoe shape with an occlusal platform covering six or eight maxillary anterior teeth (eg, Hawley, Sved, Shore). They minimize clenching forces because posterior teeth are not engaged in closing or in parafunctional activities. Due to their design (lack of contact among posterior teeth) there is a possibility of over-eruption of posterior teeth, which is extremely unlikely if worn only during sleep. However, due to the TMJ being a Class III lever contact only on the anterior teeth increases loading on the TMJ thereby potentially amplifying an already painful joint disorder.[27,28]

3. Mini-Anterior Appliance

The concept of making an OA that engages only a small number of maxillary anterior teeth (usually two-four incisors) was first introduced in the mid 1900's as the Lucia jig. There have been several variations that have appeared on the market. They include the Nociceptive Trigeminal Inhibition Tension Suppression System (NTI), the Best Bite, and the Anterior Midline Point Stop (AMPS) devices. They are made of hard acrylic and can be fabricated commercially or at chair side. Advocates of this design use this to disengage the posterior teeth. Due to their design, there is the potential for development of an anterior open bite from either over-eruption of the unopposed posterior teeth, intrusion of the maxillary anterior teeth or from the combination of the two.

4. Anterior Repositioning Appliance

The anterior repositioning appliance (also known as an orthopedic repositioning appliance) purposefully alters the maxillomandibular relationship so that the mandible assumes a more anterior position. Originally, this type of OA was supposed to be used to treat patients with internal derangements (usually anterior disk displacements with reduction). Currently, it is recommended that repositioning appliances should be used primarily as a temporary therapeutic measure to allow for symptomatic control of painful internal derangements, but not to permanently recapture the TMJ disk. This type of appliance should be used with discretion, and only for short periods of time.

5. Neuromuscular Appliance

Advocates of so-called neuromuscular dentistry have claimed that the use of electronic instrumentation enables the production of an OA at the ideal vertical and horizontal position of the mandible relative to the cranium. These OA direct the mandible into an anterior position often following 30–45 minute of TENS stimulation. Advocates believe this leads to the mandible being in a directed position where muscular relaxation is verified by the use of surface electromyography recordings from the muscles of mastication. After using these appliances to treat a TMD patient, proponents of this methodology usually recommend dental reconstruction at the newly established jaw relationship.

6. Posterior Bite Plane Appliance

Posterior bite plane appliances (also known as mandibular orthopedic repositioning appliances - MORA) are customarily made to be worn on the mandibular arch. The purpose of this OA is to produce changes to the vertical dimension and alter the horizontal maxillomandibular relationship. The major concern regarding this appliance design is that occlusion only occurs on posterior teeth, thereby allowing for over-eruption of the unopposed anterior teeth and/or intrusion of the opposing posterior teeth, resulting in an iatrogenically created posterior open bite.

7. Pivot Appliance

The pivot appliance is constructed with hard acrylic resin that covers either the maxillary or mandibular arch and incorporates a single posterior occlusal contact in each quadrant. This contact is placed as far posteriorly as possible. The purpose of this design is to reduce intra-articular pressure by condylar distraction as the mandible fulcrums around the pivot, resulting in an unloading of the articular surfaces of the TMJ. This OA was recommended for patients with internal derangements and/or osteoarthritis. Due to design and force vectors created by this OA, a potential adverse effect may be occlusal changes manifesting as a posterior open bite where the pivot was placed.

8. Hydrostatic Appliance

This uniquely designed OA known as the Aqualizer® is a self-adjusting device that has a fluid-filled chamber device that is placed between the maxillary and mandibular posterior teeth. The concept is that the mandible automatically locates its ideal position because the appliance is not influencing the jaw as to where it should rest. It is mainly used in the short term as a temporary appliance.

Since there are a number of different designs, based upon various philosophical beliefs, it is easy to understand as to why the clinician may be confused on what design to use for TMJ disorders. Two major areas of interest regarding the relationship between OA and the TMJ are the effects of an OA on TMJ loading and the influence of an OA on the disc/condyle relationship.

1. Orthotics and TMJ Loading

There exists a belief that an OA can unload the normal pressure existing inside the TMJ. However, due to the physics of the TMJ (Class III lever), it is unable to fulcrum around any point anterior to the

masticatory muscles. However, it is plausible that loading the TMJ could be reduced or redirected by the presence of an OA. This may occur due to a reduction in the amount and intensity of masticatory muscle activity and/or due to the shifting of the condylar loading area to another location (eg, region where there is no inflammation).[29]

2. Orthotics and Intraarticular Pain and Dysfunction

Historically, many clinicians believed that internal derangements, symptomatic or asymptomatic, with or without sounds, were problematic and had the potential to be a precursor to osteoarthritis and chronic painful dysfunction. Hence, some advocated to ''recapture the disk'' to a normal position with some clinicians further recommending major occlusion-changing procedures to ''stabilize the recaptured disk'' in its new anterior position.[30] Over time, it was shown that a more conservative approach (and even untreated) patients would undergo fairly predictable adaptations in the majority of cases.[31] Therefore, this resulted in clinicians treating patients with symptomatic relief from painful episodes, as well as providing proper counseling about their TMJ biomechanics.[1] Overall, there appears to be three indications for the use of OA. First, in those patients with acute arthralgia, an OA may reduce masticatory muscle activity and redirect loading to the TMJ; second, for sleep bruxers who awaken with arthralgia, an OA worn during sleep might be of assistance in reducing pain and dysfunction; and third, for patients whose TMJ become ''locked' during sleep, but who are able to successfully ''click' open while awake, an OA may reduce the frequency of these episodes or prevent their occurrence.

PHARMACOTHERAPIES

The use of pharmacotherapeutics for the management of TMD may be as a monotherapy in some cases but are mostly used in conjunction with other therapeutic options such as behavioral therapy, physical therapy, oral appliance therapy or surgical interventions. Ideally, the role of pharmacotherapy in TMD should be viewed as an adjunct to assist patients in managing their pain, when present, to a point where it decreases to a level at which it no longer restricts their day-to-day endeavors. There is no single pharmacotherapeutic agent or class of agents that has been shown to be efficacious for all patients who suffer with TMD presentations. In fact, current pharmacotherapy recommendations for patients with TMD tends to be mostly empirical. While there are several agents that tend to be commonly prescribed for the treatment of TMD, most lack robust evidence.[32] In light of this, the clinician needs to discern, as best as possible, the etiopathology of the patients presenting pain complaint within the various categories of TMD. With an understanding of the mechanisms involved in the pain presentation, the clinician will be better prepared to choose the most appropriate therapeutic agent. As most pain presentations involving the joint structures will be inflammatory in nature, using agents that either address the inflammation directly or via reducing loading forces on the articulating structures would tend to make the most sense. The clinician should also have a comprehensive knowledge of the therapeutic agents contemplated for use for each pain presentation. This would include the therapeutic agent's mechanisms of action (MOA), any potential adverse events (AEs) and all possible drug to drug interactions. The prescriber should also consider the most appropriate route of drug administration for the specific individual and pain presentation being managed. In most pain presentations, dosing schedules should be advised as opposed to ''as required for'' (PRN) dosing. This protocol has been shown to be more advantageous in preventing persistent and breakthrough pain episodes.[33] A discussion regarding the more common agents as well as some newer pharmacological approaches utilized to manage TMD is presented. The routes of administration will be confined to topical and systemic (oral) preparations.

Topical Formulations

With the growing concern of the adverse risks of medications such as the non-steroidal anti-inflammatory drugs (NSAIDs), non-systemic approaches to pharmacotherapy have become more attractive for clinicians as well as patients. Also, there are patients who may be hesitant or unwilling to take systemic medications that may be more amenable to a topical preparation. Topical medications allow for the distribution of a pharmacologic agent in a very concentrated delivery area. The site of action is the soft tissue and peripheral nerves at the area of application. These applications may be more advantageous for patients already on polypharmacy where there may be concerns for drug-drug interactions. In some cases, topical preparations can be utilized in conjunction with the systemic equivalent of the same agent allowing for the reduction of the systemic delivery dose needed in the hope of reducing the propensity for AEs. Topicals are usually prepared as a cream, ointment, gel or in a patch (with a reservoir design) and must have the ability to penetrate the skin. Factors that may influence the ability of the preparation to penetrate the skin include the integrity of the skin, the patient's age and the presence of any dermatologic disorders or disease. The ability of the preparation to absorb effectively is facilitated by using carrier materials that are highly lipid soluble. Topical preparations should be avoided in patients

with broken skin, skin lesions or atrophy, or who have a known sensitivity to the drug or carrier being used.

a. Non-steroidal anti-inflammatory drugs

Topical agents for pain management typically involve the addition of a local anesthetic agent and/or an analgesic but may include other agents. Topical NSAIDs have been shown to be effective in relieving muscle pain with a low incidence of adverse effects.[34] Topical preparations that include diclofenac, ibuprofen, ketoprofen, piroxicam, and indomethacin have all been shown to be significantly more effective than placebo with subjects reporting at least a 50% decrease in their pain scores for various musculoskeletal pains. Diclofenac in particular has been shown to reach local concentrations adequate enough to inhibit proinflammatory prostaglandin E2 production as well as inhibit the NMDA subtype of the glutamate receptor seen in TMJ nociceptors.[35]

b. Capsaicin

Capsaicin, a derivative of chili pepper, has been shown to be beneficial in the management of osteoarthritis and neuropathic related pains. It is believed to selectively stimulate the unmyelinated C-fiber afferent neurons which results in the release of substance P. Repeated application of capsaicin appears to deplete substance P and potentially other neurotransmitters, from the sensory nerve endings.[36] Over the counter preparations are available in concentrations of 0.025-0.1%. When compounded, a local anesthetic is often added to diminish the burning sensation experienced when applied to the skin. The burning sensation has made placebo-controlled trials a challenge. Recent work has supported capsaicin's analgesic properties in various pain conditions.[37,38]

SYSTEMIC MEDICATIONS

a. Non-steroidal Anti-Inflammatory Drugs

NSAIDs have traditionally been prescribed for the management of acute and in some instances chronic pain. They are the most prescribed and recommended pharmacotherapeutic agents for the management of pain, in part due to their availability in both prescription and over-the-counter formulations. As a class, they are structurally diverse yet have very similar effects. They tend to be mostly well tolerated but do include cardiovascular, gastrointestinal, and renal risks. NSAIDs will block arachidonic acid conversion into prostaglandins by blocking cyclooxygenase enzymes (COX-1 and COX-2). COX-1 is an enzyme that plays a vital role in normal homeostatic functions including platelet aggregation, renal blood flow, and the protection of the gastric mucosa in the gut. COX-2 is an enzyme that is stimulated by proinflammatory mediators that will induce inflammation and the formation of prostaglandins that facilitate inflammation, pain, and fever. NSAIDs are classified as being nonselective in which they will inhibit both COX-1 and COX-2 enzymes, semi selective COX-2 inhibitors, or highly selective COX-2 inhibitors that are seven times or greater selective in their COX-2 enzyme blocking activity.[39] In 2015, the United States Food and Drug Administration (FDA) requested that manufacturers of all NSAIDs make labeling changes to their products. The changes are to include a boxed warning, highlighting the potential for increased risk of cardiovascular events as well as the potentially life-threatening gastrointestinal bleeding associated with their use.[40] The risk of bleeding and adverse cardiovascular events has been shown to increase when the patient is also using selective serotonin receptor uptake inhibitors (SSRIs). Also of concern is the concomitant use of alcohol or herbal supplements such as gingko biloba and St. John's Wort. NSAIDs are most effective if used for a period of several weeks.[41] NSAIDs may be used as a stand-alone drug or formulated in combination with other analgesic agents such as acetaminophen or opioids. Some of the more commonly prescribed NSAIDs for TMJD include ibuprofen, naproxen, diclofenac, ketoprofen, meloxicam and piroxicam. Of note, a recent investigation found that NSAIDs taken for acute low back pain led to a more prolonged pain duration despite analgesia in the short term.[42] The authors found that the NSAIDs blocked the transient neutrophil-driven up-regulation in the inflammatory response that appears to be protective against the transition to chronic pain. This response was not observed when other analgesics were utilized.

b. Acetaminophen

Acetaminophen, known as paracetamol, outside of the United States, is a non-opioid analgesic that, like the NSAIDs, may be used as a single agent or as a combination preparation with other agents such as opioids. Unlike NSAIDs, acetaminophen is not known to be associated with adverse cardiovascular events or gastrointestinal bleeding and is typically better tolerated than NSAIDs.[38] While it is usually considered a weaker analgesic than the NSAIDs, when it is combined with an NSAID, the efficacy exceeds either agent used alone.[43] Acetaminophen also differs from the majority of NSAIDs in that it lacks significant anti-inflammatory activity. The mechanism of action leading to analgesia for acetaminophen has not been fully described but may be due to the inhibition of central prostaglandin synthesis and an elevation of the pain threshold.[38] Other postulated mechanisms of action may include the facilitation of the serotonergic descending inhibitory pathways and activation of cannabinoid receptors.[44] Hepatotoxicity is the most serious

AE associated with the use of acetaminophen, even at the recommended doses of a maximum of 4 grams per day.[45] A recent recommendation from the FDA asks that providers not prescribe or dispense combination analgesic agents containing more than 325 milligrams of acetaminophen due to its potential for life threatening hepatotoxicity.[46]

c. Corticosteroids

Corticosteroids are potent anti-inflammatory agents often used for acute and in some cases, chronic pain. They act to reduce prostaglandin synthesis by inhibiting both phospholipase enzyme and COX-2 but have only a minor activity on COX-1.[47,48] Furthermore, they inhibit tumor necrosis factor alpha, interleukin 1 and six proinflammatory mediators.[49] The effect of corticosteroids tends to occur early in the inflammatory process which appears to result in a more profound impact on the inflammatory mediators as compared with NSAIDs. Corticosteroids may be injected directly into the TMJ, taken orally or applied topically in an attempt to reduce the pain and dysfunction associated with TMD. The analgesic effects of corticosteroids have been mostly demonstrated in post-surgical and osteoarthritic pain models and have not been well studied for muscular pain.[50-52] Corticosteroids should be utilized judiciously due to the potential for endogenous adrenal suppression, limiting the body's ability to respond normally to a stressful event. These agents may also lead to suppression in the normal immune response resulting in patients becoming more susceptible to a secondary infective process. The prolonged use of corticosteroids may increase the risk of adverse events such as osteoporosis and avascular necrosis.[53] Limiting the cumulative dose over the course of therapy appears to reduce the risk of adverse effects.[54] However, new data suggests that even short courses of corticosteroids may lead to significant adverse events such as increased rates of sepsis, venous thromboembolism, fractures, gastrointestinal bleeding, and cardiac failure.[55,56] Additionally, caution or even avoidance should be considered in patients with diabetes mellitus due to its effect on glucose levels and utilization. Gurwitz and colleagues found that subjects receiving prednisone at a daily dose of 30 mg or more had a significant risk of developing new diabetes onset as compared to controls not taking corticosteroids.[57] The use of corticosteroids should be avoided in patients with an ongoing infective process as they may prevent or decrease the inflammatory response, masking the underlying pathological process involved in the infection. For the above-described reasons, the course of treatment with corticosteroids should be limited to episodic use in known inflammatory processes and utilized for the shortest duration possible.

d. Opioids

Opioids is the term used for the entire class of drugs derived from opium and used for legal purposes. Often incorrectly referred to as narcotics, these agents have traditionally been considered to be highly useful and efficacious in the management of both acute and chronic pains. However, the use of opioids in chronic nonmalignant pains remains controversial due to the potential for tolerance, dependence, diversion and addiction. The prolonged use of opioids has also been linked to the potential for worsening of depression that is frequently experienced in chronic pain patients.[43] The phenomenon of opioid induced hyperalgesia (OIH) was reported as early as 1870 in morphine-addicted patients. OIH is a paradoxical occurrence where patients who are exposed to opioids experience a decline in their pain thresholds and an increase in their pain sensitivity. In many cases, the pain experienced in OIH is of a greater intensity than the initial presenting pain complaint. OIH can occur with even brief durations of therapy. The pathophysiology of this phenomenon appears to be different from that of tolerance.[58] However, in some cases tolerance may be a consequence of OIH.[59] Other studies have demonstrated that with persistent opioid exposure, cholecystokinin (CCK) is up regulated in the rostral ventromedial medulla (RVM), which will produce both anti-opioid and pro-nociceptive effects via activation of descending RVM pain facilitation. This will then increase pain transmission and produce hyperalgesia.[60] The role that opioid induced glial activation may play in the pathophysiology of pro nociception as well as tolerance, dependance and reward has been described.[61,62] Ultimately, the use of opioid analgesics in the TMD pain patient should be limited to carefully screened individuals who have failed other, more proven and conservative modalities. Patients prescribed opioids require careful monitoring to ensure compliance, patient safety and efficacy of the prescribed dose.

e. Tramadol

Tramadol is an opioid-like analgesic that weakly binds to the opioid receptors and functions as a mu-opioid agonist. The more prominent MOA of tramadol is to inhibit the uptake of both serotonin and norepinephrine in the dorsal horns of the spinal cord similar to the action of tricyclic antidepressants.[63,64] While there are no recent reports concerning the use of tramadol in patients with TMD pain, a study by Kaneko and colleagues demonstrated an improvement in experimentally induced neuropathic and myogenous pain in a rat model.[65] Another study demonstrated an anti-inflammatory affect when tramadol was injected directly into the rat TMJ.[66] Other human studies would seem to support its use in chronic widespread

pain, chronic low back pain, and osteoarthritis.[67-70] The combination of tramadol with acetaminophen has also been demonstrated to be effective in the fibromyalgia patient population.[71,72] It should be noted that while the potential for abuse and dependence is less for tramadol than that seen in the more traditional opioid agents, there is evidence to suggest that it does exist.[73,74]

f. Tapentadol

Tapentadol is another opioid like analgesic similar to tramadol but has a higher, yet still weak, affinity for the mu-opioid receptor as compared to tramadol. The most prominent mechanism of action of tapentadol is inhibition of the reuptake of norepinephrine. Tapentadol is not a prodrug and therefore does not require metabolic activation. The potency is generally considered to be in between that of tramadol and morphine. Tapentadol is scheduled by the Drug Enforcement Agency (DEA) as a Schedule 2 controlled agent.

g. Muscle Relaxants

Muscle relaxants comprise a group of pharmacologic agents with differing MOAs that appear to act on the central nervous system (CNS) to disrupt nociceptive signaling. The specific MOA of this class of drugs is still poorly understood. Sedation and analgesia are potentially responsible for the beneficial effects in the TMD population.[75] A Cochrane review identified only one relevant study showing the efficacy of muscle relaxants in TMD presentations.[32] They found that cyclobenzaprine was statistically superior to placebo when combined with self-care protocols for jaw pain presenting at awakening.[76] In another more recent systematic review, cyclobenzaprine was shown to have a favorable effect, specifically on the myogenous component of TMD.[77] As with most muscle relaxants, the MOA of cyclobenzaprine has not been fully described and is thought to be of benefit due primarily to its sedative and analgesic properties.[78] As cyclobenzaprine is a tricyclic amine and structurally similar to amitriptyline, ventricular dysrhythmia is a possible AE with this agent.[79]

Other common agents of this class include carisoprodol, metaxalone, methocarbamol, baclofen and tizanidine. Carisoprodol undergoes hepatic biotransformation to three primary metabolites: hydroxy carisoprodol, hydroxy meprobamate, and meprobamate. Meprobamate is a potent anxiolytic with significant abuse and dependency potential.[80] Withdrawal from extended use of carisoprodol may result in severe reactions including seizures and coma. The MOA of metaxalone has not yet been fully described. Any direct effect on skeletal muscles or nerve fibers has not been established. However, CNS depression may be partially responsible for its beneficial effects. Compared with other muscle relaxants, metaxalone has a relatively low risk of drowsiness or cognitive defects.[81] Methocarbamol is a centrally acting agent that is a derivative of guaifenesin. The MOA of methocarbamol is mostly unknown but is thought to be due to sedation. One interesting side effect of this agent is the production of brown or green urine discoloration. Baclofen is a natural analog of gamma-aminobutyric acid (GABA) that will bind to GABA B receptors. It will act both presynaptically and postsynaptically to inhibit spinal reflexes. This effect may ultimately serve to reduce loading of the TMJ structures. Common AEs include dry mouth and transient sedation that tend to subside with prolonged use. Caution is advised when discontinuing this drug as withdrawal symptoms have been reported with abrupt discontinuation.[82] These symptoms may include auditory and visual hallucinations, agitation, delirium, anxiety, fever, tremors, tachycardia, and in some cases, seizures. As with most muscle relaxants the precise MOA of tizanidine has not been fully described. However, it is thought to be related to central alpha 2-adrenoceptor agonist properties.[83] It appears to inhibit presynaptic release of excitatory neurotransmitters, reducing the excitability of postsynaptic a-motor neuron. In addition, it has been demonstrated to reduce abnormal co-contractions of opposing muscle groups. Again, any potential for decreasing adverse loading of the TMJ structures may prove beneficial when treating TMJD. Caution is advised when prescribing this agent to patients with impaired renal/liver function or who may have cardiac disease.

h. Benzodiazepines

Benzodiazepines are considered as neuro psychoactive agents used primarily for their anxiolytic, sedative-hypnotic, muscle relaxant, and anticonvulsant properties. These agents primarily act at the GABA A receptors in the CNS. While there is limited evidence for their efficacy in TMJD, benzodiazepines may be considered an alternative to the more traditional skeletal muscle relaxants. However, these agents do carry a potential for abuse.[84] In a study of 20 TMD patients with both joint and muscle pain, clonazepam was found to be significantly more effective than placebo.[85] Though it is widely recommended to limit benzodiazepine use to low doses over a short duration, Schenck and colleagues found that long-term, nightly use of clonazepam for the treatment of significant injurious parasomnias proved to have sustained efficacy with a low risk of dosage tolerance, adverse events or even abuse.[86] Some reports have suggested that benzodiazepine use is associated with an increased

risk of Alzheimer's disease as well as other dementias, however, other reports dispute these conclusions.[87,88]

i. Antidepressants

Due to their inhibition of the reuptake of serotonin and norepinephrine, antidepressants are commonly used for many painful conditions.[89] Antidepressants can be grouped into four main categories: tricyclic antidepressants (TCAs), monoamine oxidase inhibitors (MAOIs), selective serotonin reuptake inhibitors (SSRIs) and the dual selective norepinephrine and serotonin reuptake inhibitors (SNRIs). The SNRIs demonstrate similar properties to those of the older TCAs but do not block cholinergic, histamine or the alpha 1 receptors which tends to decrease their undesirable side effect profile as compared to TCAs. The TCAs and the SNRIs have both been shown to have the greatest efficacy of the antidepressant class in chronic pain conditions.[90] While depression is often comorbid with chronic pain, the TCAs imipramine and amitriptyline have both demonstrated efficacy in the depressed and non-depressed subjects.[91,92] This would seem to suggest that a unique MOA other than the antidepressant qualities is responsible for their efficacy in painful conditions. In addition, the analgesic effect of these agents is typically seen to occur more rapidly and at lower doses than the mood stabilizing effect.[93,94] Several trials have explored the efficacy of amitriptyline in patients with TMDs. Two studies have shown a clinically significant reduction in pain with amitriptyline as compared to placebo.[95,96] Some common side effects of TCAs and SNRIs include nausea, sedation, psychomotor impairment, xerostomia, and constipation. These agents should be avoided in patients taking MAOIs and any other serotonergic drugs as the combination may lead to serotonin syndrome, characterized by cognitive, autonomic and neurologic symptoms.[97]

j. Anticonvulsants

The most prescribed anticonvulsants in the orofacial pain population are carbamazepine, oxcarbazepine, gabapentin, and pregabalin. These have been shown to inhibit neuronal excitation at several sites: voltage gated calcium channels, sodium channels, glutamate receptors, N-methyl-D-aspartate (NMDA) receptors, and GABA receptors.[98] A trial utilizing gabapentin for masticatory muscle pain found it to be clinically and statistically superior to placebo in reducing the report of pain, muscle hyperalgesia, and the impact of pain on day-to-day functioning.[99] Gabapentin and pregabalin, which are structurally very similar, are viable considerations in TMJD patients due to their relative low occurrence of side effects compared to other agents in this class and their proven efficacy in trials involving various chronic pain syndromes.[100-102] It is important to note that in 2008, the FDA announced that all anticonvulsant drugs must display a clear warning that their use increases risk of suicidal thoughts and behaviors.

k. Cannabinoids

The cannabinoid receptors have recently emerged as a therapeutic target of interest in pain management, particularly in patients where more conventional therapies have failed. The antinociceptive effect of cannabinoids (CBs) may be mediated by either the CB1 receptor and/or the CB2 receptors depending on the nature of the of pain presentation. CB1 receptors are expressed at high levels in the CNS as well as the spinal cord, the dorsal root ganglion and in the periphery. Analgesia at the CB2 receptor appears to occur via peripheral mechanisms without apparent CNS effects.[103] CB1 receptors appear to be more involved with acute pain whereas chronic pain appears to be mediated by both CB1 and CB2 receptors.[104] Most data would seem to suggest that CBs appear to be better suited for chronic pain conditions.[105] However, a study by Bagues and colleagues found that tetrahydrocannabinol (THC), a CB natural derivative with reported therapeutic use in humans, reduced the nociceptive behaviors in two models of acute muscle pain in rats.[106] While some other studies on the use of CBs for multiple sclerosis and spasticity related pain report positive outcomes, overall, robust evidence for use in acute or chronic musculoskeletal pain presentations is still lacking.[107,108] Future research and utilization of CBs in pain management will ultimately be affected by the legal climate involved with these agents.

l. Propranolol

TMD has been shown to have a significant sympathetic component which can be demonstrated in part by the rich sympathetic innervations of the TMJ structures.[109,110] In one recent report utilizing a carrageenan-induced TMJ hyperalgesia model in female rats, the analgesic effect of the non-selective b-adrenergic receptor antagonist propranolol was positively associated with a reduction in joint inflammation. In a recent trial involving patients with TMD (both arthralgia and myalgia pain), propranolol was found to be effective in achieving significant pain index reductions after 9 weeks of treatment.[111]

PHYSICAL THERAPY

The goals in the management of patients includes:

- The elimination or reduction of pain that is satisfactory to the patient. Pain intensity is documented using the visual analog scale (VAS)
- Increase maximal interincisal opening (MIO) to >30 millimeters, ideally between 34-38 mm.

MIO is documented using a millimeter ruler and MIO does not include overbite.
- Limit MIO if there is a risk of condylar subluxation or dislocation
- Unrestricted jaw function that is satisfactory for the patient. Jaw function includes but not limited to chew, talk, yawn, brushing of teeth, smiling, and laughing.

An important clinical point to recognize is that masticatory myalgia, headache attributed to TMD (HTMD) and TMJ arthralgia may represent separate mutually independent manifestations from other coexisting TMD arthrogenous conditions such as Disc Displacement (DD) and Osteoarthritis (OA).[7,112] Clinicians should initially assume painful conditions such as masticatory muscle pain, HTMD and TMJ arthralgia may not be related to DD or DJD. Reducing pain, reducing joint loading, and increasing joint mobility are more important than relocating a displaced disc or being overly concerned about the bony structures of the joint.

Treatment Strategies

Physical therapists diagnose all common TMD diagnostic subsets by following Axis I of the Diagnostic Criteria for TMD (DC/TMD).[7] It has also been shown that a patients' cognitive, emotional, and behavioral responses to pain can affect treatment outcomes. Clinicians can assess a patient's psychosocial distress by following Axis II of the DC/TMD guidelines. Realizing clinicians are not psychologist, the DC/TMD recommends several valid questionnaires to assess the patient's depression and anxiety.

The majority of TMD patients can be classified as having no, minimal, mild or moderate depression and/or anxiety.[113] Physical therapists are trained to address biopsychosocial distress that fall in the minimal, mild or moderate range. Physical therapy can reduce biopsychosocial distress by reducing pain, by promoting aerobic exercise, by improving general relaxation through behavioral modification/self-regulation techniques, by reducing fear of movement, and by addressing misinformation and conflicting information from family, friend, internet and unfortunately from healthcare professionals that often results in enhancing depression and anxiety.[114-119] Physical therapists recognize their limitations in managing psychosocial distress and will consult with the referring doctor regarding other treatment strategies for depression and anxiety.

Treatments offered by the physical therapist for all diagnostic subsets of TMD will be a patient specific multimodal treatment approach involving various treatment strategies. (Table 2). A systematic review and meta-analysis was done from randomized controlled trials that assessed the effects of physical therapists' management of TMD.[120] Unlike other review articles, this review excluded studies that investigate a solo modality and exercise and studies that mixed treatments ie, modality/exercise combined with other forms of treatment ie, an oral appliance, medication and injections. Focus was on interventions performed by physical therapists (ie, manual therapy, dry needling, exercise therapy). The conclusion was physical therapist's interventions are more effective than other treatment modalities and sham treatment in the management of TMD pain reduction and improved active mouth opening. PT treatment strategies for each TMD diagnostic subset often vary (Table 3). Most patients are diagnosed with several concurrent diagnostic subsets of TMD.[121,122] PT treatment strategies will vary from treatment session to treatment session based on the diagnostic subset(s), patient's response to the previous treatment session, while considering the patients' psychosocial distress. Evidence suggests that when a physical therapist is included as a conservative treatment option for patients suffering from TMD, this results in a high patient satisfaction.[123,124]

Cervical Spine Considerations in the Management of TMD and Headache

Neck pain may originate from tissues other than muscles such as cervical spine discs, facet joints and central or peripheral nerves.[125-129] Cervical spine disorder (CSD) is the term representing anyone or combination of the previously mentioned tissues. Other terms such as mechanical neck pain or nonspecific neck pain have been used to describe pain of cervical origin as well as the simple term ''neck pain''.

Evidence Linking CSD to TMD

Females are diagnosed having TMD more than males.[130] Females are also as diagnosed having more CSD than males.[131] It has been reported that 70% of females diagnosed with TMD have CSD.[122] Patients with jaw and neck pain share common risk factors. Risk factors that increase both neck and jaw pain include strenuous lifting (lifting weights, groceries or a young child), focused activity such as driving or working at a computer, resting the chin on one's hand while sitting at a desk, and daily, weekly or monthly stressors (work stress, home stress, divorce, loss of a loved one, etc). Psychosocial distress can increase masticatory myalgia but has also been documented to increase cervical myalgia in adolescence and adults.[132,133]

Table 2. PHYSICAL THERAPY MODALITIES

Patient Education	Patient education involves many facets. Fundamental to patient education is an explanation of their diagnosis(s) and the source of their symptoms. Treatment objectives, expectations of treatment, frequency, costs and expected number of treatments will be a part of patient education. Patient education that addresses misinformation is essential in order to achieve optimal treatment outcomes while reducing any unnecessary psychosocial distress resulting from misinformation. Patients will be educated on other treatment options other than physical therapy. This includes an overview of medications, injections, oral appliances, and surgical interventions. Possible complication of all treatments, to include PT, are discussed based on the available scientific evidence. Patients must be a part of the decision-making process of their care.
Behavioral Modification	Behavioral modification (BM) or self-regulation is defined as the direct changing of unwanted behavior. Unwanted behaviors include but not limited to parafunctional activities to include bruxism. Though the etiology of bruxism is unknown, patients can apply self-regulation strategies to control bruxism. Being mindful of such strategies will help the patient to take ownership by focusing on relaxation of their jaw muscles in response to pain and feelings of fear, anger, depression and anxiety. Self-regulation strategies consist of but are not limited to · jaw relaxation exercises consisting of teeth apart (*TA) and wiggle at will (**WW) with tongue relaxed. Patient to apply TA and WW when confronted with triggers for bruxism and pain such as physical triggers (lifting >10 pounds), focused triggers (reading and watching TV), emotional triggers (daily life events) and psychosocial distress triggers (depression, anxiety, anger and fear). Being mindful of TA and WW will reduce or eliminate diurnal bruxism. · body relaxation techniques pertaining to head/neck posture when standing, walking, sitting and sleeping · diaphragmatic breathing to reduce unnecessary use of upper chest and neck muscles while promoting relaxation · 30 minutes of aerobic exercise/day · Sleep hygiene: 7-8 hours of sleep, go to bed and wake up at the same time and sleep with proper pillow support · eliminate other harmful parafunctional behaviors to including gum chewing, chewing ice, biting pencils, fingernails, lips and inside of cheeks. · modify miscellaneous activities that places stress on the jaw muscles and TMJs such as hard/chewy food, avoid playing a musical instrument that applies pressure on the jaw and limit excessive mouth opening when singing and yawning. * TA all day, unless chewing or swallowing which requires only very brief moments of tooth-to-tooth contact. ** small amplitude of lateral movement without pain or clicking performed with few repetitions but repeat many times/day
Therapeutic Exercise	· Therapeutic exercise is defined as any exercise performed with the aim of improving a single parameter, such as strength, range of motion (ROM), flexibility, endurance and coordination. Pertinent to TMD and depending on the TMD diagnostic subset, therapeutic jaw exercise consist of active, active assistive, passive and/or static jaw stretching exercises. The goal is to improve active mandibular range of motion to achieve functional mandibular dynamics regardless of DD and DJD. Pending the degree of myalgia and especially arthralgia, jaw exercises will need to be closely monitored so to not increase any unnecessary muscle or joint pain · Jaw strengthening exercises are seldom, if ever, needed. · As mentioned under behavioral modification, patient will be encouraged to do aerobic exercises. Aerobic exercise is essential to general relaxation and has been shown to reduce depression and anxiety. Aerobic exercises assist in managing other comorbidities (fibromyalgia, low back pain, somatic pain, sleep deprivation) not to mention weight control, high blood pressure and cholesterol. Physical therapist will not only address the patients TMD and cervical spine complaints but other musculoskeletal conditions that interfere with aerobic exercise.
Neuromuscular Reeducation	Neuromuscular reeducation (NR) is defined as the reeducation of movement, balance, kinesthetic sense, posture and proprioception. Mandibular kinesthetic and proprioceptive exercises are used to enhance self-awareness of jaw movement during opening, closing, lateral and protrusive movements. NR will help to regain midline opening as a result of excessive muscle activity contributing to mandibular deviation or deflection. NR exercises will control excessive condylar translation that occurs with subluxation and mandibular deviations that may be due to a disc displacement with reduction (DDwR).

Table 2. Cont'd

Manual Therapy	Joint Mobilization – the act of moving articular structures to facilitate the intended movement. Joint mobilization is a passive intraoral procedure performed by the physical therapist but can also be performed with active participation from the patient. The clinician's thumb is placed intraorally over the back molars on the same side of the involved joint. The other hand will stabilize the head in proper position. Intraoral joint mobilization (IJM) direct forces towards the head of the condyle to assist in improving arthrokinematics movement of joint distraction, condylar translation and lateral glide (a joint play movement). IJM is done for the diagnosis of a DDwR with intermittent locking and for a disc displacement without reduction with limited opening. IJM techniques are not designed to either deliberately or inadvertently relocate a displaced disc which may or may not occur while performing intraoral techniques. The objective for IJM is to increase condylar translation regardless of disc position to regain functional mandibular dynamics. IJM is indicated for capsular tightness and intraarticular adhesions that may occur post trauma, post TMJ arthrotomy or post orthognathic surgery. Physical therapist will be mindful of necessary tissue healing and patient characteristics that may slow the healing process, ie, smoker, diabetic, etc. IJM will be modified based on the type of surgery, ie, discoplasty, autogenous graft, total joint. Patient's tolerance will be factored in while doing IJM. Soft tissue mobilization – the movement of contractile or inert tissues in such a way as to effect change in that structure or its related elements. Targeted tissues are the muscles of mastication. Dry Needling – Is the insertion of a solid filiform needle into latent or active myofascial trigger points (MTrPs). Many studies have confirmed the existence of MTrPs and the therapeutic response a MTrPs has to dry needling (DN) which is a reduction in muscle pain and an increase in mobility. Masticatory muscles that can be DN are the temporalis, masseter, lateral/medial pterygoid and anterior/posterior digastric muscles. Physical therapist must be certified in DN. (DN courses that maintain high levels of competency require extensive hours in class and lab sessions followed by passing an oral, practical, and written examinations before receiving a certification in DN.)
Modalities	Continuous Ultrasound energy is absorbed in tissues with high collagen content. Used to heat tissue that has shorten or scarred down. Therapeutic exercises can be done during or after US to improve flexibility. US is used for myalgia with or without limited opening. US is indicated for capsular tightness due to prolong immobilization. Prolong immobilization occurs when patients are not placed on a passive/active jaw exercise program post trismus, TMJ arthrotomy or orthognathic surgery. Chronic immobilization occurs when a non-reducing disc with limited opening is not diagnosed or if diagnosed the patient was not referred to a physical therapist for the necessary treatments. Pulsed Ultrasound facilitates healing in the inflammatory and proliferative phase and is used for transdermal transport of anti- inflammatory medications (ketoprofen) referred to as phonophoresis. Primary use is for the diagnoses of TMJ arthralgia or arthralgia/capsulitis post TMJ arthrocentesis, arthroscopic and arthrotomy. Iontophoresis is the process by which drugs, usually anti-inflammatory in nature, are introduced to a small body part via direct electrical current. It is non-invasive, painless and eliminates potential side effects and adverse reactions which can occur with medications delivered orally or by injection. Iontophoresis is used primarily for the diagnoses of TMJ arthralgia but can be used to treat myalgia/ MTrPs of the masseter muscle. Interferential Stimulation is a type of electrical stimulation used for the control of pain. Interferential stimulation (IF Stim) is believed to penetrate to deeper tissues better than other forms of electrical stimulation such as transcutaneous electrical nerve stimulation (TENS). At higher frequencies, there is a decrease in skin resistance with IF Stim allowing the patient to tolerate interferential current better than TENS especially when applied over the masseter and/or TMJs. To avoid the cross over effect as with true interferential, premodulated IF Stim is used. When applied over the masseter muscles, a premodulated IF Stim with an intermittent setting of 10-15 seconds on and 10-15 seconds off is favored by this author. The intermittent current cues the patient when to perform active, active assistive, passive and/or neuromuscular exercises while, at the same time, receiving the benefits from premodulation IF Stim of pain reduction related to myalgia and/or arthralgia.

Table 3. PHYSICAL THERAPY TREATMENT STRATEGIES AND TREATMENT OBJECTIVES

Diagnostic Subset	Treatment Strategies	Treatment Objectives
1. Myalgia with or without limited opening 2. Local myalgia 3. Myofascial Pain 4. Myofascial pain with referral	· Patient education · Behavioral modification · Modalities: heat, US and IF · Manual therapy: STM and DN · Therapeutic exercise	· Reduce pain · Increase function · Lifelong behavioral modification
5. Headache attributed to TMD	· Patient education · Behavioral modification · Modalities: heat and IF · Manual therapy: STM and DN · Therapeutic exercise	· Reduce headache · Increase function · Lifelong behavioral modification
6. Arthralgia	· Patient education · Behavioral modification · If diagnosed reduce myalgia to reduce joint loading	· Reduce pain · Increase function · Lifelong behavioral modification
7. Subluxation	· Patient education · Behavioral modification If diagnosed reduce myalgia to reduce joint loading · If diagnosed eliminate arthralgia	· Reduce/eliminate subluxation · Lifelong behavioral modification · Therapeutic exercise to limit opening
8. Disc displacement with reduction	· Patient education · Behavioral modification · If diagnosed reduce myalgia to reduce joint loading · If diagnosed eliminate arthralgia	· Non painful click · Quiet click · Lifelong behavioral modification
9. Disc displacement with reduction and intermittent locking	· Patient education · Behavioral modification · If diagnosed reduce myalgia to reduce joint loading · If diagnosed eliminate arthralgia · If above is unsuccessful progres to manual therapy and therapeutic exercise	· Re duce frequency of locking · Non painful click · Quiet click · Lifelong behavioral modification · Progress to disc displacement without reduction without limited opening
10. Disc displacement without reduction with limited opening	Patient education Behavioral modification If diagnosed reduce myalgia to reduce joint loading · If diagnosed eliminate arthralgia · Manual therapy · Therapeutic exercise	· Lifelong behavioral modification · Progress to disc displacement without reduction without limite opening
11. Disc displacement without reduction and without limited opening	· Patient education · Behavioral modification · If diagnosed reduce myalgia to reduce joint loading · If diagnosed eliminate arthralgia	· Lifelong behavioral modification

Table 3. Cont'd

Diagnostic Subset	Treatment Strategies	Treatment Objectives
12. Osteoarthritis	• Patient education • Behavioral modification • If diagnosed reduce myalgia to reduce joint loading • If diagnosed eliminate arthralgia	• Lifelong behavioral modification

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A bidirectional relationship exists between neck pain and masticatory muscle pain. The cervical spine influences mandibular movement and masticatory muscle activity. Low-to-moderate co-contraction of neck muscles and the muscles of mastication occur during eating, clenching and different body positions (sitting and supine). An increase in neck muscle activity may therefore have a negative effect on jaw function.[134,135] Limited neck mobility and poor head/neck posture influences mandibular movements during jaw opening-closing and is likely to compromise natural jaw function.[136-138] Patients with neck pain report more masticatory pain and limited mouth opening compared to controls without neck pain.[139] This interaction is based on the trigeminocervical nucleus (TCN).[140] The TCN is tightly integrated into functionally connecting jaw and neck muscle activity.[141] (Fig 15) The trigeminocervical nucleus receives input from trigeminal nerve afferents but also receives sensory input from structures not supplied by the trigeminal nerve, in particular the neck muscles.[142] This organization allows for a bidirectional interaction between jaw and neck muscles. Afferent nociceptive input from neck structures can influence TCN neurons to modulate (excitatory and inhibitory) masticatory muscle activity.[143,144] For example, an increase in jaw muscle activity occurs when an inflammatory agent is injected in cervical muscles.[145] Similarly, a reduction in masticatory muscle activity occurs when trigger points located in the trapezius are injected with lidocaine.[146] The cervical spine must be considered when evaluating and treating patients with TMD.

Cervical Spine and Headaches

The TCN provides the same pathway to explain the mechanism by which the cervical spine tissues innervated by cervical nerves C1, C2 and C3 can refer pain to areas innervated by the trigeminal nerve.[147] Nociceptive afferents from C1, C2, and C3 innervated tissues converge onto second order neurons located in the TCN.[148] This convergence provides the pathway for cervical spine tissues to refer pain into the parietal, frontal, and orbital regions.[149] The most notable of the cervical tissues innervated by C1, C2 and C3 the sub occipital, trapezius, sternocleidomastoid, splenius capitus, and semispinalis muscles.[125] (Fig 16A) Equally important are the upper cervical discs, cervical facet joints and the greater occipital nerve (C2) all can refer pain to the parietal, frontal, and orbital regions.[126,127,129,150] (Fig 16B-D)

A headache originating from the cervical spine is referred to as a cervicogenic headache (CGH).[151] A CGH can be unilateral or bilateral, radiating from the neck and occipital region to project to the forehead, orbital region, temples, vertex, or ears.[152] CGH is one of four headache types that can result in a chronic headache. The other 3 headache types are Headache related to TMD (HTMD), Tension Type headache

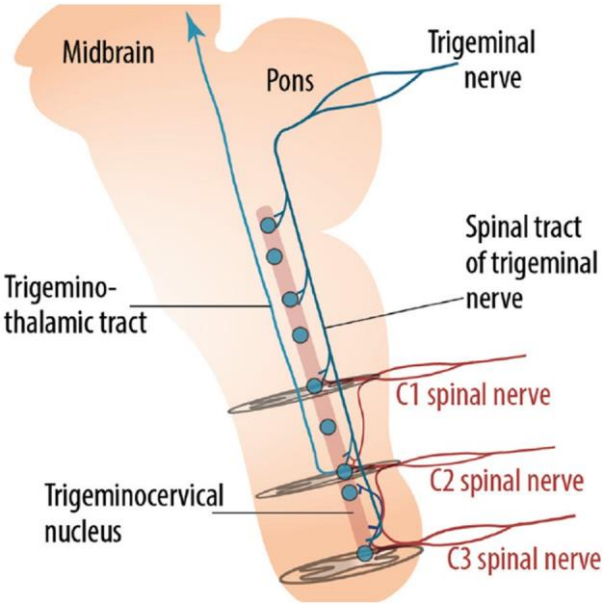

FIGURE 15. Anatomical relationship between the upper cervical nerves and the trigeminal spinal nucleus. Reproduced with permission from Young AC. Occipital Neuralgia & Cervicogenic Headache. Practical Neurology (US) 2021;20(4):43-46.

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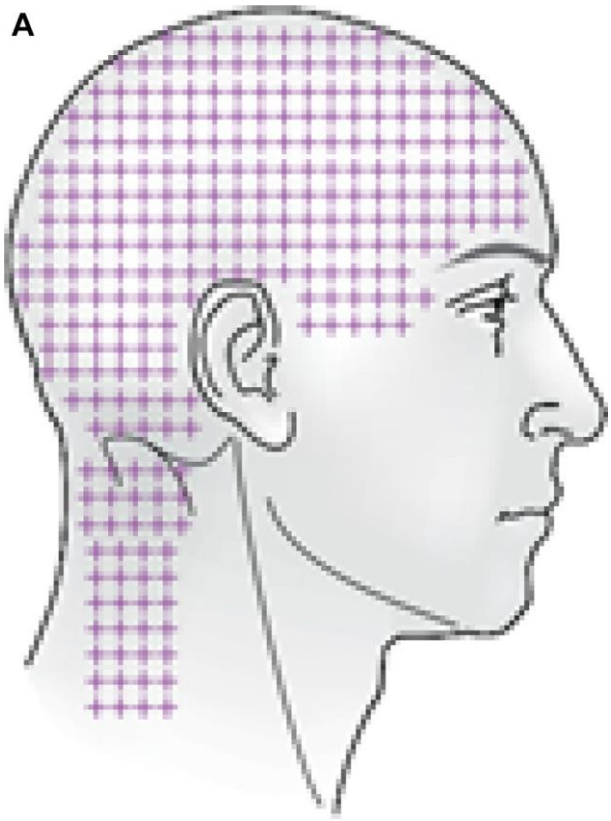

FIGURE 16. *A*, Patterns for pain referral from the cervical muscles (Used with permission and adapted from Fernandez-de-Las-Penas et al.[125]).
(Fig 16 continued on next page.)

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(TTH) and migraine. The differential diagnosis for these 4 headaches remains problematic as there is considerable overlap with symptoms, risk factors, and pathophysiology. The following highlights CGH overlapping with HTMD, TTH and Migraine.

HTMD, by definition, is a headache located in the temple area(s) either being referred to the temple area by TMJ arthralgia or by myofascial trigger points (MTrPs) located in the temporalis muscle.[7] MTrPs located in the sub occipitals, trapezius, sternocleidomastoid, splenius capitus, and semispinalis muscles also refer pain to the temple area.[125] Patients complaining of a headache located in the temple(s) must have their cervical spine evaluated as part of the differential diagnosis.

TTH is the most common of all headaches. Muscle tenderness to palpation is the most significant abnormal finding for TTH. TTH is reproduced or increased by palpation of common muscles to include the frontalis, temporalis, masseter, pterygoid, and pertinent to this discussion, the sternocleidomastoid, splenius and trapezius muscles. Cervical myofascial pain and associated myofascial trigger points (CGH) not only can mimic a TTH but the cervical muscles may be involved in the pathogenesis of TTH.[153,154]

A simple questionnaire has been validated to diagnosis migraine.[155] The patient needs to have a positive response to 2 of the 3 following questions. A positive response to question 3 (presence of aura) is the defining feature for migraine (classic)

1 Has a headache limited your activities for a day or more in the last 3 months? (disability)
2 Are you nauseated or sick to your stomach when you have a headache? (nausea)
3 Does light bother you when you have a headache? (photophobia)

Seventy five percent (75%) of patients with migraine reported neck pain or stiffness associated with their migraine attack.[156] High EMG activity of the cervical muscles occurs during a migraine.[157] Migraine is more likely to be accompanied by neck pain than by nausea and neck pain is a common trigger for migraine.[158,159] According to The International Classification of Headache Disorders, migraine features such as nausea, vomiting and photo/phonophobia may be present with cervicogenic headache. Though not

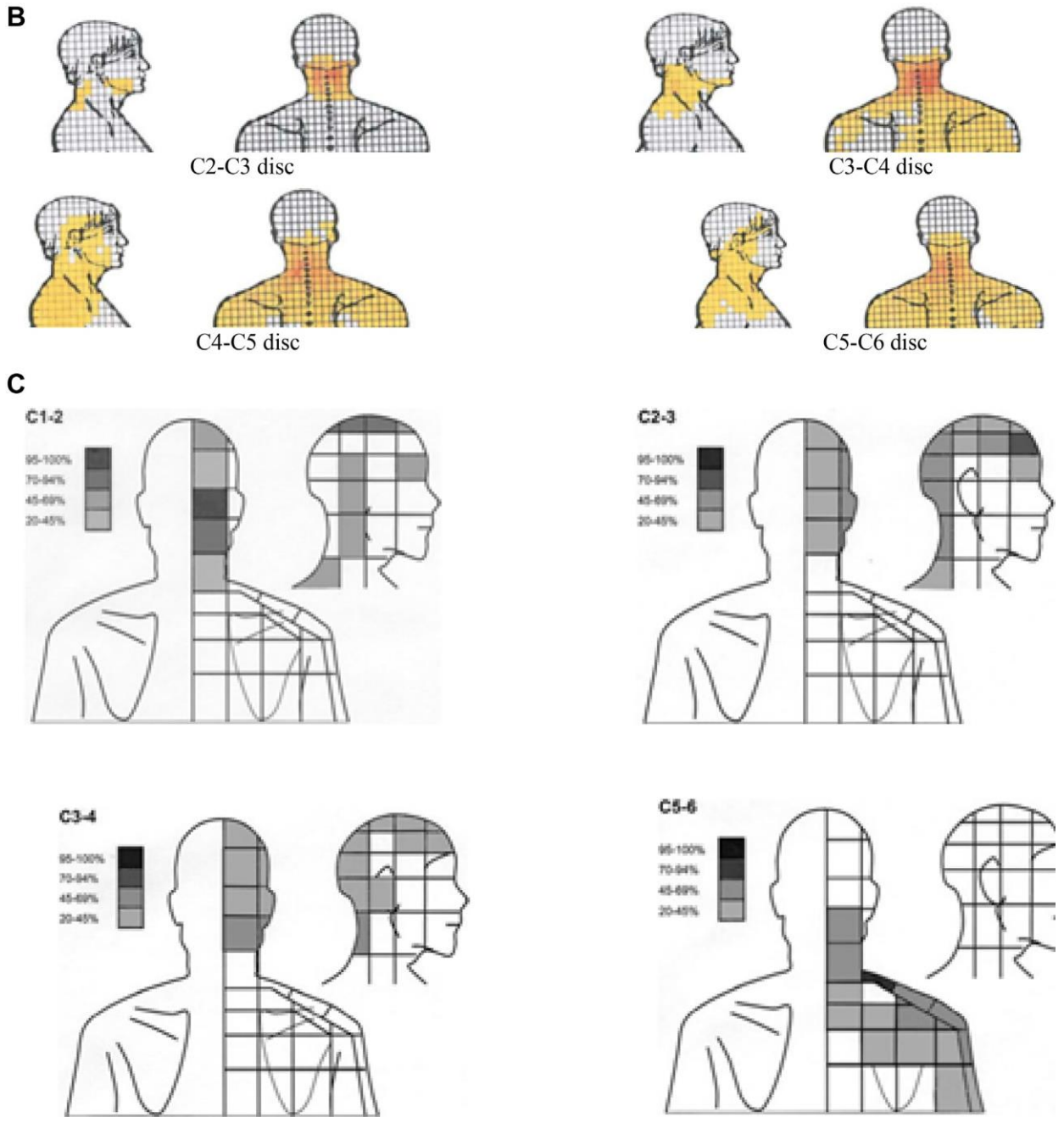

FIGURE 16 (cont'd). *B*, Patterns of pain referral from cervical discs (Used with permission and adapted from Slipman et al.[126]). *C*, Patterns of pain referral from cervical facet joints (Used with permission and adapted from Cooper et al.[127]). (Fig 16 continued on next page.)

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completely understood, migraine is believed to be a neurovascular disorder resulting from activation of the trigeminovascular system (TVS) affecting the meningeal blood vessels.[160] TVS is believed to be activated by the releases of neuropeptides, mainly calcitonin gene-related peptide (CGRP).[161] Evidence supports that nociceptive input from the cervical muscles may contribute to the activation of the TVS via an increase in CGRP.[162] Cervical muscle pain may play a role in the pathogenesis of a migraine headache.

Cervical Spine and Secondary Ear Symptoms

Ear symptoms that are not the result of a primary ear pathology areconsidered tobesecondaryear symptoms. Common secondary ear symptoms include ear fullness, otalgia, tinnitus, and subjective hearing loss. TMD has commonly been considered the primary source for

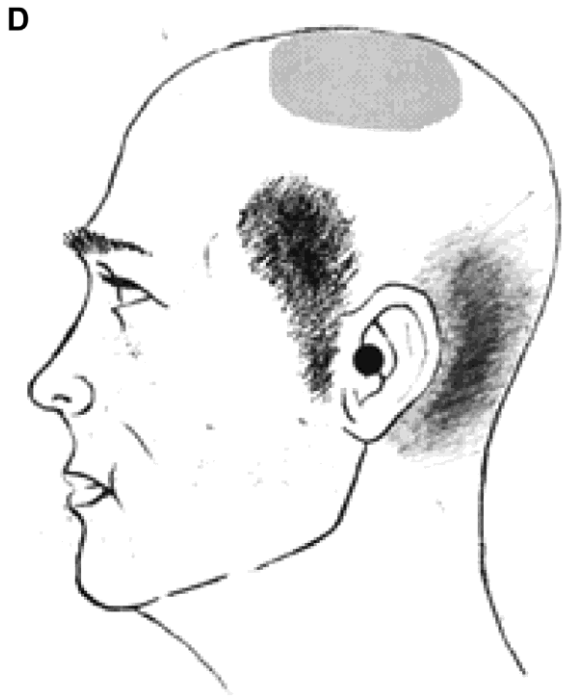

FIGURE 16 (cont'd). *D*, Pattern of pain referral from the occipital nerve (Used with permission and adapted from Son[129]).

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secondary ear symptoms. This is based on proximity of the TMJ (arthralgia) to the ear and myofascial trigger points located in the masseter muscle referring pain to the ear. Overlooked is the cervical spine as a source of secondary ear symptoms.[163] Muscle trigger points (MTrPs) located in the cervical muscles especially the SCM and peripheral entrapment of the occipital nerve can refer pain to the ear.[125,164,165] In a study of 91 patients with secondary ear symptoms, it was determined that the cervical spine was a more prevalent source for secondary ear symptoms than TMD.[166] An examination of the cervical spine should routinely be performed in the diagnostic process of patients with secondary ear symptoms.

Cervical Spine Evaluation

Screening for cervical pain is relatively simple. The physical therapist will perform a comprehension examination to differentiate between cervical muscles, facet joints, central nerve root involvement secondary to disc degeneration and herniation and peripheral nerve entrapments involving the greater occipital, dorsal scapular, suprascapular nerves, and entrapment of peripheral nerves passing through the thoracic outlet area. Physical therapists will screen for vertebral artery compression and upper cervical ligament laxity. Involvement of these tissues may be a contraindication for cervical spine treatment. If such findings are suspected, the physical therapist will consult with the patient's primary care physician.

Crucial to the management of TMD and headache is an understanding of the cervical spine as a possible source of the patient's jaw pain, headache symptom and masticatory muscle pain.[167] Physical therapists will perform a comprehensive examination of all cervical tissues followed by a multimodal treatment plan addressing all cervical spine tissues. Reducing neck pain, improving cervical posture, and increasing cervical mobility and strength may:

1 reduce masticatory myalgia and increase mouth opening resulting in improved jaw function.
2 reduce frequency and intensity of HTMD, TTH, migraine & secondary ear symptoms.

Botulinum Toxin

Botulinum Toxin (Botox) is derived from a gram-positive anaerobic bacteria Clostridium Botulinum. There are seven different serotypes (A- G) with a biological effect on neurotransmission. The mechanism of action of Botox involves the following four basic steps:

- binding of Botox on neuronal presynaptic membrane via synaptic vesicle protein 2 (SV2) and/or synaptotagmin depending on the serotype
- Internalization of the Botox via endocytosis

- Translocation of Botox from the endocytosed vesicle to the neuronal cytosol
- Cleavage of specific proteins involved in neuroexocytosis including SNAP-25 cleaved by Botox A, E and C; VAMP/synaptobrevin cleaved by Botox B, D, F and G; and syntaxin cleaved by Botox C.

The net result of cleavage of these proteins is inhibition of the formation of the soluble N-ethylmaleimide-sensitive factor attachment protein receptors (SNARE) which are critical for the fusion of the acetylcholine (Ach) containing synaptic vesicles to the presynaptic neuronal membrane. This effectively prevents the release of Ach from the motor end plate at the neuromuscular junction. The net result is muscle paralysis that persists until the internal neuronal proteins (SNAP-25, VAMP and Syntaxin) are regenerated over the following 3-6 months.

Botox was originally thought to reduce orofacial pain through inhibition of muscle activity. Current evidence suggests that Botox also possesses analgesic properties as a result of its ability to inhibit the release of Substance P (SP), Calcitonin Gene Related Peptide (CGRP) and transient receptor potential (TRP) vanilloid receptor type 1 (TRPV1). This translates into reduced pain and neurogenic inflammation in the peripheral and central nervous system. This maybe synergistic with the ability of Botox to block neuromuscular transmission.

GENERAL INDICATIONS

Although Botox was initially approved by the FDA in 1989, the list of approved indications has grown to include overactive bladder, headaches, spasticity, dystonia, blepharospasm and hyperhidrosis. It can be administered using an intramuscular or intradermal technique. The use of electromyographic (EMG) monitoring during intramuscular injection is beneficial to facilitate location of motor endplates during injection. The maximum dose for adults is 400 units in a 3-month period or the lesser of 300 units or 8 units/kg for pediatric patients. The dose of Botox for any given patient is dependent on the indication for which it is being used. Complications following the use of Botox are exceedingly rare but may include distant spread resulting in asthenia, generalized muscle weakness, diplopia, ptosis, dysphagia, dysphonia, dysarthria, urinary incontinence and breathing difficulties. Botox should not be used in patients with an allergy to Botox or with overlying skin infections.

ADMINISTRATION TECHNIQUE

Botox comes as a 50-, 100- and 200-unit vial. It needs to be reconstituted with sterile normal saline. Typically, this requires the addition of 1 mL, 2 mL or 4 mL of NS for the 50-, 100- and 200-unit vials, respectively. This results in a uniform concentration of 5 units of Botox per 0.1 mL. Vials are single use and should be used within 24 hours of reconstitution. Reconstituted vials should be stored at 4° Celsius if not used immediately after preparation. The injection site for muscles should target the anatomical location of the greatest density of motor end plates.

INDICATIONS IN ORAL AND MAXILLOFACIAL SURGERY

There are several uses for Botox in helping to reduce orofacial pain.[168]

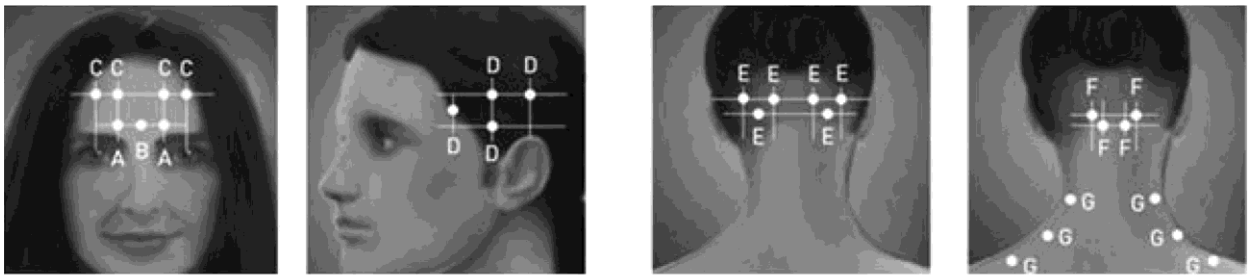
a) Chronic Migraine

Chronic migraines are defined as headache $ 15 days per month with headache lasting more than 4 hours. Botox has been shown to reduce the frequency and intensity of headaches. The exact mechanism of action is unclear. One thought is that Botox reduces the mechanical sensitivity of cranial muscle nociceptors secondary to reduced glutamate and CGRP. Another potential mechanism is the reduction in Substance P and CGRP within the trigeminovascular system. There are specific muscles and injection sites that have been shown to be beneficial (Fig 17).

b) Myofascial pain

According to the DC/TMD the two validated muscle related Axis I diagnoses that can be diagnosed with confidence are myalgia and myofascial pain with referral. They are diagnosed by history and physical examination.

Local myalgia, myofascial pain and headache attributed to TMD remain additional myogenous Axis I diagnoses but the patient's history and physical examination is not generally sufficient to provide an adequate sensitivity or specificity for these conditions. The pathophysiology of Axis I myogenous pain remains unclear with neurological, myogenous and psychological theories proposed. Botox has been shown to reduce pain in the myogenous Axis 1 diagnoses. Pain reduction appears to be even greater when bruxism and/or psychiatric stress related comorbidities exist. The most ideal location for injection is at the junction of the lower and middle third of the masseter muscle. The most ideal location for injection of the temporalis muscle is at the midpoint of the belly of the muscle. A total of 50 units of Botox is injected into each masseter and temporalis muscle with an equal number of units at each injection site. This results in a typical dose of 200 units for bilateral masseter and temporalis muscles (Fig 18).



A. Corrugator
B. Procerus
C. Frontalis

D. Temporalis

E. Occipitalis

F. Cervical
G. Trapezius

Head and Neck Area	Dose
Frontalis	20 units divided at 4 sites
Corrugator	10 units divided at 2 sites
Procerus	5 units at 1 site
Occipitalis	30 units divided at 6 sites
Temporalis	40 units divided at 8 sites
Trapezius	30 units divided at 6 sites
Cervical	20 units divided at 4 sites

Adapted from BOTOX® (onabotulinumtoxin A) Product Insert

FIGURE 17. Botox Injection sites for migraine. Adapted from BOTOX (onabotulinumtoxin A) Product Insert.

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c) Orofacial Movement Disorders

Orofacial Movement Disorders include orofacial dystonia and orofacial dyskinesia. These conditions can affect multiple orofacial muscles including platysma, orbicularis oris, buccinator, genioglossus, geniohyoid, digastric and intrinsic tongue muscles. The net result can be involuntary jaw opening, clenching, tongue thrusting and facial tics. They have the potential to result in TMD when the masseter, temporalis or medial pterygoid muscles are involved. It may also result in temporomandibular joint dislocation when the lateral pterygoid muscle is involved. The etiology of these movement disorders is unclear, and examination of the central and peripheral nervous systems is typically unremarkable. The treatment is often medications including benzodiazepines, anticholinergic medication, levodopa, bromocriptine and baclofen. Botox remains an effective treatment particularly for focal dystonia and dyskinesia.

The technique for injection is as described for Myofascial Pain when the temporalis and masseter are involved. Injection for the medial pterygoid is more challenging. There are two basic options both

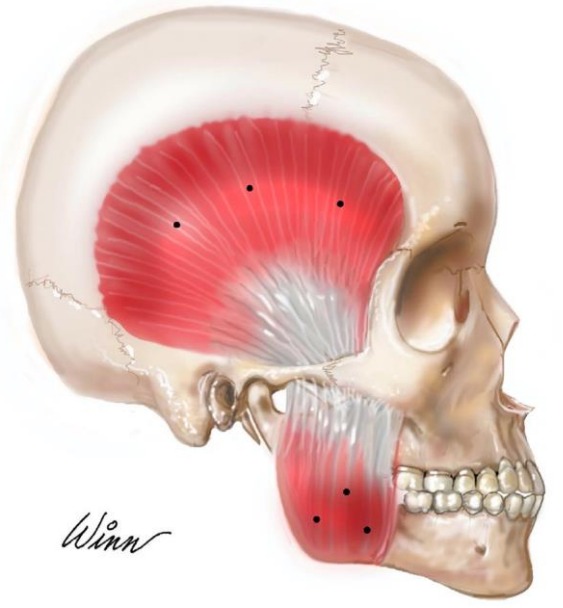

FIGURE 18. Botox injection site for the masseter and temporalis.

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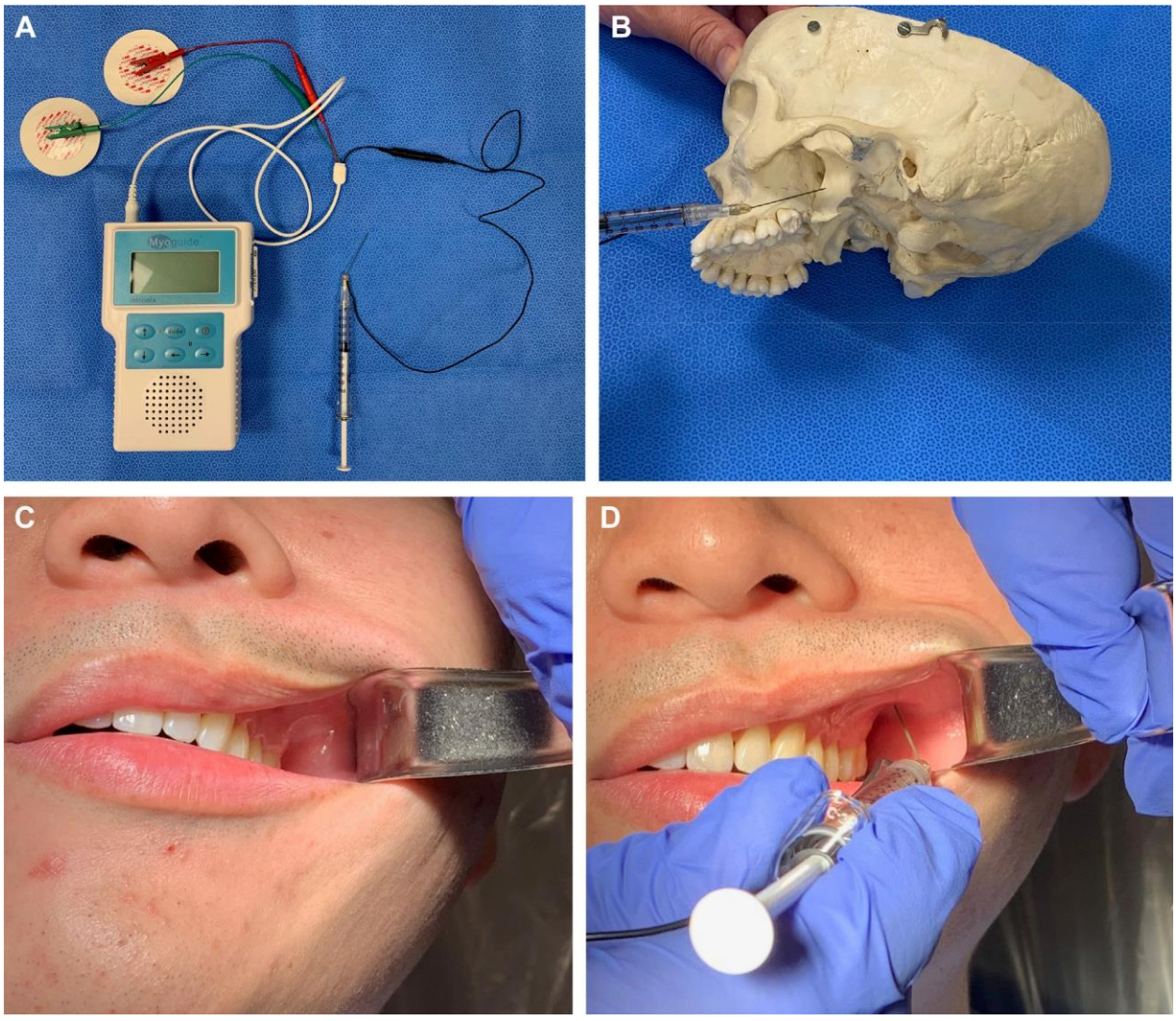

FIGURE 19. A-D, Botox administration to the lateral pterygoid muscle.

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requiring EMG guidance. The first option involves an intra-oral injection using 1.5-inch needle that is inserted just lateral to the pterygomandibular raphe and parallel to the ramus of the mandible. This is similar to an Inferior alveolar nerve block except that the needle is intentionally directed parallel to the inner aspect of the ramus. The dose of Botox is typically 20 units. An alternate technique that may be considered in thin patients is an extraoral approach from beneath the antegonial notch. It requires the same 1.5-inch needle. The skin and soft tissue immediately beneath the antegonial notch is displaced medially with the index finger of the non-dominant hand. This allows the Botox needle to be inserted from an inferior direction. The needle is kept parallel to the medial aspect of the ramus to ensure the medial pterygoid muscle is correctly targeted. It is technically relatively simple to perform this injection particularly if the needle is bent at about 30º at the hub to allow the hand holding the syringe to be kept lateral with respect to the neck. Complications after Botox injections are uncommon, however with the medial pterygoid muscle being within the infratemporal fossa there is potential for diffusion with resultant dysphonia and dysphagia.

Botox injection for the lateral pterygoid muscle is technically easier. As with the technique for the medial pterygoid muscle there are two approaches and EMG guidance is required. The first and most simple is an intraoral injection immediately behind the maxillary tuberosity. A tongue blade is used to displace the cheek laterally and with the teeth slightly separated and a 30-degree bend of the needle, the needle is directed medially and superiorly behind the tuberosity until the lateral pterygoid plate is encountered. Typically, 15-25 units of Botox is adequate (Fig 19A-D).

The extraoral technique is more challenging. It requires a straight needle to be inserted immediately beneath the articular eminence and through the sigmoid notch. The needle is kept horizontal but directed slightly anteriorly.

d) Neuropathic Pain

Botox is also known to inhibit neuromodulator and transmitter secretion that may reduce central neuropathic pain. The proposed mechanism of action includes a reduction in the release of excitatory glutamate, substance P, and CGRP. Furthermore, Botox reduces the expression of transient receptor potential vanilloid type 1 (TRPV1) that is associated with capsaicin-evoked and calcium channel responses.

The injection technique in the management of neuropathic pain is unique in that the Botox is inject subcutaneously in the dermatome overlying the neuropathic pain. The dose varies but generally 5 units of Botox per cm^2 of skin is adequate. This does not require EMG guidance and can be completed with a tuberculin needle to reduce discomfort during the injections.

e) Trigeminal Neuralgia

Trigeminal neuralgia (TN) is not synonymous with neuropathic pain. The etiology of TN remains unclear although compression of the trigeminal nerve root by the superior cerebellar artery is thought to result in focal demyelination and paroxysms of pain. Additional causes include tumors, multiple sclerosis and postviral syndromes. The mainstay of treatment is medication including carbamazepine, oxycarbamazepine, phenytoin, gabapentin, pregabalin and baclofen. Surgical treatment involves gasserian ganglion rhizotomy, gamma knife and microvascular decompression.

The mechanism of action of the Botox is thought to be similar to that of neuropathic pain. The injection technique is also identical. The time to maximal pain reduction for both neuropathic pain and TN appears to be 4-8 weeks. This is much longer than the 1 -2 weeks for muscular injection and may reflect the need for retrograde axonal transport to the Central Nervous System (CNS)

f) Bruxism

The beneficial effect of Botox on sleep related bruxism has been reported. It appears to reduce the intensity of muscle activity within the masseter and temporalis muscles with a reduction in self-reported pain and jaw stiffness. The injection technique and dosing is identical to that for myofascial pain.

g) Frey Syndrome

Frey syndrome is a rare complication following parotid and temporomandibular joint surgery. It results in gustatory sweating during eating. The parotid gland receives parasympathetic secretory innervation via a complicated neural pathway that involves the auriculotemporal nerve and the release of acetylcholine.

The sweat glands of the skin are innervated by the sympathetic fibers originating from superior cervical ganglion travelling via the same auriculotemporal nerve. Parotid and temporomandibular joint surgery are thought to cause an injury to the auriculotemporal nerve leading to inappropriate parasympathetic innervation of the overlying sweat glands. This can result in profuse sweating during mastication. The diagnosis of Frey syndrome can be made using the Minor iodine/starch test. This test will also delineate exact area of skin that is affected. This should be outlined with a skin marker. Botox can be injected subcutaneously using a tuberculin syringe/needle with a dose of 5 units per square centimeter (cm^2) of skin involved. The therapeutic effect will be apparentin aweek and should last 3-6 months.[169]

Our understanding of the mechanism of action of Botox has grown considerably and now extends well beyond s simply inhibition of neuromuscular transmission. The anti-nociceptive property of Botox relies on multiple factors including a reduction in substance P, CGRP and TRPV1. This has the potential to see a more widespread application for Botox for various pain conditions. Local myalgia, myalgia, myofascial pain, myofascial pain with referral and headache due to TMD are likely to benefit from Botox. Current limitations to the Food and Drug Administration (FDA) list of approved indications combined with the relative expense of Botox largely limits its use for most of these conditions. Further clinical research into the efficacy of Botox outside of current indications is warranted to provide a sound scientific rationale for its use.

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Chapter 7: Intraarticular Pain and Dysfunction (IPD)

Objectives:

- To provide clear and organized steps to select optimal treatment options for common TMJ related conditions.
- To provide a range of treatment options for each common TMJ related condition.
- To provide a structured framework for managing various TMJ related surgical conditions.
- To provide the levels of evidence that support various surgical procedures and medications

Surgical algorithms are dynamic and evolve over time as new evidence and technologies emerge. The

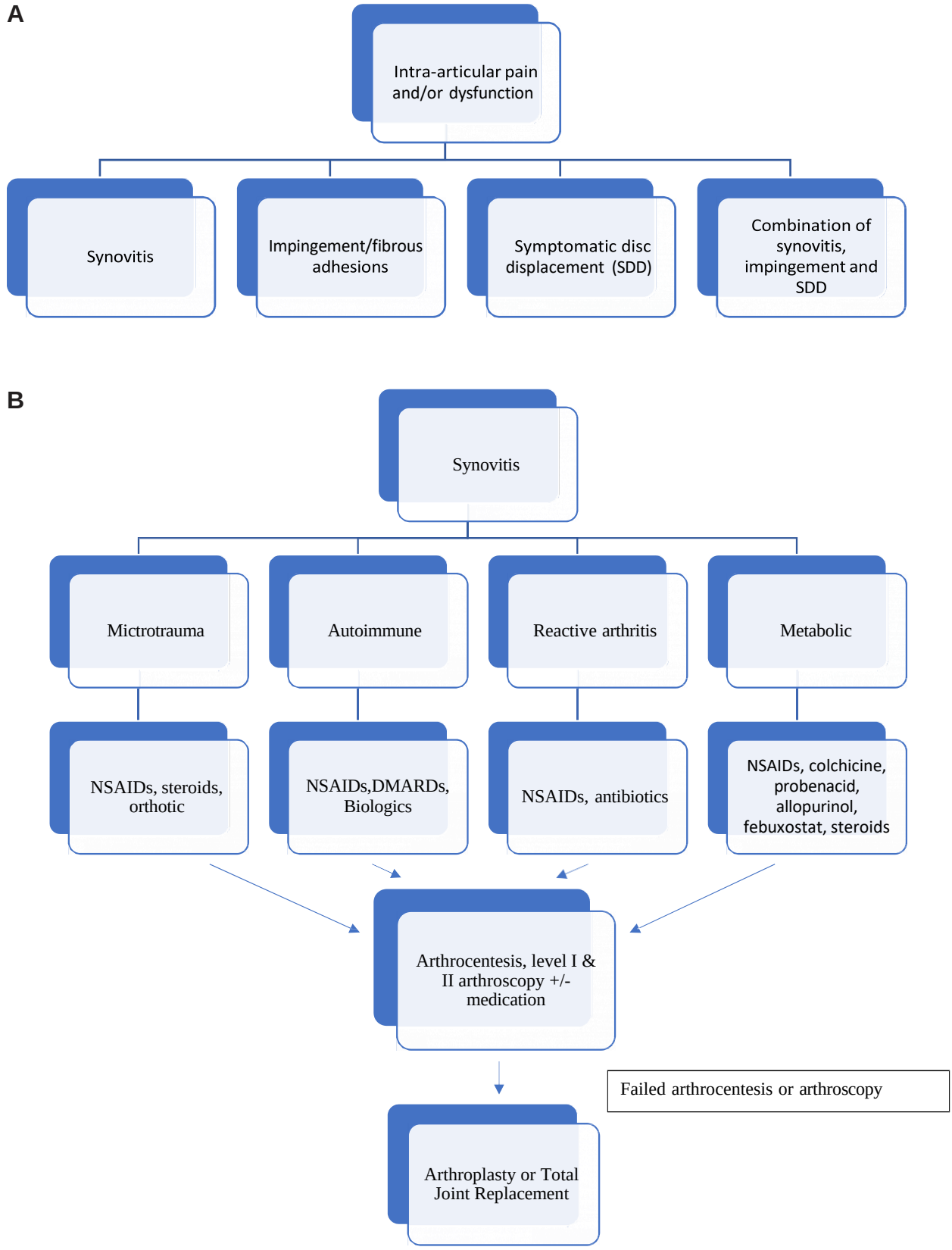

FIGURE 20. *A*, Algorithm for intraarticular pain and dysfunction. *B*, Algorithm for synovitis.

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following algorithms are developed by incorporating the current research evidence and expert consensus and may serve as a resource for both experienced and novice surgeons. However, it is important to note that surgical algorithms are meant to assist and guide surgeons, but they should not replace clinical judgment and individualized patient care. Surgeons should adapt algorithms to suit the unique needs of each patient and consider any factors that may require deviation from the algorithmic approach.

INITIAL SURGICAL OPTIONS

The initial approach to symptomatic intra-articular pain and restricted joint rotation/translation should consider either arthrocentesis or arthroscopy. Factors that may influence the choice of procedure include the differential diagnosis, procedure goals (biopsy, pain reduction and increased range of motion), surgeon skill, previous surgical history, psychosocial factors and medical history. The advantages of selecting a minimally invasive initial approach include good outcomes with minimally invasive procedures, minimal complications and, providing sufficient time to identify psychosocial factors that may adversely affect outcomes. The causes of intraarticular pain and dysfunction are many but can generally be classified into one of four etiologies. (Fig 20A).

It is generally unclear in any given patient which of the above potential sources of intraarticular pain and dysfunction is the key etiological factor. The causes of synovitis are multiple, and more than one cause may be present at any point in time. The suggested algorithm for the treatment of synovitis will depend on the etiology. (Fig 20B).

Arthrocentesis and arthroscopy both treat synovitis through removal of inflammatory mediators and degraded intraarticular proteins. In addition, arthroscopy provides an opportunity to directly treat synovitis through the use of coblation, laser, mechanical instrumentation and subsynovial injection of medication (Fig 21A and B).

The suggested algorithm for the treatment of impingement is simpler than that of synovitis (Fig 22A).

The presence of disc displacement in the general popluation approaches 25%. The vast majority are asymptomatic with respect to pain and limited function although an audible click may be present.[170] This suggests that disc displacement may be present in a given patient but not responsible for intraarticular pain and dysfunction. It is therefore suggested that a minimally invasive approach be adopted initially (Fig 22B).

As stated previously, the potential sources of intraarticular pain and dysfunction are numerous, and the ability to discern the specific diagnosis in a given patient is problematic. It is therefore reasonable to initially adopt a non-surgical and minimally invasive approach for all patients (Fig 22C).

SPECIAL CONSIDERATIONS

On rare occasions the initial surgical intervention may be an arthroplasty when certain patient specific situations are present. This may be appropriate

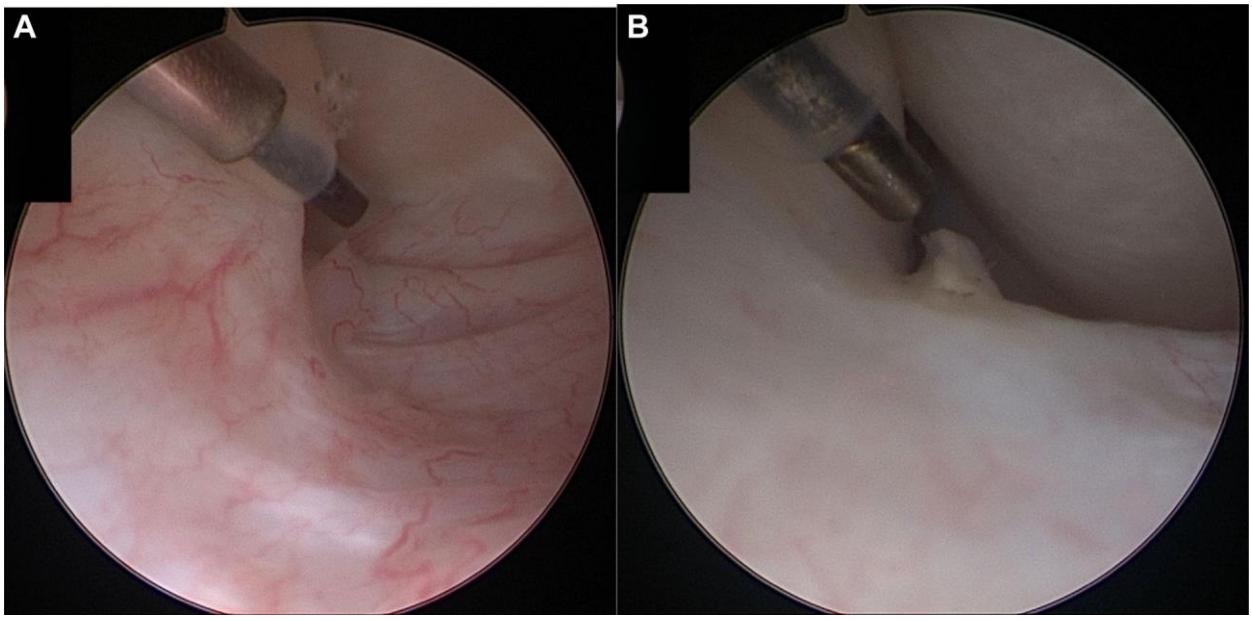

FIGURE 21. *A-B*, Coblation of synovitis and synovial plica.

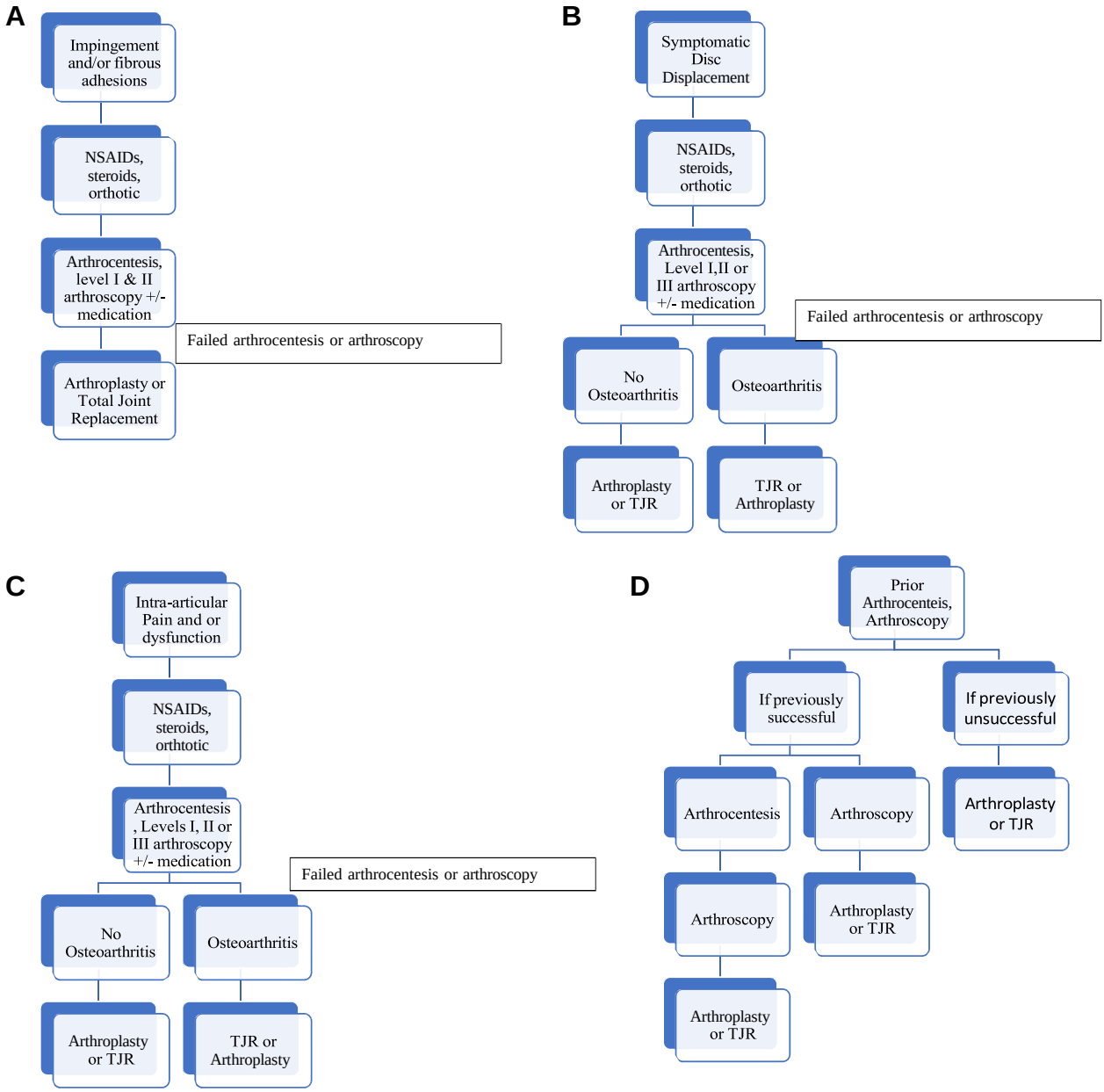

FIGURE 22. *A*, Algorithm for treatment of impingement and fibrous adhesions. *B*, Algorithm for treatment of symptomatic disc displacement and osteoarthritis. *C*, Summary Algorithm for treatment of intraarticular pain and dysfunction. *D*, Algorithm for patients with a history of prior arthrocentesis or arthroscopy. (Fig 22 continued on next page.)

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when the patient requires orthognathic surgery including a bilateral sagittal split osteotomy advancement and has concomitant TMJ disc displacement (with or without reduction) as post orthognathic surgery stability/relapse can be adversely affected by disc displacement. In addition, when a painful click is present, the success of arthrocentesis and level I and II arthroscopy may be less than satisfactory.

The short and long-term benefit of adjunct medications during arthrocentesis and arthroscopy to improve outcomes (pain and range of motion) remains unclear. Some studies have shown some short-term benefit with hyaluronic acid (HA) and corticosteroid. However, it is worth noting that the use of corticosteroids in the growing patient is not recommended unless all other treatments (medications and minimally invasive procedures) have failed dueto concerns for altered growth. The use of Platelet Rich Plasma (PRP), Platelet Rich Fibrin (PRF) and Platelet Derived Growth Factor (PDGF) for osteoarthritis has gained popularity with improvement in pain, range of motion and several patient-reported outcome measures. The initial data would seem to

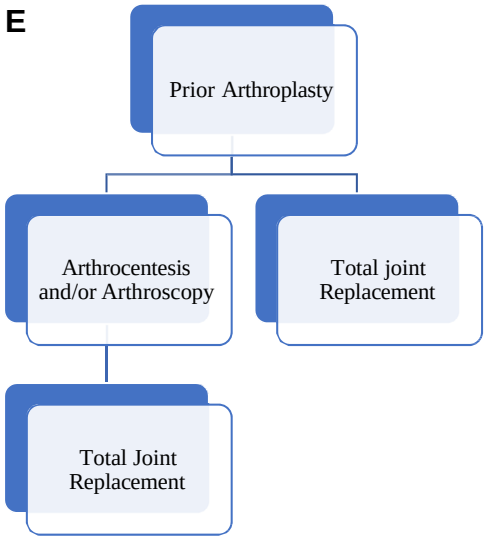

FIGURE 22 (cont'd). *E*, algorithm for patients with a prior history of arthroplasty.

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suggest that PRP, PRF and PDGF result in a better long-term outcome for osteoarthritis when compared to HA or corticosteroid.[171-182] Furthermore, the injection of Microfragmented Adipose tissue into the TMJ has shown some promising preliminary results, particularly for Wilkes IV and V.[183] At present although the levels of evidence to support any adjunct medications following arthrocentesis or arthroscopy remains low, several options exist. (Table 4).

SPECIAL CONSIDERATIONS IN PATIENTS WITH PRIOR SURGICAL PROCEDURES

Patients who have had prior arthrocentesis, arthroscopy and arthroplasty but present with recurrent or persistent intraarticular pain and dysfunction are challenging. An identical approach to history taking and physical examination is required to help develop a differential diagnosis to ensure the correct diagnosis is made. Non-surgical management, arthrocentesis and arthroscopy should be all be considered in these patients. Repeat arthrocentesis and/or arthroscopy should be considered if the initial procedure was beneficial. Patients who have undergone prior arthroplasty and are deemed good candidates for an additional open surgical procedure (based on the diagnosis of intraarticular pain and/or dysfunction) are best treated with total joint replacement. This is based on data that shows that outcomes are inversely proportional to the number of prior open procedures. This is a result of many factors including the development of

Table 4. CONSIDERATIONS FOR ADJUNCT MEDICATIONS DURING ARTHROCENTESIS OR ARTHROSCOPY

Hyaluronic Acid	Corticosteroid	Platelet Rich Plasma/ Platelet Rich Fibrin/PDGF	Microfragmented Adipose Tissue
Anti-inflammatory; increase viscosity and lubrication; decrease ROS, cytokines, vascular permeability and neutrophil migration	Anti-inflammatory; decrease elastin and collagen production	Release IL-1b, IL-8, TNF-alpha, PDGF, PDEGF, TGF-B1, IGF-1, FGF-2, VEGF-A resulting in cell differentiation, proliferation and migration	Mesenchymal Stem Cells and reparative capability
Use with Caution in patients with significant synovitis	Avoid repeat injections and use in the growing patient	Better long-term outcomes that HA or CS	May rarely result in intravascular thrombosis

Abbreviations: FGF-2, Fibroblast Growth Factor 2; IL-1b, Interleukin 1 beta; IL-8, Interleukin 8; IGF-1, Insulin like growth Factor 1; PDGF, Platelet Derived Growth Factor; PDEGF, Platelet Derived Epithelial Growth Factor; PDEGF, Platelet Derived Growth Factor; ROS, Reactive Oxygen Species; TNF-alpha, Tumor Necrosis Factor-alpha; TGF-B1, Transforming Growth Factor 1; VEGF-A, Vascular Endothelial Growth Factor A.

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Table 5. NUMBER OF PRIOR OPEN SURGICAL PROCEDURES BEFORE TJR AND REPORTED OUTCOMES

Number of prior Open Procedures	Increase Maximum Incisal Opening %	Decreased Pain %	Increased Function %	Increased Diet %
0-2	28	75	54	56
3-5	14	49	38	38
6-8	47	66	52	68
9-28	70	21	32	29

Adapted from: Mercuri, LG. Subjective and objective outcomes in patients reconstructed with a custom-fitted alloplastic temporomandibular joint prosthesis. J Oral Maxillofac Surg 1999 Dec; 57(12):1427-30.

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peripheral and central sensitization as well the development of neuropathic pain which can follow any open surgical procedure. The development of chronic pain is unpredictable but has been shown to be proportional to the number of open TMJ surgical procedures. Therefore, the number of prior surgeries should not only influence the surgical procedure chosen but it should also direct a very transparent conversation with the patient regarding realistic expectation in terms of reduced pain, improved range of motion and quality of life.[184] (Table 5)

Patients who have undergone previous arthrocentesis or arthroscopy may still be good candidates for repeat arthrocentesis or arthroscopy particularly when they was an initial positive response to the procedure. If prior arthrocentesis or arthroscopy was performed recently with only temporary improvement in pain and/or dysfunction, patients may benefit from moving to arthroplasty or total joint replacement (Fig 22D)

Patients who have undergone an arthroplasty may still be candidates for arthrocentesis or arthroscopy depending on the type of arthroplasty. Prior disc plication will usually lend itself to either procedure. Prior discectomy without replacement or with replacement with fat, temporalis muscle flap, silastic pullout or other interpositional material will make both arthrocentesis and arthroscopy more challenging. If arthrocentesis or arthroscopy are not indicated, or fail to reduce pain and improve function, consideration should be given for total joint replacement. Only in rare circumstances is repeat arthroplasty indicated as

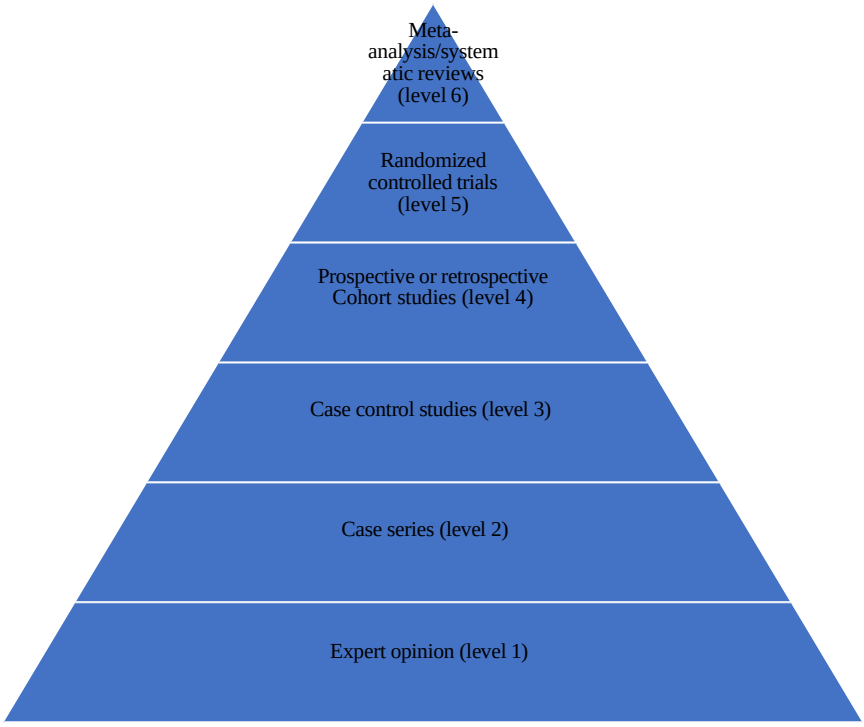

FIGURE 23. Levels of evidence.

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Table 6. LEVELS OF EVIDENCE FOR SURGICAL PROCEDURES

Surgical Procedure	Arthrocentesis	Arthroscopy	Arthroplasty	Total Joint Replacement
Levels of Evidence	4	4	2	4
Recommend based on consistent outcomes (Level A)	X	X		X
Recommend based on inconsistent outcomes (Level B)			X	
Do not recommend (Level C)				

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the prognosis is less than that on total joint replacement. (Fig 22E).

The levels of evidence with regard to the purported efficacy of various surgical procedures should be considered when recommending any particular treatment. There two important questions that must be addressed. The first is what is the level of evidence with regard to any particular surgical procedure. The second question should address what the efficacy is of particular surgical procedure given the level of evidence. The levels of evidence can be categorized. (Fig 23).

The current levels of evidence supporting or refuting the efficacy of the use of various surgical procedures for IPD vary depending on the procedure (Table 6)

Current levels of evidence supporting or refuting the efficacy of various intraarticular medications following arthrocentesis or arthroscopy.[185-188] (Table 7)

PROGRESSIVE CONDYLAR RESORPTION (WITH INTRA-ARTICULAR PAIN AND/OR REDUCED RANGE OF MOTION; MANDIBULAR RETRUSION, APERTOGNATHIA OR A COMBINATION)

The evidence to support the use of medical management to arrest progressive condylar resorption is largely theoretical and anecdotal. The theory behind medical management is to reduce Tumor Necrosis Factor alpha (TNF-alpha), inflammatory cytokines and metalloproteinases (MMP) which are thought to result in increased condylar resorption. Medications to be considered include the following.

- Omega-3 fatty acids
- Vitamin D
- Tetracyclines
- Non-steroidal anti-inflammatory drugs
- Statins
- TNF-alpha inhibitors (adalimumab, certolizumab, etanercept, golimumab, infliximab etc.)
- Rank ligand inhibitors (denosumab)
- Bisphosphonates

Condylar resorption that results in joint related pain and/or decreased function requires treatment. Although arthrocentesis and arthroscopy can address some pathology within the superior joint space resulting in pain reduction and improved range of motion, they cannot address continued condylar resorption. Temporomandibular joint reconstruction is a valuable and predictable treatment that can be combined with orthognathic surgery to achieve pain reduction, improved function and esthetic facial harmony. (Fig 24).

Table 7. EFFICACY OF INTRAARTICULAR MEDICATIONS

Medication	Hyaluronic Acid	Corticosteroid	PDGF, PRP, PRF, growth factors	Micro fragmented Fat
Levels of Evidence	6	6	2	2
Recommend based on consistent outcomes (Level A)				
Recommend based on inconsistent outcomes (Level B)	X			
Do not recommend (Level C)		X	X	X

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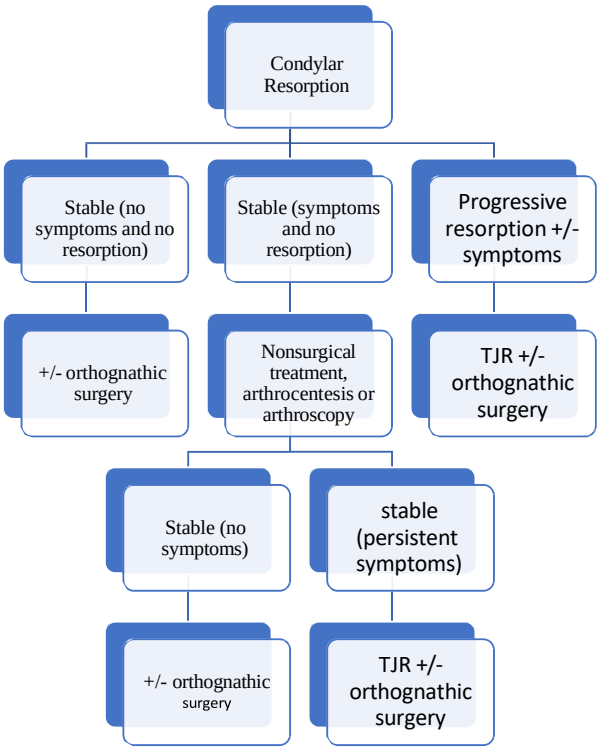

FIGURE 24. Progressive condylar resorption.

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FIBROUS AND BONY ANKYLOSIS

The distinction between fibrous and bony ankylosis can be made based on the maximum incisal opening, the presence of a hard or soft end feel and, imaging. There is a tendency for fibrous ankylosis to progress to bony ankylosis over timeinsomepatients. The tendency to reankylosis is significantly higher with autogenous reconstruction versus TJR. This is due to several factors including the creation of a critical bone gap (2 cm), fat grafting and earlier mobilization with the latter. The need to perform unilateral/bilateral coronoidectomy at the same time as addressing the temporomandibular joints is not uncommon. The compliance with post-operative jaw exercises is also critical to the long-term outcome. (Fig 25).

HYPERMOBILITY

Hypermobility can result in subluxation with little impact on a patient's quality of life. It may also result in pain, limited opening and a reduced quality of life. Dislocation requires manual reduction, often under sedation, and when recurrent or chronic has a significant negative impact on quality of life. Patient age, comorbidities and the frequency of dislocating episodes may all play a role in determining the most ideal treatment. The options for managing dislocation are numerous. (Fig 26).

Patients who have failed surgical management and continue to experience recurrent dislocation can be effectively managed with custom TJR which provides the opportunity to avulse the lateral pterygoid attachment at the mandibular fovea as well as design a large anterior lip within the ultra-high molecular weight polyethylene fossa. TJR maybe the initial treatment of choice in patients with connective tissue disorders such as Ehlers Danlos syndrome.

CONDYLAR HYPERPLASIA

The diagnosis of condylar hyperplasia can be challenging depending on the degree of hyperplasia and the associated mandibular and maxillary asymmetry. Even more challenging is determining whether the condylar hyperplasia is active. Non-active condylar hyperplasia does not require temporomandibular joint surgery unless in of itself it is associated with joint pain and or reduced MIO and has failed to respond to non-surgical management. Serial clinical examinations: panoramic, lateral and PA cephalometric radiographs and changes in the occlusal relationship

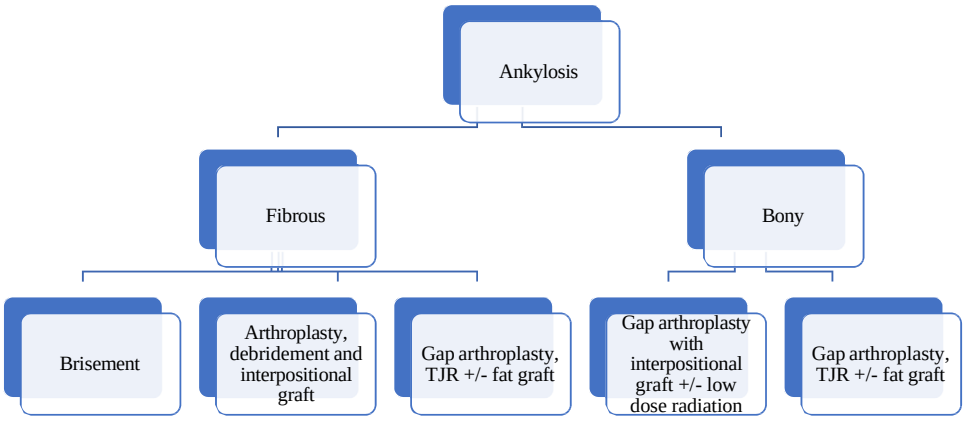

FIGURE 25. Fibrous and bony ankylosis.

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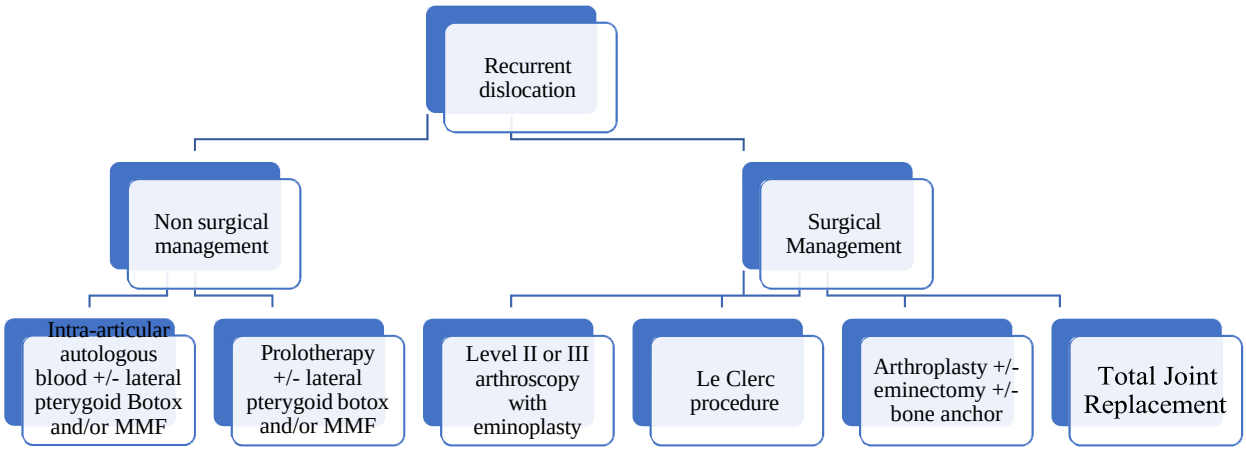

FIGURE 26. Hypermobility.

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overtime are all helpful to assess growth activity. Bone scintigraphy is also helpful although the test specificity is often not high enough to separate active condylar growth from a non-growing but enlarged condyle. Active condylar hyperplasia will result in progressive mandibular asymmetry which may also be associated with concomitant maxillary asymmetry and requires intervention in order to prevent continued growth and worsening asymmetry. (Fig 27).

Inflammatory Arthropathy

Rheumatoid arthritis, systemic lupus erythematosus, psoriatic arthritis and ankylosing spondylitis are the most common auto-immune conditions affecting the temporomandibular joints. Medical management will often result in a good response that limits pain and maximizes range of motion. Despite optimal medical management some patients will progress with joint pain, limited range of motion and joint destruction. The temporomandibular joint is not spared from inflammatory arthropathy and often requires treatment. It is not always possible to determine which TMJ signs and symptoms are the result of the patients inflammatory arthropathy versus osteoarthritis which may coexist. MRI with or without gadolinium is helpful to identify synovitis which has a relatively high positive predictive value for inflammatory arthropathy (Fig 28).

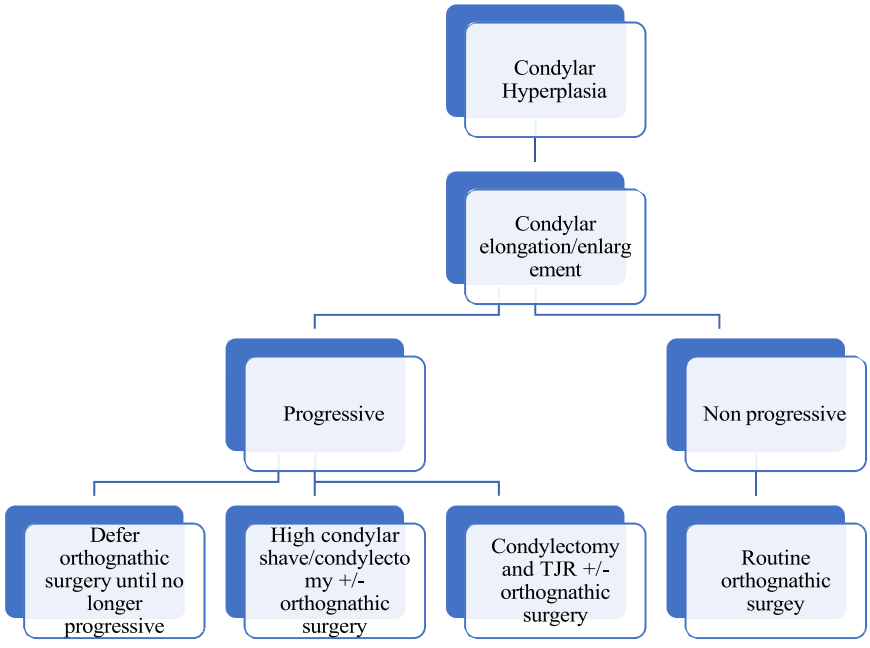

FIGURE 27. Condylar hyperplasia.

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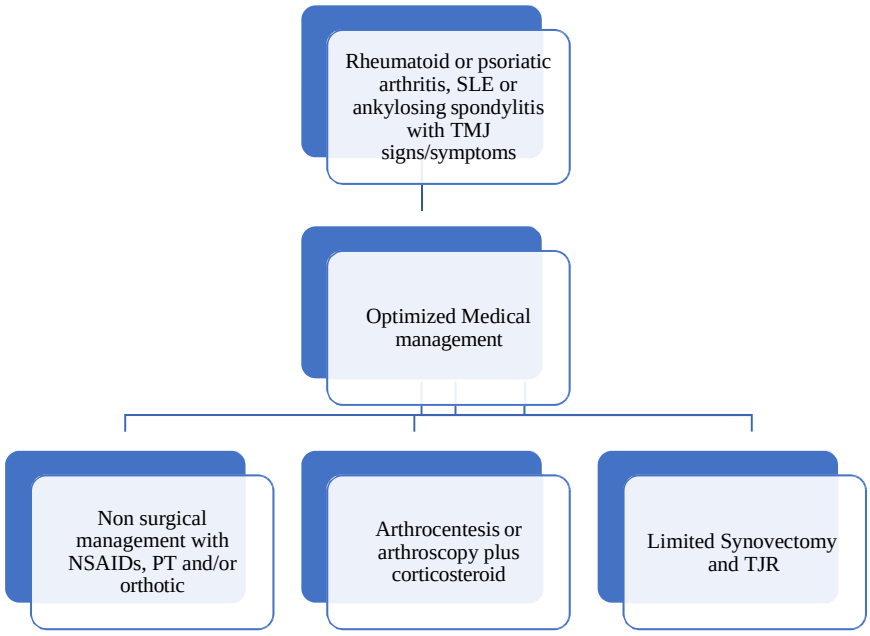

FIGURE 28. Inflammatory arthropathy.

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Hemifacial Microsomia/Goldenhar Syndrome

Hemifacial microsomia has the potential to affect the orbit, mandible, ear, facial nerve and soft tissues. Treatment will be influenced by many factors including the severity of the disorder in any particular patient. Pruzansky (1969) classified patient according to the degree of the bony abnormality. This was further modified by Kaban (1988) (Fig 29).

Condyle Fractures

Fractures of one or both mandibular condyles presents a challenge in management. Fractures may be treated with closed reduction, ORIF or a combination of these. Although the majority of extracapsular fractures can be treated satisfactorily with ORIF, this is less predictable for intracapsular fractures. This is the result of the relatively small fracture segments and soft medullary bone which combine to make

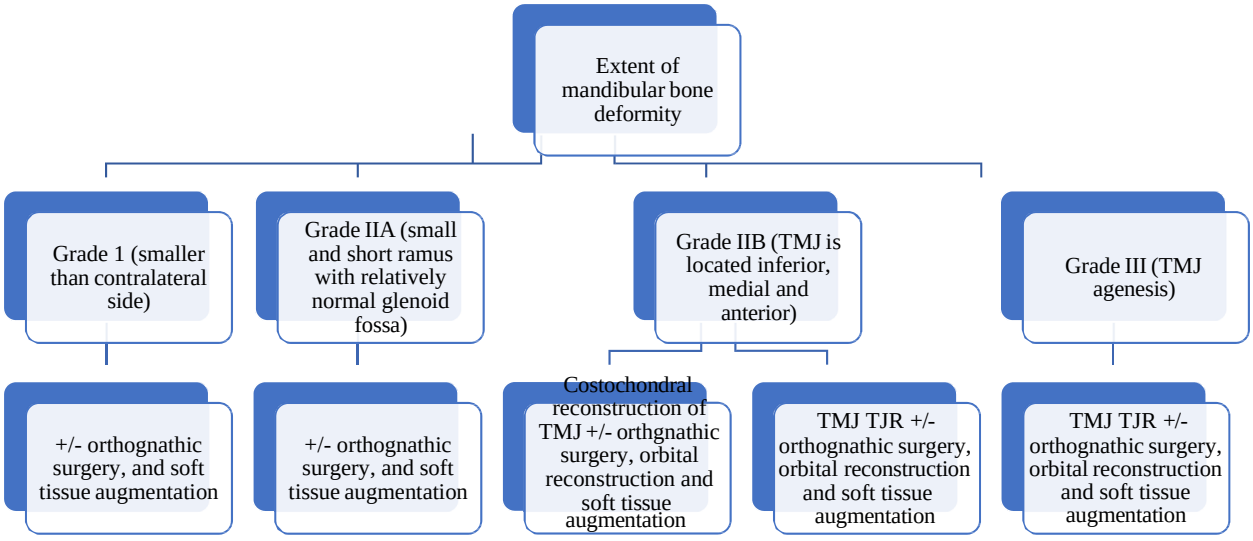

FIGURE 29. Hemifacial microsomia.

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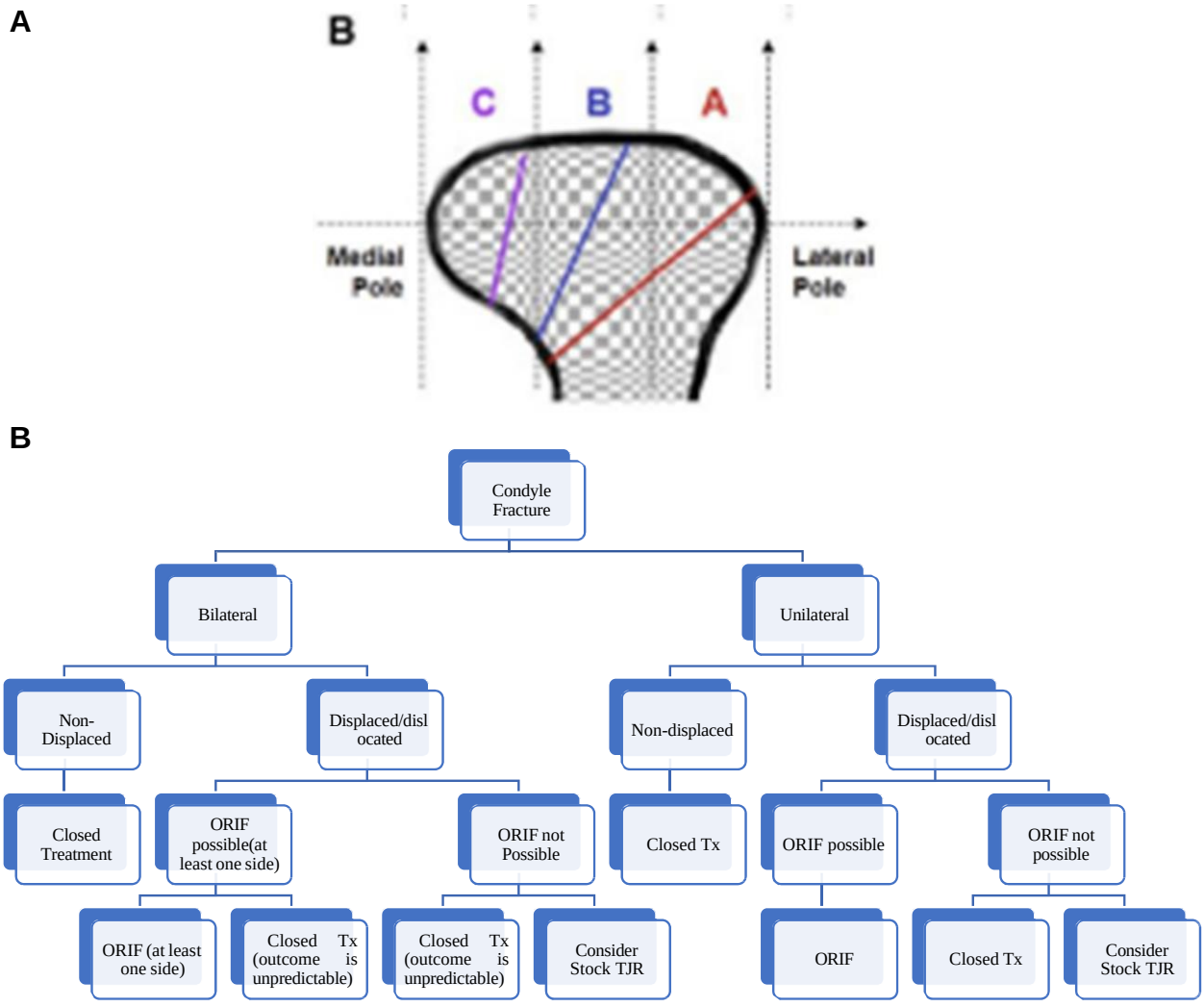

FIGURE 30. *A*, Intracapsular fracture patterns. *B*, Adult condyle fractures. Used with permission and adapted from: He D, Yang C, Chen M, Jiang B, Wang. Intracapsular condylar fracture of the mandible: our classification and open treatment experience. J Oral Maxillofac Surg. 2009;67(8):1672-9.

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ORIF challenging despite the use of position screws and/or small titanium plates. Intracapsular fractures that only involve the medial one or two-thirds of the condylar head will typically not result in a loss or ramal height due to the intact lateral pole (Fracture lines B and C below). This will often maintain the occlusion and eliminate the need for ORIF. Fractures involving both medial and lateral poles may result in a loss of ramal height with corresponding malocclusion (Fracture line C) (Fig 30A).

Historically the approach to displaced or dislocated condyle fractures in adults has been controversial with some advocating closed treatment and others open reduction and internal fixation. However, several systematic reviews and metanalysis over the last 10 years has shown clearly that when long-term outcomes including pain, function and deviation are measured, ORIF is superior to closed Tx.[189,190] (Fig 30B).

Important Considerations which *Favor ORIF*

- Bilateral displaced fractures
- Displacement into the external auditory canal, middle cranial fossa or lateral displacement
- Loss of posterior facial height
- Concomitant midface fractures (maxillary)
- Unstable occlusion
- Poor dentition
- Aspiration risk or seizure disorder
- Long-term outcomes for pain and function

Chapter 8: Pediatric Considerations

Objectives:

- Appreciate growth and development of the facial skeleton in the pediatric patient

- Appreciate the different diseases and disorders that affect the pediatric patient
- Appreciate differences in treatment needs and timing of intervention in the pediatric patient.

Growth of the Mandible and Maxilla

The mandible is the last bone in the human body to reach skeletal maturity. Any condition that affects the condyle of the mandible prior to skeletal maturity may result in an alteration of normal mandibular growth. Normal growth of the mandible is considered to be in a downward and forward direction in relation to the skull base and the maxilla. Appositional bone formation occurs in the condyle in an upward and backward direction to allow maintenance of the condyle in the glenoid fossa leading to the normal vertical height of the ramus/condyle unit. The two condyles of the mandible function together as one unit. Alteration in the growth of one condyle will lead to facial asymmetry.[191] When the vertical growth of the mandible is restricted, the downward growth of the maxilla is also restricted resulting in both maxillary and mandibular asymmetry.

Craniofacial growth including growth of the mandible is a result of genetic and epigenetic factors. Epigenetic factors include all the extrinsic, extra-organismal, macro environmental factors impinging on vital structures (for example, food, light, temperature, mechanical loading, and electromagnetic field), and all the intrinsic, intra-organismal, biophysical, biomechanical, biochemical, and bioelectric micro-environmental events occurring in and between individual cells, extracellular materials and extracellular substances. As a result of these epigenetic factors, functional loading of the craniofacial skeleton including the maxilla and mandible leads to appositional growth that is not reliant on a primary growth center (condyle). This is thought to occur as a result of deformation of the extracellular matrix and cell shapes which leads to changes in gene expression, increased cellular activity and ontogenesis.[192-195] It is therefore possible for mandibular growth to occur despite genetic or acquired abnormalities of the temporomandibular joint. This should be taken into consideration during the reconstruction of the mandibular condyle in the growing patient.

History and Physical Exam in the Pediatric Population

Although physical examination of the adult and pediatric is similar, the presence of facial pain in the pediatric patient necessitates that the parent or caregiver be present. This allows for the establishment of trust and openness. However, the potential for the parent or caregiver to adversely influence the history and physical examination should be kept in mind and may necessitate reevaluation at another time without the parent or caregiver present.[196,197]

The evaluation of children with craniofacial pain should include the use of a standard survey form that elicits nature, timing, and triggers of the pain. Triggers of the pain may differ in children. Changes in sleep hygiene, meals, nutrition, lights, noises, weather, or menstrual cycle should be considered as potential triggers.[198] Assessment for learning disability and possible social stressors such as bullying should be noted. Changes in sleep pattern, school performance, school attendance may also be markers of pain.[199] The physical exam in the pediatric population serves to rule out focal, neurologic, or intracranial source of pain or pinpoint the organic source of pain and follows the adult examination.[200,201]

Conditions Affecting the TMJ in the Pediatric Population

CONGENITAL ABNORMALITIES

1. Treacher Collins

Also known as mandibulofacial dysostosis is an autosomal dominant dominate genetic disorder that affects the structures of the first and second branchial arches bilaterally. Both male and females are affected equally, and the incidence of the condition is 1 in every 25,000-50000 live birth.[202]

The facial structure anomalies develop during the 12th day and the 20th week of gestation and the degree of malformation at birth is stable and does not progress with age. Clinical findings in these patients include colobomas and hypoplasia of the lower eye lid and lateral canthi leading to down slopping slant of the eyes, hypoplastic orbits, dystopia, malformed ears to variable degrees, hypoplastic zygoma, maxilla and mandible. The TMJ and muscles of mastication are variably affected.

2. Hemifacial Microsomia

Also known as first and second branchial arch syndrome, otomandibular dysostosis, craniofacial microsomia, lateral facial dysplasia is hypothesized to be an aplasia or hypoplasia of the structures of the first and second branchial arches due to disrupted blood supply to the brachial arches due to hematoma of the stapedial artery.[203,204] The skeletal, soft tissue and neuromuscular components of the lower face are affected asymmetrically. The incidence of the condition is 1 in 3500 to 5600 births.[13,14]

The clinical findings could be classified using the OMENS classification: O (orbital distortion), M (mandibular deficiency), E(ear anomaly), N (neural

involvement), S (soft tissue deficiency). The Kaban classification system specifically define the degree of the TMJ and mandibular deficiency in Hemifacial Microsomia.

3. Oculoauriculovertebral Syndrome (OAVS)

OAVS is linked to multiple chromosomal abnormalities including but not limited to 5q deletion, trisomy 18, and 7q duplications. It occurs in 1 in 5600 live births.[15] It is characterized by mandibular hypoplasia, mandibular asymmetry, epibulbar dermoid and vertebral anomalies and is consider a variation of hemifacial microsomia.[205]

4. Oculomandibulodyscephaly (OMD)

Also known as Hallermann-Streiff Syndrome. It is rare (1 in 200,000 live births) and occurs sporadically. It is characterized with bird like craniofacial features such as prominent, thin, pointed nose, bilateral congenital cataracts, blue sclera, nystagmus, bilateral microphthalmia, proportionate nanism, small stature, hypotrichosis, cutaneous atrophy, dental anomalies, frontal and parietal bossing, cranial suture dehiscence with open fontanelles, high arched palate, microstomia, and mandibular hypoplasia with anterior displacement of the mandibular condyles.[206]

5. Nager Syndrome

Nager Syndrome is a form of acrofacial dysostosis. It shares with Treacher Collin Syndrome the craniofacial findings in addition to upper limb abnormalities. The upper limb abnormalities are limited to the anterior forelimb. Nager Syndrome may be sporadic or autosomal dominate or recessively inherited.[207]

ACQUIRED ABNORMALITIES

Trauma

Intracapsular and subcondylar fractures of the mandible are the most common type of mandibular fractures in the pediatric population. Growth disturbances may occur after the traumatic injury. In unilateral injury, premature posterior contact in the affected side may occur with loss of posterior facial height, deviation of the chin point to the affected side and limited lateral excursion to the contralateral side. Bilateral injury may result in retrognathia and anterior open bite.[208]

The treatment of the fracture depends on the age of patient, stage of dental development, type of fracture, degree of fracture displacement and patient cooperation. Non-surgical or closed treatment is favored for those less than 10 years of age. Restitutional remodeling of the mandibular condyle is common, and growth is typically normal. Early mobilization and function are the most critical factor in this patient populations. Patients older than 18 years of age may be treated with closed or open reduction and internal fixation. ORIF is generally associated with better outcomes in terms of pain and function. The management of factures in pediatric patients between ages of 10 and 18 years is less clear. It is reasonable to approach fractures in this age group by favoring closed treatment for those closer to 10 years of age and open treatment for those closer to 18 years of age.[209]

Tumors

Tumors of the TMJ are uncommon in the pediatric population. Patient may present with classic signs and symptoms of TMJ dysfunction. Diagnosis requires a biopsy. Once diagnosis is confirmed, surgical management is dictated by the diagnosis.[210]

JUVENILE IDIOPATHIC ARTHRITIS (JIA)

JIA is the most common rheumatologic disease in the pediatric population. It consists of a group of systemic inflammatory conditions including oligoarthritis, rheumatoid factor positive, rheumatoid factor negative polyarthritis, psoriatic arthritis, enthesis-related arthritis, and undifferentiated arthritis. It is characterized by inflammation of the synovium of a single or multiple joints leading to the destruction of hard and soft tissues of the joint/joints.[211]

TMJ involvement occurs in 40% of patient diagnosed with JIA.[212] Patients with JIA with at least 2 of the following signs/symptoms involving the TMJ may have TMJ involvement even without radiologic findings: joint swelling, joint pain with motion, limited joint motion and heat over the joint for more than 6 weeks.[213]

Imaging studies helpful in the diagnosis of TMJ involvement in JIA.[214]

1 MRI with contrast (gadolinium)-increased synovium enhancement indicates synovitis and joint involvement.
2 Plain radiograph-skeletal abnormalities may not be evident early stages.
3 CT-bony erosion and joint space narrowing may be seen.

Goals of Treatment

Maintain optimal joint function, reducing joint symptoms and preventing permanent anatomic damage using systemic (NSAIDs, disease modifying agents, biologics) measures as first line treatment. Local measures such as intra-articular steroid injections as second line treatment.[215] Judicious use of intra-articular steroid injection is recommended as inhibition of growth, heterotopic ossification and joint destruction

may occur. In severely affected TMJ, open joint surgery with total alloplastic TMJ replacement is recommended.[216]

TMJ ANKYLOSIS

When one joint is ankylosed, restricted mandibular movement with deflection to the affected side occurs due to intra-articular fibrous adhesions or bony fusion of the mandible to the skull base. Fibrous ankylosis often occurs in the superior compartment.[217] TMJ ankylosis is commonly an acquired condition as result of trauma at birth or during childhood, septic arthritis, or systemic infections such as smallpox, syphilis or tuberculosis.[28] Congenital ankylosis is rare.

Pain and joint sounds are often absent. Patients with ankylosis presents with progressive limited mouth opening, limited laterotrusion to the contralateral side or no laterotrusion if condition is bilateral. In addition, the ankylosis results in undergrowth of the mandible if it occurs in growing patient. Unilateral ankylosis results in deviation of the chin point to the affected side.

Early surgical intervention is recommended in children to minimize the progressive dentofacial deformities that results from ankylosis. Surgical intervention in children includes total resection of the ankylotic mass, ipsilateral or bilateral coronoidectomy to allow for adequate MIO, reconstruction of the temporomandibular joint either with autologous grafts such as interpositional temporalis myofascial flap and costochondral grafting, alloplastic total joint replacement, or distraction osteogenesis of the reshaped mandibular stump. Early post-operative mobilization and aggressive physical therapy is essential in preventing re-ankylosis.[218-220]

POST RADIATION THERAPY DEFORMITIES

Radiation therapy for malignant tumors may affect the soft tissue and bony tissue of a growing patient. Radiation treatment, including to the condyles, may result in micrognathia, facial asymmetry, limited jaw movement, TMJ ankylosis, soft tissue fibrosis, and dental anomalies.[221,222] Due to the negative effects of the radiation therapy to the vasculature and angiogenesis, surgical reconstruction may require free vascularized flaps.[223,224]

PEDIATRIC CONSIDERATION IN THE NON-SURGICAL MANAGEMENT OF TMD

Non-surgical modalities may be employed in the management of intraarticular pain and dysfunction (IPD) and myofascial pain dysfunction in the pediatric population. The modalities available include patient education, behavior modification, biobehavioral therapy, physical therapy, oral appliances, and pharmacotherapy. These options are similar to the non-surgical management of adult patients.

Caution should be taken when oral appliances are utilized. Hard material devices may interfere with craniofacial and odontogenic development and growth of children with primary and mixed dentition. Short term use of oral appliances made with soft material minimizes adverse tooth movement in children with primary and mixed dentition.[225]

PEDIATRIC CONSIDERATION IN THE SURGICAL MANAGEMENT OF TMJ PATHOLOGY AND TMJ RECONSTRUCTION

1. Arthrocentesis and arthroscopy remain excellent minimally invasive surgical modalities for the management of intra-articular pathology including IPD and JIA. The use of adjunctive medication such as corticosteroids is not contraindicated in the pediatric patient. However, the potential for cartilage and bone destruction, particularly with repeated use is a concern. In addition, extravasated corticosteroid may result in lipolysis leading to temporal hollowing.
2. Mandibular and Maxillary Osteotomies
   a Conventional osteotomies are recommended for the correction of TMJ related deformities if the mandibular condyle and disc are functional and a reproducible centric relationship can be accomplished.
   b Osteotomies such a sagittal split osteotomy, extra or intraoral vertical ramus osteotomy and inverted L osteotomy are employed and allow for preservation of the growth potential of the mandible, though normal growth may not be expected, depending on the condition that leads to the reconstruction.
   c When ramus lengthening and rotation is required on one side, an osteotomy should also be made on the contralateral side if excessive torquing of the contralateral normal joint will result without an osteotomy.

3 Distraction Osteogenesis
   a Distraction Osteogenesis is recommended when significant soft and bony tissue reconstruction are required in a growing child. It avoids a donor site.
   b There are many devices available for mandibular distraction osteogenesis, single vector, multi-vectors and curvilinear, the device used depends on the treatment goal.

4. Costochondral Grafts and Free Fibula Flaps
   a Costochondral grafting is the most common autologous reconstruction used for the TMJ. The non-vascularized graft is subject to resorption over time and especially when there is also a deficient soft tissue envelope such as in hemifacial microsomia.
   b Free tissue transfer with vascularized flaps such as the free fibula flaps are more resistant to resorption and recommended when large soft tissue and bony defects are present.
   c Both non-vascularized and vascularized reconstruction are subject to ankylosis or unpredictable growth/resorption. The risk of ankylosis can be mitigated by maintaining the articular disc or the use of a pedicled temporalis muscle/fascia flap. Costochondral grafts and free vascularized fibular reconstructions will permit mandibular growth secondary to appositional growth. Costochondral grafts can result in excessive growth even when the cartilage cap is limited to 2- 4 mm of cartilage.
5. Alloplastic Total joint Replacement
   a Alloplastic total joint replacement is recommended for progressive resorptive conditions of the TMJ or when reproducible centric relationship cannot be achieved with conventional osteotomy.
   b Historically, alloplastic total joint reconstruction in the pediatric population was considered less than ideal due to the perceived lack of growth of the prostheses and potential for asymmetry. This can be managed by replacement of the condylar component or with the use of conventional osteotomies (eg, sagittal splint osteotomy). Interestingly, recent data have shown that alloplastic total joints placed in skeletally immature patients may not lead to skeletal deformity and asymmetry. This is likely the result of establishing normal TMJ function combined with epigenetic factors and the functional matrix theory.

Chapter 9: Temporomandibular Joint Procedures

The actual surgical techniques for each of the following procedures will differ depending on the individual patient and the individual surgeon's personal technique. The techniques described below are surgeon specific examples of how to safely perform these procedures. It is expected that the actual techniques used will vary between surgeons and the descriptions of the surgical technique below are NOT intended to represent the only acceptable technique or standard of care for arthrocentesis, arthroscopy, arthroplasty or total joint replacement. It is recommended that consideration be given to perform an otoscopic examination immediately before and after arthroscopy, arthroplasty and TJR to document the health and patency of the tympanic membrane and external ear canal prior to and following procedures.

TMJ Arthrocentesis[226]

Basic Armamentarium
Iodine swab (2)
Local anesthetic such as 2% lidocaine with 1:100,000 epinephrine (dental cartridge) with 27-gauge hypodermic needle
22-gauge 1.5-inch hypodermic needle (2)
Lactated Ringers Solution
60 cc Syringe
Intravenous Tubing (12 inch)
Small round band-aid (2)

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PROCEDURE

Arthrocentesis can be performed under local anesthesia either with or without deep sedation/general anesthesia. The consent should include the potential for temporary weakness of the facial nerve. There are many technical variations for performing the procedure, butall revolve around the same basic steps. The use of the Holmlund-Hellsing (HH) line has traditionally been advocated to identify the posterior and anterior needle puncture sites. A line is drawn from the lateral canthus of the eye to the mid-tragus. The posterior puncture site is located 10 mm anterior to the posterior edge of the tragus and 2 mm inferior to the HH line. The anterior puncture site is an additional 10 mm anteriorly along the HH line and 10 mm inferior. These puncture sites correspond to the posterior recess and apex of the articular eminence but are much more suited for arthroscopy. A relatively simple technique for arthrocentesis is:

1 Place a disposable surgical head cap of the patient to hold the hair out of the surgical field. The additional use of surgical tape to secure the edge of the cap to the skin at a level near the pinna of the ear is very helpful (Fig 31A).
2 Use the nail of the index finger to identify the lip of the glenoid fossa and posterior slope of the articular eminence by pressing moderately firmly against the skin. Mark the outline carefully with a skin marker ensuring accuracy (Fig 31B).

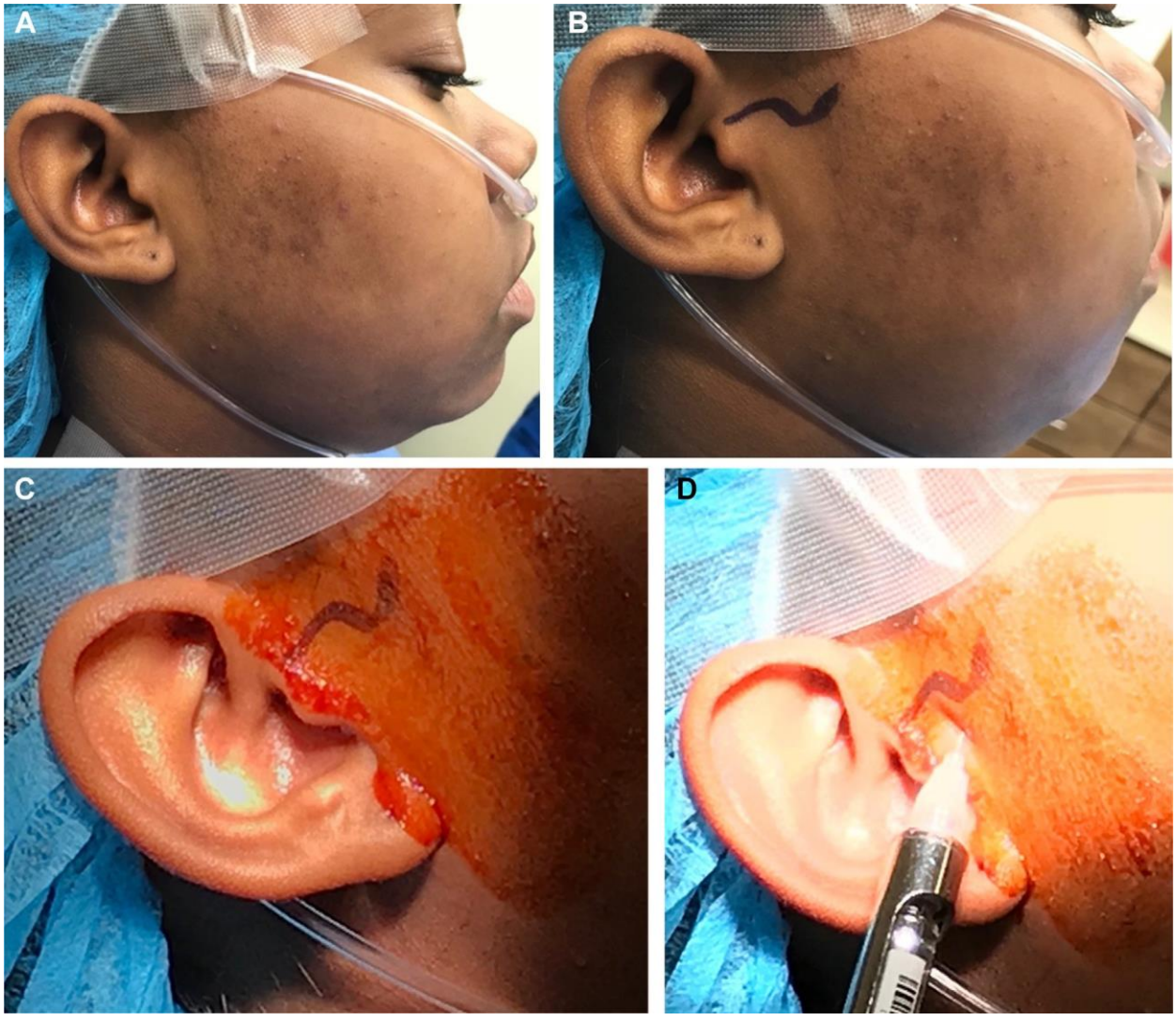

FIGURE 31. *A-I*, arthrocentesis. (Fig 31 continued on next page.)

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3 Place a mark at the junction of the midpoint of the tragus and outlined lip of the glenoid fossa. This is the initial puncture site for the posterior needle.
4 Paint the skin with iodine (Fig 31C).
5 Place the tip of the local anesthetic needle over the posterior puncture site. Angle the needle approximately 15º anteriorly and superiorly. Gently puncture the skin and begin to inject 2% lidocaine with 1:100, 000 epinephrine (1 dental cartridge) immediately under the skin to form a weal. Allow the anesthetic to diffuse and then gently advance the needle while depositing local anesthesia. Resistance will be felt when the joint capsule is encountered. Gently puncture the capsule and advance the needle gently until the glenoid fossa roof is encountered. Be gentle to avoid scuffing the fossa. A 1.5-inch needle should be at a minimal depth of approximately $^{3}/_{4}$ to 1 inch to ensure correct positioning. The remaining half of the dental cartridge should be injected into the superior joint space before removing the needle (Fig 31D).
6 If the local anesthetic needle encounters bone too prematurely based on the depth of the needle the most likely cause is an excessive superior angle striking the lip of the glenoid fossa or an excessive inferior angle striking the lateral pole of the condyle. Adjust the needle accordingly.
7 Wait several minutes to allow adequate anesthesia.
8 Insert a 22-gauge needle using the same puncture site and trajectory that was used for the local anesthesia. This needle has a larger

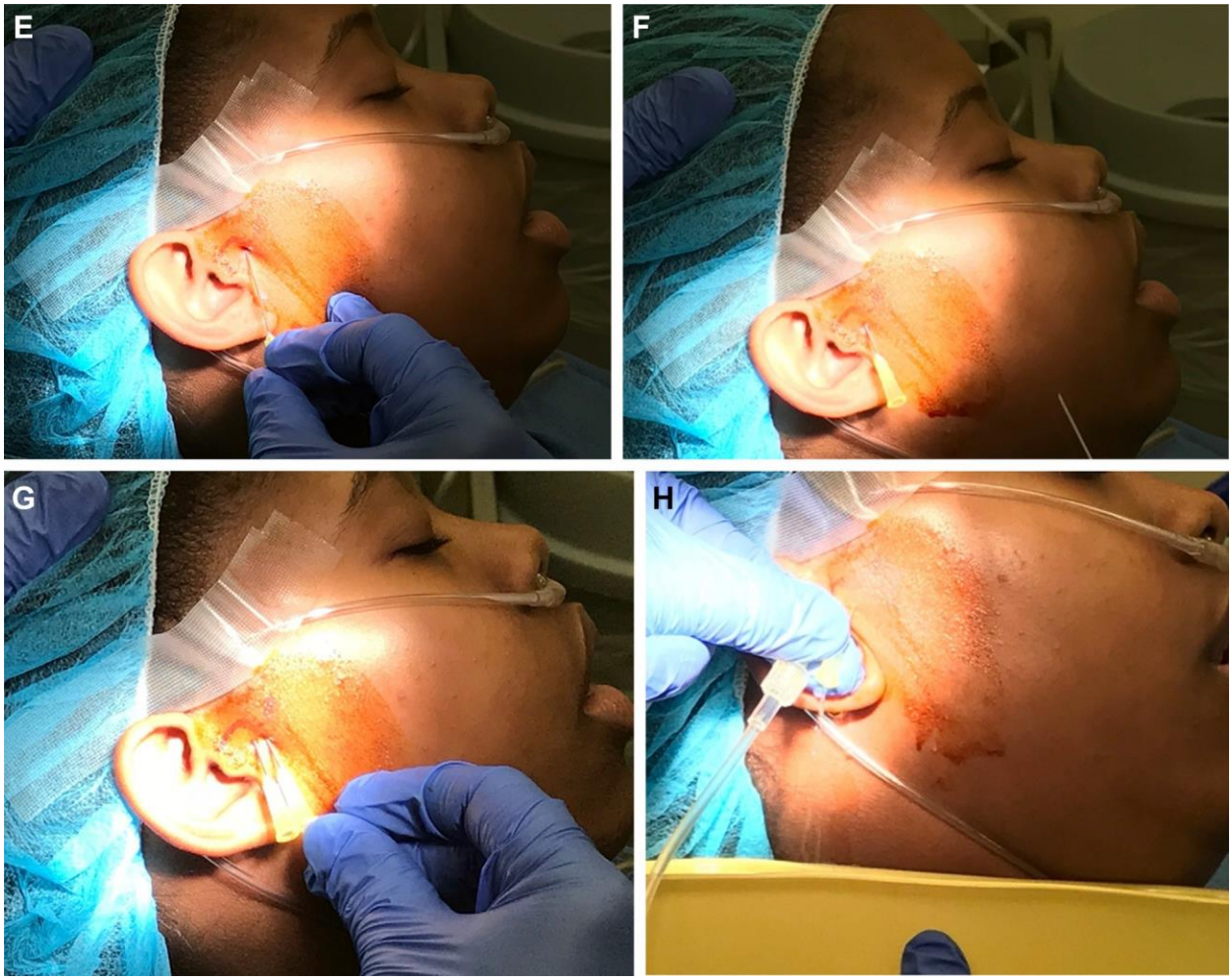

FIGURE 31 (cont'd). (Fig 31 continued on next page.)

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diameter but should also pass through the capsule and gently strike the roof of the glenoid fossa (Fig 31E and F).

9 Take a second 22-gauge needle and insert it 3-4 mm anterior to the posterior needle being sure to keep the needle parallel to the first needle at all times. The needle will also encounter resistance as it passes through the capsule before gently striking the roof of the glenoid fossa (Fig 31G).

10 Take a 60-cc syringe/12-inch tubing filled with Lactated ringers' solution and connect to the hub of the one needle. Hold the hub firmly to prevent undue movement which can scuff the fossa.

11 Irrigate the full 60 cc of LR while ensuring the outflow from the other needle. If irrigation is difficult take a moment to advance both needles to ensure they are resting on bone. Additionally, gently rotate one needle at a time to ensure the bevel is not pressed against intraarticular tissue. It is sometimes helpful to move the irrigating tubing between needles if irrigation is difficult (Fig 31H and I).

12 Irrigation should be with intermittent pressure to ensure a variation in intraarticular pressure which can disrupt a stuck disc and immature fibrous adhesions.

13 Manipulate the jaw to obtain maximum incisal opening and translation towards the end of the procedure prior to the last 10 cc of irrigation.

14 If unable to irrigate the majority of the 60 cc despite adjusting needles and manipulating the jaw, remove the anterior needle and instill 0.5 cc of corticosteroid through the posterior needle before removing.

15 Manipulate the jaw again to ensure maximal mouth opening and joint translation.

16 Place a small Band Aid over the puncture sites.

17 Ensure patients receive a written program of jaw exercises to be performed several times per day over several weeks.

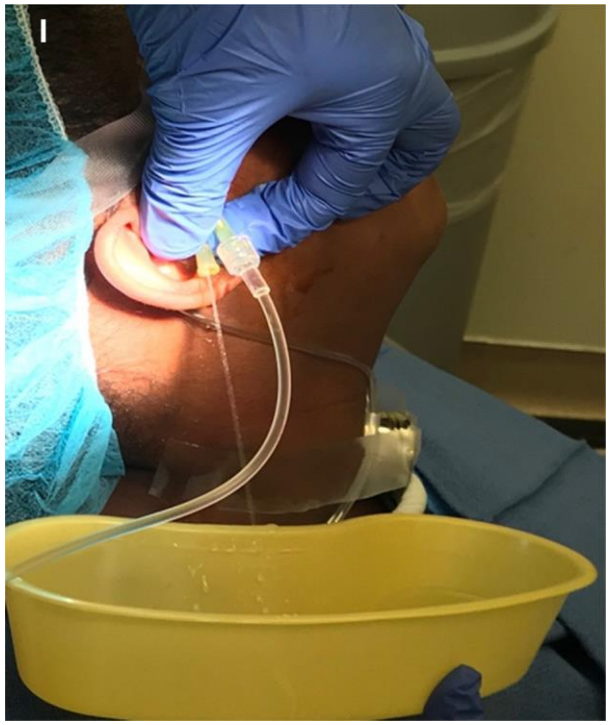

FIGURE 31 (cont'd).

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Arthroscopy[227]
Basic Armamentarium
1.9-mm TMJ arthroscope 30-degree angle
Arthroscopy tower: light source, HD camera, monitors
Two similar 2-mm scored cannula
Sharp trocar
Blunt obturator
Straight probe
French #3 myringotomy suction tip
IV Line, stop cock
Lactated ringer/1: 300,000-epinephrine irrigation fluid
Lavage needle, 22 gauge x 1.5 inches
Local anesthetic with vasoconstrictor
Appropriate sutures
Advanced Armamentarium
Basic armamentarium and the following:
2.4-mm suction punch
Biopsy forceps and needles
Meniscus mender set, with appropriate suture/wire, button
Other hand instruments: as bone rasps and bone curettes
Radiofrequency coblation micro debridement
Holmium YAG laser
Monopolar and bipolar electrocautery probes
Motorized instruments: full radius shaver

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Technique: Basic TMJ Arthroscopy (Single-Puncture Arthroscopy)

STEP 1

Patient Preparation

Primary arthroscopy is performed under local anesthesia or under local anesthesia and intravenous sedation or monitored anesthesia care (MAC). The patient is brought to the operating suite, placed in the dorsal supine position, and brought to the proper anesthesia plane for MAC. Examination under anesthesia should be performed to confirm or modify the preoperative diagnosis. The patient's head should be turned to the contralateral side to ensure the operative field is horizontal. The hair is placed in a bouffant cap and secured by silk tape. After digital localization of the greatest concavity of the glenoid fossa, the preauricular skin is prepped with Betadine and draped in the usual manner for TMJ arthroscopy, and ear wick is placed for protection. (Fig 32A).

STEP 2

Local Anesthesia

After standard landmarks have been marked, local anesthesia is deposited using 2% lidocaine with

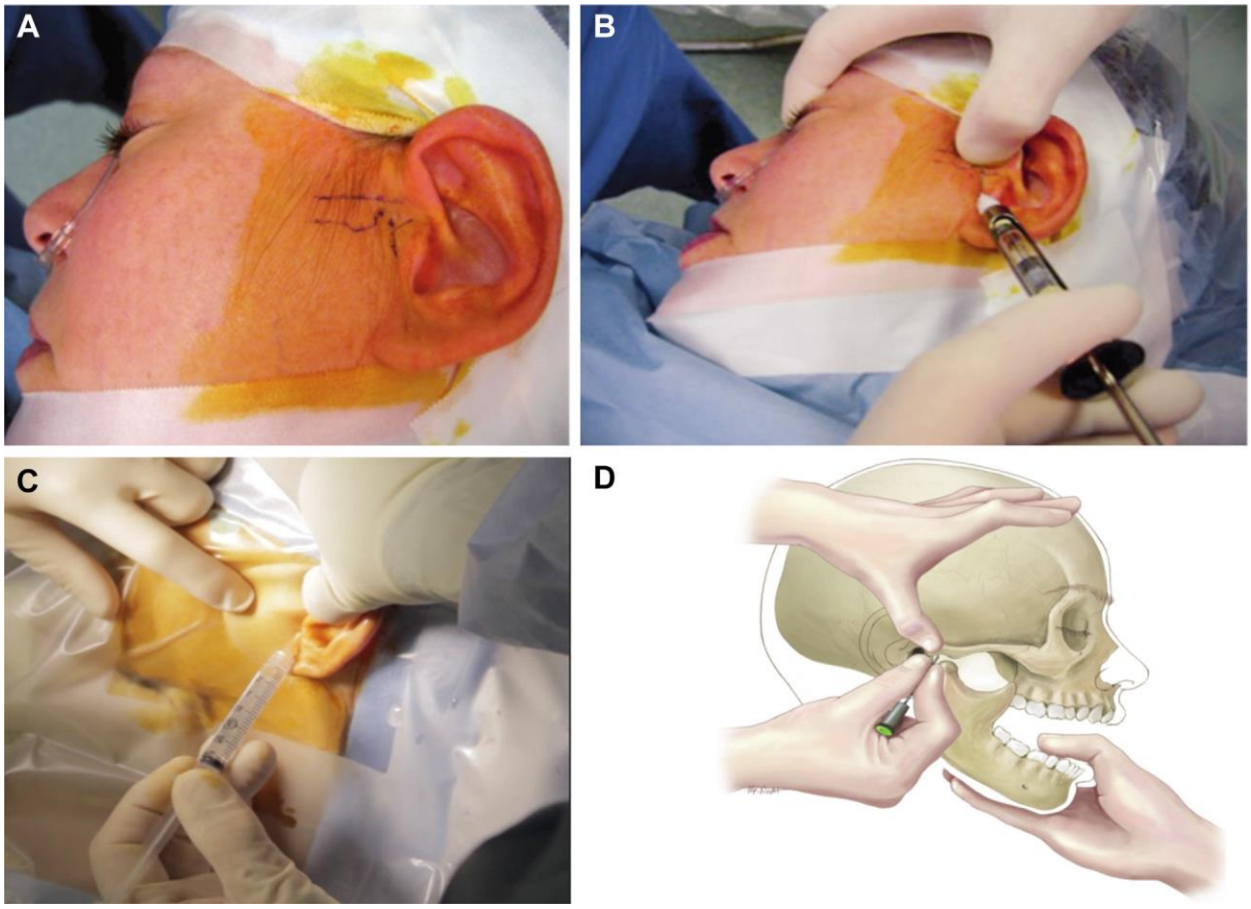

FIGURE 32. *A-G*, Single puncture arthroscopy. (Fig 32 continued on next page.)

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1:100,000 epinephrine using a 30-gauge needle in the preauricular area. (Fig 32B).

STEP 3

Insufflation

The superior joint space is then insufflated via an inferior and lateral approach utilizing a 30-gauge needle and insufflated with 2.5 cc of the local anesthesia ensuring good plunger rebound. (Fig 32C).

STEP 4

Fossa Portal Puncture

The first puncture is always placed at the maximum concavity of the glenoid fossa while the mandible is in protruded position. Hold the cannula/trocar in the dominant hand and grip the cannula with an index finger controlling the tip and the base of the cannula in the palm of the hand. Experienced TMJ arthroscopists may hold the trocar and puncture with the nondominant hand. Using the trocar, penetrate the skin at the fossa puncture site with a slow rotational motion. The fossa puncture should be made deliberately and carefully in an attempt to pass one time through the lateral capsule and into the joint space to avoid extravasation problems. Advance the trocar until contact is felt with the lip of the glenoid fossa. Always sound this bony landmark; never pass the instrument straight through the capsule without locating the bone. Allow the trocar to feel the junction of the lateral lip of the glenoid fossa and the superior aspect of the capsule. Advance the cannula completely into the joint space. At this point, the cannula should be inserted approximately 20 to 25 mm as measured from the skin to the center of the joint, which is known as the safety zone. Then remove the trocar and attach the scope. (Fig 32D).

STEP 5

Insertion of Outflow Needle

With mandible maintained in a protrusive position, instruct the surgical assistant to insufflate the joint with approximately 2 to 3 mL of fluid using the direct irrigation syringe and to maintain pressure on the plunger to retain distention of the joint. Then the surgeon inserts a 22-gauge, 1.5-inch needle approximately 5 mm anterior and 5 mm inferior to the fossa

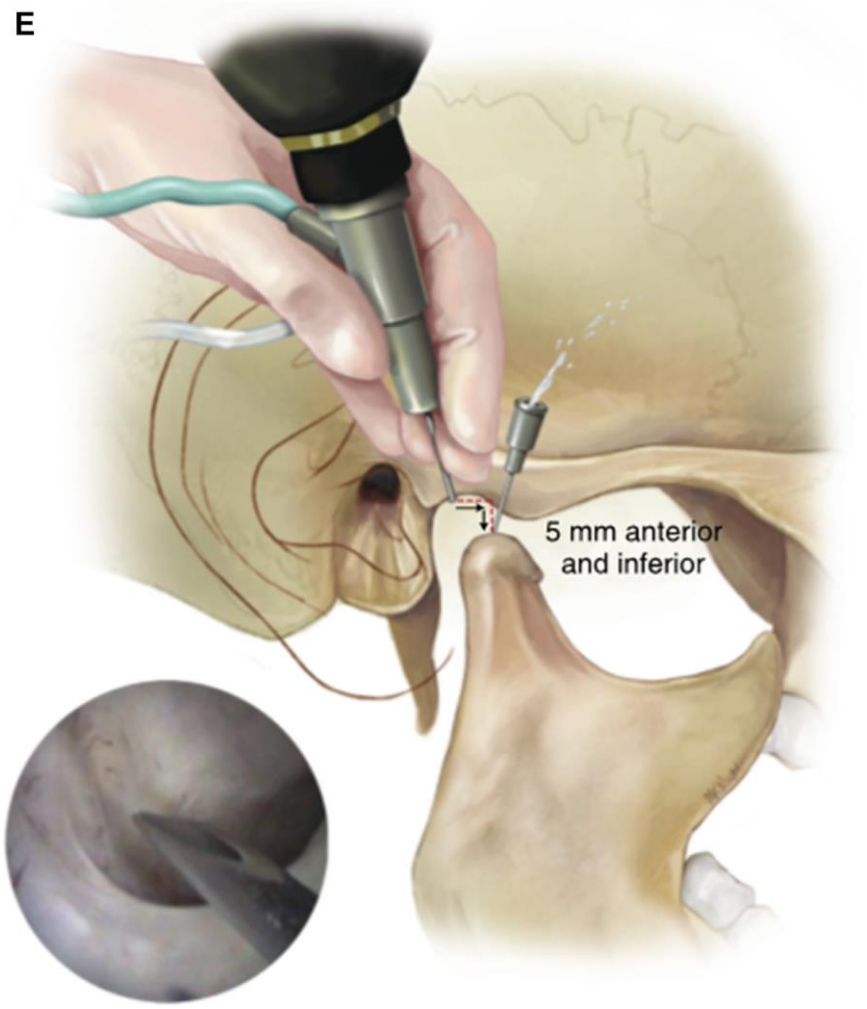

FIGURE 32 (cont'd). (Fig 32 continued on next page.)

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puncture site while observing the flow of irrigation fluid through the needle consisting of lactated Ringer's solution with 1:300,000 epinephrine. (Fig 32E).

STEP 6

First Level of Treatment

The first level of treatment begins with an arthroscopic arthrocentesis, which is done with a minimum of 120 cc of irrigation fluid with turbulent flow in order to remove inflammatory substrates as well as lysis of any small adhesions present.

This is followed by the diagnostic sweep providing an accurate diagnosis of normal and pathologic states in the superior joint space through describing the seven points of interest as well as joint motion. (Fig 32F).

STEP 7

Second Level of Treatment

The second level of treatment involves the instillation of intra-articular medications, as indicated in certain cases, including the following:

- Injection of sclerosing agents into the retrodiscal tissue crease in cases of dislocated discs, using the lavage needle.
- Injection of steroid agents into a severely inflamed retrodiscal tissue (Fig 32G).
- Intra-articular injection of hyaluronic acid acting as a Band-Aid for micro bleeders and synovial fluid replenishing agent as well as lubricant.
- Intra-articular injection of regenerative medicine agents. (platelet-rich plasma [PRP], stem cells, etc.)

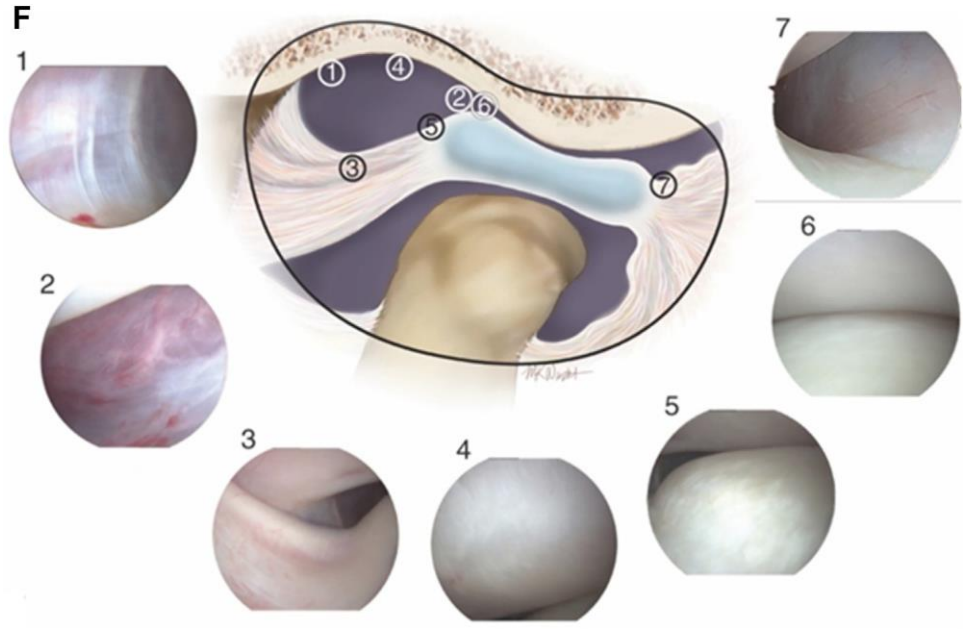

FIGURE 32 (cont'd). (Fig 32 continued on next page.)

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STEP 8

Closure

Once the procedure is complete, all instruments are removed while maintaining direct pressure over the puncture sites, and then the patient's head is elevated slightly to aid in hemostasis. The fossa puncture site is closed using single suture of 6-0 nylon and then covered with bacitracin ointment and a spot Band-Aid. Finally, the ear wick is removed.

STEP 9

Jaw Manipulation

With the patient's head faced upward, the surgeon manipulates and stretches the mandible, and the range of motion is recorded afterward.

Technique: Operative TMJ Arthroscopy (Double-Puncture Arthroscopy)

STEP 1

Intubation

The patient is placed in a dorsal supine position and brought to the proper plane of general anesthesia via nasotracheal intubation.

STEP 2

Patient Examination and Preparation

The patient is examined under anesthesia. Then the patient is positioned, prepped, and draped in the usual manner for TMJ arthroscopy. A sterile ear wick is placed in the ear for protection.

STEP 3

Fossa Portal Puncture with Lysis and Lavage

Steps 2 through 6 of the primary arthroscopy technique are repeated.

STEP 4

Second Cannula Puncture

After completion of the diagnostic sweep, the second puncture needs to be placed exactly in the most anterior and lateral corner of the superior joint space to ensure maximum flexibility of the operative cannula. While the condyle is being seated in the fossa, the irrigation needle is removed and then the puncture site is located according to triangulation principles, creating an equilateral triangle in the following manner:

- A second measuring cannula is positioned flat against the tegument with the tip (0-mm marking) contiguous with the scope at the point of entry (skin) and continuous (in a straight line) with the plane of the arthroscope. The depth of the scope penetration is now translated to the cannula.
- The site for the second puncture has now been established. In a fashion similar to that used for the fossa puncture, the assistant insufflates the joint with 2 mL of irrigation fluid. The sounding 22-gauge needle then trocar/cannula penetrate perpendicular to the tegument then continue in

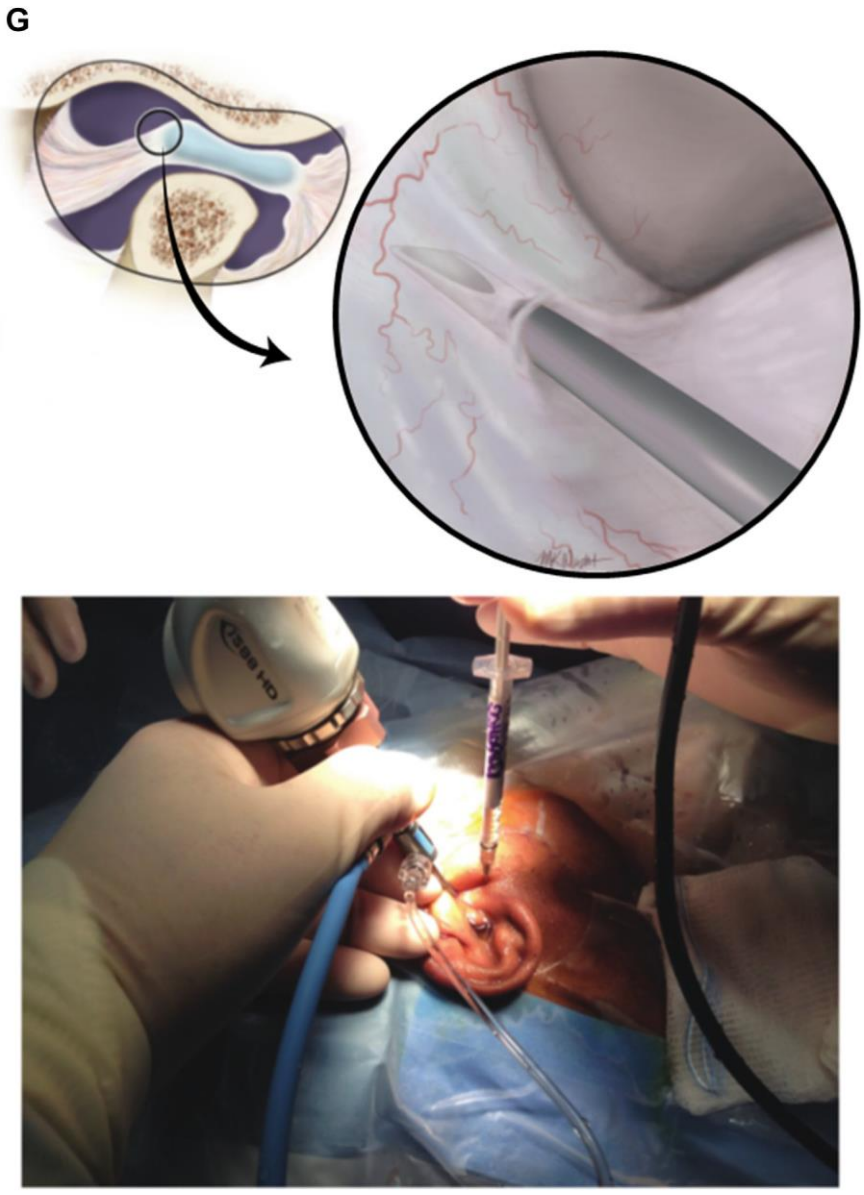

FIGURE 32 (cont'd).

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the same direction. The trocar is rotated through the skin and advanced until encountering bone at the junction between the anterior aspect of the anterior slope of the articular eminence and the continuation of the zygomatic arch. Next, the trocar/cannula is rotated through the capsule and synovium.

- The trocar is observed on the monitor entering the joint space. Once intra-articular, the trocar is removed, and drainage of the irrigating fluid is noted through the cannula. (Fig 33A).

STEP 5

Advanced Arthroscopic Procedure

At this stage of the operation, different additional procedures can be performed as needed, including the following:

Synovial biopsy: Either using cup forceps punch full-thickness biopsy or soft tissue biopsy needles. (Fig 33B).

Arthroscopic debridement: done in cases of arthrofibrosis, synovial hyperplasia, chondromalacia stages III and IV, synovial plica and ankylosing osteoarthritis.

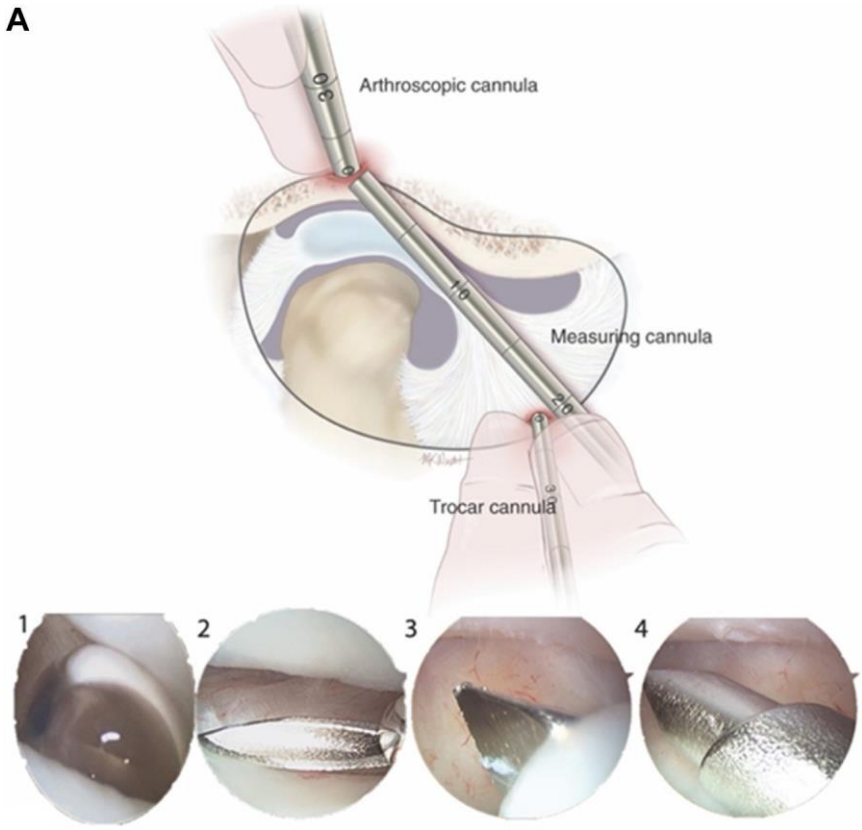

FIGURE 33. *A-D,* Double and triple puncture arthroscopy. (Fig 33 continued on next page.)

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It is advisable to start debridement in the anterior recess and do as much of the debridement as efficiently as you can in the anterior recess, then work your way to the intermediate zone, and then finally the posterior pouch. An effective joint debridement is one where the instruments can pass freely from the front to the back of the joint. You have increased joint space once the debridement has been completed. This is done by different instrumentation and motorized devices that can be inserted through the second working cannula to ablate or coagulate any adhesions or redundant tissue, which include the following:

- Hand instruments such as a straight probe, curved probe, cup biopsy forceps, bone curettes, bone files, banana blade
- Holmium laser fiber tip
- Motorized mini shaver with full radius or barrel tips
- Coblation therapy
- Bipolar and monopolar electrocautery
- Ultrasonic aspirator

Arthroscopic discopexy: This technique is used for both the reducing and the nonreducing disk. It consists of the following steps:

a Anterior release (pterygoid myotomy): The incision is made with electrocautery, coblation, or laser through the synovial membrane extending laterally to the vascular hump, penetrating the synovial membrane, and slicing the capsule. The pterygoid muscle is identified then cut; the intention is to release the superior belly of the lateral pterygoid muscle from its insertion into the articular disc.

b Disc reduction: Once the anterior release has been completed, the operative cannula and the scopeare then walked back in the lateral sulcus to the posterior pouch. Once these two instruments reach the peak of the articular eminence, the condyle is pulled forward and then both instruments can drop into the posterior pouch. The disc is reduced by compressing the retrodiscal tissue laterally with a straight probe while the condyle is in a forward or forward and contralateral position. Occasionally the disc will hold its position, but most of the time it will slip back forward which will require deepening of the anterior release. (Fig 33C).

c Retrodiscal scarification or contracture: The target area of the retrodiscal contracture is generally the boggy and redundant synovium found lateral to the oblique protuberance. Low-voltage laser, bipolar, or coblation is used to weld that

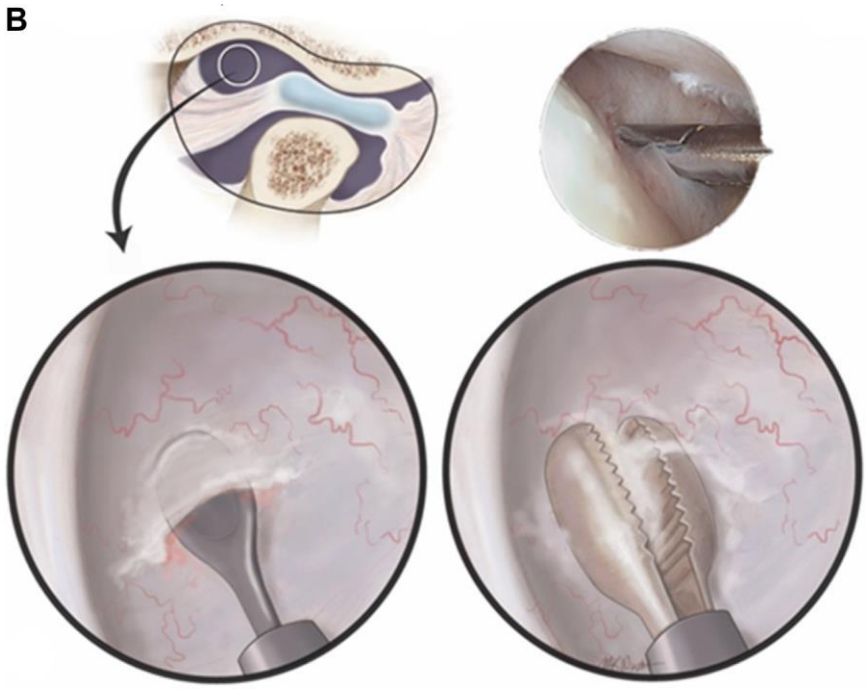

FIGURE 33 (cont'd). (Fig 33 continued on next page.)

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tissue, accomplishing synovectomy superficially and then penetrating deeper into the bilaminar zone, causing scar contracture.

d Disc fixation: A disc fixation can be accomplished in one of two ways. The first and more traditional way is the suture or wire/button discopexy. A second way is by rigid fixation with either resorbable or titanium screws. Regardless of the methodology of fixation, the disc is held in reduction during the course of the fixation. The target area of fixation is the posterior lateral corner of the disk-condyle assembly, the area of the lateral pole where the disc attaches to the condyle. (Fig 33D).

e Arthroscopic contracture: This procedure is indicated in cases of recurrent TMJ dislocation cases. Once the second puncture is established, then the scope and the working cannula are moved to the posterior pouch. Utilizing a Holmium laser on weld mode, coblation, electrocautery, or chemical sclerosing agent injection, multiple lesional burns or scars are created in the oblique protuberance. The posterior synovectomy is completed in order to enact scar contracture and then penetrate the retrodiscal tissue into the bilaminar zone with laser, coblation, or electrocautery and again enact multiple regional burns to contract that retrodiscal tissue of the posterior capsule. Then the patient is taken through jaw function to check the existence of any dislocation; if no dislocation is present, then the procedure is done.

Avoidance and Management of Intraoperative Complications

The potential intraoperative complications are numerous but should be reduced through the use of careful and meticulous surgical technique. Injury to the facial nerve remains a significant potential complication. Prevention of this injury requires proper placement of cannulas and care in joint lavage to avoid extravasation. Injury to other significant trigeminal nerve branches can also occur. These include the auriculotemporal nerve, the lingual nerve, and the inferior alveolar nerve. Prevention requires exact puncture measurement, anterior to the tragus. The surgeon must also avoid medial drape perforation that can occur by overextending the cannula by more than 35 mm. Care must also be taken to avoid medial extravasation by careful puncture technique, observation of surgical site, gentle pressure to irrigation, and patent inflow/outflow system, and routine postoperative parapharyngeal space examination using laryngoscope. Fortunately, these injuries are rare, and most patients regain nerve function within 6 months postoperatively.

Injury to the vestibulocochlear nerve and dysfunction of the auditory system can also occur during arthroscopy. Attention should be paid to angulate the trocars anteriorly, with same angulation of the tragus to avoid any perforation into the middle ear. Not advancing the arthroscope past 25 mm is also

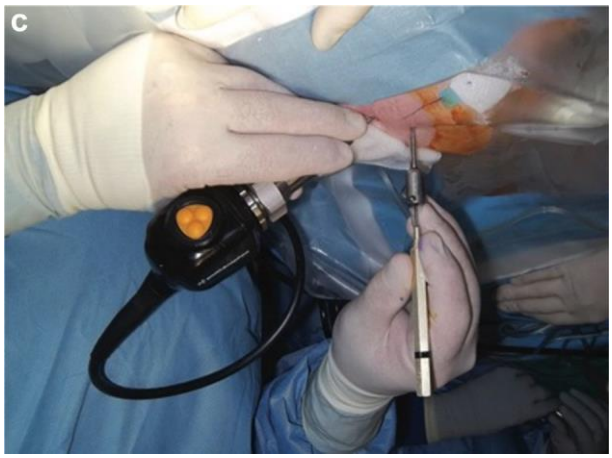

FIGURE 33 (cont'd). (Fig 33 continued on next page.)

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prudent. If the tympanic membrane is punctured, the surgeon should immediately stop the procedure, avoid irrigation, and obtain an intraoperative otolaryngology (ENT) consultation. Usually if the tympanic membrane injury is less than 30% of the drum surface, healing should occur with no sequela. Any minor ear hemorrhage is controlled by bipolar cautery, while the external auditory meatus is treated with hydrocortisone drops for up to 2 weeks.

Scuffing of the fibrocartilage lining can also occur intraoperatively. This is mitigated by avoiding repeated attempts of irrigation needle insertion. During glenoid fossa puncture, attention to the direction of trocar insertion as well as intra-articular manipulation should be considered. If scuffing is minor fibrocartilage regenerates with little consequence. Injury to major vascular structures can also occur during arthroscopy. The vertical distance of the maxillary artery from the usual arthroscopic approaches 20 mm. Although a rare complication, it may result in an arteriovenous (AV) fistula with a pathognomonic patient complaint of a persistent hissing sound, which requires medical attention and treatment. Injury to the overlying superficial temporal vessels may also occur. This is managed by applying controlled pressure without sequela. On rare occasions, injury to these vessels results in an AV fistula or pseudoaneurysm, which should be managed surgically.

Perforation of the glenoid fossa during arthroscopy is a severe complication of the procedure. Prevention focuses primarily on controlling the direction of instruments toward the tubercle and away from the fossa. Extreme caution during attempting triangulation must also be observed. If the skull base is perforated, an intraoperative neurosurgical consultation and the use of prophylactic antibiotics is recommended. A post-operative CT with IV contrast must be obtained to help identify any extradural or subdural hematoma. Most Cerebrospinal Fluid (CSF) leaks will heal spontaneously. If CSF leak persists, a pressure dressing should be applied with patient hospitalization and head elevation. A leak that has persisted for longer than 48 hours is an indication for lumbar subarachnoid drain placement. Surgical repair of the middle cranial fossa dura, although an option, is rarely necessary.

Injuries to the disc may also occur during the procedure. The surgeon should avoid any deviation from standard technique of capsular puncture to prevent this complication. If disc perforation occurs, small perforations should be sutured using a 20-gauge needle. The avascular area of the meniscus shows a fibrous tissue healing response 4 to 6 weeks postoperatively. Hemarthrosis can be a difficult intraoperative problem to manage, so it should be prevented by not tearing the superficial temporal artery. It may also be caused by excessive bleeding from severely inflamed synovium/retrodiscal tissue upon joint entrance and by bleeding from the pterygoid artery when the surgeon is performing a myotomy for an anterior release procedure. Minor hemorrhage can be tamponaded by pressure irrigation. Excessive hemorrhage can be difficult to manage. Initially, increasing pressure in the irrigation bag can be attempted. Other initial maneuvers may include injecting a small amount of hyaluronic acid intra-articularly, using cautery or a laser to the bleeding area, or injecting local anesthetic with vasoconstrictor into the bleeding site via spinal needle or as insufflation. Additional methods to apply pressure to stop the bleeding include insufflating the entire joint under pressure while all cannulas are obturated for 5 minutes or removing all instruments while direct palmar external pressure is applied for 5 minutes. For added pressure, the condyle is seated in the fossa if the

D Discopexy technique for disk repositioning.

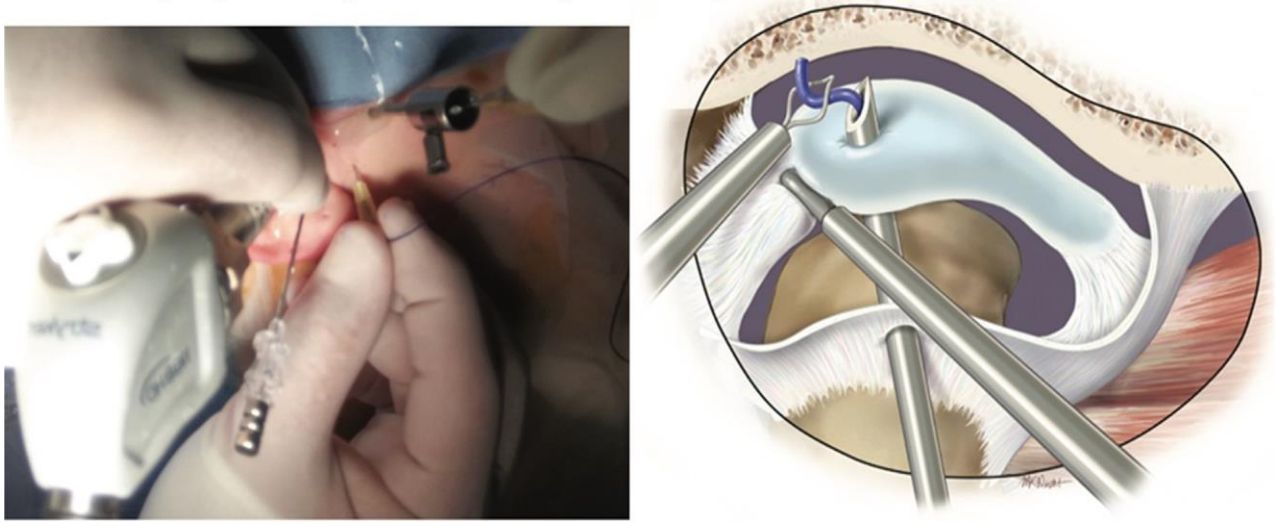

FIGURE 33 (cont'd).

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source of the bleeding is located posteriorly, and it is protruded if bleeding anteriorly. It is also possible to apply pressure through the use of a #4 catheter balloon inserted through a working portal and inflated with normal saline, which is left in place for 5 minutes as well. Certainly, if all measures are not successful, the joint is approached via open technique.

Like all surgical procedures, infection remains a risk with TMJ arthroscopy. Adherences to a proper sterile operating environment and technique, proper perioperative antibiotic prophylaxis, high-volume irrigation, and absence of any adjacent skin infections are all important considerations. If an infection occurs, administration of cephalosporin for 7 days is prudent. If the infection persists, exploration of the area under local anesthesia with removal of any residual suture and further cephalosporin administration for 7 days may be helpful. In the presence of frank purulence, arthrocentesis or incision and drainage are necessary with copious antibiotic solution lavage, debridement with placement of a drain (to be removed in 3 days), and intravenous antibiotic coverage. Noninfectious effusions of the joint may also occur. Prevention of this complication is aimed at instructing patients to avoid overzealous use of the jaw or bruxing habits. If it does occur, initial management is directed at joint rest and a soft diet with heat application and nonsteroidal anti-inflammatory drugs (NSAIDs) for pain management. If the effusion persists after 6 weeks, aspiration can be performed, and if this fails, a steroid injection will help to settle underlying inflammation. If the effusions develop some months after surgery, the surgeon should view this as an indication of degenerative disease progression.

Any minimally invasive procedure in a tight space carries with it the risk of instrument breakage and loose bodies. TMJ arthroscopy is no exception. Avoid instruments with manufacturing defects. Care must be exercised to not misuse any instruments, and if an instrument has worn out, it should be replaced. All instruments with flexible parts should be tested prior to the insertion into the joint. One must never apply excessive force or bending during the procedure. The use of ferromagnetic instruments is also recommended. If an instrument fracture occurs, immediately stop the procedure and maintain the position of arthroscope and cannulas. Keep the broken instrument in view and ensure the joint space is distended for optimal visibility. Next, measure the depth of the instrument with a scored cannula. Consider using fluoroscopic assistance to localize the piece if it cannot be visualized. Finally, remove the fragment. It may be desirable if this particular complication occurs to switch systems to a larger, 3-mm working cannula with a switch stick technique and retrieve the broken fragments with a golden retriever.

Postoperative Considerations

Postoperatively, patients are placed in a self-regime of physical therapy that includes stage II exercises or jaw motion rehabilitation device exercises at 20 repetitions, four times a day. The goal of management is to achieve a normal range of motion. Immediately postoperatively, patients will present with decreased range of motion, probably secondary to pain. However, patients need to be mobilized if an adhesive

phenomenon is to be prevented. Later, the physical therapy is modified to prevent excessive range of motion.

Patients are maintained on a soft consistency diet, which is to be maintained for 3 to 6 months followed by regular compromised consistency, and they are given analgesics. They are also instructed to wear the flat occlusal orthotic. Follow-up is maintained at 1 week, 1 month, 3 months, 6 months, and then every 6 months thereafter. Preoperative and postoperative assessment parameters include the improvement index measured with a visual analog scale, range of motion measured vertically in millimeters, and diet consistency. The patient should maintain the immediate postoperative opening, and pain should be limited to the first postoperative week. In cases of mandibular dislocation, four brackets are to be applied on cuspids, on which medium elastics are applied to each side, holding jaws in fixation.

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Arthroplasty[228]

Armamentarium
#15 scalpel blade
Needle electrocautery/PEAK® Plasmablade[1]
Local anesthetic with vasoconstrictor
#9 periosteal
Cotton pellet or Merocel® ear packing[2]
Senn retractor
Kitner retractors
TMJ retractors
Freer elevator
Osteotomes
Round bur
Bone file or reciprocating rasp
0.02-inch silastic sheet
702 carbide bur
Right angle vascular clamp
4-0 vicryl® [3], 4-0 monocryl® [3], 5-0 prolene® [3] or 5-0 fast absorbing suture
Nerve stimulator/Checkpoint® 9394 Stimulator[4]
Bone clamp
Headlight for operating surgeon

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Technique 1: Arthroplasty

STEP 1

Prepping and Draping patient

Open TMJ surgery is performed under general anesthesia. The patient is placed supine on the operating table with the head turned to expose the side to be operated. It is usually unnecessary to remove hair unless it will evidently enter the wound during surgery and increase likelihood of infections. Prior to prepping the skin an antibiotic ointment impregnated cotton pellet, petroleum gauze, or an impregnated Merocel[1] ear packing is placed into the external auditory canal. This minimizes entry of fluid into the ear during surgery which could lead to postoperative irritation and pain of the canal or tympanic membrane. The skin is then prepped and draped to include the external ear, in case of need for cartilage harvest, preauricular skin including the zygomatic arch to the lateral canthus, and inferiorly to the mandibular border. A sterile adhesive border is then applied with the ear exposed and sterile drapes are placed.

STEP 2

Incision

The endaural approach, a modification of the preauricular incision placed on the tragus instead of the pretragal crease, will be described herein. If further access is required, Al-Kayat and Bramley describe an extension into the temporal area about 1 cm above the superior margin of the palpable zygomatic arch. After marking out the incision, care must be taken to avoid injected local anesthetic too deep or it may anesthetize the facial nerve. If a nerve stimulator will be used, it is also crucial to communicate this to the anesthesia team and avoid use of neuromuscular blockade. Newer and more sophisticated instruments like Checkpoint[2] have been studied and found to be equally reliable to electromyography in thyroid surgery.

An incision is made from the uppermost point of the auricle inferior to the attachment of the ear lobule with the middle portion of the incision hidden in the posterior tragus. Care is taken to avoid tragal cartilage and minimize the risk of perichondritis, although some surgeons transect through the tragal cartilage. (Fig 34A). The incision is through the skin and subcutaneous tissue down to superficial temporalis fascia. A blunt instrument is then used to open a pocket anterior to the external auditory canal. The superficial temporal artery and vein are located deep to the subcutaneous tissue and may be encountered and ligated or retracted forward. Dissection is bluntly continued toward the superficial temporal fascia until the smooth white well-defined surface of the superficial surface of the temporalis fascia is encountered. (Fig 34B). When a small pocket has been developed a Senn retractor is placed into the wound down to this layer. Another Senn is placed in the pretragal incision. The soft tissue in the depth of the wound has clearly defined landmarks that can then be sharply released using scissors.

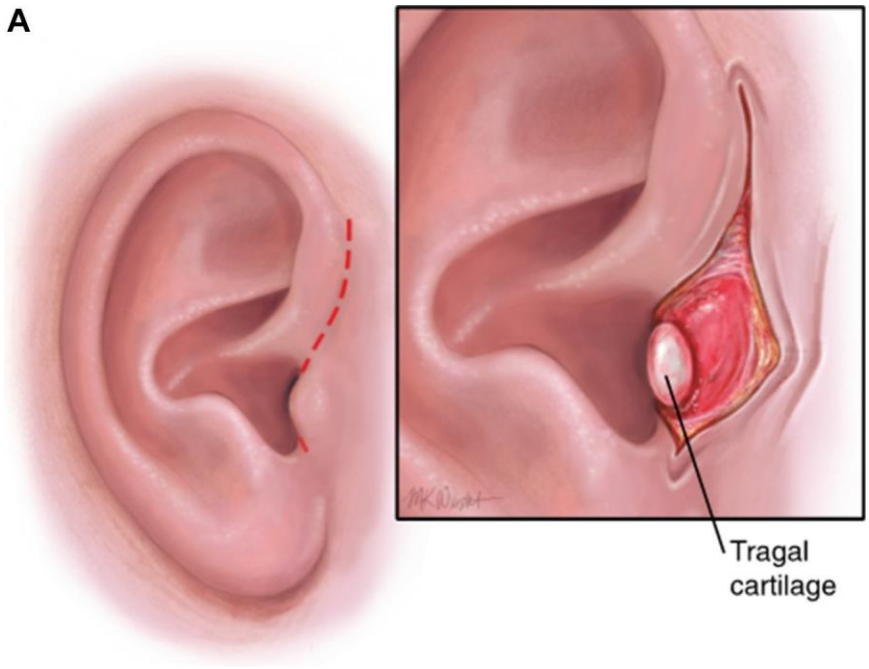

FIGURE 34. *A-E,* arthroplasty. (Fig 34 continued on next page.)

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STEP 3

Dissection to the Temporomandibular Joint Capsule

The superficial layer of the deep temporal fascia is now visible the entire length of the wound. A scalpel is used to incise this layer just anterior to the plane of dissection and parallel to the skin incision. A Molt periosteal elevator is then utilized to dissect over the periosteum anteriorly. Dissecting in this layer protects and allows retraction of branches of the facial nerve. (Fig 34C). This plane is superior to the lateral capsule of the TMJ and the posterior attachment of the parotidomasseteric fascia. This attachment is cut just anterior to the auditory canal and the fascia retracted anteriorly to expose the lateral capsule to view. (Fig 34D). Kitner retractors are particularly useful and cause minimal soft tissue injury during this process and if performed in the correct layer, bleeding is minimized.

STEP 4

Exposing the Intraarticular Spaces

The lateral capsule is now well exposed and entry into the joint may be accomplished by making a 4-5 mm incision through the capsule parallel to the zygomatic arch and 2-3 mm below it. (Fig 34E). A Freer elevator is then used to enter the superior joint space and break up adhesions between retrodiscal tissues, disc, glenoid fossa and articular eminence. The Freer is then turned vertically into the lateral recess. At this point the first decision for surgical management is made: if the superior surface of the disc is intact without perforations or evidence of condyle exposure, or cartilaginous degeneration, an additional disc sparing incision is performed instead of a disc replacement incision. A disc sparing incision is parallel to the incision described above but is placed below (about 1 cm) and through capsule, disc and ligament down to the lateral bone of the condylar head. A subperiosteal dissection is done and a retractor is placed onto the lateral cortex of the condyle and a subperiosteal dissection is directed superiorly. Simultaneously, the mandible is retracted inferiorly by a surgical assistant so that the head of the condyle is exposed and inspected. This also allows examination of the articular disc through both the upper and lower TMJ compartments without cutting through the disc. A disc replacement incision is placed vertically from the middle of the superior joint access incision described above in a descending direction onto the lateral cortex of the condylar head and neck completing a T shaped incision over the lateral capsule. This incision transects the lateral and superior portions of the articular disc and allows excellent access to the internal components (condylar neck, head, articular cartilage and retrodiscal tissue) with the intent to resect/remove the articular disc (or condylar neck, etc).

Technique 2-Eminectomy

The authors prefer to perform an eminectomy in addition to meniscoplasty. Ideally, the eminectomy is

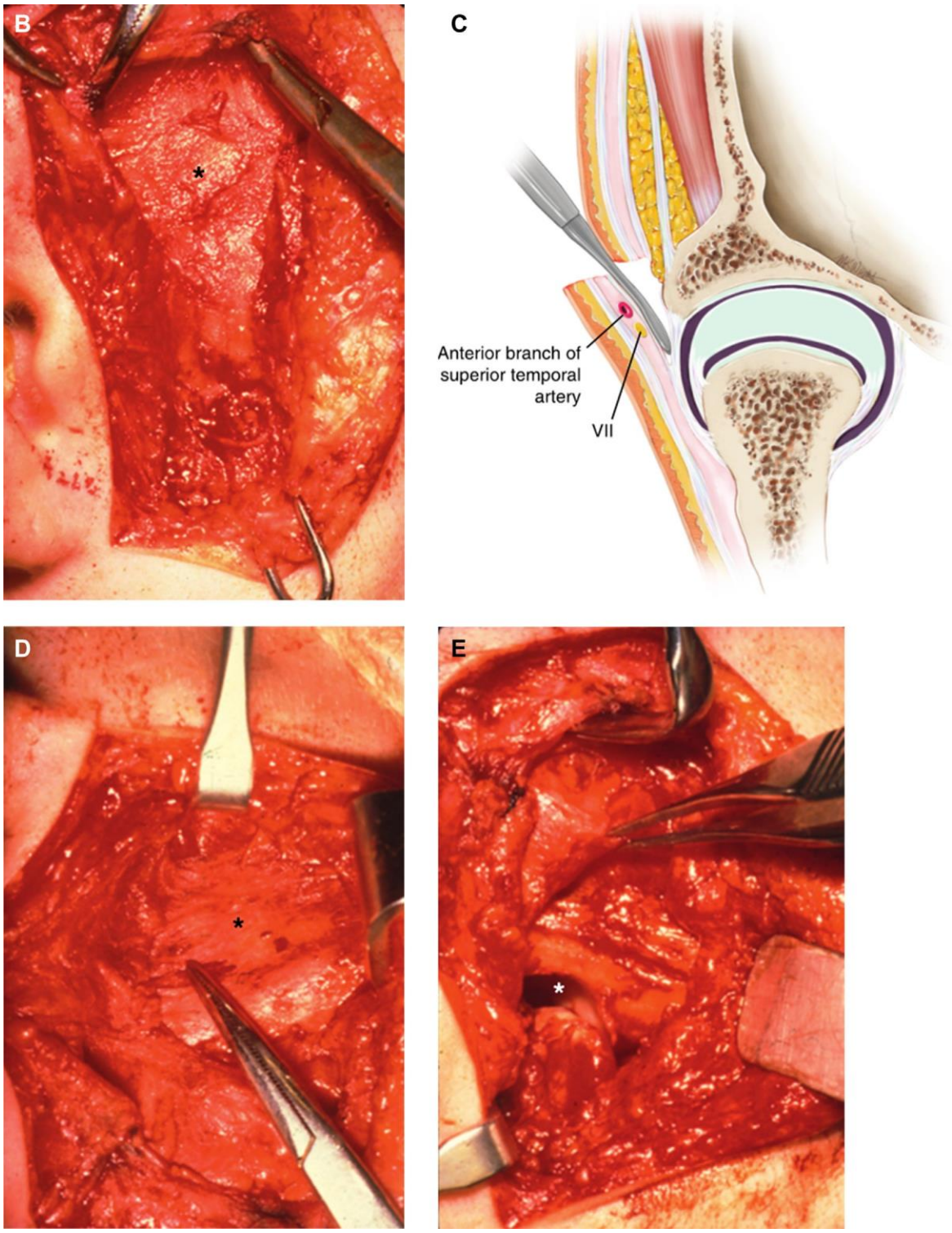

FIGURE 34 (cont'd).

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done prior since it significantly enhances the access to the disc during meniscoplasty. If surgical management requires meniscectomy, eminectomy is avoided to allow the autograft of choice to adapt and fixate to the unaltered fossa and eminence. Meniscectomy eliminates the possibility of disc recapture that may occur when the disc is repositioned with meniscoplasty. The eminence determines the movement of the disc during translation and rotation but does not affect the condylar head movement. Thus, the eminectomy has

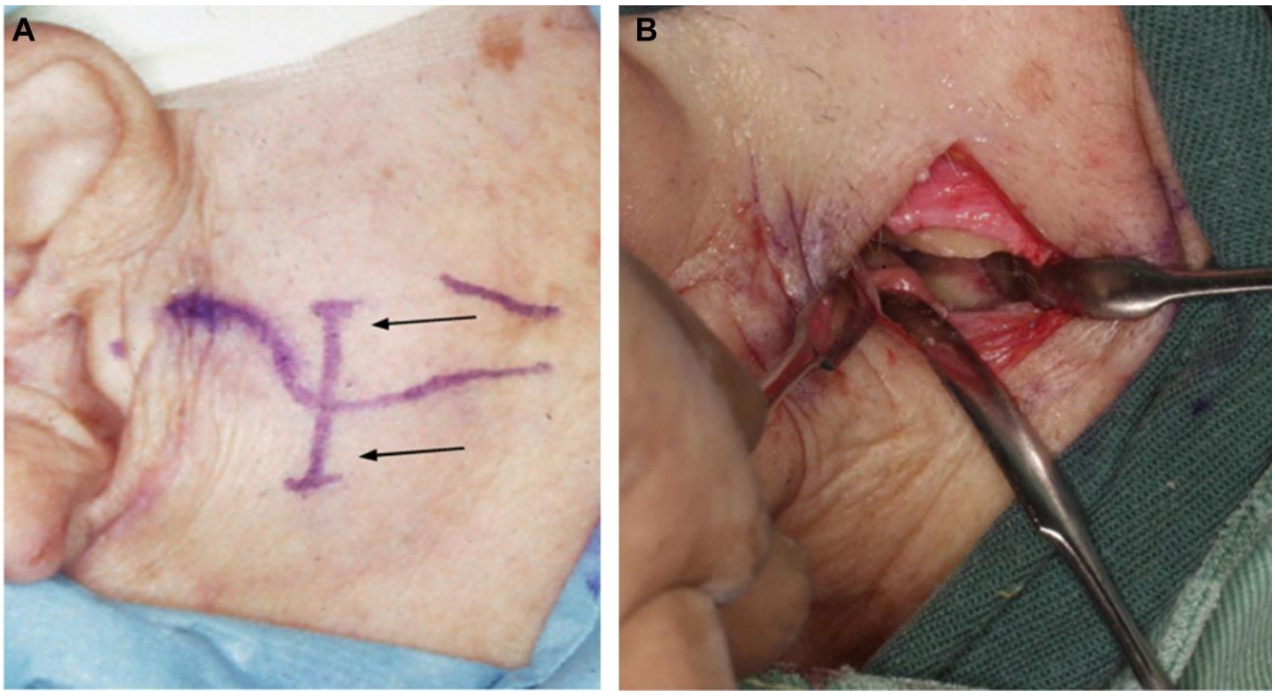

FIGURE 35. *A-D*, eminectomy. (Fig 35 continued on next page.)

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a positive outcome on disc movement postoperatively without effecting condylar movement. Post-eminectomy patients demonstrate normal opening, excursive movements and do not deviate on opening or protrusion since condylar pathways are not affected.

STEP 1

Exposure

After access to the TMJ has been accomplished eminectomy is performed with the aid of chisels, rotating burs, reciprocating rasps, or piezo surgery device. Reducing the eminence as far medially as possible is important to a successful surgical outcome. An alternative exposure is described by Segami as a 2 cm vertical incision anterior to the pretragal crease for more direct access under local anesthesia. (Fig 35A and B).

STEP 2

Reduction of the eminence

Once the joint is exposed and the articular eminence is identified a 1 mm fissure bur is utilized to make a horizontal cut into the lateral tubercle of the eminence that will allow for an unobstructed path of condylar translation. (Fig 35C). Complete bone removal is continued, with the surgeon ensuring at all times that the plane of the bur is directed inferiorly approximately ten degrees as it proceeds medially, to follow the natural slope of the eminence toward its medial base. Approximately 90 percent of the cut in the eminence is made utilizing the 1 mm fissure bur. An osteotome positioned with an inferior angulation is then used to complete the cut and avoid the skull base. A large round bur or reciprocating rasp smooths the eminence and completes the eminectomy as far medial as possible. (Fig 35D). During the cuts a broad retractor is used to depress and protect the meniscus and condyle. At the end of the procedure the bony surfaces should be well rounded and smooth.

STEP 3

Closure

After completion of the eminectomy it has been recommended to reattach the lateral joint capsule by drilling holes into the lateral rim of the zygomatic arch and reinserting the joint capsule with resorbable sutures. Arthroscopic eminoplasty has also been described. The amount and location of bone removed from the eminence is subject to debate. Some advocate reducing the height and contouring the eminence, whereas others recommend complete resection.

Technique 3 Disk Imbrication, Plication, Mitek Implant, Meniscectomy

Reestablishment of the anatomic disc-condyle relationship has been described by utilizing plication, imbrication as well as the use of implants. Typically, discs are displaced anteromedially therefore a posterior lateral vector will return the disc to a normal

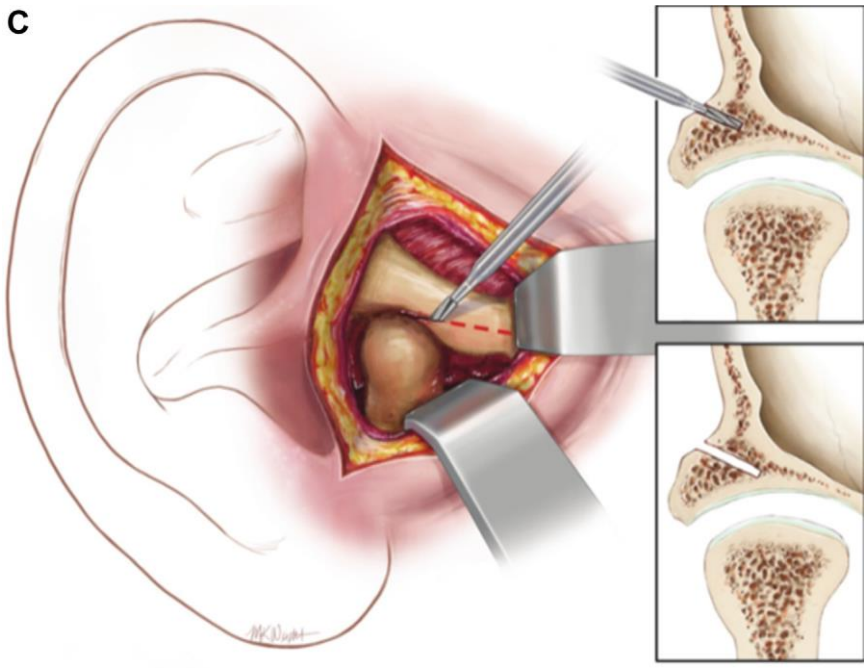

FIGURE 35 (cont'd). (Fig 35 continued on next page.)

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position. The directional relocation of the disc is the primary objective in these procedures.

Meniscocondylar imbrication, also known as discopexy, is suturing of the disc to the lateral condyle, thus preventing the classic anteromedial displacement while attaining an appropriate vector to reduce the disc to its anatomic location. Utilizing a 701 fissure bur a hole is drilled in the lateral condyle in a sagittal plane from posterior to anterior. The meniscus is freed up from adhesions so that it can passively lay over the height of the condyle. A 4-0 non resorbable (Mersilene)[3] suture is first passed through the hole in the lateral condylar neck from posterior to anterior. The suture is then placed through the meniscus from inferior to superior and as far medial as possible through the intermediate band and posterior attachment. The knot is tied in the posterior lateral aspect. The lateral border of the disc is then secured to the lateral capsular attachment utilizing four to six 4-0 Vicryl sutures. Adequacy of the sutures is then assessed by opening and closing the mandible.

Disc plication is the partial or complete removal of retrodiscal tissue and suturing the remaining tissue to the posterior ligament. A partial meniscoplasty is performed by removing a wedge and repositioning the disc posterior and lateral. Utilizing right angle vascular clamps, the anterior and posterior portion of the disc may be clamped for better control of wound edges and hemostasis. (Fig 36). The disc is then sutured together utilizing 4-0 resorbable suture to recreate the natural position of the disc. A wedge plication can be tailored to control different vectors to return the disc to the appropriate position. After plication, range of motion exercises are performed in the OR to evaluate for locking, catching, or obstruction. Often eminectomy is performed in conjunction with this procedure.

The use of mini anchors (Mitek)[5] is commonly used to reposition the disc in its anatomical position. This implant is a bone anchoring system that is placed into the most posterior, superior, lateral condylar neck. The implant secures itself in bone with a cleat system that is activated upon insertion. The anchor has an eyelet that a non-resorbable suture which is then passed through posterior band of the disc. This allows the disc to be securely sutured in place preventing relapse. The lateral capsule is the closed with 4-0 Vicryl.

When the disc is perforated, deformed, irreparable, causing pain, or inhibiting smooth pain free function a meniscectomy may be required. Meniscectomy removes the central avascular portion as well as the posterior attachment that may be perforated. Care must be taken to minimize injury to the retrodiscal tissue, which is usually hyperemic; if it begins oozing, it is difficult to achieve hemostasis without extensive cauterization. Some literature suggests that the PEAK PlasmaBlade facilitates better post op healing than traditional electrocautery. The medial attachment of the disc is generally the most challenging to remove.

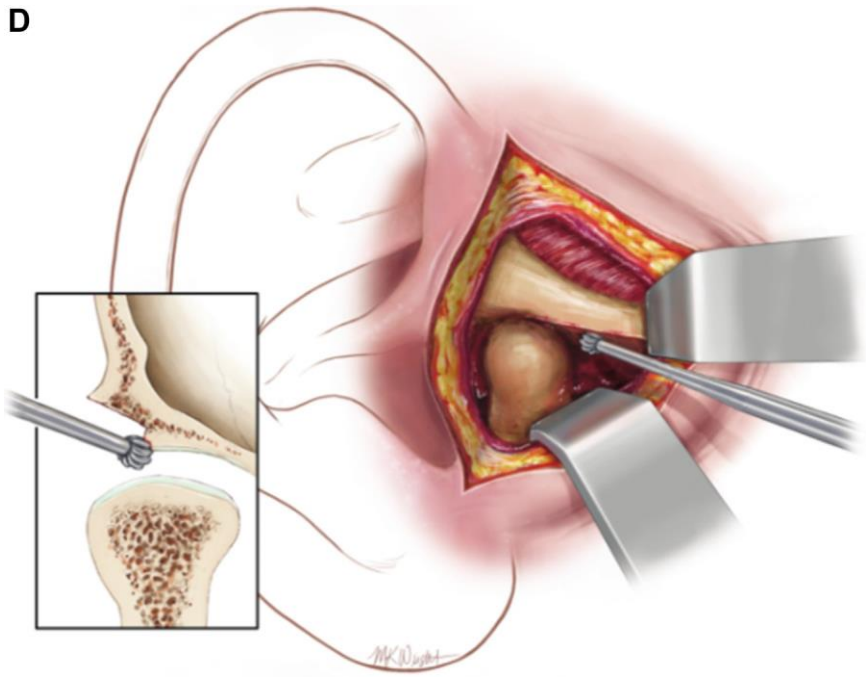

FIGURE 35 (cont'd).

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The condyle must be retracted anterior and inferior to allow access to the medial attachment which can then be incised using a curved TMJ scissor or a 15 blade. It is important to remove the entire meniscus and to trim any loose or ragged edges to prevent adhesion or fibrosis. Care must be taken to avoid perforating the medial capsule which may result in bleeding due to injury of the middle meningeal or internal maxillary artery, or the pterygoid venous plexus. Depending on disc and joint morphology it may be prudent to

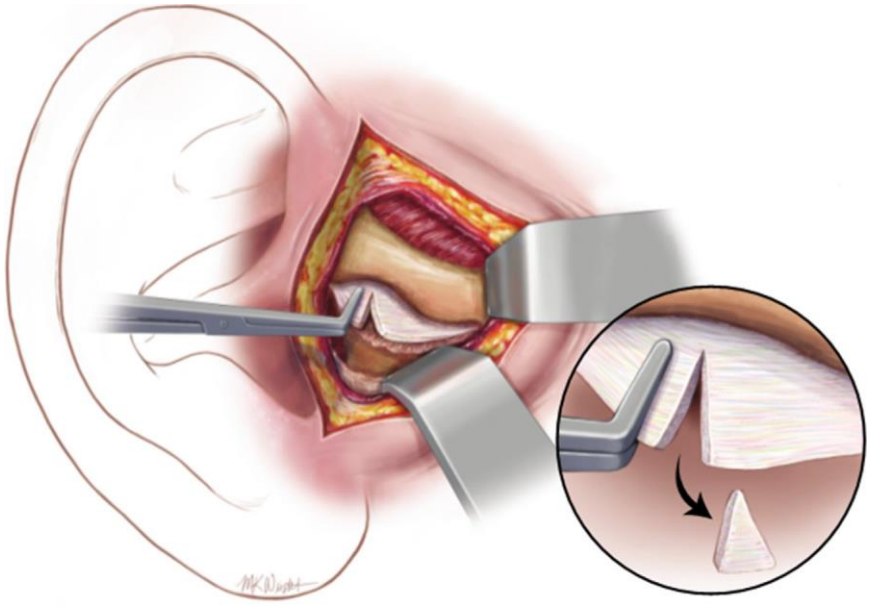

FIGURE 36. Disc plication.

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Table 8. INTERPOSITIONAL MATERIALS

Material	Challenges
Silastic pullout	Temporary (6 weeks) and not FDA approved
Cartilage	Migration, fibrous ankylosis, fragmentation
Fat	Remote surgical site, predictability
Temporalis muscle	Pain, temporal hollowing, fibrosis
Dermis	Remote surgical site, epidermoid cysts

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remove the disc in pieces starting with the easily accessible lateral portion and proceeding deeper into the joint with improved vision and access.

Post meniscectomy imaging shows flattening of the anterior-superior slope of the condyle while the anterior lip of the condyle might show sclerosis or some beaking. Other potential post-operative changes include crepitus and fibrosis.

Technique 4-Interpositional Grafting

Following meniscectomy some advocate a temporary silicone implant while others recommend autogenous reconstructive options include temporalis muscle flap and fat. Matrix associated chondrocytes have also been transplanted as a means of tissue regeneration with promising results. Studies are equivocal, and most patients do well without reconstruction but there is still concern about crepitus and regressive remodeling. (Table 8).

TEMPORALIS MUSCLE FLAP

Temporalis fascia is an inadequate graft however the temporalis myofascial flap is the most commonly used interpositional material because of minimal donor site cosmetic and functional morbidity. This flap is harvested by extending the endaural incision into the temporal region about 3 cm. The superficial temporalis fascia is then identified. Utilizing a 15 blade a full thickness inferiorly based, pedicled flap including superficial fascia, temporalis muscle, and deep temporalis fascia is harvested. The distal edge of the flap needs to be wider than the joint space it is going to fill to account for contraction. In general, the length of the flap from the superior edge to the zygomatic arch is 5 to 6 cm and approximately 3 cm in width. (Fig 37). The flap is then rotated laterally over the zygomatic arch and positioned in the joint space so that the temporalis periosteum is against the glenoid fossa. One issue with this rotation is the asymmetry of the ipsilateral face caused by the bulk of muscle that is lateral to the zygomatic arch. Alternatively, the posterior zygomatic arch may be osteotomized in two places to allow for the flap to rotate into the joint space. The segments of the arch are then returned and secured in place with rigid fixation. The flap is held in position with two nonresorbable sutures that are passed through holes drilled in the posterior fossa and anterior eminence.

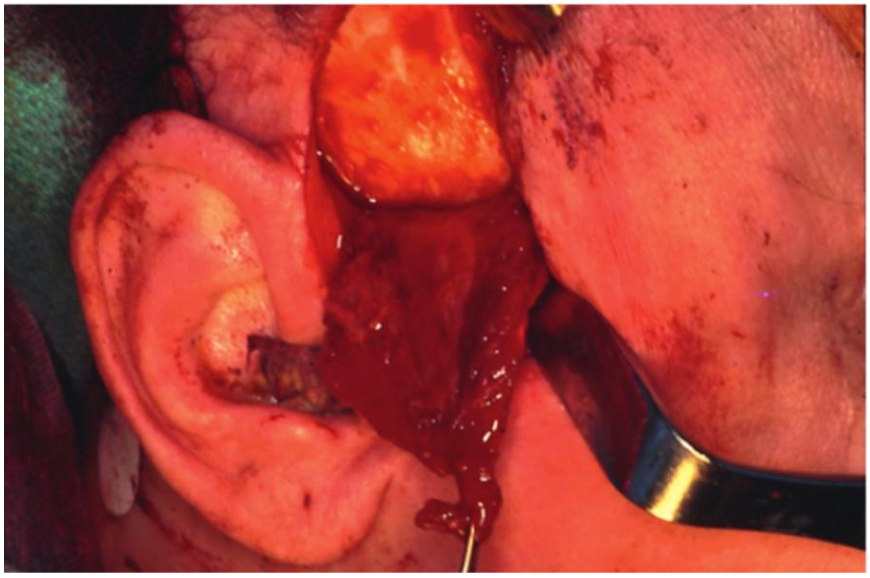

FIGURE 37. Temporalis flap.

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The flap may also be raised in the same manner as described above but passed through the infratemporal space passing it from the articular eminence posteriorly into the joint space. It is sutured in place as above.

MATRIX-ASSOCIATED CHONDROCYTE TRANSPLANTATION (MACT)

While tissue regeneration has been employed in other joints limited literature exists of its application in TMJ arthroplasty. The technique described by Undt et al discussed the use of collagen scaffold combined with a suspension made from harvested rib chondrocytes and autologous blood. This was secured with a 1 mm thick silicone sheet that was retrieved 4-5 months later and the graft could be analyzed for differentiation.

Avoidance and Management of Intraoperative Complications

The major associated intraoperative complications with TMJ surgery include damage to cranial nerve VII, bleeding associated with terminal branches of the external carotid artery, retromandibular vein, and damage to the parotid gland. Facial nerve injury of the temporal branch (occasionally the zygomatic branch) is the most significant complication that is associated with open surgery. Although total facial nerve paralysis is possible, it is rare. Inability to raise the eyebrow is the most commonly observed finding occurring in 5% of the cases and usually resolving in 3 months; it is permanent in <1% of the cases. Other complications are limited opening and minor occlusal changes. Avoidance of the facial nerve as well as anatomic variation and location has been well described. Al-Kayat described the distance utilizing many landmarks. From the lowest point of the external bony auditory canal to the bifurcation of the facial nerve. it was found to be 1.5 to 2.8 cm (mean 2.3 cm), and the distance from the post glenoid tubercle to the bifurcation was 2.4 to 3.5 cm (mean 3.0 cm). The most variable measurement was the point at which the upper trunk crosses the zygomatic arch. It ranged from 8 to 35 mm anterior to the most anterior portion of the bony external auditory canal (mean 2.0 cm). (Fig 38).

Prior to eminectomy, imaging of the articular eminence is extremely important to prevent possible perforation into the middle cranial fossa, exposure of the temporal lobe and possible CSF leak. It is important to identify the lateral tubercle of the temporal bone on CT or MRI and to estimate the thickness both laterally and medially to prevent penetration into the middle cranial fossa. Hall conducted an anatomic study of 38 cadaver heads to establish a guideline for eminectomy and found the average distance from the lateral tubercle to the temporal bone is 9 mm with a range of 5-14 mm. The antero-posterior length of the eminence averaged 11 mm ranging from 9-18 mm while the width was 21 mm ranging from 16-25 mm. After eminectomy is completed, the mandible is manipulated to ensure unobstructed condylar motion during normal range of motion.

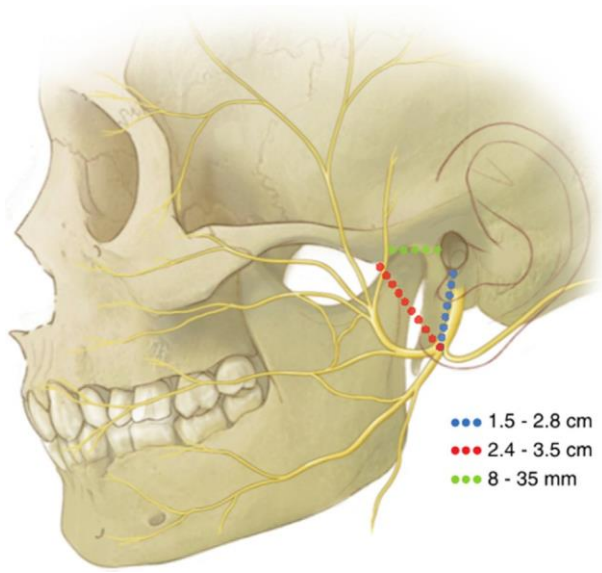

FIGURE 38. Facial nerve.

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Post-Operative Considerations

Before closure of the capsule an angiocath is placed in the surgical site which will be connected to an On-Q[6] pain pump after closure. The pump delivers a continuous infusion of anesthetic intraarticularly and has been shown to be effective in controlling postoperative pain. This is usually left in place for 3 days postoperatively. A pressure dressing is also placed and is usually removed 24 hours later. Routine wound care is performed on the incision with application of antibiotic ointment twice per day until the sutures are removed 5 days later. A no chew diet is prescribed for 3 weeks, and the patient is slowly advanced. Aggressive and early joint mobilization is required for any intraarticular joint surgery to succeed. Range of motion exercises begin on post-operative day 4 with the goal of improving range of motion and mobilizing post-surgical edema. Liposomal bupivacaine (Exparel)[7] may also be used as an alternative to the use of the On-Q pump.

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Footnotes

1 Medtronic Merocel – Dublin, Ireland.
2 Medtronic PEAK – Dublin, Ireland.
3 J&J Ethicon – Sommerville, NJ, USA.
4 Checkpoint Surgical – Cleveland, OH, USA.
5 DePuy Mitek – Raynham, MA, USA.
6 Avanos Medical On-Q – Alpharetta, GA, USA.
7 Pacira Biosciences Inc. Exparel – Parsippany-Troy Hills, NJ, USA

Custom Total Temporomandibular Joint Replacement[229]

Armamentarium
Temporomandibular joint surgical instruments
Basic oral and maxillofacial surgery instruments
Custom TMJR instrument and fixation kit
SL model and screw length diagram in OR for reference
Power equipment, burs, saws, and Jacob's chuck
Piezosurgery instrumentation
Maxillomandibular fixation instrumentation
Fat graft harvest surgical instruments
Vancomycin solution to soak implants.
Hair shears and elastic adhesive tape.
Plastic adhesive and surgical isolation drapes.
Eye Lubricant and eye Protection Devices
Otoscope, cotton pledgets, and mineral oil.
Marking Pen, Cautery
Local anesthetic with vasoconstrictor.
Change of gown and gloves.
Penfield Elevator to place Fat Graft
Appropriate suture material
Otic speculum, drops, and cotton pledgets.
Barton head dressing materials
Jaw-exercising device

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The indications for total joint replacement (TJR) include:

- Osteoarthritis and inflammatory arthritis
- Ankylosis, re-ankylosis and heterotopic bone formation
- Failed autogenous reconstruction
- Failed alloplastic TMJ reconstruction
- Condyle fracture
- Functional Deformity
- Benign neoplasms and cysts
- Malignant pathology when not irradiated
- Developmental abnormality

Special Considerations

FAILED AND FAILING TMJR DEVICES

Failed and failing TMJR devices require either revision or replacement. Revision means maintaining the same device in place, whereas replacement means the complete removal of the TMJR and implanting a new one. Revision should be considered forcases with acute infections, dislocation, some malpositioned implants, malocclusion, and early heterotopic ossification. On the other hand, replacement should beconsidered for TMJR failures due to device component fracture, an adverse local tissue response, component loosening, late/chronic infections, or documented material hypersensitivity. However, patient selection and surgeon experience play a critical role in successful patient revision and replacement outcomes.

Due to the osteolysis that occurs around failed TMJR components, substantial host bone anatomic architectural discrepancies occur. Therefore, it is difficult to adapt and stably fixate autogenous tissues or stock TMJR components to the hostbone. Further, the adverse local tissue responses associated with failed or failing devices provide an unfavorable environment for the introduction of an autogenous graft. Therefore, custom TMJR components provide more predictable outcomes than do autogenous tissue or stock TMJR devices.

DOCUMENTED HYPERSENSITIVITY TO TMJR MATERIALS

Documented hypersensitivity to the materials typically used in the production of TMJR device components, commercially pure titanium (cpTi), titanium alloy (Ti6V4Al ELI), cobalt-chrome-molybdenum alloy (CoCrMo), and ultra-high molecular weight polyethylene (UHMWPE), is rare. Although approximately 10% of the population can be sensitive to the nickel, (<1%) of the CoCrMo alloy, far fewer reports of such allergic reactions have been reported in the orthopedic literature. Historically, there have been two testing modalities that have been used to diagnose hypersensitivity to implanted metals: The skin patch testing (SPT), and the lymphocyte transformation test (LTT). Furthermore, SPT tests for hypersensitivity utilizing macrophages (Langerhan cells) within the dermis, which are much different than the lymphocytic response cells found in deeper tissues (T lymphocytes) where joint implants are located. Further, the interpretation of SPT results is purely subjective as there is no verified standard scoring system. Additionally, skin testing using intradermal metal disks has been determined to be unreliable and is not recommended.

The limitations of SPT led researchers to find another test to confirm pre-implantation metal allergy. It has been demonstrated that the lymphocyte transformation test (LTT) measures the ability of lymphocytes to proliferate in the presence as well as the absence of a metal ion stimulus cultured with a patient's peripheral blood lymphocytes. Investigators have also used the LTT to evaluate orthopedic implant patients with symptoms indicating potential

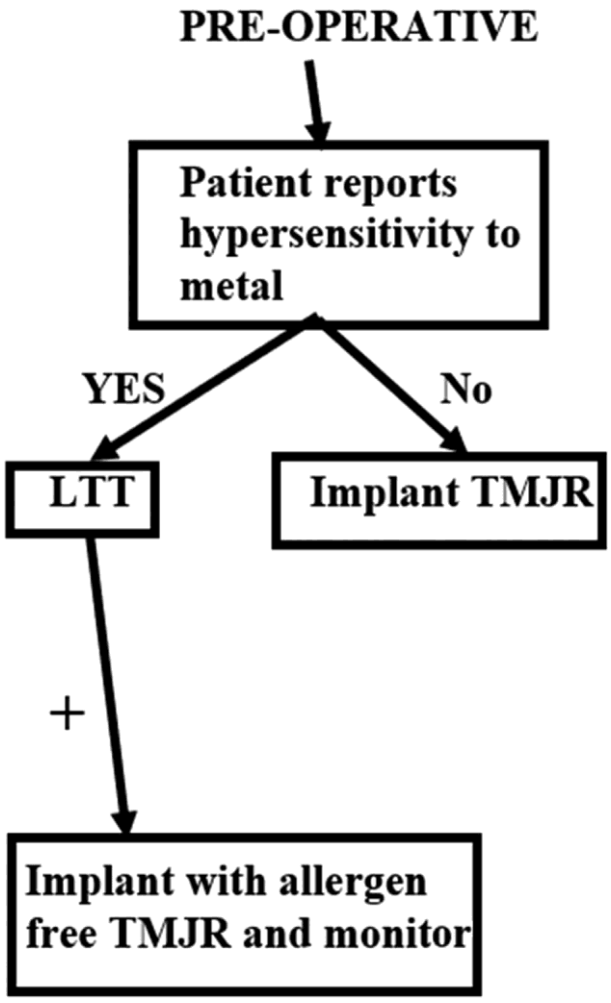

FIGURE 39. Preoperative hypersensitivity testing.

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hypersensitivity to their implants to identify patients who might benefit from implant removal and replacement with components composed of non-reactive materials. Currently, the diagnosis of alloplastic joint replacement material hypersensitivity appears to be one of exclusion and there are no available validated management algorithms for the management of such cases. However, based on a review of the available orthopedic literature, a practical workup and management for hypersensitivity algorithm for TMJR was developed. (Fig 39).

Hypersensitivity testing prior to primary TMJR can be helpful when a patient reports a history of intolerance to jewelry or of an allergic reaction to a prior metal implant. However, to date, routine testing is not supported by the literature.

ACTIVE INFECTION AT THE IMPLANTATION SITE

As with any alloplast material, implantation into an infected or contaminated area can result in failure of the components of the device to stabilize, leading to micromotion and catastrophic failure under functional loading. Cutibacterium acnes (nee: Propionibacterium acnes) has increasingly become recognized as a causative agent of periarticular TMJR infection. C acnes is a Gram-positive bacterium that forms part of the normal flora of the skin, oral cavity, large intestine, the conjunctiva, and the external auditory canal. Although primarily recognized for its role in acne, C acnes is an opportunistic pathogen, causing a range of postoperative and device-related infections. Surgeons should consider C acnes as a risk for infection in patients especially those with a history of chronic acne or acne vulgaris.

Technique

PATIENT AND OR PREPARATION

- After the proper diagnosis has been made indicating the need for TMJR and the patient's well-informed consent is obtained, the process of designing and manufacturing custom TMJR components can begin. The patient undergoes a protocol computed tomography (CT) scan from which a stereolithographic acrylic (SLA) model will be developed. This model serves as the template for the design and manufacture of the patient fitted custom TMJR components. (Fig 40A).
- The surgeon and design engineer use the SLA model for planning the design for the TMJR device and any applicable host bone modifications (Fig 40B).
- In some cases, especially those involving combined TMJR and orthognathic procedures, a virtual surgical planning (VSP) conference can be scheduled to coordinate the case planning. The surgeon approves the surgical plan, implant design, and the custom TMJR components are manufactured, sterilized and forwarded to the hospital.
- A computerized tomography arteriogram (CTA) is not necessary for every TMJ ankylosis case. CTA should be considered when the preoperative CT demonstrates possible involvement of the internal maxillary artery itself, or any of its major branches in the planned surgical procedure.
- Patients should be advised to wash their hair with mild shampoo before surgery and not use any hair sprays, gels, or facial makeup.
- The use of prophylactic antibiotics is the most important factor in preventing a periprosthetic joint infection (PJI). In order to reach the minimum inhibitory concentration in the end organs

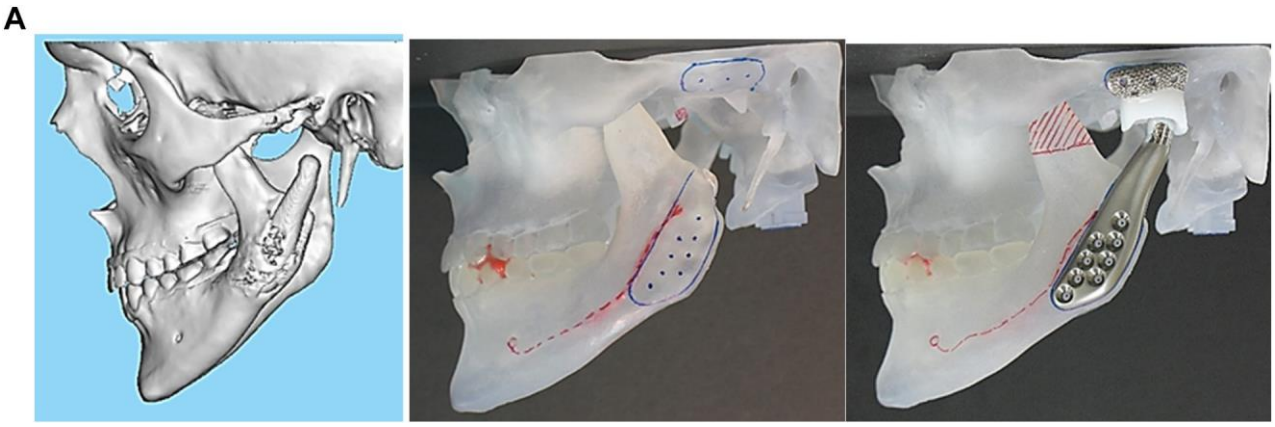

FIGURE 40. *A-B,* SLA models, planning and component design. (Fig 40 continued on next page.)

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during surgery, the optimum time for weight-adjusted prophylactic antibiotic administration is 1 hour prior to the surgery. A first- or second-generation cephalosporin is suggested. The timing can be extended up to 2 hours for vancomycin and fluoroquinolones.

- Hair in the area of the preauricular incision should be sheared, not razored. Any remaining hair, the eyes, the auditory canal(s), and mouth should be appropriately isolated from the surgical field using adhesive iodine-impregnated drapes.
- Any maxillomandibular fixation (MMF) system can be positioned after general anesthesia induction and before the patient is prepped and draped for the sterile implantation procedure. The patient should not be placed into MMF at this stage of the procedure to allow free movement of the mandible during the implantation procedure.
- All instruments and rotatory equipment used intraorally to apply MMF appliances, remove teeth, or to harvest autogenous abdominal fat must be strictly isolated from those used to implant the TMJR device components.
- After removal from their sterile packaging under sterile conditions, it is the authors recommendation that the TMJR components be placed in a 1 g vancomycin/500 cc saline solution to soak during the procedure. After implantation of the TMJR components, before wound closure the vancomycin solution should then be used to irrigate the preauricular and retromandibular incisions thoroughly before closure.

CUSTOM TMJR IMPLANTATION

- The standard preauricular and retromandibular incisions, dissections, and steps for the implantation of the temporal fossa and ramus/condyle components of the TMJ Concepts custom TMJR system have been well documented and described.
- To determine the anterior extent of the preauricular dissection, refer to the SLA model that should be available in the operating room. Sterilizing the anatomic bone model and handling during surgery in the sterile field is not recommended.
- Custom TMJR ramus/condyle and fossa components are patient fitted. Therefore, to avoid implantation errors, it is essential that the surgeon prepare the fossa and ramus host bone properly so that each component interfaces with the host bone as planned. (Fig 41). This is especially important at the medial aspect of the fossa; otherwise, the medial aspect of the fossa will lie away from the bone, resulting in an inappropriate or more lateral bearing relationship of the ramus/condyle component condylar head to the fossa. Using the fossa seating tool is important during fossa seating to assure it is fully seated against the bone and does not rock. (Fig 42).
- The retromandibular incision not only provides good access to the mandibular ramus for implanting the TMJR ramus/condyle component, but also provides access to the external carotid artery should uncontrollable hemorrhage arise from the internal maxillary artery or any of its branches. Therefore, this incision and dissection to bone should be completed before condyle resection, especially in ankylosis cases.
- A subparotid rather than a transparotid dissection should be used to prevent contamination of the ramus/condyle component implant site by saliva and prevent potential post-operative sialocele formation.
- There must be a minimum of 15 mm between the mandibular condylar resection and the height of the articular eminence area to accommodate the

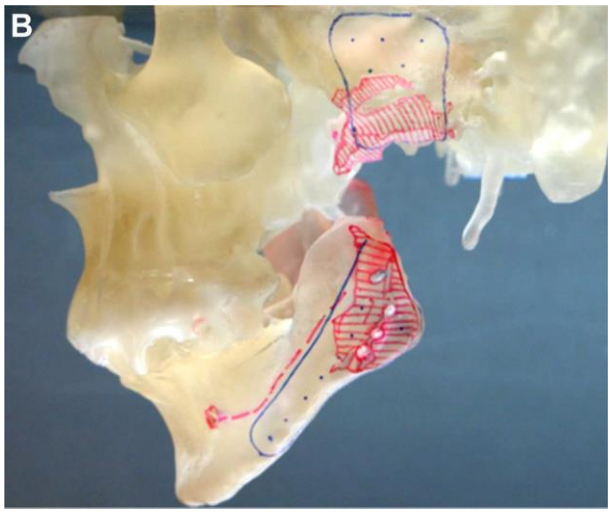

FIGURE 40 (cont'd).

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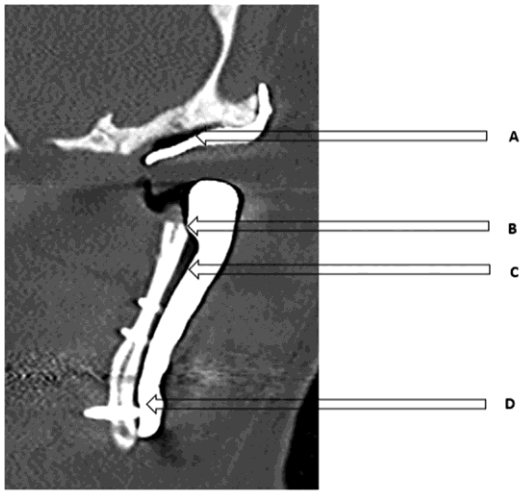
anterior flange of the TMJ Concepts fossa component. (Fig 43A).

- Custom TMJR ramus/condyle and fossa components are designed and manufactured to have a precise articulation. Therefore, the surgeon must be sure to fixate the components with the patient in the proper planned occlusion.
- Finite element analysis has confirmed that the maximum functional forces placed on a TMJR condyle/ramus component during function are concentrated at the most superior screw hole. Therefore, the most superior screw is important for stabilization during function.
- The screw lengths are recommended to enable bicortical fixation and to avoid functional irritation of the medial pterygoid muscle or temporalis muscle by overextended screw tips. If a screw hole should happen to strip-out, or the quality of the host bone is poor, rescue screws provided in the instrumentation kit should be used. Loose screws



A. Incomplete removal of soft tissue from the medial aspect of the fossa preventing the fossa component from seating properly.
B. Inadequate removal of bone at the superior aspect of the mandibular ramus preventing the ramus/condyle component from seating properly.
C. The most superior screw is not placed.
D. Improper screw length

FIGURE 41. Implant seating.

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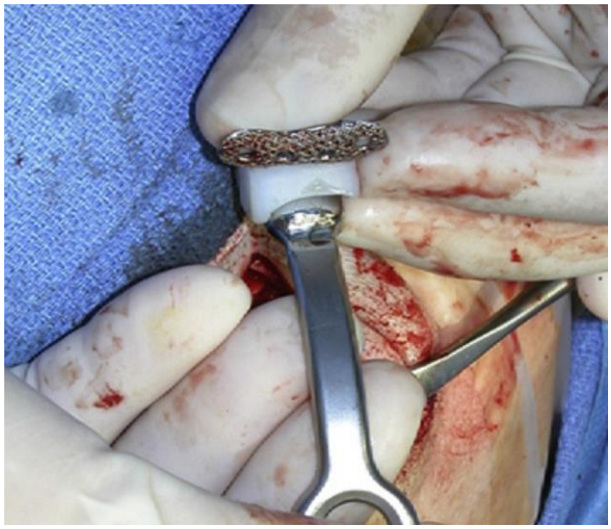

FIGURE 42. Fossa seating device.

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should never be left in place. All screws should be placed and retightened before closure. (Fig 43B).

- When drilling the pilot holes for the self-tapping fixation screws, the drill guide must be used to ensure that each drill hole is centered properly, otherwise there is the possibility that the shoulder of the screw will prematurely contact the ramus condyle plate causing the screw to fracture at its collar as the screw is hand tightened into its recessed position in the plate. Slow speed, utilizing copious irrigation is essential as these holes are drilled to assure that the bone retaining the screws remains viable. (Fig 44A).
- If an autogenous fat graft is utilized, it must be packed all around the articulation (Fig 44B). A Penfield neurosurgical elevator works well to maneuver the fat medially.
- All wounds should be copiously irrigated with the remaining vancomycin solution and closed carefully in layers. Drains are typically not required.
- While the patient is still under anesthesia, the auditory canal(s) and tympanic membrane(s) should be inspected with an ear speculum to ensure there was no intraoperative accumulation of irrigation fluid, blood, or inadvertent communication created between the TMJ area and these structures. This inspection should be documented in the operative notes. Blood clots should be removed with gentle, warm irrigation and careful suction. Instillation of antibiotic/steroid otic drops and occlusion of the external auditory canal with a cotton pledget are recommended to decrease the potential for the development of infection or inflammatory pain.
- Due to obscure involvement of the pathology with the auditory canal, perforation, tearing of the cartilaginous auditory canal or the tympanic membrane can occur. Should any of these occur, or be discovered on inspection, consultation with an otolaryngologist is advised to determine the best management option.
- In cases where bilateral hyperplastic coronoid processes are removed, light training elastics should be maintained for 1 week to prevent potential TMJR dislocation.

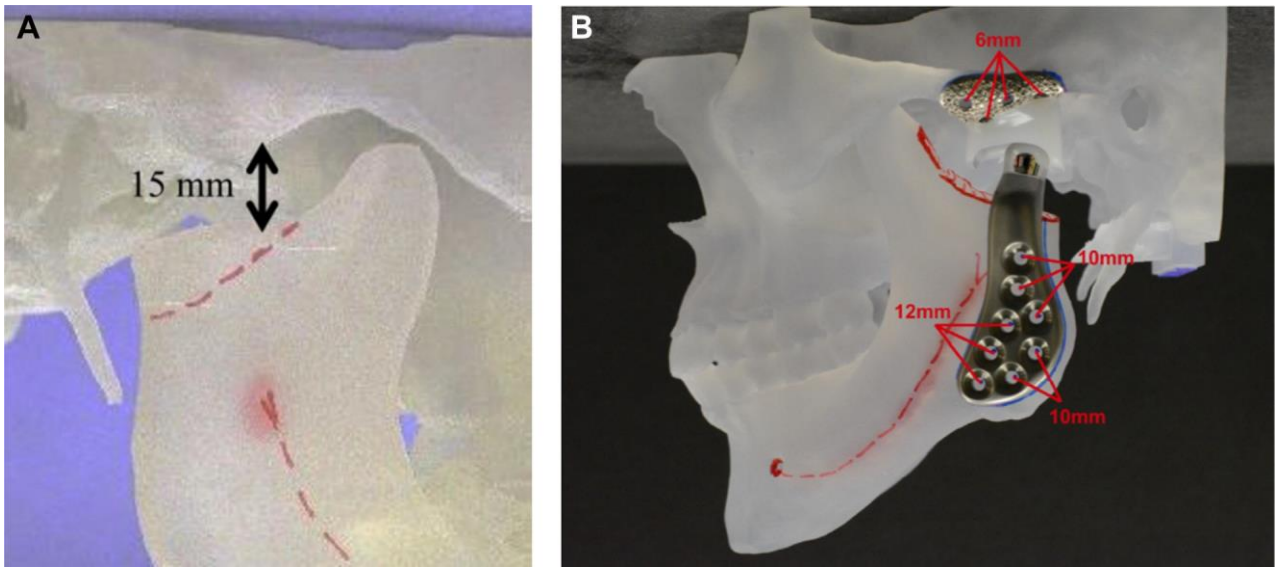

FIGURE 43. *A-B*, Condylectomy and implant design.

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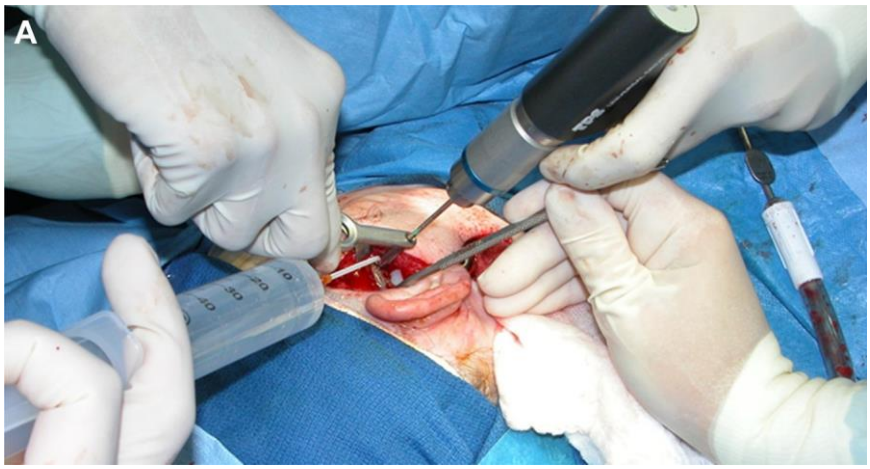

FIGURE 44. *A-B*, Placing screws and fat grafting. (Fig 44 continued on next page.)

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- A pressure dressing should be applied and kept in place for 24 hours. When the pressure dressing is removed, the wounds should be cleansed at least twice daily with a 50: 50 mixture of H_2O: H_2O_2 using a sterile cotton swab followed by a light coating of antiseptic ointment until the sutures are removed.

POSTOPERATIVE CONSIDERATIONS

- Early active physical therapy, either with commercially available jaw exercising devices or other methods, is essential to successful future function, especially in ankylosis cases.
- While the American College of Surgeons stated there was no evidence that post-operative antibiotic decreased surgical site infections, an exception was made for orthopedic joint replacement. Therefore, an antibiotic (first- or second-generation cephalosporin) that covers the spectrum of potential skin, ear, and saliva contaminants is recommended for 7 to 10 days postoperatively.
- Surgeons should educate the patient and relatives regarding proper wound care, personal hygiene, the early signs of a surgical site infection, and the importance of reporting symptoms to the surgeons as soon as they arise. Preprinted post TMJR instructional and FAQs encompassing these issues should be provided on discharge.

MANAGEMENT OF COMMON COMPLICATIONS

Infection

The infection rate for TMJR devices has been reported to be 1.86%.

Three types of TMJR postoperative infections can occur:

1 Superficial infections. It is Recommended that ‘‘Stitch Abscesses’’ and seromas be aggressively managed before the organisms involved affect the deeper tissues and TMJR components
2 Early deep infections. The earlier the diagnosis is made after TMJR surgery (2 to 5 days) and managed, following an aggressive early management protocol (Fig 45A), the greater the chance of salvaging the TMJR device.
3 Late deep infections. When signs and symptoms of infection appear weeks to months after TMJR implantation, a biofilm infection is the most likely culprit.[101] Management using the modified late management protocol (Fig 45B) is recommended.

Heterotopic Ossification

The incidence of heterotopic ossification associated with a TMJR is reported to be 0.58%. Heterotopic bone formation is the presence of bone in the soft tissue surrounding a joint replacement device where bone normally does not exist, leading to decreased joint mobility and pain. History and imaging are used to distinguish it from other diagnostic possibilities. As prophylaxis, a nonsteroidal anti-inflammatory drug (such as indomethacin), a diphosphonate (such as ethane-1-hydroxy-1, 1-diphosphate), or local radiation therapy have all been recommended. Surgical removal of the heterotopic bone has been endorsed to preserve joint mobility, but heterotopic bone formation is likely to recur and possibly progress. Therefore, as prophylaxis in TMJR, it is recommended that an autogenous fat graft be packed around the TMJR articulation to decrease potential recurrence.

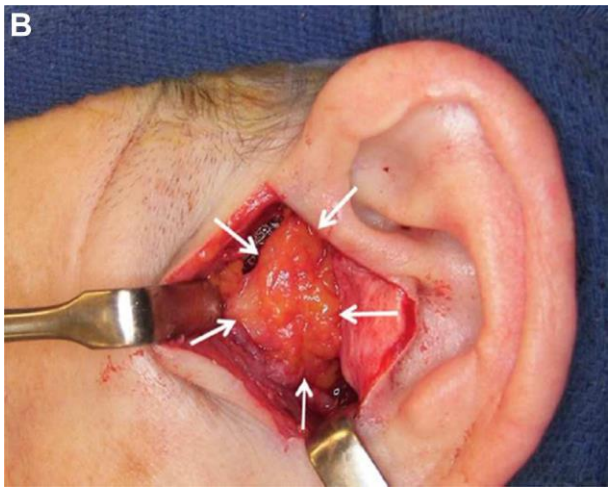

FIGURE 44 (cont'd).

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A

Early Protocol
1. Infection identified
2. Broad-spectrum antibiotics started
3. Infectious disease consult
4. Surgery a. I/D, C&S, debridement b. Prosthesis scrubbed with toothbrush and Betadine solution c. Placement of irrigating catheters/drains for 4-5 days
5. Irrigation of catheters Q4h with DAB for 4-5 days, then catheters/drains removed
6. PICC line placed
7. IV antibiotic therapy based on C&S
8. Outpatient IV antibiotics for 4-6 weeks
9. Outpatient oral antibiotics for 10 days

B

Late Protocol
1. Infection identified
2. Broad-spectrum antibiotics started
3. Infectious disease consult
4. Surgery Stage I a. I/D, C&S, debridement, device removed b. Placement of PMMA/Tobramycin spacer
5. PICC line placed
6. IV antibiotic therapy based on C&S
7. Outpatient IV antibiotics for 6-8 weeks
8. Surgery Stage II Replacement with new device at 8-10 weeks
9. IV antibiotics until discharge

FIGURE 45. *A-B*, Early and late infection protocols.

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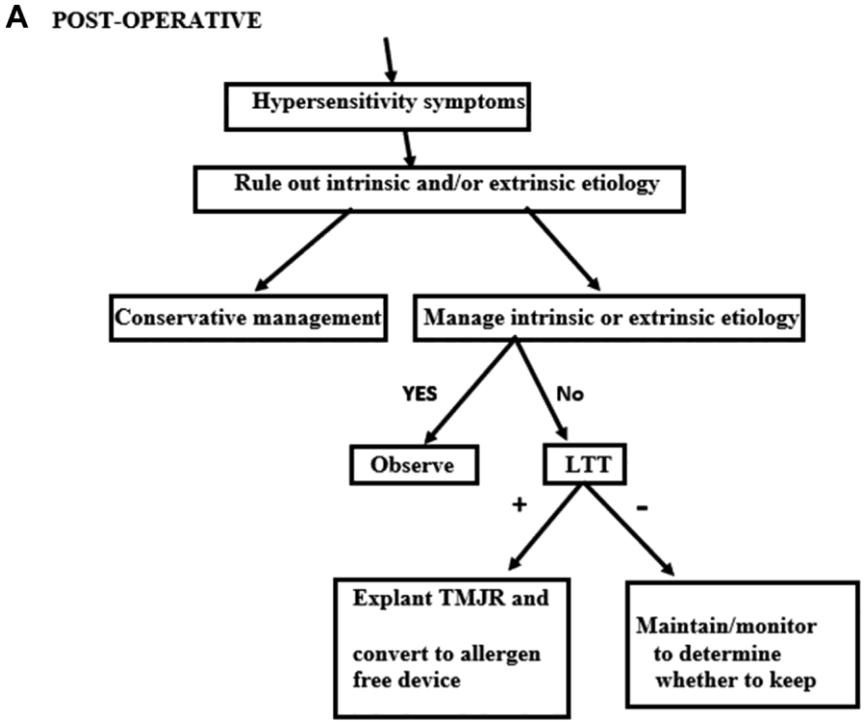

FIGURE 46. *A,* Postoperative hypersensitivity testing. *B,* Post TJR pain. (Fig 46 continued on next page.)

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Material Hypersensitivity

Long-term TMJR follow-up data reveal a reported post-operative material sensitivity of 0.14%. Should a post-TMJR patient be documented by LTT as sensitive to any nickel containing alloy metal (CoCr alloy) used in the manufacture of a TMJR device (Fig 46A), the metal components of a replacement TMJR should be manufactured from a non-reactive material.

Post-TMJR Chronic Pain

Persistent or chronic post-surgical pain can become both a significant clinical and economic issue. While the estimated mean incidence varies between 10 and 50% relative to a variety of surgical procedures, to date it has been rarely reported post TMJR (0.43%). Acute pain almost always originates from nociception in somatic or visceral tissues (intrinsic pain); however, not every pain sensation originates from nociception (extrinsic pain). After TMJR, there may be both intrinsic and extrinsic causes for pain. The surgeon must rule each out in a systematic manner to manage the etiology appropriately. (Fig 46B).

Intrinsic Causes for Post TMJR Pain

The Biologic Response to Metal Implants report states that when working up a patient with a painful total joint, hypersensitivity should be the last item on the list, since the literature clearly demonstrates that 1% or less of joint replacement device failures are causally related to material hypersensitivity. Therefore, the most common causes for post TMJR pain are infection, heterotopic ossification, micromotion and loose hardware. However, there are two other potential intrinsic causes of post TMJR chronic pain, synovial impingement, and an adverse local tissue response to material wear, both of which mimic hypersensitivity and should be ruled out before TMJR removal and replacement.

The synovial impingement syndrome has been reported to be a cause of pain and dysfunction after orthopedic joint replacement. After any joint replacement, a pseudo synovium develops. Westermark and Monje demonstrated this in TMJR patients. Further, Murakami et al demonstrated synovial plicae as invaginations of synovial tissue in the TMJ similar to those found in the hip and knee. Davis presented arthroscopic images of inflamed synovial plicae entrapped between the bearing surface of TMJR devices in patients with post TMJR pain and dysfunction and demonstrated that the early clinical outcomes of arthroscopic management with decreasing pain and increasing the MIO. Larger studies with longer follow-up are needed to further classify the different causes of prosthetic failure and advance the approaches to management. TMJR arthroscopy should be reserved only for surgeons with Level 3 arthroscopy skills.

Although rare in TMJR due to minimal functional TMJ loading compared to hips and knees, material

B

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

FIGURE 46 (cont'd).

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wear results in the release of material wear particles and ions leading to synovial and periarticular soft tissue inflammation. Therefore, this local tissue response may act as another source of post TMJR pain and dysfunction. Diagnostic local anesthesia infiltration may be diagnostic and careful debridement can be therapeutic.

Extrinsic Causes for Post TMJR Pain

It is noteworthy that many of these painful and dysfunctional post TMJR patients have been multiply operated and/or misdiagnosed muscular TMD patients with multiple comorbidities and persistent centrally mediated pain. A multicenter cross-sectional study to identify pre-operative risk factors for pain at rest and with activity after total hip and knee replacement stated that moderate to severe pain was reported by 20% at rest and 33% with activity. Among the significant predictors for post-operative pain at rest were female gender, increased severity of pre-operative pain in the hip or knee area, and preoperative use of opioids. Predictors for post-operative pain with activity were severity of the pre-operative hip and/or knee pain, pre-operative use of anticonvulsants and antidepressants, and prior previous hip/knee surgery.

A prospective study of knee replacement patients identified possible predictors of outcome 6 months post-operatively. The strongest predictors of outcome were pre-operative pain with function: those with less severe pre-operative disease obtained the best outcome; diagnosis: those with rheumatoid arthritis did better than those with osteoarthritis; social status: economically needier patients had worse outcomes; and mental status: anxiety/depression were associated with poorer pain symptom relief. The orthopedic literature also reveals that the greater number of pre-operative co-morbidities, the poorer the outcomes. These data are consistent with TMJ disorder data that demonstrated that the presence of co-morbid conditions may perhaps explain why 50% of patients seeking care for TMJ pain, some of whom were multiply operated and/or exposed to failed materials or devices, still report experiencing pain 5 years later, and 20% of chronic TMJ pain patients experience long-term disability from their pain.

The appropriate overall management of patients requiring TMJR requires the surgeon make the correct diagnosis and understand the patient's associated predictors of outcomes, especially any comorbid conditions. Then the surgeon must perform the surgery at the right time, correctly and aseptically, utilizing the appropriate TMJR system. This assures the results will be professionally satisfying for the surgeon, and most importantly provide the best outcome for the patient.

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Stock Alloplastic Temporomandibular Joint Reconstruction[230]
Armamentarium
24-Gauge Surgical Wire
Obwegeser Periosteal Elevator
Basic Soft Tissue Set
PDQ Zygoma retractors
Army-Navy retractors
Surgairtome
1-mm side cutting fissure bur
Dunn-Dautrey retractors
Dingman Bone Holding forceps
T-bar osteotome
Molt Curette
Nerve stimulator
Oscillating Saw
Nasal freer
Flat Diamond Rasp
Antibiotic Irrigation
Needle-tip Bovie
Malleable retractors
Suture – 3-0, 4-0 Vicryl (Ethicon Inc, J & J Company - J494 G), 5-0 Fast gut

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The indications for total joint replacement (TJR) include:

- Osteoarthritis and inflammatory arthritis
- Ankylosis, re-ankylosis and heterotopic bone formation
- Failed autogenous reconstruction
- Failed alloplastic TMJ reconstruction
- Condyle fracture
- Functional Deformity
- Benign neoplasms and cysts
- Malignant pathology when not irradiated
- Developmental abnormality

Technique

STEP 1—PREP AND POSITIONING

The patient should be intubated with a nasal Rae with the endotracheal tube either sutured in place or secured to the head wrap to allow for head and mandible manipulation. A preoperative antibiotic should be given during this time to ensure adequate tissue levels prior to incision. Hair should first be removed from the proposed incision site, typically to the superior portion of the helix. A head wrap is then applied and secured with skin staples. The skin is prepped. The patient is then draped, and a urologic drape is then adapted and used as a sterile barrier to manipulate the mandible during the operation. Minimizing contamination from the oral cavity is critical for reducing postoperative infections. Attention to sterile technique, especially when alternating between the surgical site and the oral cavity, is the most important step in preventing prosthetic infection. (Fig 47). Finally, the surgeon should address the external auditory canal to minimize potential contamination of the surgical filed. This can be accomplished by irrigation of the canal with antibiotic saline or isolation of the canal through the use of mineral oil impregnated cotton or suturing.

STEP 2—ENDAURAL INCISION

An endaural incision is the preferred approach to the mandibular condyle in alloplastic joint reconstruction. This approach provides an excellent cosmetic outcome. In addition, it allows for a stepped approach to the joint, which improves tissue coverage and increases the distance of the prosthetic device from the incision. (Fig 48).

STEP 3—Retro-MANDIBULAR INCISION

The retro-mandibular incision is marked by placing a gloved finger from the lobule of the ear to the angle of the mandible. The incision is marked approximately a 1-cm below the lobule of the ear to the pre-masseteric notch. (Fig 49A). A nerve stimulator is used during this dissection to identify and avoid the marginal mandibular nerve. Once dissection through the superficial layer of the deep cervical fascia is completed, the angulation of the dissection changes to parallel the sternocleidomastoid muscle. A Kelly hemostat is used to define the plane between the submandibular gland and sternocleidomastoid muscle. This will expose the posterior belly of the digastric muscle, marking the deepest point of the dissection. An army-navy retractor is used to retract the facial artery anteriorly exposing the inferior border of the mandible and anterior portion of the pterygomasseteric sling. The sling is incised with a 15-blade along the avascular aponeurosis. A Molt curette can be used to strip any remaining periosteal attachment. This allows a clean dissection of the masseter from the lateral aspect of the mandible with the aid of an Obwegeser periosteal elevator. Once this dissection is complete, communication between the endaural and retromandibular incisions exists in a safe subperiosteal plane. (Fig 49B). Care should be taken to minimize trauma between the small cuff of tissue between the two incisions, as the facial nerve travels in this plane.

STEP 4—CONDYLECTOMY

It is essential to complete the retromandibular incision prior to the condylar osteotomy, to ensure rapid

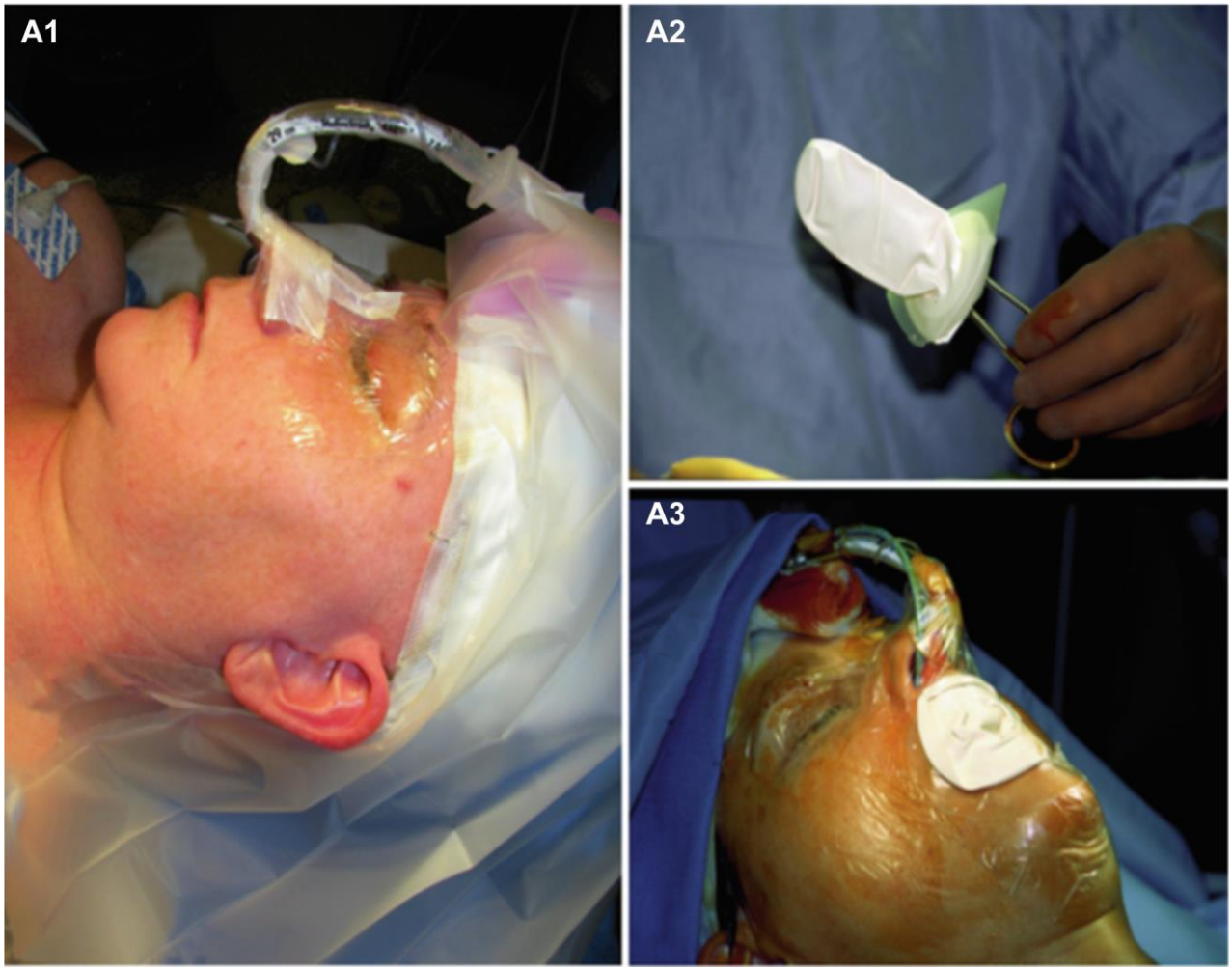

FIGURE 47. Sterile Preparation.

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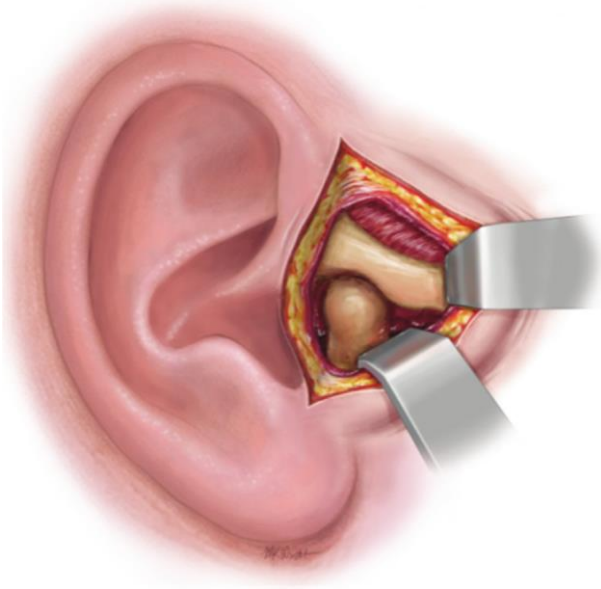

FIGURE 48. Endaural incision.

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access to the underlying vasculature, in case difficult-to-control bleeding is encountered. In cases of ankylosis or previous surgeries where altered anatomy may be encountered, thrombin-soaked sponges, collagen, or flow seal should be available to control bleeding following removal of the condylar segment. A two-step osteotomy has been developed to minimize risk to the internal maxillary artery and ensure adequate bone removal for the fossa component. Two Dunn-Dautrey retractors and a condylar neck retractor should be placed in the subperiosteal plane at the level of the neck of the condyle. This allows improved visualization and protects adjacent soft tissue structures. Once the neck of the condyle is fully exposed, a 1-millimeter fissure bur is used to perform the condylectomy. (Fig 50A). An osteotomy is performed by first starting at the midpoint of the condylar neck sparing the medial cortex. The cut is then extended both anteriorly and posteriorly toward the Dunn-Dautrey retractors. A T-bar osteotome is then used to complete the osteotomy. (Fig 50B).

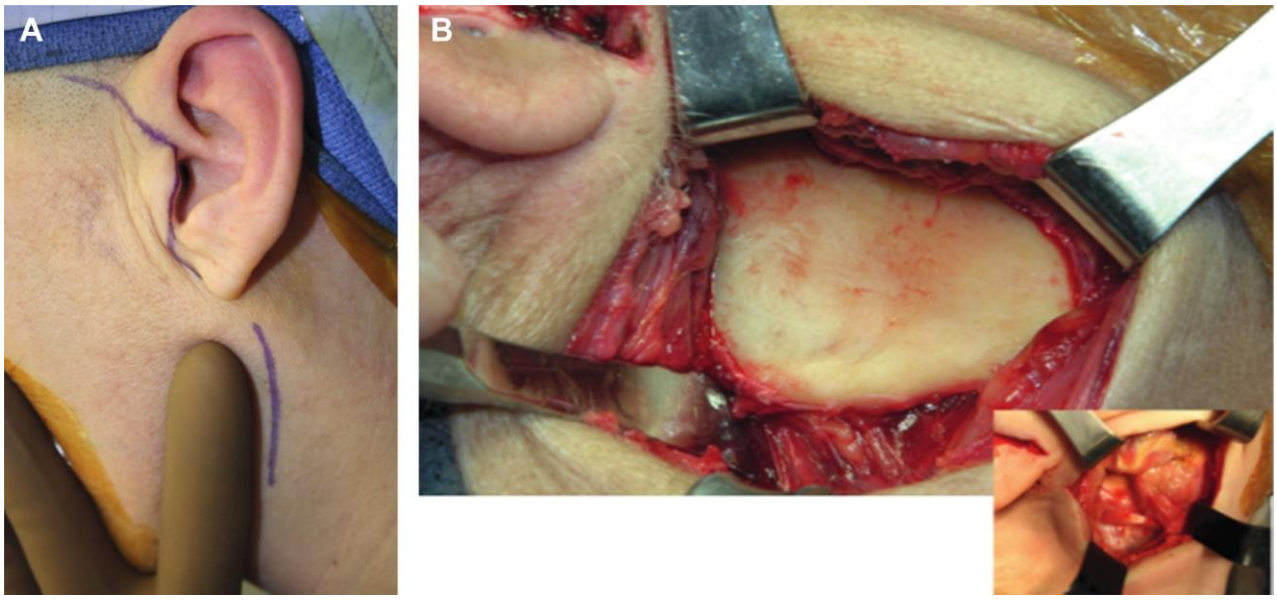

FIGURE 49. *A*, Incision Sites. *B*, Retromandibular access.

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Alternatively, a piezoelectric saw may be used. The condyle is then grasped with a bone-holding forceps and the lateral pterygoid muscle is then carefully dissected free. Significant bleeding may occur during this portion of the procedure due to the rich vascularity in the region. (Fig 50C). With the condyle removed, the mandible is superiorly repositioned by grasping the inferior border through the retromandibular incision with a bone-holding forceps. This allows for the second osteotomy, at the level of the inferior portion of the sigmoid notch, to occur at a safer distance from the internal maxillary artery.

STEP 5—FOSSA PLACEMENT

Once adequate bone is removed from the mandible, attention is then directed to the articular eminence. The eminence is reduced to allow for tripod stability of the fossa component. This is best accomplished with a diamond rasp. While reducing the eminence, it is important that the rasp parallels the superior aspect of the zygomatic arch. This ensures correct angulation of the fossa and minimizes potential dislocation. Once the eminence is modified, fossa sizers should be placed to determine the correct implant size. If the fossa flange does not sit flush with the eminence, the rasp can be used to reduce any irregularities along the lateral aspect of the zygomatic arch. (Fig 51). It is important to note, the system utilizes trial sizers, which can be placed multiple times until satisfactory position and seating is obtained. Once satisfactory seating and angulation of the fossa has been accomplished with the trial sizer, the fossa implant is soaked in antibiotic impregnated saline and then inserted into the modified glenoid fossa. This minimizes the potential for contamination. The fossa can then be initially secured with two screws until the adequacy of the fossa position can be confirmed although 5 screws are typically used once this is verified. (Fig 52). An assistant should then place the mandible into occlusion and the clearance between the mandible and fossa is checked. A nasal freer should easily pass between the fossa and the stump of the condylar neck. If resistance is met, a hand-held rasp or fissure bur should be used to further reduce the mandible.

STEP 6—INTERMAXILLARY FIXATION

The patient is placed in intermaxillary fixation. This is best accomplished, in a stable and efficient manner, with the use of Ivy loops placed on the premolars. Alternatively, Erich arch bars, IMF screws or pigtail wires can be used. (Fig 53A). A separate intra-oral instrument set should be utilized to minimize contamination of the joint. Re-prepping and draping of the patient, as well as glove changes before returning into the surgical wounds should also occur.

STEP 7—CONDYLAR PLACEMENT

The condylar component is placed through the retromandibular incision. Sizers are available to determine the correct size mandibular component. The condyle head should sit in a slight posterior direction and at the midpoint of the fossa in the medial-lateral dimension. (Fig 53B). This will ensure good matching and function of the prosthesis. In order to achieve optimal placement, any irregularities in the lateral aspect of the mandible interfering with seating can

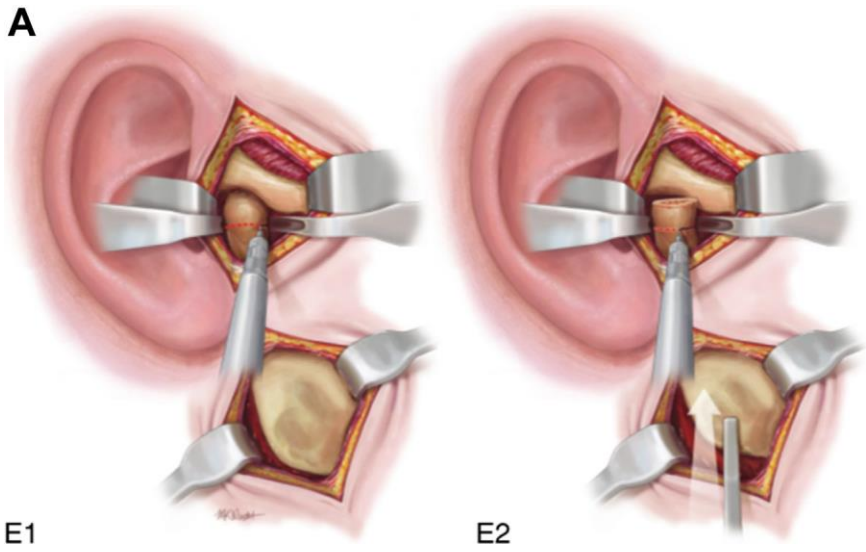

FIGURE 50. *A*, Condylectomy. (Fig 50 continued on next page.)

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be reduced with the reciprocating diamond rasp. Once satisfactory placement of the mandibular component is achieved, it should be secured with two screws.

STEP 8—RANGE OF MOTION

With the mandibular and fossa component in place, the intermaxillary fixation should be released and the range of motion and function should be checked. It is important to ensure that the prosthesis does not dislocate. Finally, the maximal opening should be measured. If a passive opening of less than 30 mm is found, a coronoidectomy should be considered. The osteotomy should occur through the endaural incision.

STEP 9—FINAL SCREW PLACEMENT

Once optimal range of motion and position of the prosthesis have been achieved, final screw placement can occur. Care should be taken to ensure that the screw holes do not encroach on the inferior alveolar nerve when placing the mandibular screws. This is easily accomplished with the aid of standard radiographs.

STEP 10—CLOSURE

Closure begins once all wounds have been thoroughly irrigated with antibiotic-impregnated saline. Hemostasis is confirmed and the endaural incision is closed in layers. The retromandibular incision is then irrigated, hemostasis confirmed and closed in layers beginning with the superficial layer of the deep cervical fascia. The pterygomasseteric sling is left to passively reattach. The skin is then closed, and dressings are placed. Once the wounds are dressed, the IMF wires or screws are removed.

ALTERNATE OR MODIFIED TECHNIQUE

Ankylosis

Ankylosis of the temporomandibular joint presents several additional challenges to reconstruction and restoration of temporomandibular joint function. Patients with massive ankylosis should have a CT angiogram to identify any potential vasculature that may result in bleeding. If the internal maxillary artery is found to be adjacent to the bony mass, selective preoperative embolization can be utilized to minimize the potential for massive hemorrhage.

Intubation can be problematic with minimal mouth opening and may necessitate fiberoptic intubation. Close communication and planning with the anesthesia team is essential for the safe induction of anesthesia in these patients. Additional measures including awake intubation, or tracheostomy, may also be considered. The ankylotic bony mass often extends beyond the boundaries of the joint capsule. This presents three unique problems. Firstly, the alteration of local anatomy and proximity to adjacent vasculature increases the risk of bleeding. Secondly, the bony mass renders the separation of the mandible from skull base more difficult and increases the risk of middle cranial fossa exposure or perforation. Lastly, limited movement of the mandible may cause fibrosis of musculature or elongation of the mandibular coronoid process, resulting in a secondary cause of trismus. In order to minimize or avoid the first two potential

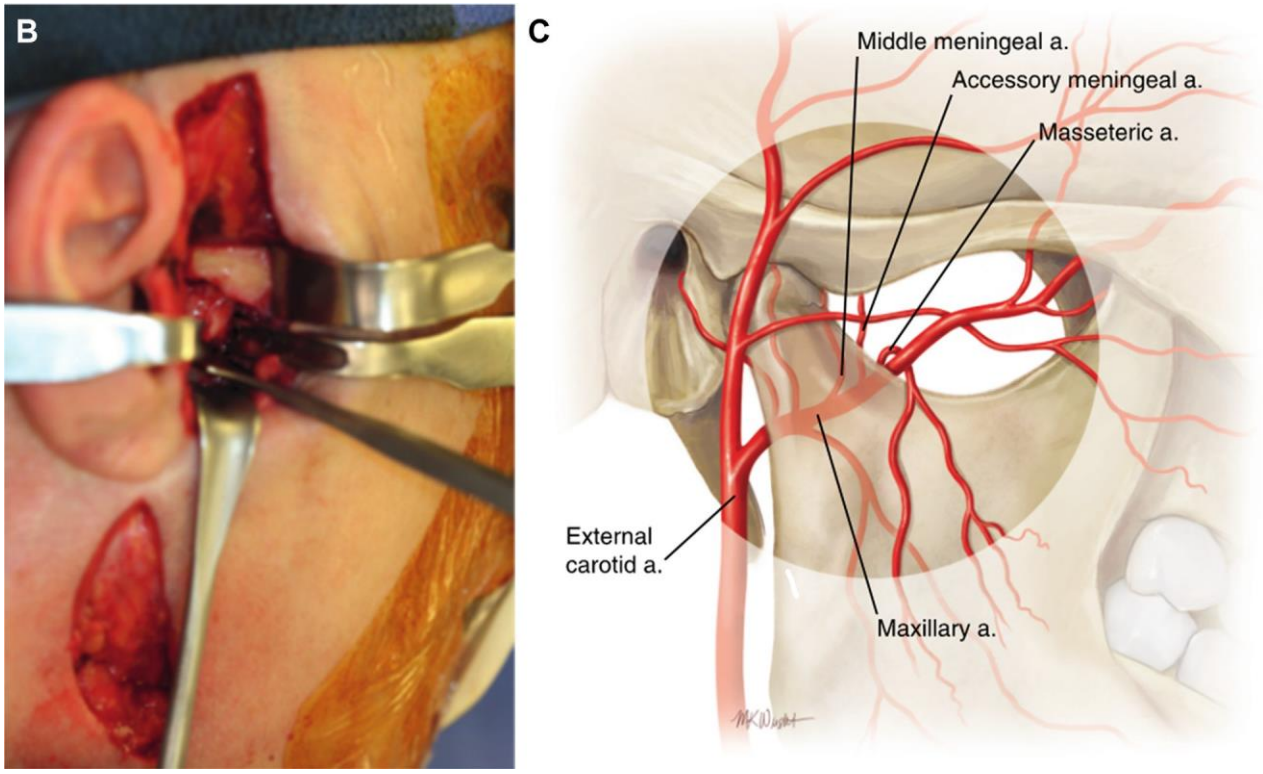

FIGURE 50 (cont'd). *B,* Completing the osteotomy. *C,* Vascularity.

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problems, the osteotomy for the condylectomy should be modified. The initial osteotomy is the placed at the most inferior aspect of the ankylosis to be removed. Once this lower osteotomy is completed and the mandible separated from the skull base, attention should be directed to removing the ankylotic mass. Often, a pseudoarthrosis, or evidence of previous meniscal anatomy is present. A 1-mm fissure bur should be used to initiate the osteotomy, with a slight inferior angulation. A curved T-bar osteotome can then be used to separate the remaining bony mass from the skull base. (Fig 54). The remaining bone can be carefully and judiciously removed with the aid of a diamond rasp or pineapple bur. Alternatively; the osteotomies may be aided by the use of virtual surgical planning and custom cutting guide fabrication. (Fig 55A and B). In this scenario, a saw may be considered for the osteotomy. The use of CT-guidance may also be considered during this aspect of the procedure to minimize iatrogenic entry into the middle cranial fossa. Once bone removal is complete, jaw function should be evaluated. If opening is less than 30-mm, a

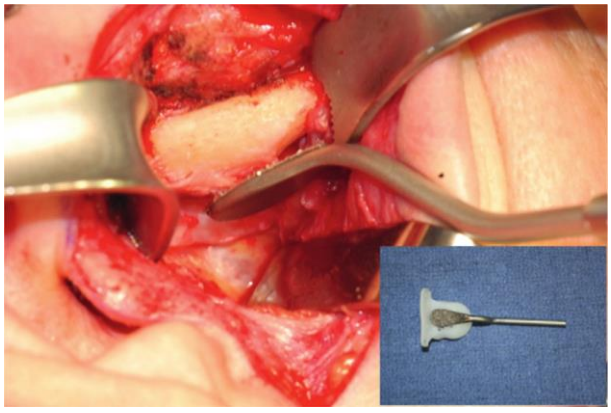

FIGURE 51. Eminectomy.

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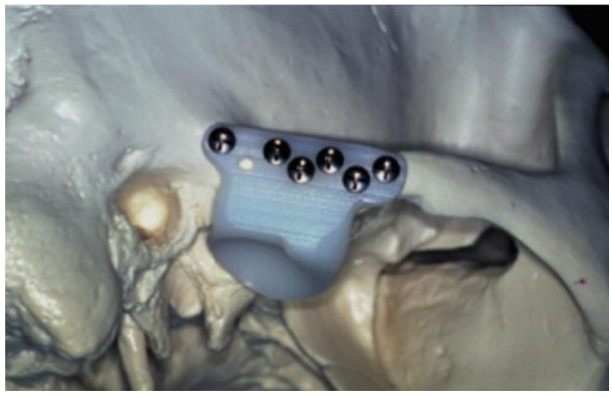

FIGURE 52. Fossa position.

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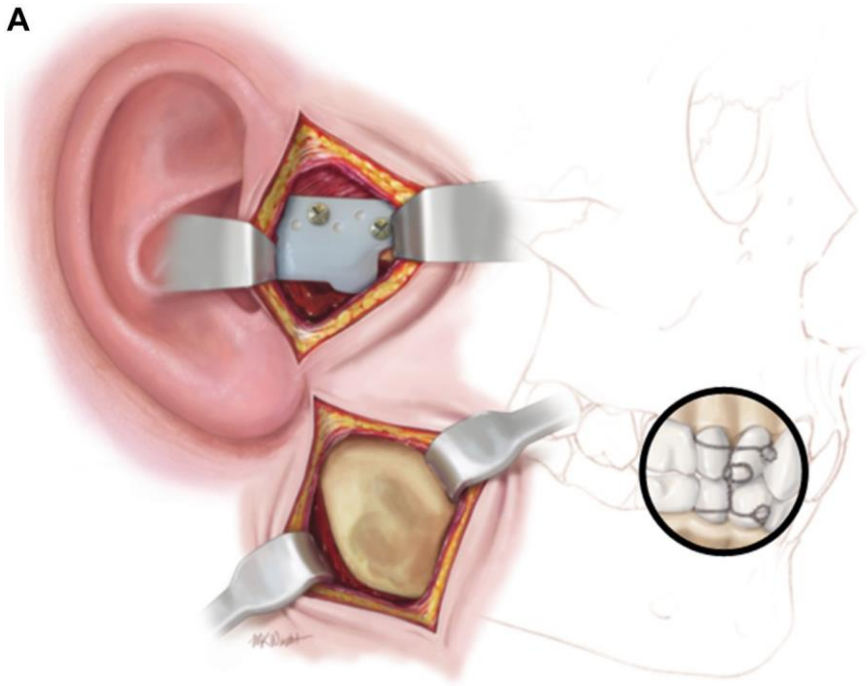

FIGURE 53. *A*, Initial Fossa Fixation. (Fig 53 continued on next page.)

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coronoidectomy should be considered. It is also helpful to actively stretch the mandible with the aid of two molt mouth props or a Bell-retractor. Once the prosthesis is placed to prevent re-ankylosis, fat grafting should be utilized to minimize dead space and prevent adhesions and heterotopic bone formation.

Correction of Facial Asymmetry

Growing patients affected with temporomandibular joint disease will often have a secondary facial asymmetry. In cases where the disease process results in loss or destruction of the native mandibular condyle, alloplastic joint reconstruction can serve to replace the non-functioning joint and be used to correct the pre-existing dentofacial discrepancy in the skeletally mature patient. In conjunction with a LeFort I osteotomy, the skeletal asymmetry can be corrected in a single-stage surgery. (Fig 56). The use of an alloplastic joint further provides a stable platform for orthognathic reconstruction. The protocol for the reconstruction of facial asymmetry secondary to temporomandibular joint disease begins with a standard LeFort I osteotomy with repositioning of the maxilla utilizing an intermediate splint based on the position of the native mandible. Once the maxilla is secured in its new position, the total joint reconstruction proceeds with the condylectomy as described above. The mandible is then placed into its new position utilizing a final splint and the prosthesis is placed. If a midline correction or yaw correction is needed, a contralateral sagittal split osteotomy can be performed prior to placing the patient in intermaxillary fixation. The prosthetic device is the placed, and function checked. Once complete, a genioplasty may also be performed at the same time, if necessary.

Avoidance & Management of Intra-operative Complications

Surgical complications can be divided into failure of technique, failure of diagnosis, failure of device, failure secondary to patient disease, and relative failure secondary to patient expectation. Fortunately, TMJ prosthetic infection is rare with an infection rate of 1.6%. Prosthetic joint infection can be difficult to treat and is often secondary to the formation of biofilms. The microbiology of TMJ device infection is often polymicrobial and typically consists of skin flora. Early infections (<3 weeks) may be treated with antibiotics, but intermediate or late infections often require device removal, prolonged IV antibiotics and, a new prosthesis. In 2010, Wolford published the results of his protocol for treating infections. Early prosthetic joint infections were treated by a peri-prosthetic antibiotic washout-out and debridement, placement of irrigation catheters and followed by 4-6 weeks of IV antibiotics. Patients with chronic infections were treated with a similar protocol, except the prosthesis was removed and replaced 8-10 weeks later. Wolford was able to salvage 4 of the 5 infected prostheses with the acute protocol and achieve resolution of all infections in

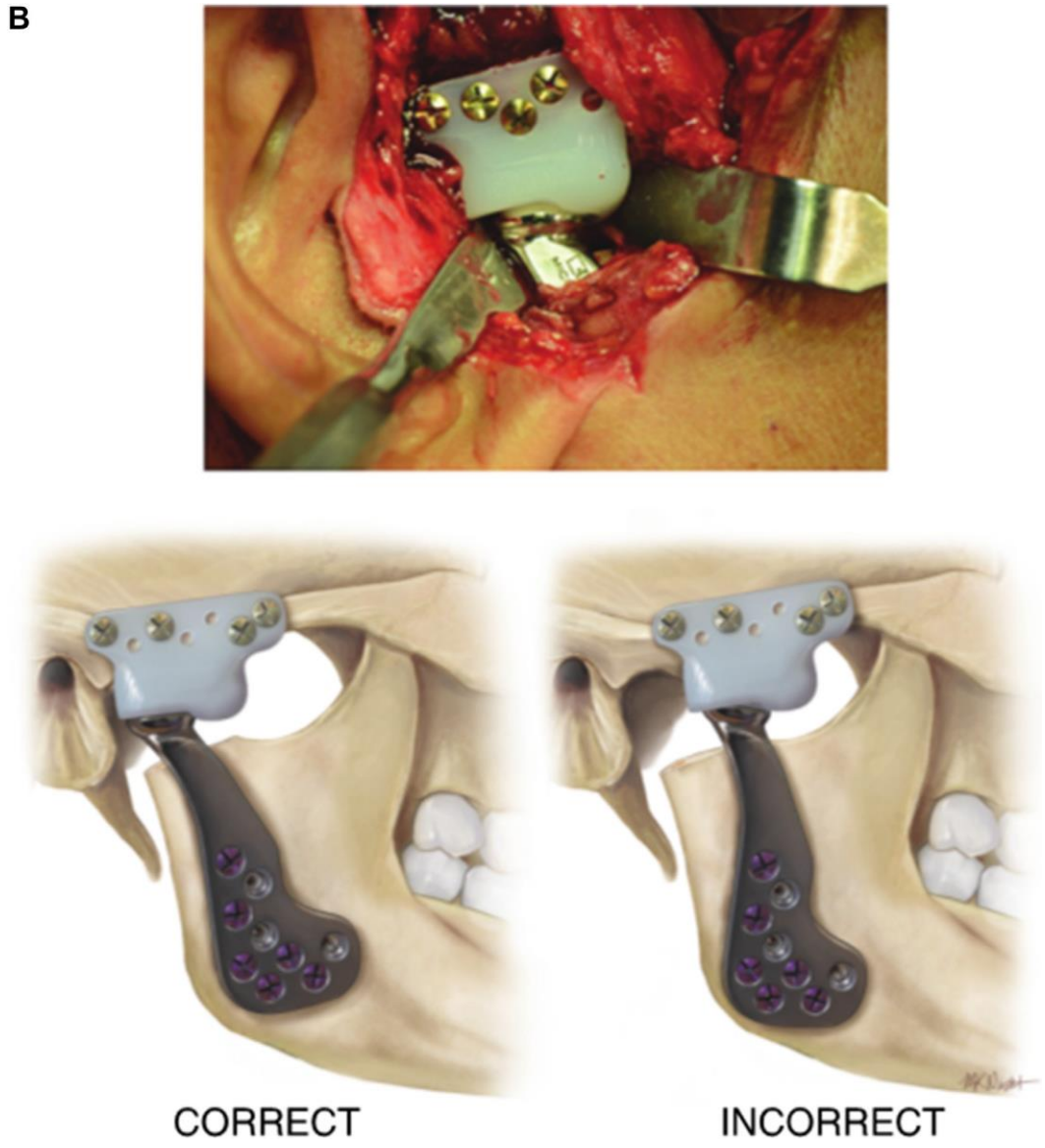

FIGURE 53 (cont'd). *B*, Final fossa and ramus positioning.

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the chronic protocol.[19] Strict attention to aseptic technique, the use of peri-operative antibiotics, as well as minimizing cross-contamination from any intra-oral procedure is key to decreasing prosthetic joint infection. Additional measures utilized include external auditory ear irrigation with antibiotic impregnated saline and soaking components in antibiotic saline prior to implantation.

Heterotopic bone formation and re-ankylosis can be a difficult problem, particularly in the multi-operated patient. In addition to excessive bone formation, there is often extensive scarring, which renders achieving acceptable maximal incisal opening difficult. Several methods to combat heterotopic bone formation have been addressed in the orthopedic literature. This includes nonsteroidal anti-inflammatory medication (indomethacin) and the use of low dose post-surgical radiation. Reported regimens include 10 Gy in five fractionated daily doses in the immediate post-operative period. Wolford reported the use of autogenous fat grafting around the prosthetic joint to prevent bone formation and scarring. In this case series of 37 joints, no patient grafted with fat formed heterotopic bone, compared to 35% of the controls.

Bleeding is most often encountered from the middle meningeal artery, internal maxillary artery, masseteric artery or the lateral pterygoid muscles. Identification and ligation of the severed vessel is clearly preferred but often difficult. Several hemostatic agents should be available to help with bleeding, particularly since

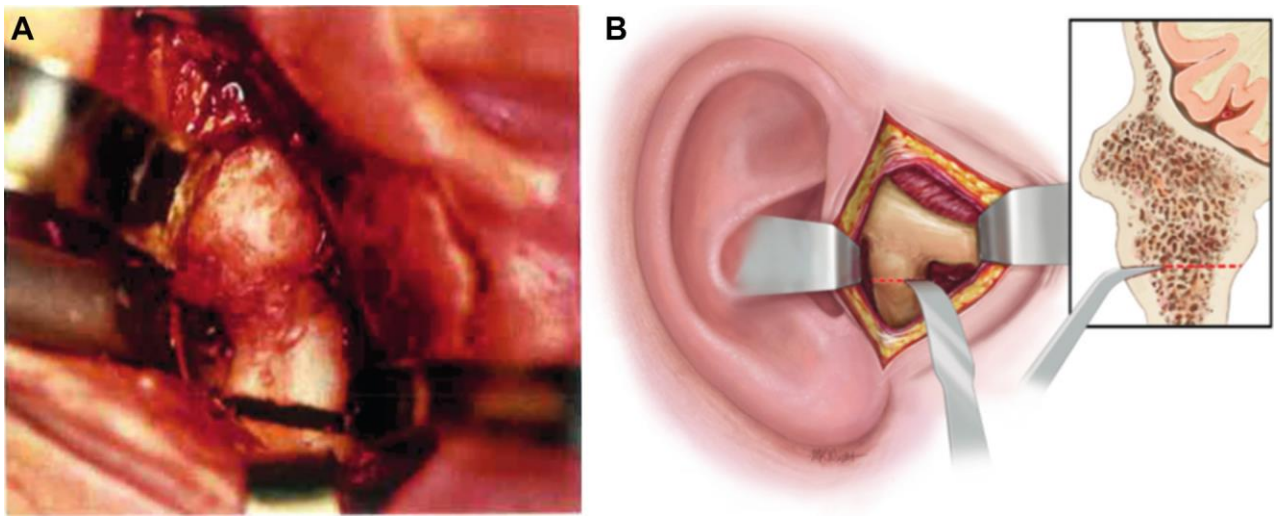

FIGURE 54. Ankylosis.

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many of these vessels can be difficult to identify through the standard approaches. It is essential to achieve hemostasis prior to closure to prevent hematoma formation. Thrombin, collagen, or flowable gelatin matrix may be utilized to decrease bleeding. For more brisk bleeding, which is difficult to control, access to the external carotid artery is possible through the retromandibular incision. It is important to identify at least three branches to ensure proper identification of the external carotid artery before it is ligated. Collateral circulation can limit the effectiveness of ligation. Lastly, interventional radiology may be considered to identify and occlude a bleeding vessel.

Malocclusion following placement of the prosthesis is ideally identified prior to final screw placement. If only two screws were placed, the mandibular component can easily be re-positioned without compromising stabilization. Prior to release of the intermaxillary fixation, occlusion should be checked to ensure correct alignment and to confirm that there was no failure of the intermaxillary wires. The clearance between the fossa and mandible should also be checked for interference, and if present, must be addressed to obtain acceptable occlusion and function. The surgeon should not hesitate to reposition the components to ensure proper occlusion.

Finally, patients with chronic or centralized pain can be difficult to manage in the acute post-operative period. Unfortunately, many patients with TMJ disease have undergone multiple operations and subsequently developed centralized pain. Consultation with a pain specialist is important in the care of these individuals. Several studies have shown that patients with fewer open TMJ procedures report significantly lower pain scores following total joint surgery compared to those with multiple operations. This information suggests that patients, following a failed open arthroplasty, may benefit from total joint replacement compared to further revision procedures. Prior to the consideration of TMJ replacement surgery, the clinician should consider a diagnosticblock to determinethe percentage of pain resolution. For patients with minimal pain reduction, additional surgery should be avoided if possible.

Post-operative Considerations

Postoperative radiographs should be obtained prior to patient discharge in order to confirm correct angulation, screw position and condylar seating. Patients are seen 10 days following discharge for suture removal and passive jaw motion is encouraged. Active jaw physical therapy may be indicated for patients and is typically initiated for 4-6 weeks following surgery, beginning on post-operative day one. Post-operative pain management should primarily consist of anti-inflammatory medication and acetaminophen with narcotic medications for rescue. The patient should be quickly titrated off narcotic medication during the immediate post-operative period. For patients taking narcotic medication prior to surgery, or a requiring narcotic pain medication for longer than 1 month, referral to a painspecialist should be considered.

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TMJ Ankylosis[231]

Armamentarium
#9 Periosteal elevator
#15 Blade
Arch bars
Adson forceps
Bipolar forceps
Channel retractor
DeBakey tissue forceps
Double Skin Hooks
Dunn-Dautrey condyle retractor
Freer elevator
Hemostats
Kittner dissector sponges
Metzenbaum curved scissors
Microdrill
Needle tip microdissection needle
Nerve stimulator
Obwegeser-Freer curved and J shaped elevators
Obwegeser right angled curved up and down retractors
Ruler
Rongeur
Sagittal saw
Scissors
Senn retractor
Seldin elevator
Ultrasonic handpiece (Piezo)
Tessier straight and curved osteotome
Virtual surgical planning cutting guides

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PROCEDURE

Virtual Surgery Planning

The managementof individuals with temporomandibular joint ankylosis can be very challenging and requires coordination of care with radiology and anesthesiology. Essential radiology includes CT with axial, coronal, and sagittal images. Consideration should be given to the use of Computed Tomography Angiography (CTA) if concern exists for the presence of blood vessels within the ankylotic mass. Measurements can be taken from the CT scan using identifiable landmarks to determine the superior and inferior boundaries of resection. Virtual surgical planning (VSP) can be an extremely helpful adjunct when doing this and allows for the development of a precise surgical plan. VSP can also provide splints to guide occlusal relationships and cutting guides for precise resection. If the patient has sufficient opening, dental models or intraoral scans can be incorporated into the CT to aid the accuracy of occlusal planning. The combination of VSP, stereolithographic models, and CT-based navigation has enhanced the safety, accuracy, and efficiency of ankylosis surgery. This approach reduces the need for intraoperative improvisation due to the accuracy of the planning and precision of surgical guides and navigation. In addition to enhancing the resection of the ankylosis, the use of VSP and models also enhances the design of the alloplastic joint replacement. Orthognathic occlusal splints are also used to facilitate functional occlusal relationships. (Fig 57A-F).

A plan for providing a safe airway should be coordinated with the anesthesiologist before surgery. Fiberoptic nasoendotracheal intubation is frequently required due to limited opening. The patient should be informed that a tracheostomy may be performed if fiberoptic intubation is unsuccessful.

Surgical Procedure

STEP 1

After the induction of anesthesia and verification of endotracheal intubation, the endotracheal tube should be stabilized in a manner thatprevents pressure necrosis of the nasal tip and forehead. Endotracheal tube stabilization should allow the head to be moved from right to left without dislodgement of the tube. The eyes should be lubricated and sealed shut with individual adhesive dressings. If adequate opening exists to place arch bars, this can be completed at this point. Complete a thorough sterile preparation of the face and neck with Betadine solution, chlorhexidine, or both. Place a sterile towel under the head and seal the mouth and nose with a rectangular adhesive dressing. Isolate the surgical field with towels, a split sheet, and a head sheet (Fig 58).

STEP 2

Outline the preauricular incision within the skin crease closest to the tragus, making sure it is within 8 mm of the external auditory meatus. A question mark shaped extension (Al Kayat and Bramley modification) into the temporal region can be utilized for better visualization of the temporalis fascia and muscle. Outline a retromandibular incision centered on the angle of the mandible between the antegonial notch and tragus in a natural fold approximately 1 cm inferior and posterior to the mandibular ramus angle inferior border outline. Preemptive analgesia and hemostasis are established by providing a subcuticular injection of local anesthetic along both incision outlines and by an auriculotemporal nerve block along the avascular pretragal plane. If surgical navigation is planned, the fiduciary system is placed, and point-to-point indexing is completed. The availability of intraoperative navigation has significantly enhanced the accuracy of reconstruction cases. This technology is well suited to the complex nature of recurrent TMJ cases. It allows safety in all aspects of the surgery, with particular attention to the superior and medial aspects of the surgery, where the anatomy can be particularly altered in patients with recurrent ankylosis. (Fig 59).

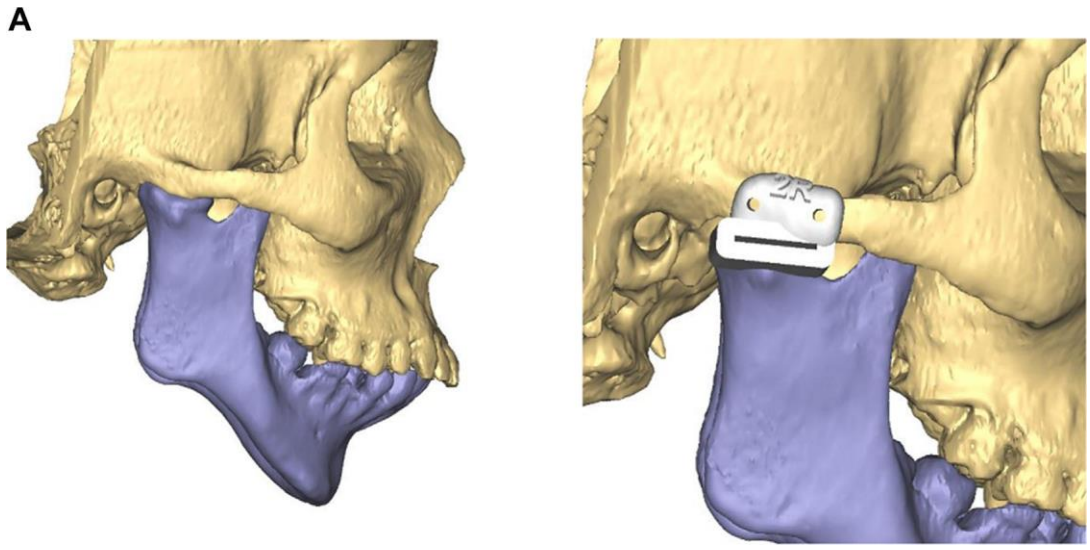

FIGURE 55. *A,* virtual Surgical Planning. (Fig 55 continued on next page.)

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STEP 3

The dissection is initiated in the pretragal fold with continuous, curvilinear skin incisions. The pretragal avascular plane is established with a mosquito hemostat. The plane of dissection is posterior to the superficial temporal artery and vein and the auriculotemporal nerve. The neurovascular structures are moved anteriorly during the dissection, using hemostats and Kittner dissectors, to avoid unnecessary neurovascular trauma. The superficial layer of the deep temporal fascia is identified, and an incision is made through it with the needle tip microdissector. The dissection is followed to the zygomatic arch, and the root of the zygoma is identified. A periosteal or Obwegeser elevator is used to expose the lateral aspect of the joint; great care is taken to remain subperiosteal to avoid trauma to the temporal branch of the facial nerve. The elevator should be lifted in a tenting motion as the tissue overlaying the zygomatic arch is dissected. If the joint capsule is present, insufflate with a local anesthetic, make an incision below the zygomatic arch, and open the capsule to inspect the ankylosis. At this point, the preauricular dissection is packed with moist gauze. (Fig 60A and B).

STEP 4

A layered retromandibular dissection is then completed with positive identification of the platysma, marginal mandibular branch of the facial nerve, and parotidomasseteric fascia. The pterygo-masseteric sling is then divided, and the mandible is exposed (Fig 60C)

The entire lateral surface of the mandible is exposed with careful protection of the retromandibular vein posteriorly. The entire posterior and anterior borders, condylar neck, sigmoid notch, and coronoid process should be visible. One toe-out retractor is placed on the anterior border at the base of the coronoid process, one into the sigmoid notch, and one above the coronoid process. If cutting guides were made during VSP, they should be applied at this time with 2-3 screws. The coronoidectomy is completed with a sagittal saw or Piezosurgery (Mectron, Carasco, GE, Italy) handpiece. The coronoid process is held with a Kelly clamp and dissected free with elevators and electrocautery. Of note, it can be helpful to ask anesthesia for brief paralysis during this time to decrease the pull of the temporalis. It is otherwise much more likely for the coronoid to be lost into the infratemporal space.

STEP 5

Once the ankylosis is well exposed, a caliper is used to score the superior line of resection based off the measurements from the CT scan in relation to an identifiable landmark, such as the superior border of the zygomatic arch. A second score line is marked approximately 1 cm inferior to the original line. Again, if VSP was used, the cutting guide can be placed and secured at this time. The initial resection is completed with a microsagittal saw, fissure bur, or Piezosurgery handpiece to a depth a few millimeters short of the dimension determined by CT measurements or to the depth determined by the cutting guides. Surgical navigation can also be used to guide the depth of resection. The resection is completed as necessary with judicious use of osteotomies. The surgeon should look for remnants of a joint space and follow that plane during final resection of bone from the fossa. The surgeon must be very careful during removal of bone from the posterior wall of the joint to avoid fracture of the bony external

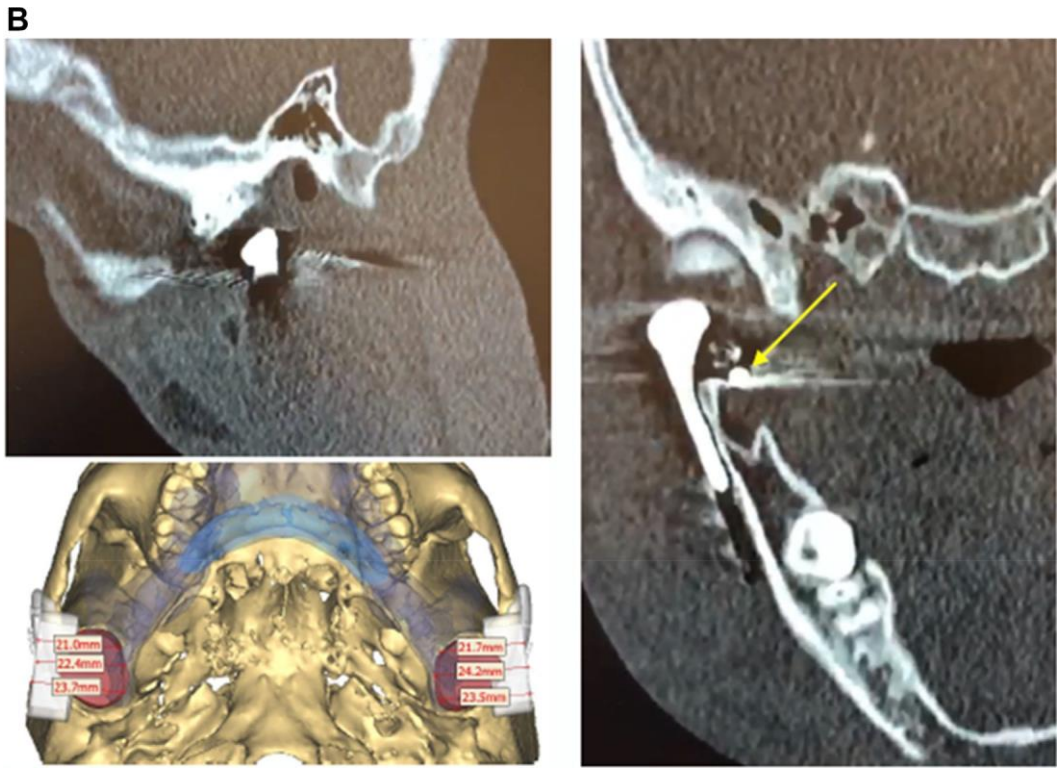

FIGURE 55 (cont'd). *B*, virtual Surgical Planning.

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auditory canal. The resected bone must be carefully dissected from soft tissue with elevators and electrocautery. If bleeding is encountered, hemostasis can be achieved with bipolar cautery, needle tip electrocautery, vascular clips, and oxidized cellulose. Once the resection is complete, it can be verified by inspection from the preauricular and retromandibular view and, if needed, by intraoperative CT (Fig 61)

STEP 6

Once the resection is complete, the reconstruction of choice is completed to maintain the gap. In adults without contraindications, the method of choice is a patient-specific joint replacement. The patient is placed into maxillomandibular fixation (MMF) prior to placement of the implant. The entire surgical team should change gloves and gowns and the

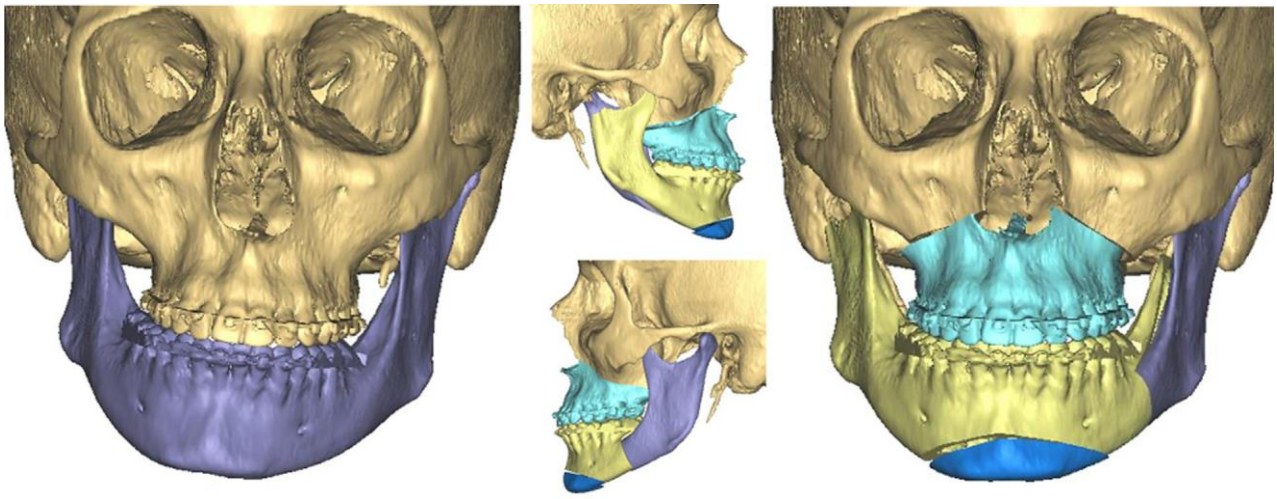

FIGURE 56. Asymmetry.

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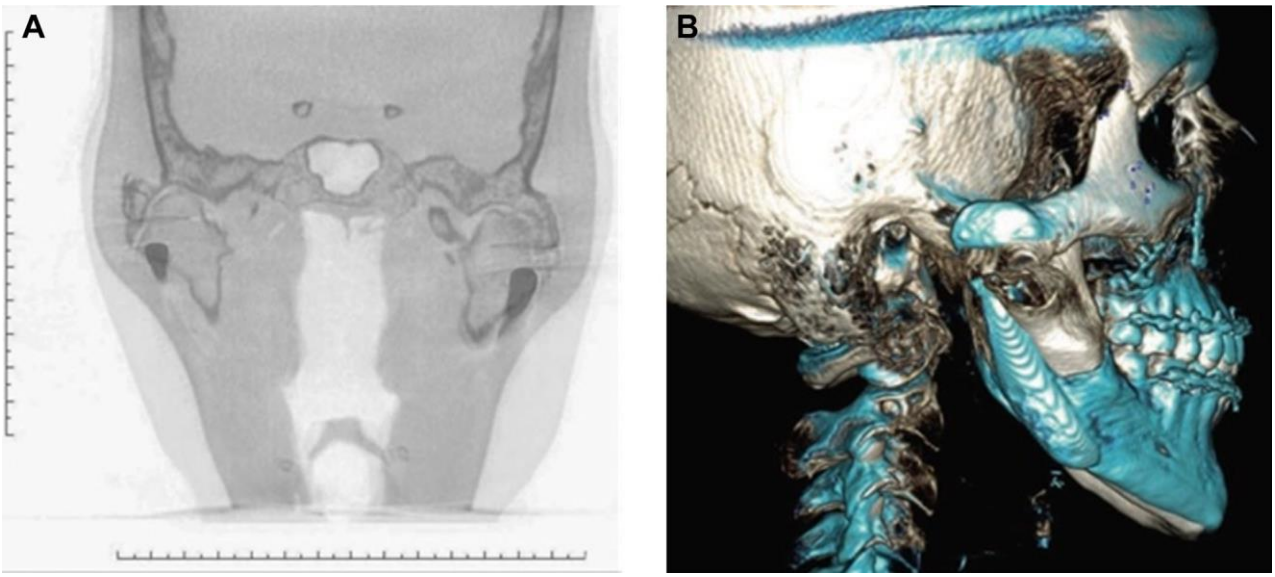

FIGURE 57. *A,* Computed tomography (CT). *B,* 3D CT image to correlate with navigation images. (Fig 57 continued on next page.)

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patient's oral cavity needs to be recovered with adhesive dressings prior to turning attention back to the surgical site. At this point, a standard approach is used to complete a total joint replacement. (Fig 62A-C). The patient is released from MMF and function of the autogenous or alloplastic joint is verified. Intraoral guiding elastics are placed. Adhesive dressings are then replaced over the oral cavity prior to skin closure.

STEP 7

A 2-3 cm inferior curvilinear periumbilical incision is made after subcutaneous injection of local anesthesia. Dissection is carried out with hemostats until adipose tissue is encountered. Approximately 2-4 cc of fat graft is harvested and placed in saline. The site is irrigated and closed with deep Vicryl (Ethicon Inc, J & J Company - J494 G) sutures and subcuticular or interrupted skin sutures. The fat graft is then placed around the condylar head of the TJR implant. It is important to tuck the graft into the medial and inferior space surrounding the implant to minimize the risk of ankylosis recurrence. (Fig 63A-C).

STEP 8

Perioperative pain control can be enhanced by placing an On-Q pain pump (Avanos, Alpharetta, GA, USA) to deliver Ropivacaine 0.2%. The On-Q set PM003 with a standard catheter delivers 2 cc/hour for 5 days. The total volume in the elastomeric pump is 270 cc. The catheter is introduced from inferior to the retromandibular incision on a subcuticular plane using the needle and plastic cannula contained in the set. It is advanced to the preauricular incision; the catheter is placed adjacent to the auriculotemporal nerve, not in the joint. It is secured with a Vicryl suture looped around the catheter. Where it exits the neck, the catheter is secured with Steri-Strips, and the full length of the tubing is covered with Opsite dressings. Closure is then carried out in a layered fashion, first closing the joint space with 3-0 Vicryl suture. Fascia and subcutaneous tissue are then reapproximated with 4-0 Vicryl suture followed by 5-0 nylon running suture to close the skin. The incisions are covered with Steri-Strips and an adhesive island dressing that is removed on the second post-operative day. (Fig 64A).

An alternative pain control modality is injection of 10 cc of Exparel (Pacira, Troy-Hills, NJ, USA) at each surgical incision site. This has been shown to decrease opioid requirements in other oral and maxillofacial procedures and helps to provide extended anesthesia post-surgery.[25] (Fig 64B)

Avoidance and Management of Intraoperative Complications

Correction of TMJ ankylosis is one of the most challenging procedures in oral and maxillofacial surgery. The medial aspect of the ankylosis is in close proximity to the middle meningeal artery, internal jugular vein, and internal carotid artery. VSP and the use of a Piezosurgery device can assist in avoidance of these structures. Access to the internal maxillary artery through the retromandibular incision is mandatory to control acute hemorrhage during resection. Temporary facial

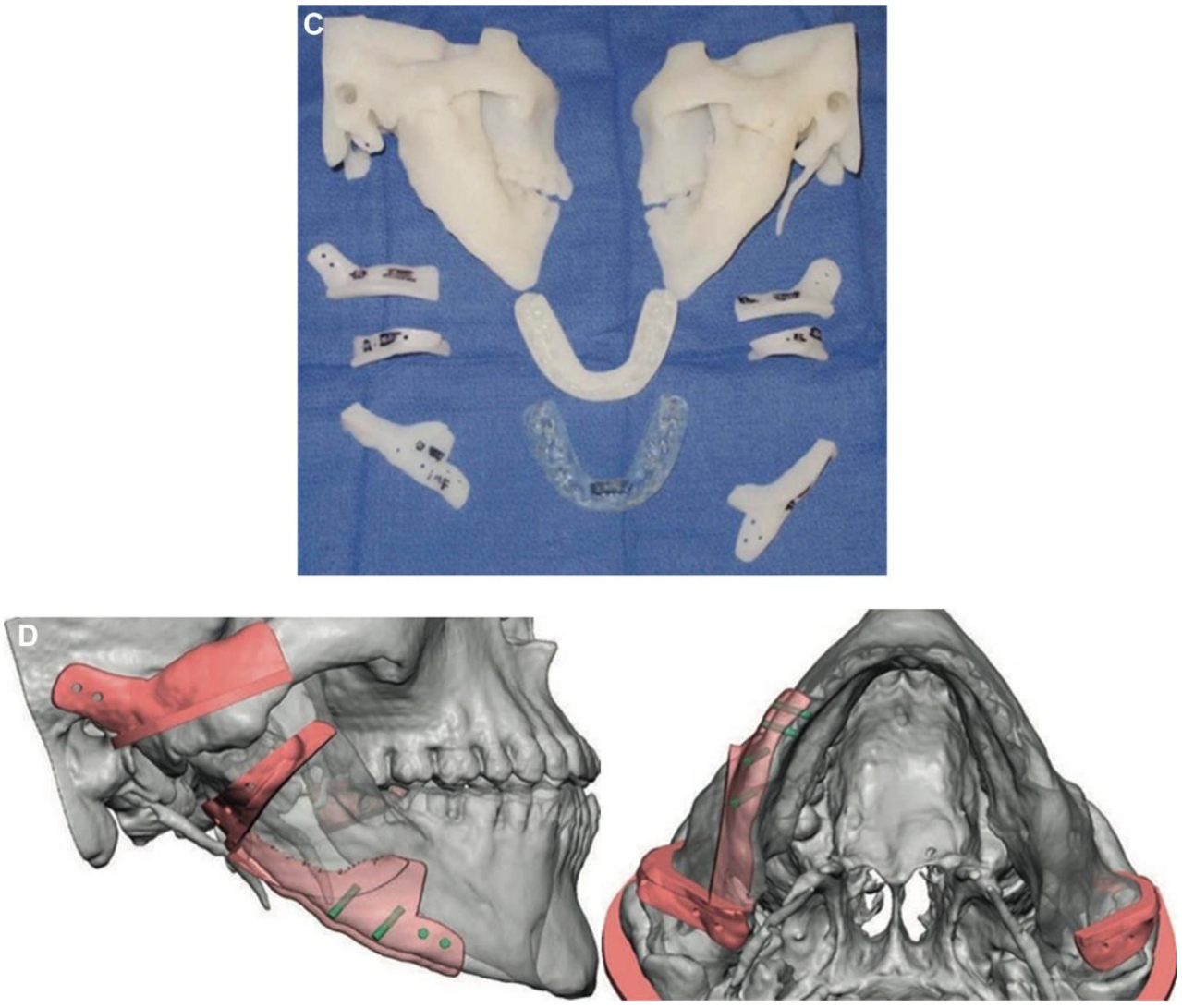

FIGURE 57 (cont'd). *C,* Model and surgical guides. *D,* Surgical guides overlaid on CT scan. (Fig 57 continued on next page.)

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nerve weakness is not uncommon after TJR. Facial mimetic exercises, as well as adequate lubrication and taping of the eye are imperative until the patient regains function. Parotid injury with sialocele or fistula formation are also possible, and these can be treated conservatively with pressure dressings, antisialagogues, and botulinum toxin. Patients may experience decreased hearing due to edema surrounding the external auditory canal. Perforation of the tympanic membrane can also occur and can be treated with antibiotic drops, placement of an occlusive ear wick and close follow up.

POSTOPERATIVE CONSIDERATIONS

Rehabilitation is the most significant concern in the postoperative period. Physical therapy should continue after the maximal incisal opening (MIO) is established to prevent relapse.[26] The greatest advantage of alloplastic material over autogenous grafts is the ability to start physical therapy immediately following surgery. Returning the jaw to normal function reduces scar tissue formation, allowing for optimal range of motion.[27] An oral orthotic appliance is necessary for patients with parafunctional habits to reduce wear and increase the functional life of the prosthesis.

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Temporomandibular Joint Dislocation

Temporomandibular joint dislocation most often present as an acute process that can be managed

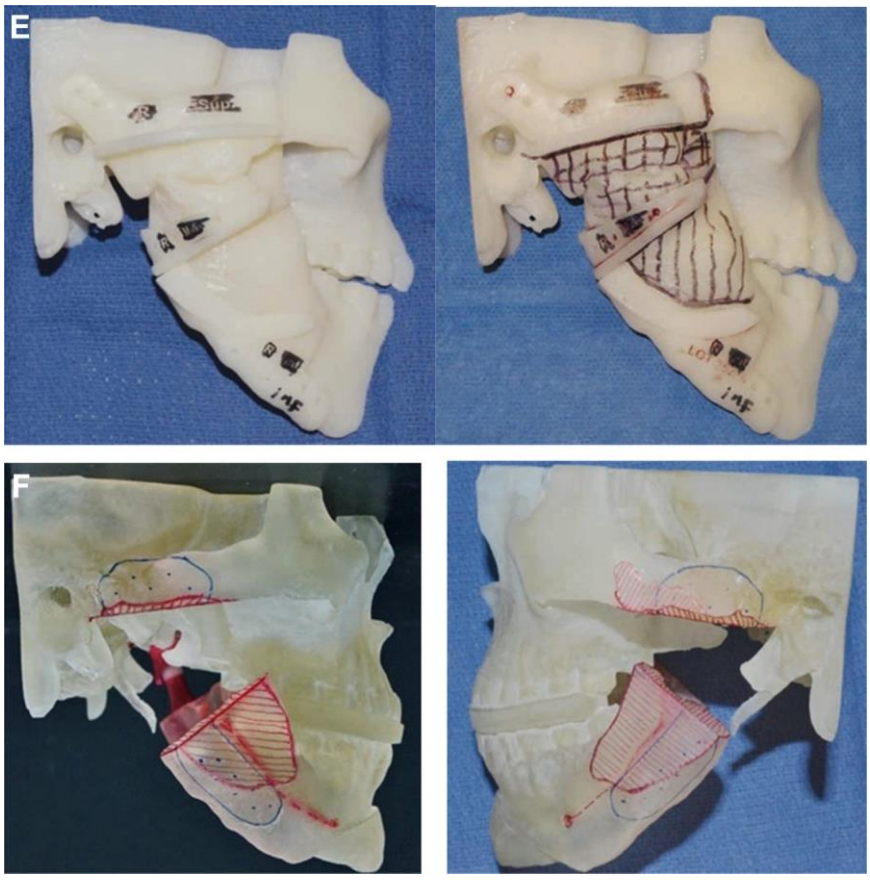

FIGURE 57 (cont'd). *E*, Ankylosis and planned Resection. *F*, Resected ankylosis, Recontoured ramus/fossa, repositioned mandible and prosthesis design.

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with closed reduction (CR). Closed reduction often requires the use of sedation to facilitate the reduction. Dislocation can also be recurrent or chronic which typically requires additional treatment including surgical intervention. Temporomandibular joint dislocation involves the displacement of the condyle, outside of its functional position within the glenoid fossa.[232] Although the most common condylar dislocation is

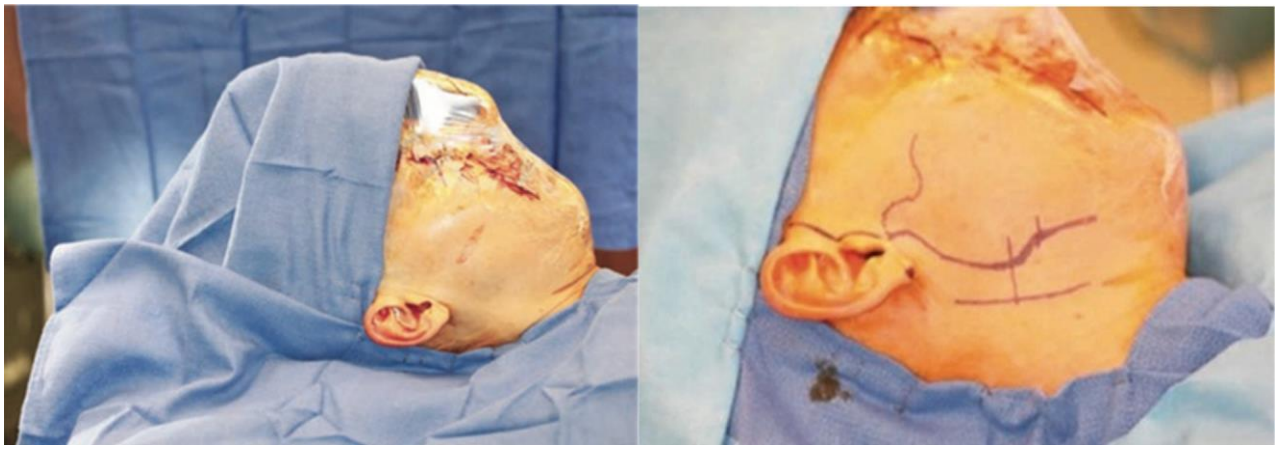

FIGURE 58. Patient Prep and draping.

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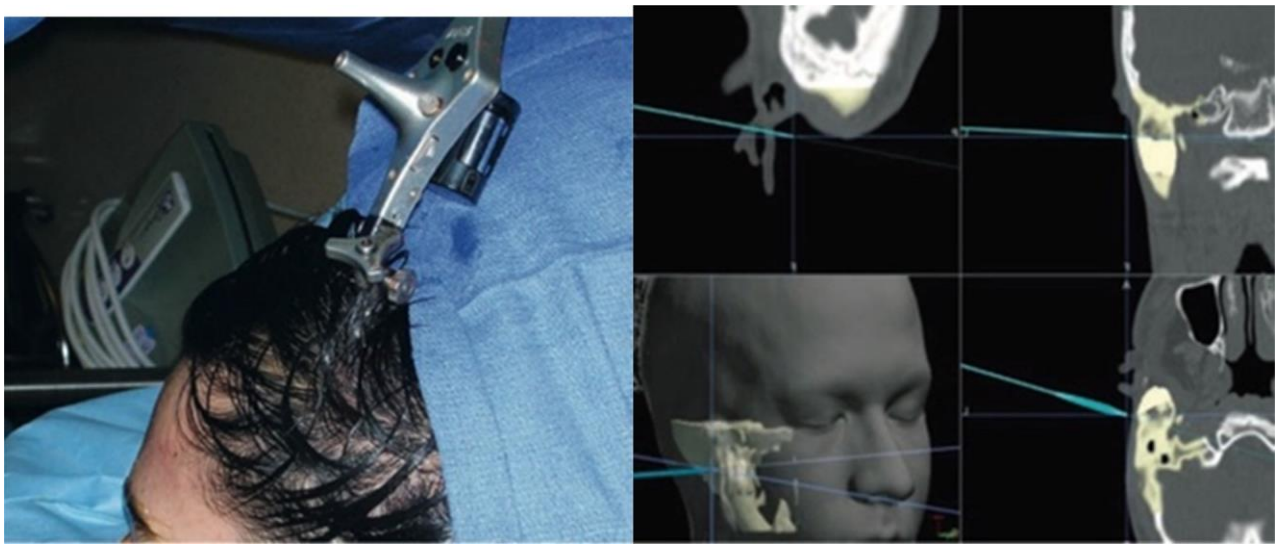

FIGURE 59. Intra-operative navigation.

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anterior to the articular eminence, dislocation can also be medial, lateral, posterior, and intracranial.[233-236] The classification of dislocation and the various treatment options have been reported.[237]

Classification

Despite a variety of classification schemes, the most common divides dislocation into three categories: acute, chronic and chronic recurrent. (Fig 65).

Acute dislocation may be associated with any number of etiologies, including prolonged mouth opening during a lengthy dental procedure, vomiting, yawning, and singing. There are also reports of acute dislocation secondary to epileptic seizures, acute facial trauma, and direct laryngoscopy. Frequent dislocation may also be seen in patients with connective tissue disease, such as Ehlers-Danlos syndrome or muscular dystonias. (Table 9). Acute dislocations are typically isolated events, which, when managed appropriately, usually have no long-term sequelae.

Chronic dislocation results from an acute dislocation that is not treated. This results in intraarticular fibrosis and masticatory muscle shortening. It is very difficult to perform CR in these cases which often require open reduction of the dislocation. Chronic recurrent dislocation refers to recurrent acute dislocations which occur regularly.

Closed Reduction

Acute dislocation is typically addressed in a non-surgical fashion. Conventional non-operative methods have been described by multiple authors.[238,239] The typical maneuver is described as bimanual intraoral traction, placing the thumbs in the retro-molar pad/external oblique ridge and pressing inferiorly and then posteriorly, manipulating the condylar head over the articular eminence, seating it back in the glenoid fossa. This maneuver is typically done asking the patient to open the mouth so that the elevators of the mandible are relaxed. In some instances, this may be accomplished utilizing local anesthesia for auriculotemporal nerve blocks. Litler has advocated for intra-articular anesthetic distribution into the empty glenoid fossa(e), aimed at minimizing myospasm prior to digital manipulation. In some cases, the acute myospasm of the temporalis, masseter and pterygoid muscles requires the use of intravenous sedation or general anesthesia.

Other less known techniques for CR include that described by Ardehali et al. This involves an external component wherein one hand creates anterior traction at the mandibular angle withfingers along the ascending ramus using the thumb at the malar eminence as a fulcrum while the other thumb places posterior directed pressure antero-superior to the displaced coronoid process, with fingers creating traction at the mastoid process. This technique is said to minimize bite risk to the practitioner.[240] Awang et al also described a technique in which a mouth mirror is used to stimulate a gag by soft palate/pharyngeal contact. Somatosensory impulses to the CNS result in stimulation of muscles that depress and protrude the mandible, reflex inhibition of the associated antagonists (elevators) and enable the mandibular condyle to relocate into the glenoid fossa.

The potential for additional dislocation can be managed in the short term with the use of a chin strap/face-lift bandage for vertical traction or intermaxillary fixation for 7 days.[241]

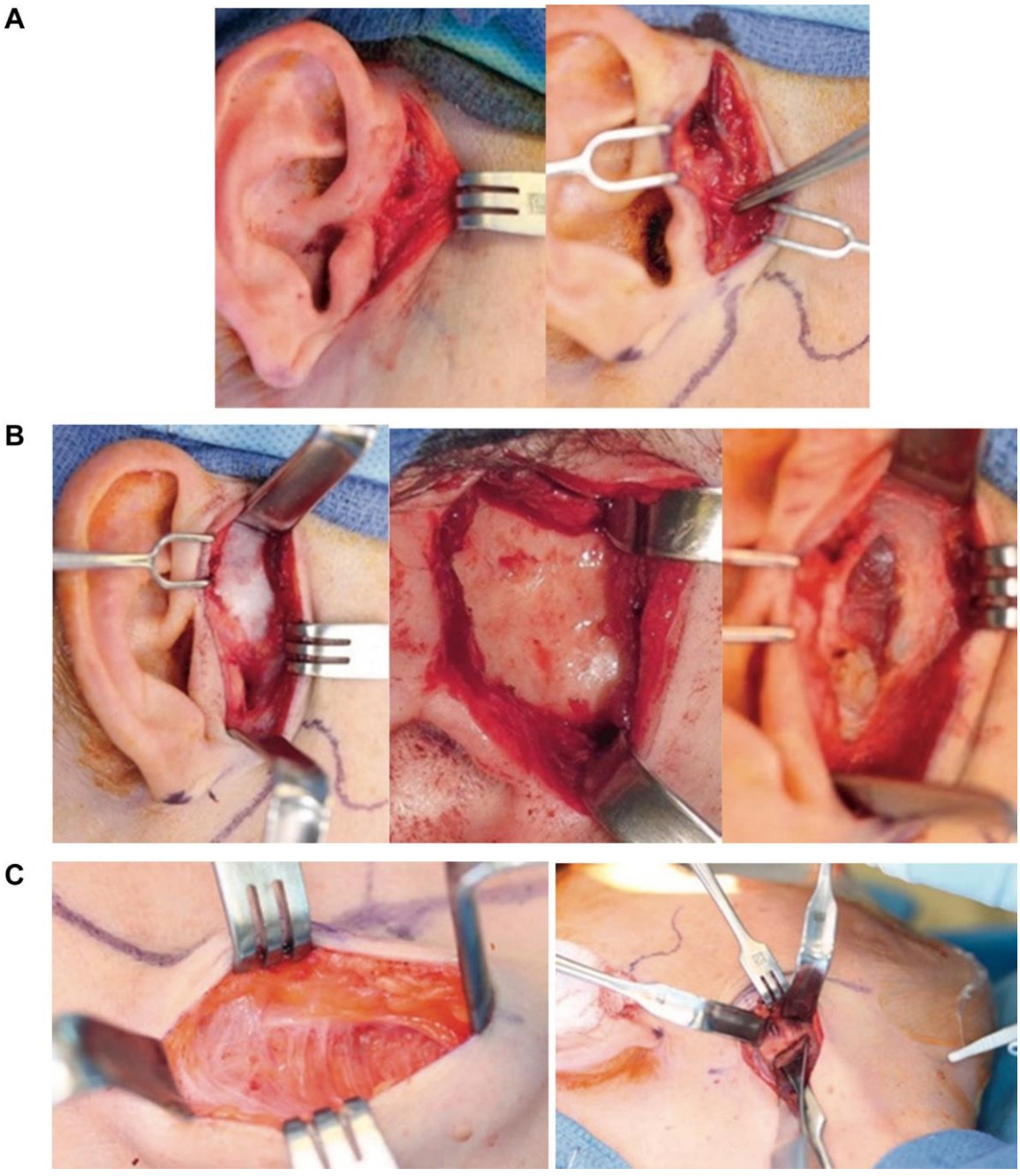

FIGURE 60. *A*, Preauricular approach. *B*, Dissection planes. *C*, Retromandibular approach.

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PROCEDURES TO PREVENT RECURRENT DISLOCATION

Autologous Blood Injection/Sclerotherapy

Autologous blood injection (ABI) was initially described by Brachmann in 1964, where he reported successful management of 60 patients with recurrent dislocation. Since that article, scattered case reports have intermittently appeared in the literature.[242-244] Intra-articular blood injections are aimed at initiating an intra and peri-capsular inflammatory response. This inflammation creates fibrosis and adhesions, as is seen in the post-traumatic hemarthrosis model. Fibrosis and cicatricial maturation cause limitation in joint translation and a decrease in range of motion. ABI has been described using a variety of techniques; however, the global methodology and endpoints are similar. ABI may be completed with local anesthesia alone, intravenous sedation, or general anesthesia.

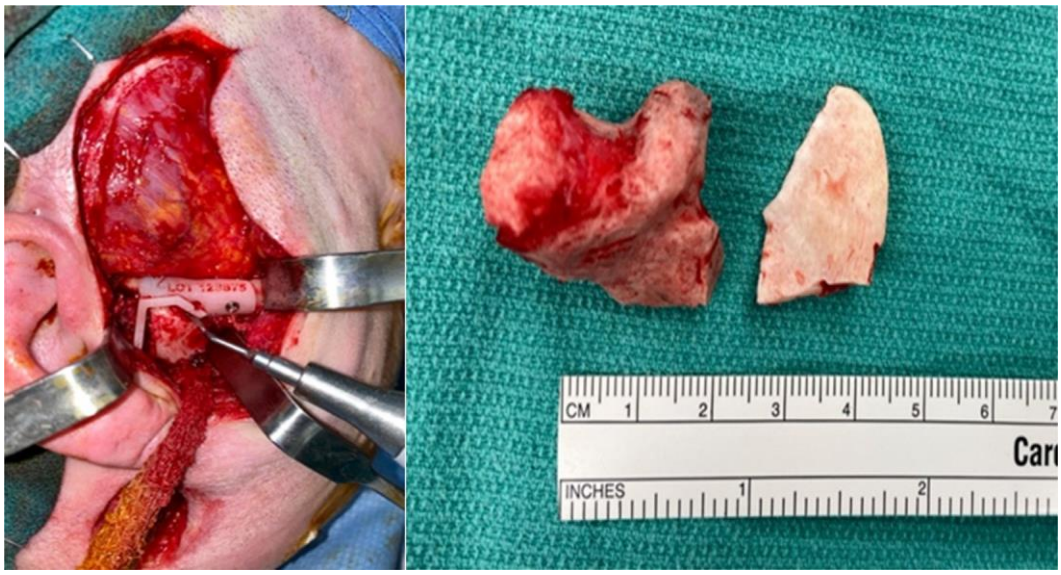

FIGURE 61. Resected ankylosis.

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Two 20-gauge needles are placed into the superior joint space in a manner identical to arthrocentesis. The space is gently lavaged with normal saline or lactated ringers. The outflow needle is removed, and whole blood is then drawn from the patient and deposited into the superior joint space and infiltrated into the pericapsular tissue. The remaining needle is removed, and patients are given specific instructions to minimize jaw function for 2 weeks post operatively.[242,245] At 2 weeks, patients can resume restricted function. Depending on clinical outcome, repeat injections may be needed. The success of ABI varies from 60 to 80% with the two factors that seem to influence outcomes positively being repeat ABI and the use of temporary maxillomandibular fixation.[246]

The intraarticular deposition of sclerosing agents also induce a robust inflammatory response, which is followed by secondary localized fibrosis, resulting in joint hypomobility. A variety of sclerosing agents have been described, including cyclophosphamide, alcohol, ethanolamine oleate, sodium tetradecyl sulfate, sodium morrhuate and OK-432(Picibanil).[247] Arthroscopy can be used to facilitate ABI and sclerotherapy. The latter can be achieved under direct vision and can be augmented with the use of laser and coblation. Outcome data following the use of sclerosing agents is scant making any recommendations for the use of these agents unlikely.

Prolotherapy

Prolotherapy has been used since the 1930s in the management of temporomandibular dysfunction.[248] Prolotherapy, or regenerative injection therapy, is described as the infiltration of a non-pharmacologic solution into pericapsular, tendinous tissues, with the aim of initiating a mixed inflammatory response.[248,249] This inflammatory process is thought to initiate the localized proliferation of fibrous connective tissue that limits condylar translation and the potential for subluxation or dislocation. Historically, a variety of solutions have been used, including dextrose, psyllium seed oil, and a variety of combinations of dextrose, glycerin, and phenol.[250]

The technique for prolotherapy is not unlike that described previously for autologous blood injection. Local anesthesia is administered followed by the injection of 2 ml of 50% dextrose into the superior joint space (as described for arthrocentesis), retrodiscal tissues, and periarticular tissues. Upon completion of the procedure, patients are put on a soft diet and jaw rest for 2 weeks. Patients are then followed, with repeat injections as needed in the event of recurrent dislocation. When using prolotherapy, the literature would suggest that although patients may only require a single series of injections, some patients require 3-5 injections for optimal therapeutic effect. Prolotherapy with the use of 50% dextrose injection into joint space and periarticular tissue is relatively simple to perform and has been reported to result in a 90% success rate in preventing recurrent dislocation, although a second and third injection may be required in 25% of patients.

Botulinum Toxin

Therapeutic use of Botox-A (BTX-A) in the head and neck region has significantly increased over the past decade. BTX-A induces a dose-related weakness of skeletal muscle by inhibition of acetylcholine release

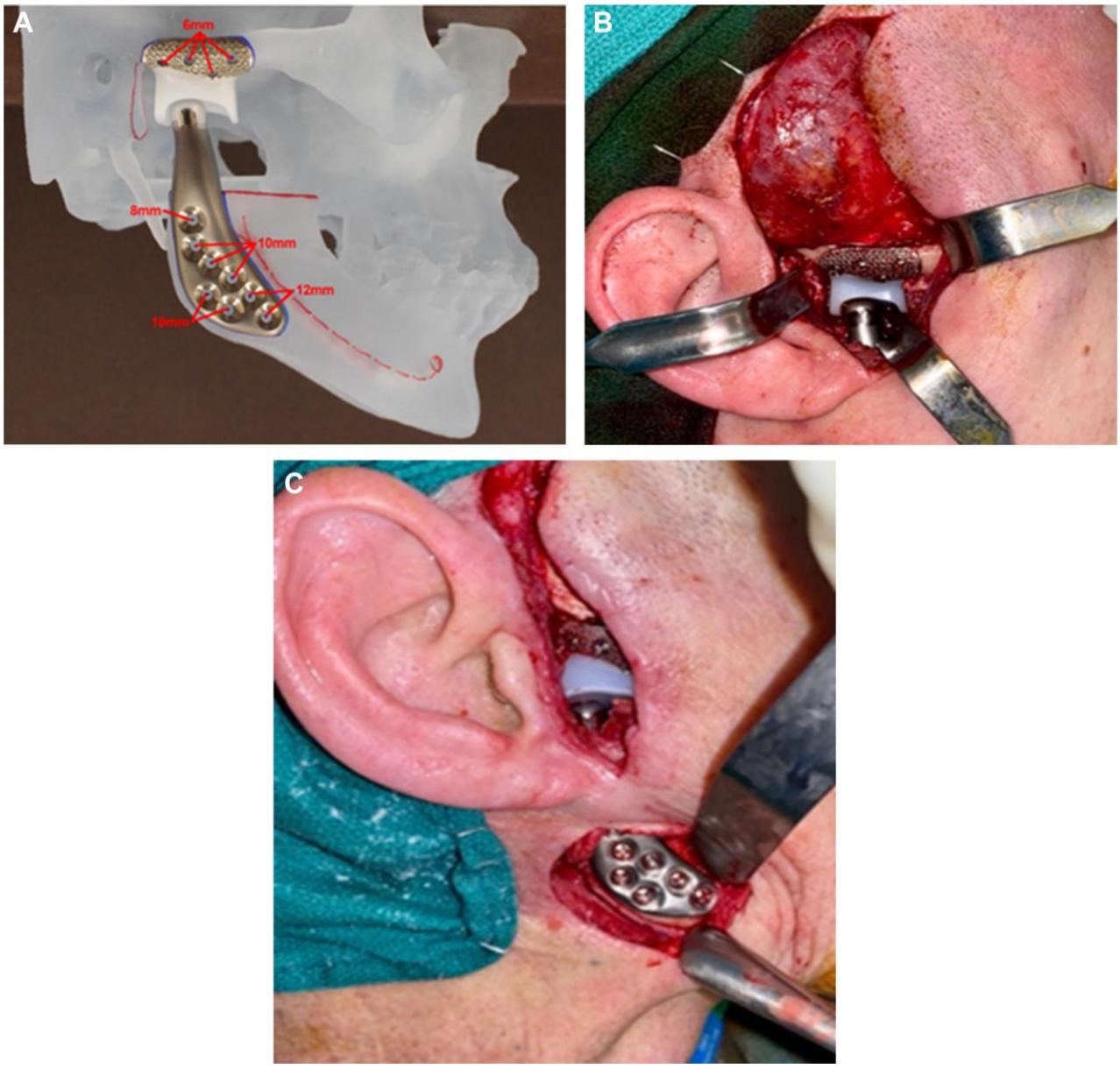

FIGURE 62. *A*, Position of Total Joint Replacement. *B*, Insertion of fossa. *C*, Insertion of ramal component.

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at the neuromuscular junction. When used in the treatment of dystonia or other neurogenic disorders, repeat injections at 3–6-month intervals are required for maximal therapeutic efficacy.[251] In the context of chronic recurrent TMJ dislocation, there are many reports of the use of BTX-A, both as a primary therapy, and as an adjunct to other reductive techniques.[252-254] It is often employed in patients that may not be candidates for surgery, based on age, medical comorbidities, etc.

The targeted muscle is the lateral pterygoid, which is often implicated in myospasm associated with dislocation. Ideally both the superior and inferior heads of the lateral pterygoid muscle must be targeted. Fu et al described accessing the lateral pterygoid percutaneously through the sigmoid notch, inferior to the zygomatic arch. This provides easy access to the superior head. Twenty-five units of Botox is deposited directly into the muscle belly, aspirating prior to injection to avoid inadvertent intravascular injection.[24] Access to the inferior head is easily achieved transorally behind the maxillary tuberosity. Electromyographic guidance (EMG) is critical to ensuring the needle/electrode is correctly positioned within

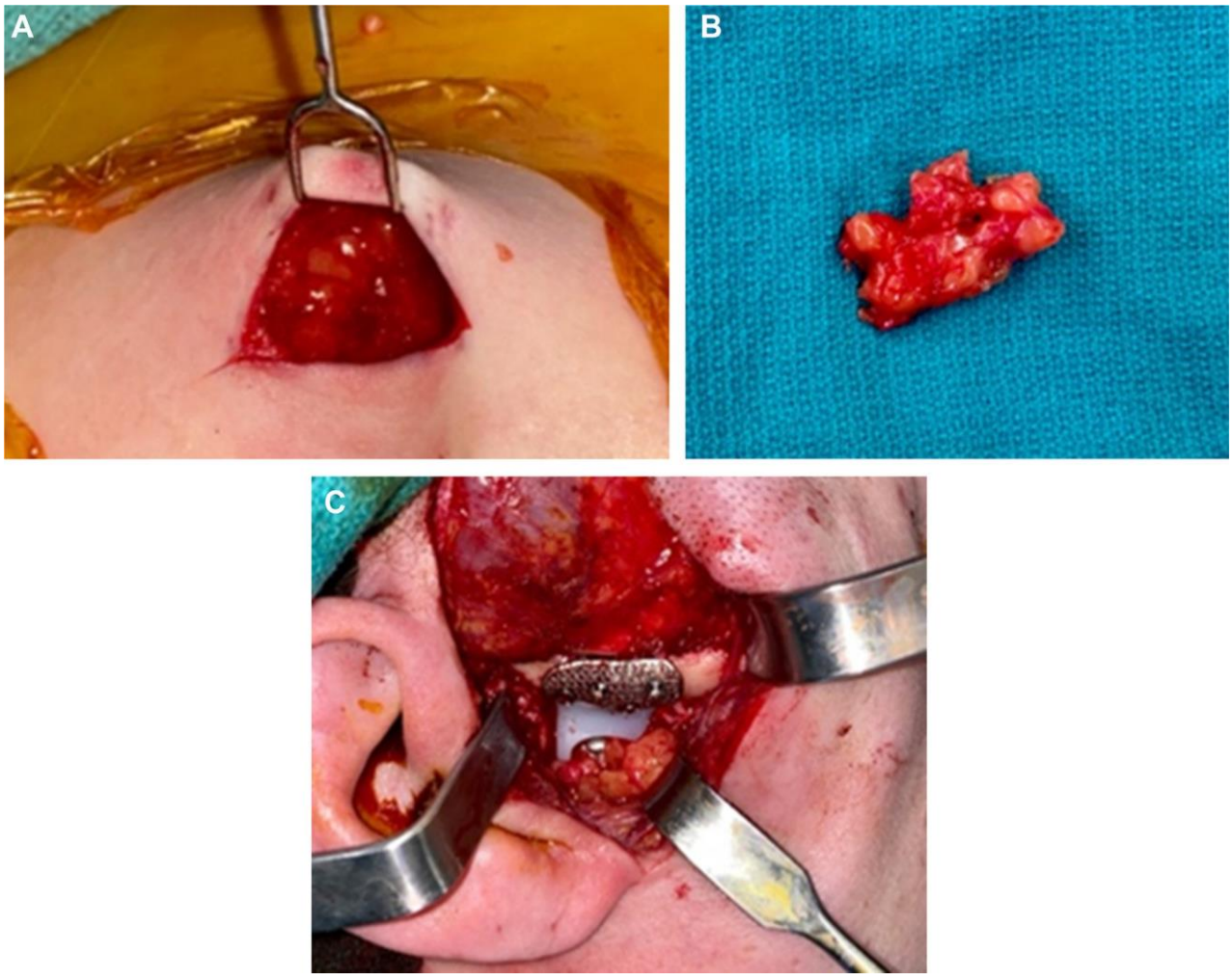

FIGURE 63. *A*, Fat graft harvest. *B*, Fat graft. *C*, Fat packed around TJR.

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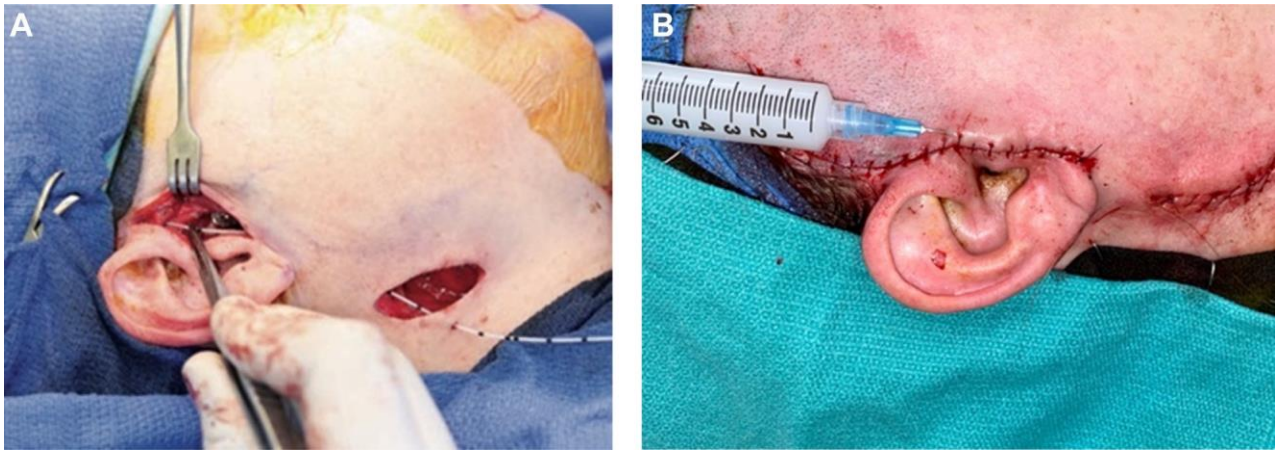

FIGURE 64. *A*, On-Q pain pump. *B*, Liposomal bupivacaine.

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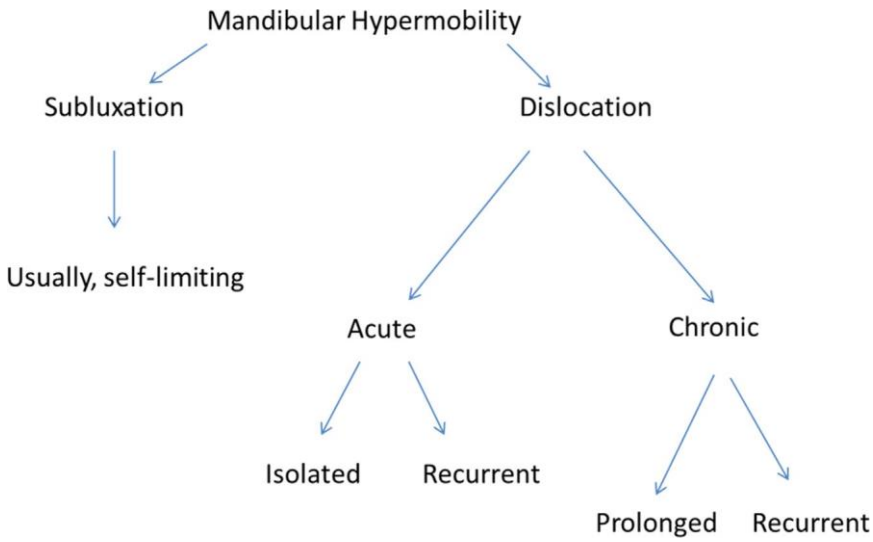

FIGURE 65. TMJ dislocation.

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the muscle. In many instances, a single injection may be sufficient. Adverse effects of injection include hemorrhage and intravascular injection. Additionally, there is risk of toxin induced transient velopharyngeal insufficiency, dysarthria, and dysphagia.[255] Fortunately, in the unlikely event that one of the above ensues, symptoms typically subside between 2-3 months. Data supporting the use of Botox for dislocation is limited. (See chapter on Nonsurgical Management, Botox)

Lateral Pterygoid Myotomy

In the context of recalcitrant chronic recurrent or chronic dislocation of the TMJ, surgery is often indication. Alteration of the juxta-articular musculature has been described as one of many surgical options, aimed specifically at alteration the implicated spastic muscle units. Ultimately, it is likely the formation of intramuscular scar tissue also facilitates hypomobility of the mandible.

Lateral pterygoid myotomy has been described as being performed transorally and via a preauricular incision.[256] The transoral approach is more challenging given the location of the lateral pterygoid insertion into the mandibular condylar fovea and disc. Patients require MMF for 7 days. Data to support lateral pterygoid myotomy is generally weak and there is no data on outcomes.

Table 9. CAUSES OF TEMPOROMANDIBULAR JOINT DISLOCATION

Iatrogenic	Intubation, laryngoscopy, dental or ENT procedure, endoscopy
Spontaneous	laughter, vomiting, yawning, singing
Trauma	Sports, interpersonal violence, motor vehicle accident
Systemic Disease	Connective tissue disease (eg, Ehlers-Danlos), neurodegenerative disease (eg, Huntington's, epilepsy, Parkinson's, multiple sclerosis, oromandibular dystonia, dyskinesia
Pharmacological	Phenothiazines, antipsychotic drugs, antiemetic drugs
Anatomic	Articular eminence, condyle deformities

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Open Surgical Treatment

EMINECTOMY

Initially described by Myrhaug in 1951, eminectomy is completed with aim of reducing the vertical height of the articular eminence, such that in the event of condylar hypermobility and dislocation, the condyle will slip posteriorly back into the fossa without significant anatomic restriction.[257]

The procedure is typically completed using a standard endaural or preauricular incision, with anterior/temporal extension. Dissection is carried to the superficial layer of the deep temporal fascia. This fascia is incised, with anterior release as needed (extending obliquely anterosuperiorly at a 45-degree angle so as to minimize trauma to the temporal branch of the facial nerve).[258] At this point, the periosteum is incised on the zygomatic arch, and dissection is carried anteriorly, to the level of the articular eminence. The

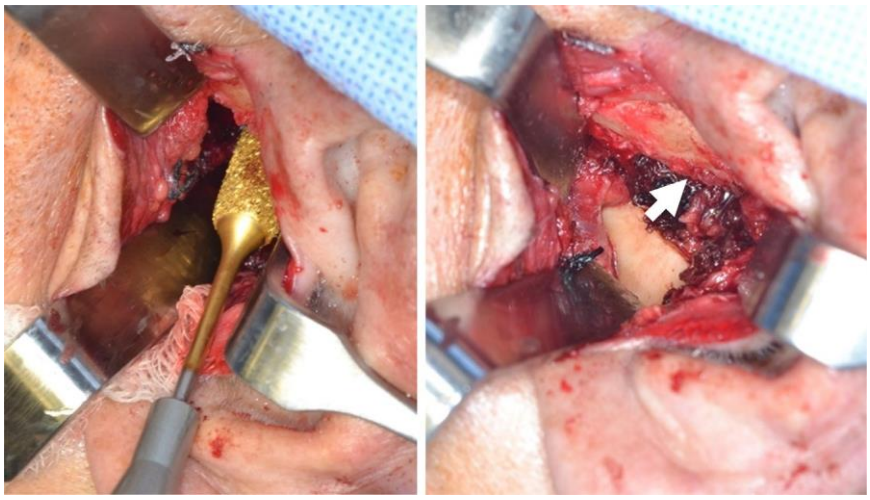

FIGURE 66. Eminectomy.

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eminence is then reduced to its medial margin with burs, osteotomes, piezo device or a reciprocating rasp. Patients are encouraged to comply with a soft diet over the first week following surgery. Eminectomy can also be performed arthroscopically using rotary instruments. Success following eminectomy has been reported to be above 90% although sample sizes were small. Furthermore, long-term data on the potential development of osteoarthritis is lacking. (Fig 66).

DAUTREY PROCEDURE

Techniques aimed at creating a mechanical interference to the condylar translation have also been described extensively in the literature. Segmental dislocation of the zygomatic arch to act as a physiologic obstruction to condylar hypermobility. The technique was later modified wherein a vertical osteotomy was created in the zygomatic arch and inserting a segment to impede the path of the hypermobile condyle.[259] Further modification by Gosserez and Dautrey described a greenstick fracture of the zygomatic arch, with displacement in inferior and medial to the articular eminence.[260]

The technique involves a preauricular incision to expose the joint capsule which is not violated. An osteotomy is then created in the zygomatic arch, anterior to the eminence, extending from posterior-superior to anterior inferior, in an oblique fashion. Gentle pressure is exerted proximally on the osteotomized arch, to create a greenstick fracture, anteriorly. The osteotomized proximal arch is then mobilized laterally and inferiorly to allow inset under the articular eminence. Once inset, the segment may be held in place by a mini plate, or simply left passively.[261-264] Once the procedure has been completed, patients are placed on a soft diet, with restriction of function over 2-3 weeks.

The immediate challenge with this technique, particularly in older individuals is fracture of the arch anteriorly (rather than green stick) which results in excessive mobility of the arch necessitation plate fixation. The efficacy of this procedure in preventing dislocation has been reported to be greater than 90%. (Fig 67).

OTHER BLOCKING PROCEDURES

Other blocking procedures involve augmentation of the articular eminence with interpositional grafts or miniplates.[265] Glenotemporal osteotomy with autogenous grafting can be completed using a variety of different graft donor sites, however, the most frequently described techniques typically use iliac crest or cranium.[266-268] In this technique, the joint is accessed as previously described, with subperiosteal dissection to the articular eminence. A sagittal saw or fissure bur is used to create a horizontal osteotomy along the eminence, which is subsequently down fractured, maintaining intact periosteum. Once the eminence has been down fractured, the harvested bone is then shaped and inset as an interpositional graft between the zygomatic arch and down fractured eminence.[269] (Fig 68). Depending on the stability of the inset graft, wires, screws, or mini plates may be used to secure the graft.

Mini plates have also been described in the context of physiologic blocking procedures. The approach to the TMJ is completed as previously described. Once the articular eminence has been visualized, an L plate is placed, with the short arm fixed laterally to the eminence with two screws and the long arm being

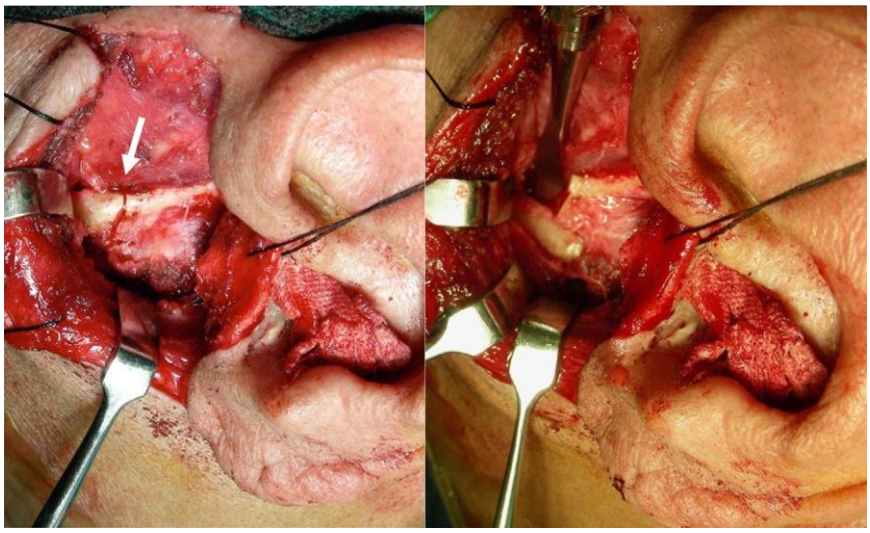

FIGURE 67. Dautrey procedure.

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contoured and placed along the eminence, inferiorly, to act as a mechanical obstruction.[270] Careful attention is given so as to remain extracapsular during the procedure. Proponents of miniplate placement advocate the procedure based on its reversibility and relatively less invasive nature. That being said, there is risk for plate fracture, in addition to a larger reduction in maximum interincisal opening.[271,272] In the event of plate fracture, a second surgery must be undertaken, to remove the hardware, and a decision must be made as to whether additional treatment might be required.

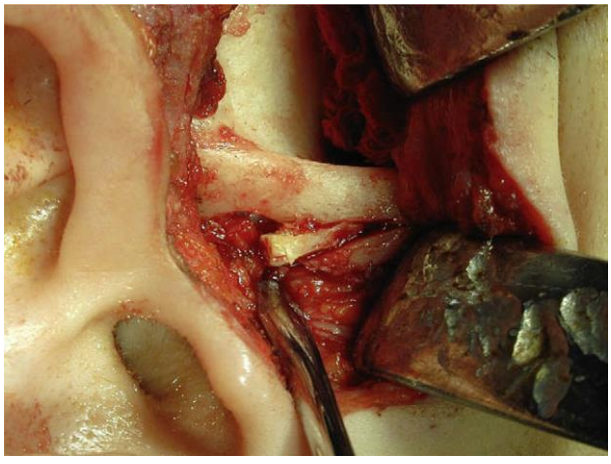

FIGURE 68. Eminence augmentation.

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WOLFORD PROCEDURE

This relatively simple procedure uses 2 Mitek (Mitek Products Inc., Westwood, MA, USA) mini bone anchors with osseo-integration potential. An endaural or preauricular incision is used for access to the TMJ and dissection completed exposing the zygomatic arch and lateral capsule. The lateral pole of the condyle is exposed, and one anchor is placed in the lateral pole of the condyle. The other anchor is placed in the very posterior root of the zygomatic arch. The 2 sutures are then adjusted for the amount of mobility desired and tied. (Fig 69). If the procedure is used to prevent dislocation anterior to the articular eminence, then the artificial ligaments can be left slack to provide translation but limit forward movement so the condyle cannot translate beyond the articular eminence. This will allow relatively normal movement of the condyle including excursive movements. Potential risks with these techniques include rupture or breakage of the sutures or failure of the Mitek anchors. Wolford et al reported good success in 5 cases treated with this technique.[273]

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Chapter 10

COMPLICATIONS

Complications following any surgical procedure are concerning for both the patient and surgeon. They are inevitable despite attention to detail and correct

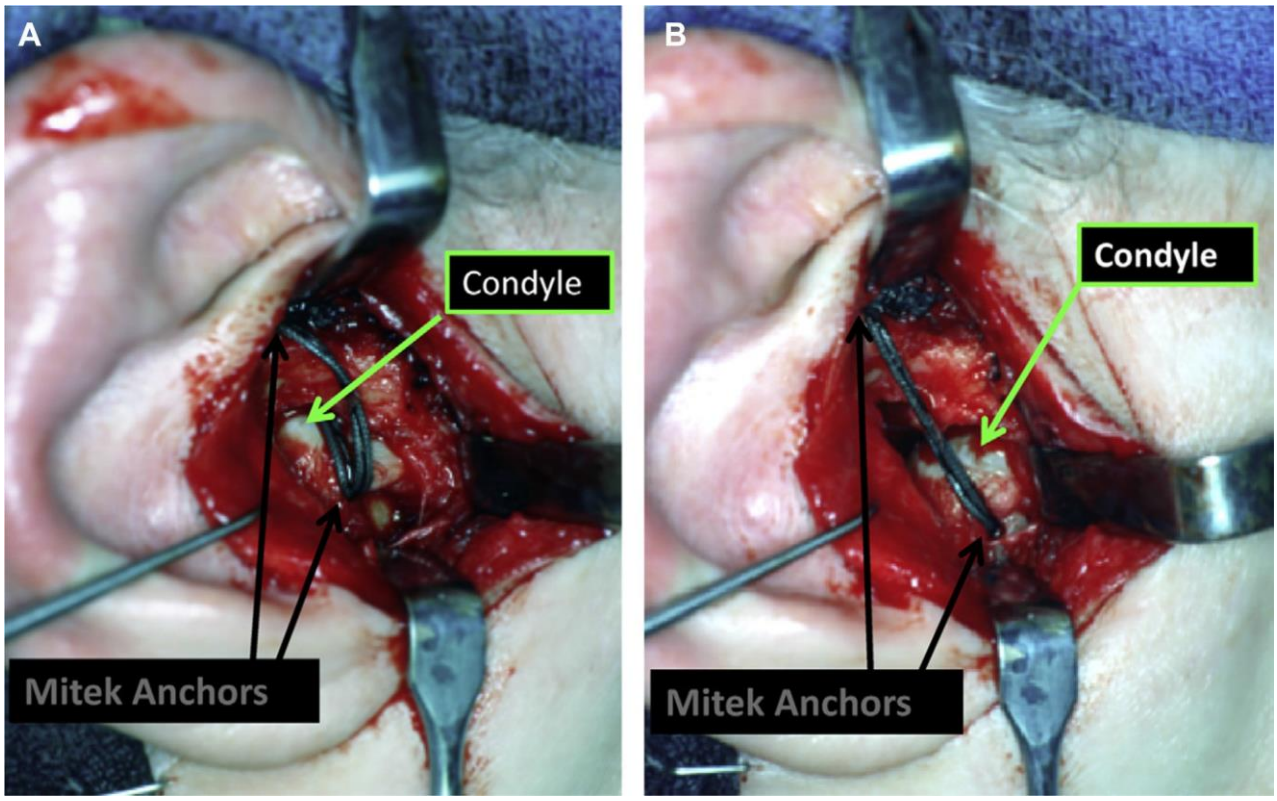

FIGURE 69. Mitek bone anchor.

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surgical technique. The goal is to prevent complications whenever possible and identify and manage them when they occur.[274]

FAILURE TO MAKE THE CORRECT DIAGNOSIS

The causes of facial pain and headaches are many. It behooves the oral and maxillofacial surgeon to consider, and be familiar with all potential causes, even those that lie outside of the practice of oral and maxillofacial surgery. One must be familiar with the varying causes of headaches including migraine, tension-type headache, chronic daily headache, trigeminal neuralgia, persistent idiopathic facial pain, cluster headache, trigeminal autonomic cephalgia, giant cell arteritis, cervicogenic headache as well as temporomandibular disorder headache. These conditions may be the cause of referred facial, jaw or temporomandibular joint pain. Failure to recognize referred pain may lead to unnecessary TMJ surgery with poor outcomes.

Although arthrogenous and myogenous sources of facial pain are relatively easy to diagnose using the DC/TMD criteria, one of the most common sources of referred pain to the face and jaws is cervical pain. It is imperative that during the history taking appropriate questions are asked to help identify cervical pain. Questions that should be considered include.

- Do you have any neck or shoulder pain?
- Does the pain radiate down the upper extremity (UE)?
- Do you have any arm weakness or tingling?

The physical examination should include UE strength; active neck flexion, extension and rotation as well as the Spurling test to identify any foraminal nerve compression. All cervical pain should be managed prior to any surgical procedure. This typically requires referral to physical therapy, physiatry or rarely orthopedic/neurosurgery. Patients should be reassessed after management of their cervical pain to ensure that any persistent facial, jaw or TMJ pain will benefit from surgery.

PERFORATION OF THE EXTERNAL AUDITORY CANAL (EAC) OR MIDDLE CRANIAL FOSSA (MCF)

Preventing perforation of the EAC is best achieved with careful attention to anatomy. The outer third of the EAC is cartilaginous while the inner two-thirds is bony. The EAC is at risk during open surgery and

care should be exercised to ensure that dissection follows the tragal cartilage to the post-glenoid tubercle. It also is important to appreciate the forward angulation of the EAC cartilage to avoid inadvertent perforation. It is also possible to perforate the EAC during arthrocentesis or arthroscopy. This can be avoided by carefully palpating the lateral lip of the glenoid fossa as well as the articular eminence. Arthroscopic trocars should be angled slightly anteriorly and superiorly, and the lateral lip of the glenoid fossa sounded before puncturing the TMJ capsule. A similar angle for needles is required although the lip is not typically sounded. Trocars and instruments should not be advanced more than 25 mm until the correct position is confirmed visually. This will prevent perforation of the tympanic membrane (TM) even if the EAC was perforated. Isolated perforation of the EAC can be easily managed with a topical antibiotic solution ± a steroid. The EAC can be packed with xeroform gauze or an ear wick for 3 – 5 days if needed. Puncture of the TM is more serious and can result in otitis media and disruption of the ossicles. Conductive hearing loss may occur particularly with involvement of the ossicles. Immediate consultation with otolaryngology is required as soon as the complication is recognized. Patients should undergo audiometry testing to quantify the hearing loss and recovery. Many small perforations of the TM will spontaneously heal. Others will need tympanoplasty.

The middle cranial fossa lies immediately above the glenoid fossa. The roof of the glenoid fossa is very thin with a mean thickness less than 1 mm. Perforation of the MCF can occur as a result of many pathological conditions of the TMJ including osteoarthritis, synovial chondromatosis, villonodular synovitis, benign tumors and trauma. It can also occur spontaneously for reasons that are unclear. MCF perforation due to pathology is usually evident pre-operatively on CT and MRI imaging. Perforation can also be iatrogenic as a result of arthrocentesis, arthroscopy and open joint procedures. The use of appropriate and gentle surgical techniques will significantly mitigate the risk. Sounding and/or visualizing normal anatomical landmarks will further reduce the risk. Ankylosis release involves abnormal anatomy and careful pre-operative planning to ensure all cuts are at least 5 mm from the MCF is recommended. Intraoperative CT and navigation can further reduce the risk. Excessive irrigating pressure during arthrocentesis or arthroscopy can also result in a MCF perforation or extravasation of irrigant into the MCF. MCF can result in epidural hemorrhage and mass effect on the temporal lobe and cerebral hemisphere with disastrous consequences. The position of the middle meningeal artery is not only medial to the TMJ but its path along the MCF places it at risk from and MCF perforation. Small bony perforations of the MCF without a Cerebrospinal Fluid (CSF) leak require no treatment. Larger bony perforation and those with an associated CSF leak require treatment. Large perforations are concerning for the potential for migration of the mandibular condyle into the MCF. The perforation can be managed with placement of an autogenous cortical graft or a custom total joint replacement. Violation of the dura with CSF leak requires dural repair with temporalis fascia, fibrin glue or other allogeneic material. A lumbar drain or, rarely an intraventricular catheter may be needed to reduce intracranial pressure (ICP) to allow the perforation to heal. An intraoperative consultation with neurosurgery should always be considered as well as an immediate post-operative non contrast CT scan. Appropriate periodic post-operative neurological evaluation should also be considered.

FACIAL NERVE INJURY

The facial nerve exits the stylomastoid foramen and enters the parotid gland before dividing into the temporal and cervical branches. The temporal branch then forms the temporal, zygomatic and buccal branches while the cervical branch gives rise to the marginal mandibular and cervical branches. The temporal and zygomatic branches are most at risk during TMJ surgery. These branches lie between the superficial temporal fascia and the superficial layer of deep temporal fascia and cross the zygomatic arch at a mean of 2 cm anterior to the bony external auditory meatus (range 0.8 – 3.5 cm). Facial nerve injury is a known complication of TMJ surgery, but the risk of injury can be significantly reduced with good surgical technique and gentle retraction using the smallest retractors. Facial nerve monitoring with a nerve stimulator is challenging as the nerve is typically with the parotid gland and most injuries are from excessive retraction rather than direct nerve injury.

The House-Brackmann grading system is a useful tool to help recognize and classify facial nerve injury. (Table 10).

Although most facial nerve injuries are temporary with full recovery over a Ttme period of several months, some patients will require treatment. Management includes

Temporal and Zygomatic Branches

- The use of artificial tears and lubricants to prevent a dry eye and the potential for corneal irritation/scarring
- The use of nocturnal taping to close the upper eyelid
- External upper eyelid weight to facilitate eye closure

Table 10. HOUSE-BRACKMANN FACIAL NERVE CLASSIFICATION

Grade	Description	Characteristics
1	Normal	Normal function
2	Mild	Rest: normal symmetry and tone Forehead motion: moderate/good Eye closure: complete with minimal effort Mouth motion: slight asymmetry
3	Moderate	Rest: normal symmetry and tone Forehead motion: slight to moderate Eye closure: complete with effort Mouth motion: slightly weak with maximal effort
4	Moderately severe	Rest: normal symmetry and tone Forehead motion: none Eye closure: incomplete Mouth motion: asymmetric with maximal effort
5	Severe	Rest: Asymmetry Forehead motion: none Eye closure: incomplete Mouth motion: slight movement
6	Total paralysis	No movement

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- Gold or platinum insertion into the upper eyelid
- Neuromuscular facial retraining including dual channel surface electromyography
- Botox 20-30 units subcutaneous for the contralateral frontalis muscle for asymmetry
- Browlift for drooping eyebrow, blepharoplasty or lower lid shortening for blepharoptosis

Buccal and Marginal Mandibular Branches

- Static reconstruction, ipsilateral nerve graft, cross face nerve graft
- Hypoglossal facial transfer
- Free muscle transfer

Frey Syndrome

Parasympathetic and sympathetic fibers innervate the parotid gland via the auriculotemporal nerve. The parasympathetic nerves also stimulate the overlying erector pilae muscles of the skin while the sympathetic fibers stimulate overlying sweat glands. Temporomandibular joint and parotid gland surgery can result in aberrant neural transmission such that the parasympathetic nerves that normally innervate the parotid gland also innervate the overlying sweat glands. This results in gustatory sweating in the preauricular region. Patients will report that eating, or even the anticipation of food, will result in sweating in the preauricular region. The degree of gustatory sweating and the exact location should be confirmed with the Minor's starch iodine test. This involves painting the skin with iodine and then lightly dusting the skin with starch. Any sweating that accompanies eating will result in discoloration of the starch producing a blue color that outlines the extent of the involved skin.

The treatment of Frey syndrome is simple with the use of subcutaneous botulinum toxin. A total of 2.5 units of botulinum toxin per square centimeter is adequate to eliminate the gustatory sweating. The procedure will need to be repeated every 4-6 months. Surgery can also be considered which typically involves elevating a skin or Superficial Musculoaponeurotic System (SMAS) flap with the insertion of autogenous fascia of acellular dermal matrix between the deeper tissue and the skin flap. This effectively prevents reinnervation of the cutaneous sweat glands.

Massive Hemorrhage

The TMJ is located in close proximity to many branches of the external carotid artery. This particularly so for the facial, maxillary and superficial temporal arteries. The maxillary artery has multiple branches and can be conveniently divided into three parts. The proximal part supplies the deep auricular, anterior tympanic, inferior alveolar, middle meningeal and accessory meningeal branches all of which traverse foramen. The middle part supplies branches to the masseter, temporalis, medial and lateral pterygoid muscles. The third part enters the pterygomaxillary fissure before dividing into the infra-orbital, posterior superior alveolar, sphenopalatine, greater/lesser palatine and pharyngeal branches. The proximal portion of the maxillary artery lies in close proximity to the neck of the condyle. The anatomy can be further complicated due to prior surgery or bony ankylosis of the TMJ.

Preventing intraoperative and post-operative hemorrhage requires in depth understanding of anatomy and good surgical technique. Careful sub periosteal dissection and gentle and well positioned retractors are crucial to prevent unintended injury to blood vessels. The use of Computed Tomography Angiography (CTA) or angiography and selective embolization may be of benefit when there is concern for altered anatomy. The latter is associated with a small risk of CVA. Ligation of the external carotid artery and its

main branches to prevent or manage bleeding from the maxillary artery is often futile due to the abundant collateral vascular supply and anastomoses.

Bleeding can nearly always be stopped with digital pressure. Ultimately good suction and lighting will allow the bleeding vessel to be identified and a vascular clip placed. Access to the TMJ from a preauricular approach is excellent although access to the maxillary artery and its branches can be more challenging particularly when the mandibular condyle is still present. The additional retromandibular approach, as is common with alloplastic joint replacement, provides excellent access for digital pressure to the medial aspect of the mandible and infratemporal fossa which will arrest bleeding immediately and allow a more controlled environment to identify and ligate the bleeding vessel. Topical agents such as Floseal, Surgiflo, Vistaseal, Surgicel, Avitene, Gelfoam and topical thrombin may also help. If bleeding remains problematic despite all attempts to control it, angiography and selective embolization remains the best choice. Rarely the wound can be packed with gauze or lap sponges and the procedure terminated.

Heterotopic Bone and Ankylosis

Heterotopic bone can form in and around the native or reconstructed TMJ as well as an alloplastic TMJ TJR. It is unclear what causes bone formation, but it can be seen in the face of trauma, inflammation, endocrine abnormalities and in the multiply operated joint. The diagnosis is often incidental based on limited jaw opening and radiographic features on a panoramic x-ray. Ultimately CT with or without IV contrast will identify and quantitate the degree and extent of heterotopic bone formation. Preventing heterotopic bone formation can be challenging. The use of NSAIDs such as indomethacin has been advocated with little evidence to support it. The use of low dose radiation (10 Gy) over 5 days immediately following surgical removal of heterotopic bone has been reported to result in a modest reduction in new bone formation. The potential for radiation scatter (post TMJ TJR) and adverse effects following radiation have limited the usefulness of this modality. The most common and least problematic treatment appears to be the creation of a critical gap arthroplasty, use of an alloplastic TJR and fat grafting. The fat graft is thought to reduce dead space and the differentiation of undifferentiated mesenchymal cells and pluripotent cells which then limits fibrosis and heterotopic calcification.

TMJ TJR Infection

Infection following TJR is uncommon with an incidence of 1.5%. It can occur in the immediate postoperative period (early) or many years after successful TJR (late). Early infections are likely to be the result of micro-organisms introduced during surgery and present within the first month following surgery. They often present with pain, swelling, fever and drainage from the incision sites. The WBC is elevated as are the CRP and ESR. A CT scan with contrast usually confirms the diagnosis. There are essentially two options for treatment. The first involves a protocol developed by Dr Larry Wolford that recommends opening all incisions, scrubbing the prosthesis with iodine using a sterile toothbrush, placement of two irrigating drains (lateral and medial to prosthesis), a Penrose drain for egress of the irrigant and a peripheral intravenous central catheter (PICC) with culture and sensitivity driven IV antibiotics for 6 weeks. The second option involves explantation of the TJR, placement of an antibiotic impregnated acrylic spacer for 3 months, PICC line with antibiotics and a new TJR at 3 months.

Late infections may be the result of less pathogenic organisms that were introduced during surgery or from hematogenous spread. The diagnosis of late infections is more challenging as both signs and symptoms are milder. Increasing pain and mild diffuse swelling may be the only presentation. The WBC, ESR and CRP are often within normal range. A CT scan with contrast should be the imaging of choice although it is often unremarkable as the infection is most often the result of an insidious biofilm on the TJR. A more sensitive test for occult prosthesis infection is Technetium-99m hexamethylpropyleneamine oxime (HMPAO) nuclear scan. An Indium-111 labelled WBC scan is also very sensitive. Late infections require TJR explantation, antibiotic impregnated acrylic spacer and a PICC line with 6-12 weeks of antibiotics followed by a new TMJTJR.

The nutritional, immune and smoking status may all adversely affect the risk of TJR infection. These should be optimized prior to surgery. Intraoperative considerations include preexisting skin infections (including acne vulgaris) and the relative challenges in obtaining and well prepped surgical site given the ear, EAC and the presence of hair. Strategies to reduce the risk of infection include.

- Shearing the hair and ensuring it is kept out of the surgical field
- Irrigate the EAC with vancomycin/normal saline solution before prepping
- Use an ear wick to occlude the EAC
- Thorough skin preparation with chlorhexidine or betadine solution
- Careful toweling and draping
- Perioperative IV antibiotics such as Vancomycin which are effective against most skin microorganisms including Methicillin Sensitive Staphylococcus Aureus (MSSA) and Methicillin Resistant Staphylococcus Aureus (MRSA).

Chronic Pain

Despite and accurate diagnosis and meticulous surgery there are patients who will continue to experience pain and/or functional issues following TMJ surgery. The causes for persistent pain following TMJ surgery are many including:

- Initial misdiagnosis
- Myofascial Pain
- Neuropathic pain
- Centrally mediated pain
- Complex Regional Pain Syndrome (CRPS)
- Axis II diagnosis including anxiety, depression, somatization disorder, catastrophizing

Persistent pain following TMJ TJR may also be the result of specific intrinsic factors including:

- Heterotopic bone formation
- Dislocation
- Hardware failure
- Synovial entrapment
- Material hypersensitivity.

Persistent post-operative pain can be stimulus evoked or stimulus independent. Stimulus dependent pain is thought to be the result of continuous post-surgery C fiber transmission that results in central sensitization. This results in allodynia, hyperpathia and hyperalgesia. The presence of allodynia alone suggests that collateral axonal sprouting and cross talk from A-fibers may be the causative factor. Stimulus independent pain is either the result of up regulated sodium channels withing the axons or sympathetically mediated as a result of the expression of alpha-adrenoreceptors. The net result is constitutional axonal firing and chronic neuropathic pain. The diagnosis of neuropathic pain in TMJ surgery requires the following steps:

1 Focused history and physical examination
2 Neurosensory testing
3 Magnetic Resonance Neurography to locate potential sites on nerve injury
4 Diagnostic local anesthetic nerve blocks.

The management of neuropathic pain is challenging due to the poor prognosis. Referral to a neurologist or pain specialist is strongly encouraged. Treatment may consider the following

- NSAIDs although the response is often poor
- Calcium channel blockers such as gabapentin and pregabalin
- Sodium channel blockers such as oxcarbazepine
- Tricyclic antidepressants (secondary amines) such as nortriptyline or desipramine
- Selective serotonin and norepinephrine reuptake inhibitors (SNRI) such as duloxetine and venlafaxine
- Topical lidocaine

The use of opioids use for the management of non-cancer pain including chronic TMJ pain remains controversial with limited data to support it. Partial opioid agonists and mixed agonists/antagonists such as tramadol, buprenorphine and butorphanol are generally better choices than full agonists. This is because the risk of Adverse Events (AE), Adverse Drug Related Behavior (ADRB) and opioid tolerance are considerably less.

There will be a small proportion of patients who develop chronic pain despite an accurate diagnosis, sound patient selection and good surgical technique. It is critical that these patients receive an appropriate diagnosis of neuropathic pain, and management. Patient abandonment must be avoided, and these individuals should be referred to other specialists that can help manage their chronic pain.

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