Clinical Handbook for Oral, Facial, and Head Pain

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Submitted August 16, 2023;
accepted October 17, 2023.

How to Cite This Handbook:
Huff KD, Benoliel R. Clinical Handbook for Oral, Facial, and Head Pain. J Oral Facial Pain Headache 2023;37(4):219–268.

doi: 10.11607/ofph.3488

Introductory Remarks from the Authors

We have compiled this material to be used as a concise summary of common painful and nonpainful temporomandibular joint (TMJ) disorders, as well as painful disorders in the face and head. It is not intended to be comprehensive, nor is it intended to be used as a standalone reference—in fact, we encourage the reader to study the references listed at the end and have provided links for open access references where possible. Our goal is for this handbook to be used as a study reference tool and a quick-reference guide in the clinical setting. The therapeutic options offered herein are backed by evidence when possible but may reflect the authors' personal opinions based on clinical experience when evidence is lacking. As such, the pharmacotherapeutic armamentarium is not intended to be all-inclusive but rather to represent current practices and first and second choices of medications for quick reference. The "differential diagnosis" row in each table lists conditions that should be considered to present similarly to the primary condition, but a true differential diagnosis should be patient-specific relative to the chief complaint. When possible, International Classification of Diseases (ICD)-10 codes have been included for clinical convenience. Where the orofacial pain (OFP) term varies from the ICD-10 terminology, those terms are included within the description of the condition.

All of the entities in this handbook are, in our opinion, within the scope of care of OFP specialists and appropriately trained dentists whose practice includes the diagnosis and management of OFP disorders. Nevertheless, we are aware that programs differ in their content and focus. This is where continuing education is irreplaceable. It is the individual professional's responsibility to remain knowledgeable and updated regarding disorders, testing that may be indicated, and evidence-based management via pharmacologic and other modalities. In the growing field of OFP, it is important to remember that the concept of evidence-based practice includes a dynamic interaction between the following elements: the available scientific literature base, patient factors (autonomy based on informed consent, physical and psychologic health, etc), and clinician experience. We acknowledge the significant relationship between sleep and pain; however, this topic was not included in this handbook because the scope of this project does not provide the attention that the topic of sleep as it relates to pain deserves.

How to Use This Handbook

The currently recognized diagnoses within the field of OFP have been grouped and categorized for ease of recognition based on clinical presentation: cutaneous pain, dental pain, periodontal pain, muscle disorders, TMJ disorders, neck pain, systemic disease, neuropathic pain, and primary headaches. Common medications for OFP conditions and appropriate serologic testing options are also included. The layout has been designed so that the pages may be printed out on a standard color printer and placed in a physical binder for desktop reference. Please note that all abbreviations used in a given section are spelled out at the end of that section. We recommend printing the document single-sided and then punching holes along the top of

each page with a three-hole punch to place in a binder for use as a flip-chart. Alternatively, the handbook may be printed and bound by any professional printer because it is open access and free for reprinting. Of course, it is also useful as a digital resource.

Within each table, the terms ***Risk*** and ***BB*** may appear in the treatment or medication sections. ***Risk*** indicates the need for caution when prescribing—not the risk of developing those conditions, but rather the potential for complications. ***BB*** indicates an FDA Black Box Warning for that treatment or medication.

Once the general type of pain has been identified by clinical examination, the appropriate color-coded section should narrow the search for conditions described by that type of pain. For example, moderate pain that is nonpulsatile and dull in character should direct the clinician quickly to the section on muscle pain. From there, the diagnosis can be further refined.

MUCOCUTANEOUS PAIN	DENTAL PAIN	PERIODONTAL PAIN	MUSCLE DISORDERS	TMJ DISORDERS	NECK PAIN	SYSTEMIC DISEASE	NEUROPATHIC PAIN	PRIMARY HEADACHES	COMMON MEDICATIONS	SEROLOGIC TESTS

Click on the color-coded category in this key and subsequent footers to hyperlink directly to that section of the handbook.

This guide was prepared using information primarily from the following sources:

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*Open access, free PMC article, or otherwise available for download.

MUCOCUTANEOUS PAIN

	Allergy (K12.1)	ANUG (A69.1)	Candidiasis (B37.0)	Lichen planus (L43.9)	Aphthous stomatitis (K12.0)
Clinical characteristics	Acute or chronic moderate pain Erythema, blisters, cracking of skin	Acute, moderate-severe pain Ulcerative gingival papillae with spontaneous bleeding Very rare; should raise concern for underlying disease	Burning, dull; patient often has history of recent antibiotic treatment or immune system suppression Multiple forms: pseudomembranous; erythematous (median rhomboid glossitis, denture stomatitis); angular cheilitis	Asymptomatic or chronic, burning, continuous pain; possible erosive lesions (very low precancerous potential) Wickham's striae or erythema on mucosa; may be ulcerated, usually generalized; accompanying erythema on skin is possible	Acute, moderate-severe continuous pain White ulcers with erythematous borders on mucosa, may be major or minor
Tests	Referral to allergist CBC with differential, CRP, MP	May need medical consultation if underlying disease suspected—rule out HIV General health work-up	Cytology	Biopsy Liver function test Hepatitis B and C antibody titer	
Treatment	Patient education and awareness training Identify and prevent cause—abort offending drug or substance Eliminate irritants Restrict function for healing Medical consultation if systemic involvement suspected	Patient education/OHI Debridement Eliminate irritants Restrict function for healing	Patient education and awareness training/OHI Eliminate irritants Medical consultation—rule out HIV or if patient is immunocompromised	Patient education and awareness training Rule out possible medication cause (ie, beta-blockers and ACE inhibitors) Minimize trauma Avoid triggers LLLT	Patient education and awareness training Identify and avoid triggers Stress reduction techniques LLLT
Medications	Diphenhydramine 25–50 mg every 4–6 h, < 300 mg/d Analgesics—avoid NSAIDs due to possible SJS Chlorhexidine rinse 0.12% 15 mL for 30 s bid	Chlorhexidine rinse 0.12% 15 mL for 30 s bid Analgesics Systemic antibiotics (eg, metronidazole 250–500 mg tid x 7–14 d)	Nystatin rinse: 4–6 mL qid for 7–14 d Daktarin oral gel Clotrimazole lozenges Fluconazole (systemic; eg, diflucan) 100–200 mg for 14 d or more Angular cheilitis: ▪ Nystatin with triamcinolone cream for cheilitis ▪ Mupirocin (eg, Bactroban) for persistent cheilitis	Fluocinonide gel 0.05% bid/qid for 2 wk Viscous lidocaine (200 mg qid, 10 mL of 2% solution) Tacrolimus ointment 0.1% tid or qid for 4–6 wk (*Risk: may be carcinogenic*) Prednisone 20 mg qd for 2–6 wk, followed by taper	Fluocinonide gel 0.05% bid-qid for 2 wk Amlexanox 5% oral paste Viscous lidocaine (200 mg qid, 10 mL of 2% solution) Dexamethasone rinse 0.5 mg/5 mL tid and then spit Prednisone 40 mg qd for 5 d
Differential diagnosis	Nutritional deficiency Autoimmune disorder	Gingival abscess Periodontal abscess Consider: leukemia, AIDS, autoimmune disease	Trauma Lichen planus Squamous cell carcinoma Consider: whether patient is immunocompromised, AIDS	Geographic tongue Aphthous stomatitis Trauma Squamous cell carcinoma Consider: Lupus erythematosus, Behçet's disease	Trauma Drug reaction (NSAIDs) Primary herpes simplex Lichen planus Geographic tongue Consider: Lupus erythematosus, Behçet's disease

MUCOCUTANEOUS PAIN

	Herpes simplex (B00.9)	Burning mouth syndrome (K14.6)	Pain due to cancer treatment	Geographic tongue (K14.1)	Trauma (K06.2)
Clinical characteristics	Ulcers on lips and intraorally on attached gingiva; not necessarily painful, generally unilateral Herpetic pharyngitis often possible; may be associated with fever and malaise Prodromal period < 6 h of tingling/itching; small tense vesicles on an erythematous base, which may coalesce; persists for 5–10 d	Persistent, continuous but variable, and superficial somatic pain; location of pain corresponds to areas of greatest movement, somewhat cyclic and increased by frictional contact Classified as primary when no causative pathology is found (thought to be neuropathic) and secondary when a local or systemic disorder may account for symptoms (see below) Present day and night, crescendos throughout day Strong psychosocial association Possible local causes: infection, chemical/mechanical trauma, GERD, radiation (considered secondary) Secondary: ▪ Systemic causes: autoimmune disorder, diabetes, hypothyroidism, medication side effects, nutritional deficiency, multiple sclerosis, HIV, sarcoidosis ▪ Local causes: *Candida*, lichen planus, etc	Postsurgical pain Mucositis from radiation or chemotherapy (would be acute in hospital or ongoing treatment settings) Neuropathy due to surgery/chemo- or radiotherapy	Benign Inflammatory Multiple, well-demarcated zones of erythema located on tongue, buccal mucosa, and lip(s) May present as burning sensation Fissured tongue May be manifestation of psoriasis	May be microtrauma (dental surgery, extractions) or microtrauma (MVAs, altercations) Acute, moderate-severe pain Varied presentation; wound may or may not be present; mobility of teeth may occur
Tests	Diagnosis via PCR available	Topical anesthetic: If it arrests pain, then primary hyperalgesia—confirm with analgesic lozenges CBC with differential, MP, CRP, arthritis panel, antinuclear antibodies, thyroid function tests, HbA1c Serum IgE and patch test for dental materials Serum iron, ferritin, transferrin, vitamins B1, B6, and B12, folate, and zinc			Analgesics Antibiotics Antimicrobials, topical and/or systemic

MUCOCUTANEOUS PAIN

	Herpes simplex (B00.9)	Burning mouth syndrome (K14.6)	Pain due to cancer treatment	Geographic tongue (K14.1)	Trauma (K06.2)
Treatment	Patient education and awareness training (reduce infection of others) Check pregnancy status Reduce triggers: sunlight, stress, unknown Sunblock for lips Stress reduction techniques	Patient education and awareness training Stress reduction techniques LLLT CBT	Manage based on presentation LLLT may work in some acute pain situations	Patient education and awareness training Avoid triggers	Intraoral radiograph or limited-volume CBCT for dental injury CBCT indicated for jaw fracture
Medications	Famciclovir 1,500 mg as one dose Valacyclovir 2 g po every 12 h for 1 d Penciclovir 1% cream every 2 h while awake for 4 d Viscous lidocaine (200 mg qid, 10 mL of 2% solution)	Clonazepam 0.5 mg tid; can also be used as "swish and spit," reducing systemic risk of liver, kidney, OSA, depression Topical benzocaine 20% Viscous lidocaine (200 mg qid, 10 mL of 2% solution) Medication carrier with analgesics, anxiolytics, artificial sweeteners	Tailored to specific pain diagnosis—musculoskeletal, neuropathic, and inflammatory- and intensity-based	Analgesics Fluocinonide gel 0.05% bid-qid for 2 wk	Patient education and awareness training Identify and prevent cause Debride if necessary Eliminate irritants Restrict function for healing Medical consultation
Differential diagnosis	Herpes zoster—rarely recurs and usually causes more severe pain and larger groups of lesions that are distributed along a dermatome Aphthous stomatitis Trauma	Consider secondary causes: ▪ Candidiasis ▪ Autoimmune disorders ▪ Nutritional neuropathy ▪ Systemic neuropathy ▪ Neuritis	Mucositis Neuropathy Neuritis	Candidiasis (median rhomboid glossitis) Lichen planus Burning mouth syndrome	Allergy Chemical, electrical, or thermal burn

ACE = angiotensin-converting enzyme; ANUG = acute necrotizing ulcerative gingivitis; BB = FDA Black Box Warning; bid/tid = twice a day/three times a day; MP = metabolic panel; CBC = complete blood count; CBT = cognitive behavioral therapy; CRP = C-reactive protein; GERD = gastroesophageal reflux disease; GI = gastrointestinal; HbA1c = hemoglobin A1c; IgE = immunoglobulin E; LLLT = low-level laser therapy; MVA = motor vehicle accidents; NSAIDs = nonsteroidal anti-inflammatory drugs; OHI = oral hygiene instruction; OSA = obstructive sleep apnea; PCR = polymerase chain reaction; po = by mouth; qd = every day; SJS = Stevens-Johnson syndrome.

ODONTOGENIC & NONODONTOGENIC DENTAL PAIN				
	Pulpitis (K04.0)	Cracked tooth (TS) (K03.81)	MFP toothache (M79.1)	Sinus toothache
Clinical characteristics	Dull, aching, throbbing, at times sharp pain; visceral; unilateral; clinical presence of etiologic factor; chief pain can be reproduced during exam; gets better or worse with time; reduced or eliminated by LA; easily localized Reversible pulpitis: hypersensitivity Irreversible pulpitis: intermittent sharp pain to stimulus—may transition to necrosis with periapical abscess Untreated decay or trauma may lead to a symptomatic or asymptomatic necrotic pulp (K041) If the tooth is painful to percussion, then there is also a periapical diagnosis of symptomatic apical periodontitis or acute apical abscess if swelling or purulence are present	Sporadic, sharp, momentary pain on biting or release, occasionally to cold stimuli Pain may be delayed minutes after chewing Easily localized Fractures may or may not be easily visualized clinically Poorer prognosis for oblique and vertical fractures	Deep, dull, aching muscle pain associated with tooth pain (masseter, temporalis, anterior digastric muscles commonly refer to teeth); nonpulsatile; not altered by stimulation of tooth Tooth pain with muscle function: temporal pattern (often late afternoon after stressful day); palpable taut bands of muscle; associated with TTH; LA does not alter; LA of muscle stops toothache	Nonlocalized maxillary alveolar pain: ▪ Bacterial: severe, throbbing, stabbing with sense of pressure ▪ Allergy-induced: chronic dull ache of the teeth Partially relieved by LA; pain to percussion of maxillary teeth that test vital; occasional cold sensitivity; sense of pressure or fullness in the infraorbital area; purulent nasal discharge if bacterial Postnasal drip is common
Tests	Percussion and vitality testing ALL TEETH ON SIDE OF INTEREST and compare to contralateral teeth LA to confirm and localize pulpal pain Radiograph Wait for transition to periodontal pain if localization not possible (a few days)	Percussion and vitality testing Selective pressure Periodontal probing Evaluate occlusion Radiograph Transillumination	Vitality testing LA of tooth LA of the taut band of muscle	Percussion and vitality testing 4% lidocaine spray (if reduces pain, sinus pain) Head dip test (increased pain when head below knees) CBCT scan
Treatment	Patient education and awareness training Remove stimulus Endodontic therapy: restore, extract	Patient education and awareness training One or combination of: ▪ Endodontic treatment ▪ Restorative treatment ▪ Extraction ▪ Occlusal adjustment	Patient education and awareness training Spray/stretch Massage Heat Trigger point injections Stabilization appliance	Patient education and awareness training PCP or ENT referral
Medications	Analgesics	Analgesics	Analgesics Cyclobenzaprine 5–10 mg tid for 3 wk (*Risk: elderly, cardio, opioids*) Amitriptyline 10–35 mg qhs (*Risk: cardio, diabetes, seizure, UT disorders*); *BB: suicide, < 25 y* Duloxetine 60 mg qd; *BB: suicide*	Bacterial: Augmentin (amoxicillin clavulanic acid) 875/125 mg bid or Bactrim (trimethoprim/sulfamethoxazole) 160 mg bid Allergy-induced: fluticasone spray and antihistamines

ODONTOGENIC & NONODONTOGENIC DENTAL PAIN

	Pulpitis (K04.0)	Cracked tooth (TS) (K03.81)	MFP toothache (M79.1)	Sinus toothache
Differential diagnosis	Periodontal pain Neuroma Neuritis Myalgia/myofascial pain Migraine	Pulpitis Neuroma Neuritis	Pulpitis Periodontal pain TTH Migraine Cardiomyopathy Lyme disease	Pulpitis Lyme disease Periodontal pain Neuritis Migraine Cardiomyopathy Trigeminal neuralgia PTTN

	Neuralgia (V) toothache	Neuralgia (IX) toothache	Neuritic toothache	PIDAP	Occlusal dysesthesia	Cardiac toothache
Clinical characteristics	Severe, shooting, electric-like pain that lasts for a few seconds followed by a refractory period; "worst pain ever"; sometimes aching in the affected zone starts several hours before attack (pre–TN); unilateral Not altered by intraoral thermal testing; V3 most affected, followed by V2; trigger zone present; often pain can be traced to a specific tooth	Severe, shooting, electric-like pain that lasts for a few seconds followed by a refractory period; "worst pain ever" Less tooth pain than TN; elicited by swallowing, chewing, or talking; pain distribution = posterior mandible, oropharynx, tonsillary fossa, and ear	Elevated threshold for pricking pain, but lower threshold for burning pain Herpes zoster = viral cause Maxillary sinusitis = bacterial cause Direct trauma can cause continuous, nonpulsatile pain consistent with duration of inflammatory process that is burning, intense, and stimulating with precisely localizable pain to a particular tooth with a "dead" or "strange" feel compared to adjacent teeth; onset of toothache after infection or trauma	Intraoral counterpart of PIFP Dull ache in tooth or teeth and/or adjacent dentoalveolar structures	Common complaint of uncomfortable and/or incorrect occlusion, usually accompanied by emotional distress Unverifiable Repeated and failed dental treatment reinforces patient perception Reassurance of no occlusal problem induces stress Often associated with extensive restorative dentistry Usually painless; when accompanied by surgery, add diagnosis of PTTN "Phantom bite"	Deep, diffuse pain that sometimes pulsates Pressure, burning quality Exacerbated by exercise Associated with neck, shoulder, and chest pain May be bilateral; may present in the left temporal region Prior history of cardiomyopathy
Tests	LA of trigger zone completely eliminates the pain and toothache; PDL injection will not reduce pain unless tooth is the trigger zone MRA w/wo contrast through cerebropontine angle; vascular loop protocol CBC with differential and platelets, urea/electrolytes, liver function, sodium level (< 136 mEq/L), and HLAb*1502 genetic testing in Asian and Indian patients; CBC + urea/electrolytes every 2–4 wk for 3 mo and then every 6 mo; liver function every 6 wk for 2 normal intervals	IA block does not affect pain Topical anesthetic to lateral pharyngeal wall may stop pain MRA w/wo contrast through cerebropontine angle; vascular loop protocol CBC with differential and platelets, urea/electrolytes, liver function, sodium level (< 136 mEq/L), and HLAb*1502 genetic testing in Asian and Indian patients; CBC + urea/electrolytes every 2–4 wk for 3 mo and then every 6 mo; liver function every 6 wk for 2 normal intervals	Identify cause: trauma, bacterial infection, viral infection	Diagnosis by exclusion There should be no sensory changes associated with the area of pain. ICOP, in the interest of research, is allowing this for now; nevertheless, pain of this type with sensory changes should be primarily diagnosed as PTTN	Bite analysis Occlusal guard test: will not be effective if occlusal dysesthesia DO NOT adjust occlusion further unless clearly contributory	Nitroglycerin test: relieves pain Ethyl chloride spray/stretch

NONODONTOGENIC DENTAL PAIN						
	Neuralgia (V) toothache	Neuralgia (IX) toothache	Neuritic toothache	PIDAP	Occlusal dysesthesia	Cardiac toothache
Treatment	Patient education and awareness training Antiepileptic medication Percutaneous balloon microdecompression (best), glycerol rhizotomy, thermocoagulation, or gamma knife Glycerol injections (short term)	Patient education and awareness training Referral to neurology Microvascular decompression surgery, glycerol rhizotomy, or gamma knife surgery (the earlier the better)	Patient education and awareness training Stress reduction techniques Reduce trauma to tooth	Patient education and awareness training Stress reduction techniques CBT	Patient education and awareness training Stress reduction techniques CBT Psychologic evaluation	Immediate referral to ER
Medications	Carbamazepine 100 mg/d + 100 mg every 2 d, < 1,200 mg/d Oxcarbazepine 300 mg + 300–600 mg/d, < 2,400 mg/d Add or alone: baclofen 5–15 mg + 5 mg q 3 d, < 30–60 mg/d Pregabalin 150 mg + 50 mg every 2 d, < 300–600 mg/d Gabapentin 300 mg, titrate to 3/d at 3-day intervals; usual dosage 1,800–2,400 mg	Carbamazepine 100 mg/d + 100 mg every 2 d, < 1,200 mg/d Oxcarbazepine 300 mg + 300–600 mg/d, < 2,400 mg/d Add or alone: baclofen 5–15 mg + 5 mg every 3 d, < 30–60 mg Pregabalin 150 mg + 50 mg every 2 d, < 300–600 mg/d Gabapentin 300 mg, titrate to 3/d at 3-day intervals; usual dosage 1,800–2,400 mg	Bacterial = antibiotics Viral = antiviral Analgesics Amitriptyline 10–35 mg qhs (*Risk: cardio, diabetes, seizure, UT disorders*; *BB: suicide, < 25 y)* Duloxetine 60 mg qd; *BB: suicide*	Topical compounded medicament containing custom dosing of medications like capsaicin, a topical anesthetic, atricyclic antidepressant, and anti-epileptic. Alternative to vacuum-formed carrier is Poly-ox bandage Consider clonazepam, similar to burning mouth syndrome therapy LLLT may be helpful Patient education Referral for psychologic therapy may be indicated	Amitriptyline 10–35 mg qhs (*Risk: cardio,diabetes, seizure, UT disorders*); *BB: suicide, < 25 y* Duloxetine 60 mg qd; *BB: suicide* Gabapentin 100 mg qd + 100 mg/d < 1,800 mg/d Pregabalin 150 mg + 50 mg every 2 d, < 300–600 mg/d Doxepin 25–75 qhs (*Risk: cardio, epilepsy, asthma, psych, heat*; *BB: suicide*)	ASA 650 mg, STAT
Differential diagnosis	Paroxysmal hemicrania Cluster headache Lupus erythematosus Pulpitis Periodontal pain Multiple sclerosis	Paroxysmal hemicrania Cluster headache Cardiomyopathy Pulpitis Periodontal pain Lupus erythematosus Multiple sclerosis	Pulpitis Lyme disease Periodontal pain Systemic arthritides Trigeminal neuralgia Lupus erythematosus Cardiomyopathy PIDAP	PTTN TN Neuroma Periodontal pain	Malocclusion PTTN Neuroma Neuritis TMD	Pulpitis PDAP Periodontal pain Neuralgia (V, IX) Migraine Somatoform TA TMD Lyme disease Neuritis Lupus erythematosus

ASA = acetylsalicylic acid; CBC = complete blood count; CBT = cognitive behavioral therapy; ECG = electrocardiogram; ENT = ear, nose, and throat; IA = inferior alveolar nerve; ICOP = international classification of orofacial pain; LA = local anesthetic; MFP = myofascial pain; OCD = obsessive-compulsive disorder; PCP = primary care physician; PDAP = persistent dentoalveolar pain disorder; PDL = periodontal ligament; PIDAP = persistent idiopathic dentoalveolar pain; PIFP = persistent idiopathic facial pain; PTTN = painful traumatic trigeminal neuropathy; qd = every day; qhs = before bed; TCA = tricyclic antidepressant; tid/qid = three times a day/four times a day; TTH = tension-type headache; TN = trigeminal neuralgia; UT = urinary tract.

PERIODONTAL PAIN

	Gingival abscess (MK05.00)	Periodontal abscess (K05.20)	Symptomatic apical periodontitis	Periapical abscess (K04.7)	Pericoronitis (K05.20)
Clinical characteristics ▪ Dull, aching, and occasionally throbbing; identifiable periodontal condition (pocket, furcation, abscess); proportional to degree of provocation; pain on biting and possibly on release; reduced or eliminated by LA ▪ More localized than pulpal pain; tooth feels "high" during chewing and sore ▪ Ranges from "itching" to "throbbing" pain	Confined to marginal gingiva Caused by foreign body or trauma, followed by infection Painful, fluctuant, erythematous swelling	Acute or recurrent inflammation in periodontally diseased site Localized swelling of the gingiva and/or alveolar mucosa Erythematous, cyanotic Purulence likely Pain ranges from deep ache to severe discomfort, exacerbated by function and percussion Affected tooth may be mobile and appear extruded Usually associated with a deep gingival pocket but could be secondary to dental condition (endodontic/periodontic lesions) Tooth tender to percussion	Acute pain attributed to inflammation of the periapical tissues Untreated may evolve into periapical abscess Associated with teeth with a necrotic pulp or acute pulpitis; can occur immediately following endodontic therapy Pain on pressure over periapical gingiva Nonvital, tender to percussion	Usually follows pulpal pain Rapid onset Spontaneous Acute percussion pain Purulence Swelling Severe cases: fever, malaise, cellulitis, and lymphadenopathy Nonvital tooth, tender to percussion, may have vertical mobility Swelling in sulcus, usually buccal (maxillary lateral incisors may have palatal)	Localized infection surrounding crown of an impacted or partially erupted tooth Erythematous, swollen, painful gingival lesion Suppuration may be present Refers to ear, throat, floor of mouth Limited range of opening Difficulty with swallowing Swelling of ipsilateral cheek Systemic symptoms possible: fever, leukocytosis, malaise Painful submandibular lymph nodes
Tests	Periapical imaging	FMX or panoramic imaging	Radiographs may not show any periapical changes	Periapical imaging	Periapical/panoramic imaging
Treatment	Incise and drain Warm salt water irrigation Remove foreign body if present Irrigate and debride lesion if necessary	Antibiotics Incise and drain Debride root surface Occlusal adjustment, only if unavoidable, for palliative purpose Endodontic treatment may be needed on follow-up Extraction Refer to ER for severe infections: ▪ Severe swelling of soft tissue spaces ▪ Difficulty breathing ▪ High fever ▪ Elevation of the floor of the mouth ▪ Neck tracks	Initially perform debridement of pulp cavity; calcium hydroxide dressing; temporary restoration After resolution, consider endodontic treatment If tooth is not restorable, consider extraction If root canal obturation is already present, consider redoing therapy or surgical endodontics	Antibiotics Incise and drain (intracoronal, if possible) Endodontic treatment Extraction Refer to ER for severe infections: ▪ Severe swelling of soft tissue spaces ▪ Difficulty breathing ▪ Fever > 101°F (≥ 38°C) ▪ Elevation of the floor of the mouth ▪ Neck tracks	Lavage with chlorhexidine Extraction of the tooth after acute episode resolved Incision and drainage if gingival abscess present Urgent referral to oral surgeon: ▪ Trismus ▪ Fever > 101°F (≥ 38°C) ▪ Facial swelling ▪ Swelling into fascial spaces

PERIODONTAL PAIN					
	Gingival abscess (MK05.00)	Periodontal abscess (K05.20)	Symptomatic apical periodontitis	Periapical abscess (K04.7)	Pericoronitis (K05.20)
Medications	Analgesics/NSAIDs	Analgesics/NSAIDs Antibiotic for typically gram-negative flora Chlorhexidine rinse 0.12%	Analgesics/NSAIDs	Analgesics/NSAIDs Antibiotics if swelling, systemic symptoms	NSAIDs/analgesics Chlorhexidine 0.12% rinse with Monoject syringe Antibiotics if cellulitis, fluctuant swelling, systemic symptoms present
Differential diagnosis	Periodontal abscess Pericoronitis Periodontic/endodontic lesion Cracked tooth	Gingival abscess Periodontic/endodontic lesion Cracked tooth Pericoronitis	Periodontal abscess Periapical abscess Cracked tooth	Pulpal pain Phoenix abscess (periodontitis near apex) Periodontal abscess	Gingival abscess Periodontal abscess Periapical abscess

LA = local anesthetic; FMX = full-mouth x-ray; NSAIDs = nonsteroidal anti-inflammatory drugs.

COMMENTARY: CUTANEOUS PAIN, DENTAL PAIN, AND PERIODONTAL PAIN

- Purulence is rare with acute necrotizing ulcerative gingivitis (ANUG); if present, underlying systemic disease must be ruled out.
- Short-term systemic antibiotic therapy in conjunction with scaling and root planing usually results in rapid resolution of ANUG; if the condition does not improve quickly, underlying systemic disease and/or a compromised immune system is likely.
- Lichen planus is considered premalignant in some texts, but the risk of conversion to carcinoma is very low. Biopsy may be initially appropriate in severe ulcerative cases or in high-risk areas like the floor of the mouth, lateral border of the tongue, or soft palate.
- Trauma can be micro or macro. Examples of microtrauma include muscle pain, joint pain, and dental injury due to parafunction. Examples of macrotrauma include dental fractures, jaw fractures, contusions, and traumatic ulcerations.
- Herpes simplex commonly presents as herpes labialis, commonly known as *cold sores*, but lesions can occur in any terminal distribution of the trigeminal nerve. Presentation on the palate often appears as unilateral multiple fluid-filled pustules or erythematous ulcerations resulting from pustule rupture. Left untreated, herpetic lesions persist for 7 to 10 days and are painful. A pathognomonic characteristic of herpetic lesions is that they are limited to keratinized mucosa (eg, attached gingiva, external surface of the lips); they do not cross the vermilion zone. Herpes is highly contagious and poses a substantial risk to those in close contact with the infected individual, as well as to dental health providers, for the entire duration of the lesions. When contracted on the hands, the condition is known as *herpes whitlow* and can be disabling to those in the dental profession. There is no known cure for herpes. Low-level laser therapy may be helpful in reducing the potential for recurrence, but there is currently no available scientific evidence to support this theory. In severe cases, antiviral therapy may be given prophylactically.

MUSCLE DISORDERS

	Local myalgia (M79.1)	Myofascial pain (M79.1), Myofascial pain with referral	Spasm (M62.838)	Fibromyalgia (M79.7)
Clinical characteristics	Nonpulsatile, variable, dull, aching, boring background pain that may escalate in intensity, occurring spontaneously or through function and/or stretching Temporal pattern varies: constant, intermittent, or recurrent with sudden onset and rapid change Compromised function Muscle tenderness on palpation without referral Pain aggravated by function Actual muscle weakness with inability to open further Pain in jaw, temple, ear, or in front of ear modified by jaw function during the last 30 days	Myalgia must be present Trigger points may be present Chief complaint is usually point of referred pain With spreading: within muscle borders With referral: beyond muscle borders to distant sites. May be particularly demonstrated on palpation of trigger points See illustration of trigger point referral patterns (Fig 1) on page 14	A sudden, involuntary, reversible tonic contraction of a muscle. Spasm may affect any of the masticatory muscles Acute malocclusion may be present Immediate onset of muscle pain modified by function Immediate limited range of motion	Widespread pain (sensitivity and specificity have not been established) with concurrent masticatory muscle pain Recent criteria are based on widespread pain report without tender point identification; used to require 11 of 18 designated painful/tender points Patients have chronic pain behavior (multiple providers, unusual dependence on others, medication overuse, etc) 42% of patients with fibromyalgia have TMD symptoms
Tests	Ethyl chloride spray/stretch	Ethyl chloride spray/stretch LA trigger point injection	Ethyl chloride spray/stretch	PSG, if indicated
Treatment Goal: Reduce pain and restore muscle function	Patient education and awareness training Eliminate source of pain input Self-care: restrict function to within pain-free limits and minimize tooth contact (“lips together, teeth apart”); soft diet; stress reduction; avoid overuse PSR Moist heat/cold Stabilization appliance (part-time use only) Passive stretching and massage Response time: 1–3 wk	Patient education and awareness training Self-care: restrict function to within pain-free limits and minimize tooth contact (“lips together, teeth apart”); soft diet; stress reduction; avoid overuse Moist heat/cold PSR Referral for PSG if indicated Spray and stretch Massage Trigger point injections Stabilization appliance Physical therapy Regular exercise	Patient education and awareness training Acute pain relief: Spray and stretch or LA into the muscle, then stretch to full length Passive stretching and massage Self-care: restrict function to within pain-free limits and minimize tooth contact (“lips together, teeth apart”); soft diet; stress reduction; avoid overuse Moist heat/cold Response time: immediate	Patient education and awareness training Refer to PCP Refer to physical therapy Treat orofacial muscle conditions Self-care: restrict function to within pain-free limits and minimize tooth contact (“lips together, teeth apart”); soft diet; stress reduction; avoid overuse Moist heat Stabilization appliance (part-time use only)

MUSCLE DISORDERS

	Local myalgia (M79.1)	Myofascial pain (M79.1) with and without referral	Spasm (M62.838)	
Medications	Ibuprofen 400–600 mg tid Cyclobenzaprine 5–10 mg tid for 3 wk (*Risk: elderly, cardio, opioids*) Amitriptyline 10 mg or nortriptyline 25 mg qhs 1% to 2% lidocaine without epinephrine or 3% mepivacaine without epinephrine	NSAIDs/analgesics Cyclobenzaprine 5–10 mg tid for 3 wk *(Risk: elderly, cardio, opioids)* Amitriptyline 10–35 mg qhs (*Risk: cardio, diabetes, seizure, UT disorders*; *BB: suicide, < 25 y)* Duloxetine 60 mg qd; *BB: suicide* Gabapentin 300 mg, titrate to 3/d at 3-day intervals; usual dosage 1,800–2,400 mg 1% to 2% lidocaine without epinephrine or 3% mepivacaine without epinephrine for trigger point injections	2% lidocaine without epinephrine or 3% mepivacaine without epinephrine for trigger point injections NSAIDs/analgesics	NSAIDs/analgesics Amitriptyline 10–35 mg qhs (*Risk: cardio, diabetes, seizure, UT disorders*; *BB: suicide, < 25 y)* Duloxetine 60 mg qd; *BB: suicide*
Differential diagnosis	Fibromyalgia Odontalgia Myositis Arthralgia	Odontalgia Fibromyalgia Lyme disease Arthralgia	Trismus Dystonia	Lyme disease Odontalgia Neuropathy Arthralgia

MUSCLE DISORDERS

	Orofacial dyskinesia (R27.0)	Oromandibular dystonia (G24)	Tendonitis (M67.90)	Myositis (noninfective M60.9; infective M60.009)
Clinical characteristics	Involuntary, dance-like movements Injury to mucosa, tongue, jaw Common with brain trauma, psychiatric conditions, and neurologic disorders "Sensory trick" may reduce movement temporarily Must have: ▪ Myalgia and/or arthralgia ▪ Nerve conduction deficit ▪ Central and/or peripheral myopathic disease ▪ EMG confirmation Ataxia, unspecified (R27.0)	Excessive, involuntary, and sustained muscle contractions that may involve the face, lips, tongue, and/or jaw Must have: ▪ Myalgia and/or arthralgia ▪ Nerve conduction deficit ▪ Central and/or peripheral myopathic disease ▪ EMG confirmation Can be: ▪ Idiopathic, familial, torsion-type (G24.1) ▪ Acute type, due to drugs (G24.02)	Pain of tendon origin affected by jaw activity Limitation of movement secondary to pain Temporalis tendon most common, refers to teeth and other structures Must have myalgia with clinical confirmation of specific tendon	Pain of muscular origin with clinical characteristics of inflammation or infection: edema, erythema, and/or increased temperature. It generally arises acutely following direct trauma of the muscle or from infection Chronic form from autoimmune disease Limitation of movement secondary to pain Calcification of muscle can occur (myositis ossificans) Must have myalgia with edema, erythema, and/or increased temperature

MUSCLE DISORDERS

	Orofacial dyskinesia (R27.0)	Oromandibular dystonia (G24)	Tendonitis (M67.90)	Myositis (noninfective M60.9; infective M60.009)
Tests	MRI	EMG		CBC, CRP, antinuclear antibodies Panoramic imaging
Treatment	Patient education and awareness training Neurology referral Self-care: restrict function to within pain-free limits and minimize tooth contact ("lips together, teeth apart"); soft diet; stress reduction; avoid overuse Moist heat	Patient education and awareness training Neurology referral Self-care: restrict function to within pain-free limits and minimize tooth contact ("lips together, teeth apart"); soft diet; stress reduction; avoid overuse Moist heat Botulinum toxin injections Myotomy in extreme cases	Patient education and awareness training Self-care: restrict function to within pain-free limits and minimize tooth contact ("lips together, teeth apart"); soft diet; stress reduction; avoid overuse Corticosteroid (dexamethasone, triamcinolone, etc) injection Stabilization appliance (part-time use only) PSR Moist heat/ice Referral for PSG, if indicated Physical therapy with isometric jaw exercises and passive stretching should be initiated after pain reduction	Patient education and awareness training Eliminate source of pain input Manage primary infection Self-care: restrict function to within pain-free limits and minimize tooth contact ("lips together, teeth apart"); soft diet; stress reduction; avoid overuse Ice Stabilization appliance (part-time use only) Referral to neurology or rheumatology
Medications	NSAIDs/analgesics Diazepam 2–10 mg tid/qid; *BB: opioids = sedation, death*	Botulinum toxin 25 U (Botox equivalent) 25 U per muscle Diphenhydramine 25–50 mg qid Gabapentin 100 mg qd + 100 mg/d, < 1,800 mg/d Diazepam 2–10 mg tid/qid; *BB: opioids = sedation, death* Topiramate 25 mg + 25 mg every 2 wk, < 100–400 mg/d	NSAIDs: ▪ Acetaminophen 350–500 mg combined with ibuprofen 200 mg (synergistic effect) ▪ Ibuprofen 400 mg qid for 2 wk Amitriptyline 10–35 mg qhs (*Risk: cardio, diabetics, seizure, UT disorders; BB: suicide, < 25*) Cyclobenzaprine 5–10 mg tid for 3 wk (*Risk: elderly, cardio, opioids*) Duloxetine 60 mg qd; *BB: suicide* Dexamethasone 4 mg injection	NSAIDs/analgesics for 5–7 d every 4–6 h: ▪ Acetaminophen 350–500 combined with ibuprofen 200 mg (synergistic effect) Cyclobenzaprine 5–10 mg tid for 3 wk; *Risk: elderly, cardio, opioids.* If secondary to infection, use antibiotics.
Differential diagnosis	Myalgia Arthralgia Dystonia	Myalgia Arthralgia Myospasm Dyskinesia	Myalgia Centrally mediated myalgia Myositis Fibromyalgia Arthralgia Odontalgia Myofascial pain	Myalgia Fibromyalgia Ondontalgia Centrally mediated myalgia Arthralgia Myofascial pain

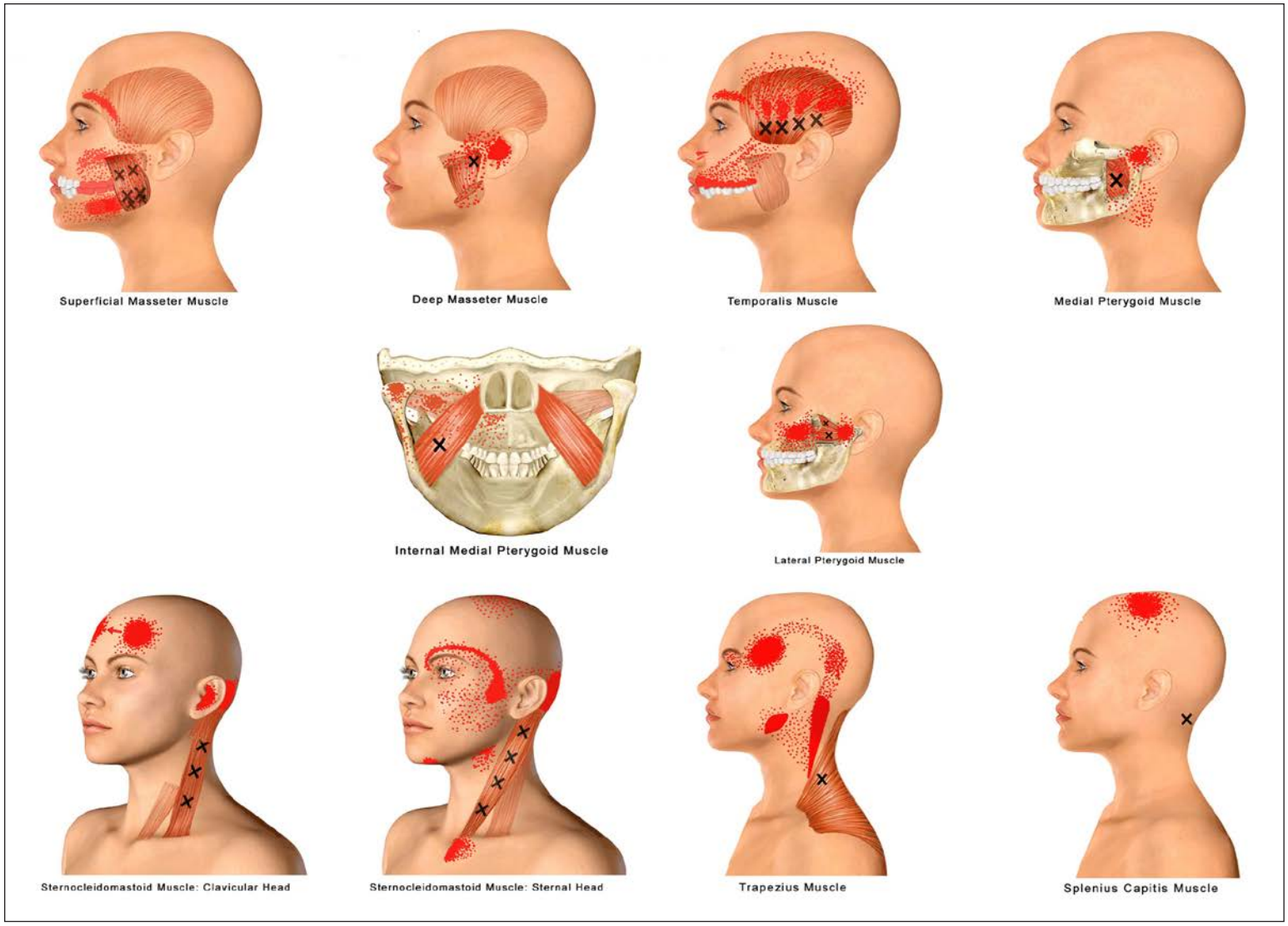

Fig 1 Pain referral patterns from the masticatory and neck muscles with myofascial pain (with referral) as originally described by Simons et al.[1] These patterns are common across patients and particularly prominent when trigger points are present and active. On examination, palpation of these points usually reproduces the referral pattern. Note that the superficial masseter muscle refers to the maxillary and mandibular molars and may be interpreted by the patient as toothache. The deep masseter refers to the TMJ, often causing a misdiagnosis of pain from the joint (arthralgia). The possibility of such a misdiagnosis would be reinforced by involvement of the pterygoid muscles. The temporalis refers pain to the maxillary teeth, causing similar diagnostic confusion. Note that in both the masticatory and neck muscles, there is pain referral to frontal, parietal, and supraorbital head regions. This reinforces the need for a coordinated approach to face and head pain. Illustrations courtesy of Dr Rich Hirschinger, the inventor of the Gentle Jaw (https://www.gentlejaw.com).

1. Simons DG, Travell JG, Simons LS. Myofacial Pain and Dysfunction: The Trigger Point Manual, Volume 1: Upper Half of Body, ed 2. Williams & Wilkins, 1999.

NONPAINFUL MUSCLE CONDITIONS

	Contracture (M62.40)	Hypertrophy (M62.9)	Muscle tumor (benign D21.0, malignant C49.0)
Clinical characteristics	The shortening of a muscle due to fibrosis of tendons, ligaments, or muscle fibers More commonly seen in the masseter or medial pterygoid muscle. Pain only on overextension Possible history: trauma, infection, radiation treatment Must have: ▪ Progressive reduction in ROM ▪ < 40 mm assisted opening ▪ Hard end-feel	Enlargement of one or more masticatory muscles as evidenced by comparison against previous records Usually painless Can be secondary to overuse and/or chronic tensing of the muscle(s) Some cases are familial or genetic in origin Diagnosis is based on clinician assessment of muscle size and needs consideration of craniofacial morphology and ethnicity	Tumors of the masticatory muscles may be benign or malignant/metastatic (uncommon) May present with: ▪ Swelling ▪ Spasm ▪ Myalgia ▪ Limited mouth opening ▪ Paresthesia
Tests	Panoramic imaging CBCT		CBCT MRI Biopsy
Treatment	Patient education and awareness training Refer to physical therapy Treat orofacial muscle conditions Self-care: restrict function to within pain-free limits and minimize tooth contact (“lips together, teeth apart”); soft diet; stress reduction; avoid overuse	Patient education and awareness training Refer for CBT if concerns Self-care: restrict function to within pain-free limits and minimize tooth contact (“lips together, teeth apart”); soft diet; stress reduction; avoid overuse	Patient education and awareness training Referral to head and neck surgeon Self-care: restrict function to within pain-free limits and minimize tooth contact (“lips together, teeth apart”); soft diet; stress reduction; avoid overuse
Medications		Consider botulinum toxin to induce atrophy; beware of potential osseous changes	Palliative posttreatment: ▪ Opiates/opioids ▪ Gabapentin 100 mg qd + 100 mg/d, < 1,800 mg/d ▪ Antimicrobial rinse ▪ Kepivance (IV only) to prevent mucositis
Differential diagnosis	Disc displacement without reduction Coronoid hyperplasia Joint ankylosis Synovial chondromatosis Myalgia Spasm	Myalgia Dystonia Arthralgia	Myoma (benign) Lipoma (benign) Rhabdosarcoma (malignant) Metastatic cancer (malignant)

CBC = complete blood count; CBT = cognitive behavioral therapy; CRP = C–reactive protein; EMG = electromyography; LA = local anesthetic; NSAIDs = nonsteroidal anti-inflammatory drugs; PCP = primary care physician; PSG = polysomnography; PSR = physical self-regulation; qd = every day; qhs = before bed; tid/qid = three times a day/four times a day; UT = urinary tract; ROM = range of motion.

TMJ DISORDERS

	Arthralgia (M26.62)	Arthritis (M26.62)	DDwR (M26.63)	DDwRwIL (M26.63)	Disc displacement without reduction with limited opening (M26.63)	Disc displacement without reduction without limited opening (M26.63)
Clinical characteristics: ▪ Self-limiting ▪ Rarely disabling ▪ Multifactorial: no single cause ▪ Pain-free noises do not need treatment ▪ Surgery only after reasonable non-surgical treatment fails and quality of life is reduced ▪ Radiographic changes should not be used as sole basis for treatment decision ▪ Arthralgia or arthritis may accompany each TMJD, but not all the time	Pain of joint origin affected by jaw movement Pain in last 30 d in jaw, temple, ear, or inside the ear Clinical exam must confirm familiar pain in the joint with at least one: ▪ Lateral pole (0.5-kg pressure) or around the lateral pole (1.0-kg pressure) ▪ Jaw opening and/or excursions	Pain of joint origin affected by jaw movement: synovitis and/or capsulitis Pain in last 30 d in jaw, temple, ear, or inside the ear No history of inflammatory or other causative systemic or local disease Clinical exam must confirm arthralgia plus one: ▪ Swelling, redness, elevated temperature ▪ Occlusal change due to inflammation may be present NOTE: Degenerative joint disease, sometimes called arthrosis or osteoarthrosis, may or may not be accompanied by arthritis	In the closed mouth position, the disc is anterior, medial, or lateral to the condyle center Correctly positions (reduces) on opening and/or in protrusion Clicking, snapping, popping during last 30 d and occurs on reduction during at least 1 of 3 opening cycles and/or excursive movements Reciprocal click present when joint closes Deviates to affected side May not be appreciated clinically; many quiet "normal" joints have DDwR	In the closed mouth position, the disc is anterior, medial, or lateral to the condyle center Occasionally, reduction does not occur, and ROM is reduced; maneuver is necessary to reduce Clicking, snapping, popping in last 30 d and occurs on reduction during at least 1 of 3 opening cycles and/or excursive movements Reciprocal click may be present when joint closes Report of locking in last 30 d Deviates to affected side	In the closed mouth position, the disc is anterior, medial, or lateral to the condyle center No reduction Limited ROM that cannot be increased by maneuver Closed lock Interferes with ability to eat Maximum assisted opening < 40 mm Deflects (uncorrected deviation) to affected side	In the closed mouth position, the disc is anterior, medial, or lateral to the condyle center No reduction Not associated with limited ROM Maximum assisted opening > 40 mm
Tests	CBCT NOTE: This diagnosis is descriptive based on clinical pain. Imaging will only assist in ruling out pathology or degenerative changes	CBCT to rule out degenerative joint disease or osteonecrosis MRI to rule out soft tissue pathology CBC with differential diagnosis, arthritis panel, C-reactive protein, antinuclear antibodies	MRI for confirmation: ▪ Maximum intercuspation: posterior band is anterior to 11:30 position ▪ Maximum opening: intermediate zone of disc is between condyle and articular eminence	MRI for confirmation: ▪ Maximum intercuspation: posterior band is anterior to 11:30 position ▪ Maximum opening: intermediate zone of disc is between condyle and articular eminence	MRI for confirmation: ▪ Maximum intercuspation: posterior band is anterior to 11:30 position ▪ Maximum opening: intermediate zone of disc anterior to the condyle	MRI for confirmation: ▪ Maximum intercuspation: posterior band is anterior to 11:30 position ▪ Maximum opening: intermediate zone of disc anterior to the condyle

TMJ DISORDERS

	Arthralgia (M26.62)	Arthritis (M26.62)	DDwR (M26.63)	DDwR with reduction with intermittent lock (M26.63)	Disc displacement without reduction with limited opening (M26.63)	Disc displacement without reduction without limited opening (M26.63)
Treatment	Patient education and awareness training Self-care: restrict function to within pain-free limits and minimize tooth contact ("lips together, teeth apart"); soft diet; stress reduction; avoid overuse Moist heat/ice Stabilization appliance (part-time wear)	Patient education and awareness training Self-care: restrict function to within pain-free limits and minimize tooth contact ("lips together, teeth apart"); soft diet; stress reduction; avoid overuse Moist heat/ice Anterior repositioning appliance, then stabilization appliance	Patient education and awareness training	Patient education and awareness training Train patient how to self-reduce Self-care: restrict function to within pain-free limits and minimize tooth contact ("lips together, teeth apart"); soft diet; stress reduction; avoid overuse Moist heat Avoid wide opening Stabilization appliance Arthrocentesis when there is persistent nonresponsive pain	Patient education and awareness training Self-care: Restrict function to within pain-free limits and minimize tooth contact ("lips together, teeth apart"); soft diet; stress reduction; avoid overuse; range of motion may improve over 3–4 mo with self-care alone If acute, attempt to manually increase range of motion by manipulation under local or IV sedation Moist heat Anterior repositioning appliance with conversion to stabilization appliance as ROM improves, if possible to capture impressions Available evidence also supports stabilization appliance Consider referral for physical therapy Arthrocentesis when there is persistent nonresponsive pain—may improve mouth opening	Patient education and awareness training Self-care: restrict function to within pain-free limits and minimize tooth contact ("lips together, teeth apart"); soft diet; stress reduction; avoid overuse Moist heat Stabilization appliance Arthrocentesis when there is persistent nonresponsive pain
Medications	Analgesics: ▪ Ibuprofen ▪ Naproxen sodium ▪ Meloxicam Glucosamine chondroitin	Analgesics: ▪ Ibuprofen ▪ Naproxen sodium Steroids: ▪ Methylprednisolone (short-term therapy) ▪ Dexamethasone injection: 4 mg/mL over joint ▪ Glucosamine chondroitin	**Analgesics and NSAIDs** 1. Acetaminophen 350–500 mg combined with ibuprofen 200 mg. Synergistic effect 2. Acetaminophen 500–1,000 qid < 4,000 g/d (*Risk: liver toxicity*) 3. Ibuprofen 400–800 tid-qid < 2,400 mg/d for 14 d; *BB: cardio, GI* 4. Naproxen sodium 220–550 mg bid < 1,375 mg/d; *BB: cardio (less likely), GI* 5. Diclofenac (Voltaren) is available in a gel that is applied topically over inflamed joints or muscle			
Differential diagnosis	Myofascial pain Lyme disease Arthritis Osteochondritis dissecans Degenerative joint disease Lupus erythematosus Systemic arthritides Eagle's osteonecrosis Synovial chondritis	Osteochondritis dissecans MFP Systemic arthritides Degenerative joint disease Trauma Lyme disease Synovial chondritis	DDwRwIL Arthralgia DDw/oR Lupus erythematosus DJD	Disc displacement without reduction with locking Arthralgia Disc displacement without reduction without locking. Synovial chondritis Degenerative joint disease Lupus erythematosus	Disc displacement without reduction without locking Arthralgia DDwRwIL Synovial chondritis Degenerative joint disease Lupus erythematosus	Disc displacement without reduction with locking Arthralgia Degenerative joint disease Synovial chondritis Lupus erythematosus

TMJ DISORDERS						
	Fibrous ankylosis (M26.61)	Bony ankylosis (M26.61)	Adhesions (M26.61)	Subluxation (S03.0XXA)	Luxation (open lock) (S03.0XXA)	Degenerative joint disease (M19.91)
Clinical characteristics	Fibrous response to trauma, especially bleeding in the joint Progressive loss of ROM Deflection (uncorrected deviation) to the affected side Limited lateral movement to the contralateral side Hard end-feel	Bony response to trauma, especially bleeding in the joint Progressive loss of ROM Severely limited or absence of jaw mobility in all movements Hard end-feel	Occur mainly in superior compartment Cause decreased movement and restriction Crepitus may be present May occur as a result of direct trauma, microtrauma, or polyarthritic disease No history of TMJ clicking Limited range of motion Deflection (uncorrected deviation) to the affected side Limited lateral movement to the contralateral side	In the open mouth position, the disc/condyle is anterior to the eminence Patient maneuver is necessary to reduce Report of locking in open position, even briefly, in last 30 d Report of inability to close from wide open without a maneuver Does not require exam confirmation	In the open mouth position, the disc/condyle is anterior to the eminence Clinician maneuver is necessary to reduce Report of locking in open position, even briefly, in last 30 d Report of inability to close from wide open without a maneuver by a clinician Exam findings: ▪ Wide open mouth ▪ Protruded jaw ▪ Jaw laterally positioned toward contralateral side	Also referred to as arthrosis or osteoarthrosis Deterioration and abrasion of articular tissue and remodeling of subchondral bone Is not painful but may be accompanied by the diagnoses of arthralgia and arthritis Loss of cartilage due to imbalance of chondrocyte activity Must have: ▪ Any joint noise with jaw function ▪ Patient report of any noise during exam ▪ Crepitus during at least one movement of jaw in ROM exam
Tests	CBCT: ▪ Decreased ipsilateral translation ▪ Joint space present between condyle and eminence	CBCT: ▪ Bone proliferation in the joint ▪ No joint space present between condyle and eminence	For definitive confirmation: MRI Arthroscopy		CBCT or MRI for confirmation: ▪ Condyle is anterior to the eminence with patient trying to close the mouth	CBCT will demonstrate at least one: ▪ Subchondral cyst ▪ Erosion of cortical bone ▪ Generalized sclerosis ▪ Osteophyte formation Flattening/cortical sclerosis not necessarily diagnostic of degenerative joint disease, but may be a sequela Tc-99m scan: evaluate activity

TMJ DISORDERS						
	Fibrous ankylosis (M26.61)	Bony ankylosis (M26.61)	Adhesions (M26.61)	Subluxation (S03.0XXA)	Luxation (open lock) (S03.0XXA)	Degenerative joint disease (M19.91)
Treatment	Patient education and awareness training Self-care: restrict function to within pain-free limits and minimize tooth contact ("lips together, teeth apart"); soft diet; stress reduction; avoid overuse Moist heat Stabilization appliance after surgery Physical therapy Arthroscopic surgery	Patient education and awareness training Self-care: restrict function to within pain-free limits and minimize tooth contact ("lips together, teeth apart"); soft diet; stress reduction; avoid overuse Moist heat Joint reshaping or may need replacement surgery Physical therapy Stabilization appliance after surgery	Patient education and awareness training Self-care: restrict function to within pain-free limits and minimize tooth contact ("lips together, teeth apart"); soft diet; stress reduction; avoid overuse Stabilization appliance Physical therapy Arthrocentesis Arthroscopic surgery	Patient education and awareness training Train patient how to self-reduce Self-care: restrict function to within pain-free limits and minimize tooth contact ("lips together, teeth apart"); soft diet; stress reduction; avoid overuse Moist heat Avoid wide opening	Patient education and awareness training Train patient how to self-reduce Self-care: restrict function to within pain-free limits and minimize tooth contact ("lips together, teeth apart"); soft diet; stress reduction; avoid overuse Moist heat Avoid wide opening	Patient education and awareness training Self-care: restrict function to within pain-free limits and minimize tooth contact ("lips together, teeth apart"); soft diet; stress reduction; avoid overuse Moist heat Stabilization appliance Arthrocentesis—added benefit of additional medication in lavage (steroids, hyaluronic acid) not established
Medications	Glucosamine chondroitin		Glucosamine chondroitin	Eminectomy Lateral pterygoid injection of botulinum toxin Injection of fibrosing substance into joint space (eg, prolotherapy) Surgical release of lateral pterygoid attachment on articular disc NSAIDs/analgesics if pain on maneuver: ▪ Ibuprofen ▪ Naproxen sodium	Eminectomy Lateral pterygoid injection of botulinum toxin Injection of fibrosing substance into joint space (eg, prolotherapy) Surgical release of lateral pterygoid attachment on articular disc NSAIDs/analgesics if pain on maneuver: ▪ Ibuprofen ▪ Naproxen sodium	NSAIDs/analgesics Glucosamine chondroitin
Differential diagnosis	Bony ankylosis Arthralgia Disc displacement without reduction with locking Synovial chondritis Degenerative joint disease	Fibrous ankylosis Arthralgia Disc displacement without reduction with locking Synovial chondritis Degenerative joint disease	Disc displacement without reduction with locking Arthralgia Fibrous ankylosis Degenerative joint disease Synovial chondritis	Luxation Disc displacement without reduction with locking Arthralgia Degenerative joint disease	Disc displacement without reduction without locking Arthralgia DDwRwIL Synovial chondritis Degenerative joint disease	Arthralgia Adhesions Arthritis Lupus erythematosus Osteochondritis dissecans Synovial chondritis

TMJ DISORDERS						
	Condylysis/idiopathic condylar degeneration (M26.69)	Osteochondritis dissecans (M93.20)	Osteonecrosis (M87.08)	Systemic arthritides (M06.9)	(TMJ) Benign (D16.5) Malignant (C41.1)	Synovial chondromatosis (D48.0)
Clinical characteristics	Idiopathic degeneration Causes loss of condylar height and progressive anterior open bite Spontaneous Mainly bilateral More common in adolescent and young adult females Low estrogen levels implicated May or may not have joint noises or pain Possibly severe form of degenerative joint disease Exam findings: ▪ Anterior open bite ▪ Evidence of progressive occlusal changes (facets that cannot be approximated by change in measurements of overbite and overjet)	Cartilage or bone fragment breaks loose and results in "loose body" within the TMJ Must have: ▪ Arthralgia ▪ Joint noises with movement or swelling ▪ Crepitus during exam or report ▪ Maximum assisted opening < 40 mm ▪ Swelling around affected joint	Painful condition of the ends of long bones Condyle is possible site Cause unknown Must have: ▪ Arthralgia ▪ Decreased signal on MRI T1 and increased T2	Inflammation with pain or structural changes caused by systemic inflammatory disease Includes: ▪ Rheumatoid arthritis ▪ Juvenile idiopathic arthritis ▪ Ankylosing spondylitis ▪ Psoriatic arthritis ▪ Infectious arthritis ▪ Reiter syndrome ▪ Gout ▪ Chondrocalcinosis Must have: ▪ Rheumatologic diagnosis ▪ TMJ pain or noises in past month or TMJ pain that worsens with episodes of systemic disease ▪ Arthritis or crepitus	New, uncontrolled growth of abnormal tissue 3% of malignancy metastasizes to the jaw; most common: ▪ Maxillofacial SCCa and nasopharyngeal tumors Adenocystic carcinomas and mucoepidermoid carcinomas may refer pain to TMJ Common symptoms: ▪ Reduced opening ▪ Crepitation ▪ Occlusal change ▪ Pain with function ▪ Swelling ▪ Midline shift	Cartilage metaplasia Cartilaginous nodules detached from synovial membrane that calcify Changes in occlusion Must have: ▪ Report of preauricular swelling ▪ Arthralgia ▪ Crepitus ▪ Degenerative joint disease ▪ Maximum assisted ▪ opening < 40 mm
Tests	Serial CBCT (yearly): Changes in sequential imaging must be documented Tc-99m scan: Evaluate disease activity CBC with differential, arthritis panel (must be negative for systemic disease)	CBCT: loose bodies present	MRI with T1/T2 CBC with differential, arthritis panel, C-reactive protein	CBCT will demonstrate at least one: ▪ Subchondral cyst ▪ Erosion of cortical bone ▪ Generalized sclerosis ▪ Osteophyte formation CBC with differential, arthritis panel, C-reactive protein Tc-99m scan: evaluate stability	CBCT and MRI	MRI or CBCT observations at least one: ▪ MRI: multiple chondroid nodules, joint effusion, and/or amorphous isointensity signals in joint space and capsule ▪ CBCT: loose bodies in soft tissues of the TMJ Biopsy: ▪ Cartilaginous metaplasia

TMJ DISORDERS						
	Condylysis/idiopathic condylar degeneration (M26.69)	Osteochondritis dissecans (M93.20)	Osteonecrosis (M87.08)	Systemic arthritides (M06.9)	(TMJ) Benign (D16.5) Malignant (C41.1)	Synovial chondromatosis (D48.0)
Treatment	Patient education and awareness training Self-care: restrict function to within pain-free limits and minimize tooth contact ("lips together, teeth apart"); soft diet; stress reduction; avoid overuse Moist heat Stabilization appliance (dual arch may be necessary to allow for anterior tongue space due to thickness) Arthrocentesis can relieve pain	Patient education and awareness training Self-care: restrict function to within pain-free limits and minimize tooth contact ("lips together, teeth apart"); soft diet; stress reduction; avoid overuse Moist heat/ice Anterior repositioning appliance, then stabilization appliance Arthroscopy	Patient education and awareness training Self-care: restrict function to within pain-free limits and minimize tooth contact ("lips together, teeth apart"); soft diet; stress reduction; avoid overuse Stabilization appliance Specific treatment unknown Based on experience with avascular necrosis in long bones, conservative management recommended	Patient education and awareness training Self-care: restrict function to within pain-free limits and minimize tooth contact ("lips together, teeth apart"); soft diet; stress reduction; avoid overuse Moist heat Stabilization appliance Consider: arthroscopic lavage with steroid, joint replacement surgery	Patient education and awareness training Self-care: restrict function to within pain-free limits and minimize tooth contact ("lips together, teeth apart"); soft diet; stress reduction; avoid overuse Moist heat Surgery	Patient education and awareness training Self-care: restrict function to within pain-free limits and minimize tooth contact ("lips together, teeth apart"); soft diet; stress reduction; avoid overuse Moist heat Stabilization appliance Arthroscopy
Medications	Glucosamine chondroitin Analgesics	Analgesics Steroids: ▪ Methylprednisolone (Medrol) ▪ Dexamethasone injection: 4 mg/mL over joint	Glucosamine chondroitin Analgesics	Analgesics Corticosteroids with PCP consultation	Palliative posttreatment: Use WHO ladder for the management of cancer pain: ▪ Opiates/opioids ▪ Gabapentin 100 mg qd + 100 mg/d < 1,800 mg/d	Analgesics
Differential diagnosis	Skeletal malocclusion Degenerative joint disease Arthralgia Systemic arthritides Lyme disease	Arthralgia Synovial chondritis Arthritis Systemic arthritides Degenerative joint disease	Arthralgia Systemic arthritides Arthritis Degenerative joint disease Osteochondritis dissecans	Arthralgia Adhesions Arthritis Osteochondritis dissecans Synovial chondritis	Arthralgia Myofascial pain Arthritis Osteochondritis dissecans Synovial chondritis Degenerative joint disease Lupus erythematosus	Arthralgia Fibrous ankylosis Arthritis Adhesions Lupus erythematosus

TMJ DISORDERS

	Fracture	Aplasia (Q67.4)	Hypoplasia (M27.8)	Hyperplasia (M27.8)	Coronoid hyperplasia (M27.8)	TMD headache (G44.89)
Clinical characteristics	Types: ▪ Closed fracture of condylar process (S02.61XA) ▪ Closed fracture most usually of subcondylar process (S02.62XA) ▪ Open fracture of condylar process (S02.61XB) ▪ Open fracture of subcondylar process (S02.62XB) Sequelae: ▪ Adhesions ▪ Ankylosis ▪ Occlusal abnormalities ▪ Joint degeneration ▪ Facial asymmetry Must have: ▪ Macrotrauma ▪ Arthralgia ▪ Preauricular swelling ▪ Maximum assisted ▪ opening < 40 mm	Unilateral absence of condyle and incomplete development of articular fossa leads to facial asymmetry Commonly associated with congenital anomalies: Goldenhar syndrome, Treacher Collins syndrome Must have: ▪ Progressive development of mandibular asymmetry or micrognathia from birth or early childhood ▪ Development of malocclusion (may include posterior open bite) ▪ Confirmation of deviated chin to affected side. ▪ Condyle cannot be palpated during movement	Incomplete development or underdevelopment of the cranial bones or mandible Growth is proportionately reduced and less severe than aplasia Can be secondary to facial trauma Must have: ▪ Progressive development of mandibular asymmetry or micrognathia from birth or early childhood ▪ Development of malocclusion (may include posterior open bite)	Overdevelopment of the mandible or condylar process Attributed to nonneoplastic increase in the number of normal cells Must have progressive development of mandibular or facial asymmetry	Progressive enlargement of the coronoid process that impedes mandibular opening Nonneoplastic increase in the number of normal cells Must have: ▪ Complaint of progressive limitation of jaw opening ▪ Reduced active and passive jaw opening ▪ Hard end-feel	Must have at least two: ▪ Headache started with onset of TMD ▪ Headache worsens as TMD worsens or resolves when TMD symptoms lessen ▪ Headache produced or exacerbated with jaw movement or on palpation ▪ Headache is on the same side as the TMD Must have both: ▪ Headache of any type in the temple region during past 30 d modified by jaw movement ▪ Palpated temporalis pain with familiar headache during jaw movements
Tests	CBCT	CBCT or panoramic imaging: ▪ Severe hypoplasia of fossa and eminence ▪ Aplasia of the condyle	CBCT or panoramic imaging: ▪ Hypoplasia of fossa ▪ Hypoplasia of condyle ▪ Shortened mandibular ramus height	CBCT or panoramic imaging: ▪ Asymmetry in ramus height ▪ Tc-99m scan: increased uptake	CBCT: must show elongated coronoid process approximating zygoma on opening	Panoramic radiograph MRI of brain if headaches persist beyond reduction of TMD symptoms
Treatment	Patient education and awareness training Self-care: restrict function to within pain-free limits and minimize tooth contact ("lips together, teeth apart"); soft diet; stress reduction; avoid overuse Moist heat Stabilization appliance after reduction Physical therapy Usually does not require surgery	Patient education and awareness training Physical therapy Joint replacement surgery Stabilization appliance after surgery appliance	Patient education and awareness training Stabilization appliance after growth has stabilized Manage occlusion	Patient education and awareness training	Patient education and awareness training Coronectomy Physical therapy	Patient education and awareness training Self-care: restrict function to within pain-free limits and minimize tooth contact ("lips together, teeth apart"); soft diet; stress reduction; avoid overuse Moist heat Stabilization appliance

TMJ DISORDERS						
	Fracture	Aplasia (Q67.4)	Hypoplasia (M27.8)	Hyperplasia (M27.8)	Coronoid hyperplasia (M27.8)	TMD headache (G44.89)
Medications	NSAIDs/analgesics Diazepam 2–10 mg tid-qid; *BB: Opioids = sedation, death* Cyclobenzaprine 5–10 mg tid for 3 wk (Risk: elderly, cardio, opioids)		Glucosamine chondroitin			Analgesics/headache medications Amitriptyline 10–35 mg qhs (*Risk: cardio, diabetics, seizure, UT disorders*); *BB: suicide, < 25 y* Duloxetine 60 mg qd; *BB: suicide*
Differential diagnosis	Disc dislocations Hemarthrosis Contusion Laceration of joint parts	Hypoplasia	Aplasia Condylysis	Acromegaly	Fibrous ankylosis	Tension-type headache Migraine Myofascial pain Fibromyalgia Cervicogenic headache

Bid = twice a day; CBC = complete blood count; DDwR = disc displacement with reduction; DDwRwIL = DDwR with intermittent lock; GI = gastrointestinal; PCP = primary care practitioner; qd = every day; qhs = before bed; ROM = range of motion; SCCa = squamous cell carcinoma; Tc-99m scan = technetium-99m scan; tid-qid = three/four times a day; UT = urinary tract.

COMMENTARY: TMJ DISORDERS

- The commonly used term *TMD* is not an adequate diagnosis; it is a classification. In fact, there are over 30 identified diagnoses within this umbrella term. Treatment cannot be adequately directed toward the term *TMD*.
- TMJ disorders include pathology or injury of bone, cartilage, and/or contiguous tissues. They can be acute or chronic, and most TMJ disorders (painful and nonpainful) are self-limiting.
- Evidence suggests that parafunctional habits can cause acute joint pain. There is also a large body of data indicating joint overload as one of the possible causative factors in joint disease. Overload may be absolute or relative. Absolute overload is due to macrotrauma or possibly due to the microtrauma caused by chronic clenching. Relative overload refers to a compromised host—there is evidence for extra-articular risk factors, such as cardiovascular disease, obesity, and nutrition. In these cases, normal loads may lead to joint disease over time. Treatment should be conservative and directed toward management of a specific diagnosis with identified outcomes. Treatment without data supporting anticipated improvement in signs and symptoms should be avoided.
- Current evidence for management of TMJ disorders overwhelmingly supports conservative care principles based on a properly performed simple clinical examination. Sophisticated diagnostic technologies to determine optimal joint positioning and occlusal stability have very low to no supporting scientific evidence.
- Diagnostic imaging should be prescribed when the anticipated findings are expected to change the outcome of the clinical examination findings or when confirmation of a diagnosis is needed. For example, early MRI of most TMJ disorders is not indicated because the likelihood that the diagnosis will differ from the clinical findings is low; however, CBCT imaging can provide valuable information about the current condition of the condyles that cannot be assessed by clinical examination. MRI may be indicated in the management of complex, recalcitrant internal derangements or suspected soft tissue pathology. Scintigraphy is used to assess activity at the TMJ before the age of ~18 years; a positive result indicates inflammation, increased metabolic activity, or pathology (eg, tumor).
- Malocclusion does not cause joint disease; however, joint disease may induce occlusal change, particularly an anterior or a contralateral open bite. This must, however, be documented and proven by comparison against a baseline image to establish causation. Simply put bad bites do not cause bad joints, but bad joints may cause bad bites.
- Treatment of occlusal and skeletal relationships is not supported as a primary therapy for orofacial pain conditions, including TMJ disorders, headaches, and neuropathy.
- Restoring teeth may be appropriate to maintain oral health. Parafunctional habits causing damage to oral structures should be attended to accordingly, eg, occlusal splints to protect teeth in bruxism.
- *Arthralgia* is a descriptive term for joint pain. Arthritis must have a diagnosis of arthralgia and signs of inflammation (rubor, calor, and dolor) with or without effusion. Degenerative joint disease on its own is not pain of joint origin and often exhibits crepitus on clinical examination; it may be accompanied by arthralgia and/or arthritis.
- Vasoconstrictors like epinephrine and norepinephrine must be avoided when giving intramuscular injections such as trigger point injections.

NECK PAIN

	Cervicalgia (M54.2)	Sprain and strain of cervical spine (S13.4)	Cervical osteoarthritis (M47.8)	Radiculopathy (M54.1)
Clinical characteristics Normal ROM: ▪ Rotation: 65–75 degrees ▪ Tilt: 35–45 degrees ▪ Flexion: 60–70 degrees ▪ Extension: 50–60 degrees	Pain in the neck Primary sites of pain: suboccipital area, SCM, and upper trapezius Referral to: frontal, temporoparietal, occipital, vertex, and orbital regions	Whiplash-associated disorder Graded due to function: I. Neck symptoms with minor limits to daily life II. Neck symptoms with substantial limits to daily life III. Neurologic signs IV. Major structural pathology May have signs/symptoms of TMD, but part of widespread pain disorder Onset immediate or up to 2 days Symptoms: ▪ Referred pain ▪ Headache ▪ Dizziness ▪ Tinnitus ▪ Dysphagia ▪ Visual disturbance Most recover in 3 mo, but some never recover	Age-related Inflammation of joint linings with osteophyte formation and exostoses C5-C6 and C6-C7 most common sites Age > 50 y, 75% display signs/symptoms of OA: ▪ Early: episodes of neck pain triggered by activity that resolve with rest ▪ Advanced: stiffness, limited ROM, crepitus, chronic neck pain Degenerative changes may not be painful	Pain and/or sensorimotor deficit caused by compression of a nerve root Potential causes: disc herniation, spondylosis, instability of the joint, trauma, or tumor C1-C3 can refer as: eye and/or ear pain, suboccipital or occipital headache, neck pain, or shoulder pain
Tests Tests for cervical cause of pain: ▪ Spurling test (passive tilt to painful side and then 7-kg vertical pressure to top of head): Does this reproduce symptoms? ▪ Neck distraction (head pulled up vertically with 14-kg pressure): Are the symptoms improved? ▪ Valsalva maneuver: Are the symptoms reproduced? ▪ Palpation of cervical muscles	CBCT, CT, or MRI	CT and/or MRI	CT and/or MRI	CT and/or MRI
Treatment	Patient education and awareness training Self-care: restrict function to within pain-free limits Moist heat Physical therapy	Patient education and awareness training I and II: rest, relative immobilization for 3–6 wk, and then PT if not resolved Cervical collar no longer recommended If not resolved in 6–12 wk or III and IV, refer to interdisciplinary team	Patient education and awareness training Mild-moderate: physical therapy When neural compression and radiculopathy are present, a neurologist or orthopedic specialist should be consulted	Patient education and awareness training Refer to neurologist Physical therapy Cervical collar not recommended
Medications	NSAIDs Muscle relaxants Glucosamine chondroitin	NSAIDs/corticosteroids Muscle relaxants	Glucosamine chondroitin	
Differential diagnosis	Myofascial pain Myalgia Tension-type headache	Spinal cord injuries Brain injury	Radiculopathy Whiplash	Cervical osteoarthritis Whiplash Lyme disease Lupus erythematosus

NECK PAIN

	Cervicalgia (M54.2)	Sprain and strain of cervical spine (S13.4)	Cervical osteoarthritis (M47.8)	Radiculopathy (M54.1)
Clinical characteristics Normal ROM: ▪ Rotation: 65–75 degrees ▪ Tilt: 35–45 degrees ▪ Flexion: 60–70 degrees ▪ Extension: 50–60 degrees	Spasmodic torticollis Sustained contraction of the neck and shoulder muscles May be spasmodic (clonic) or permanent (tonic) Bilateral SCM involvement: head in an extended position (retrocollis) and is associated with vocal and swallowing disturbances Can be idiopathic or secondary to disease, medications, or poisoning (eg, carbon monoxide) 75% of patients complain of neck pain (not consistent with other dystonias)	Classified with "painful lesions of the cranial nerves and other facial pain" (ICHD-3) Paroxysms of sharp, shooting pain that last seconds to minutes Dysesthesia and/or allodynia and tenderness of occipital nerve	Inflammation of the stylohyoid ligament Primary sites of pain: ▪ Oropharynx ▪ Neck ▪ Face Diffuse headache may be present Pain provoked by: ▪ Turning the head ▪ Digital pressure on neck over appropriate area	Headache caused by a disorder of the cervical spine and its component bony, disc, and/or soft tissue elements; usually accompanied by neck pain Must have at least three: ▪ Headache developed in temporal relation to onset of cervical disorder or lesion ▪ Headache resolves with improvement of cervical disorder ▪ Cervical ROM reduced and headache made worse with maneuvers ▪ Headache abolished by local anesthetic blockade of cervical structure ▪ Includes neck-tongue syndrome ▪ Side-locked pain that radiates forward ▪ Headache provoked by neck palpation
Tests Tests for cervical cause of pain: ▪ Spurling test (passive tilt to painful side and then 7-kg vertical pressure to top of head): Does this reproduce symptoms? ▪ Neck distraction (head pulled up vertically with 14-kg pressure): Are the symptoms improved? ▪ Valsalva maneuver: Are the symptoms reproduced? ▪ Palpation of cervical muscles		Panoramic radiograph MRI of brain if headaches persist beyond reduction of TMD symptoms	Panoramic or CBCT: elongated stylohyoid ligament	CT and/or MRI
Treatment	Patient education and awareness training PSR CBT	Patient education and awareness training Self-care: restrict function to within pain-free limits, improve posture Moist heat Physical therapy Occipital nerve blocks—may include dexamethasone 4 mg/mL or triamcinolone 10 mg/mL	Local injection of anesthetic Styloidectomy	Patient education and awareness training Physical therapy Injections of local anesthetics/steroids
Medications	Botulinum toxin Diphenhydramine 25–50 mg qid Gabapentin 100 mg qd + 100 mg/d, < 1,800 mg/d Diazepam 2–10 mg tid-qid; *BB: opioids = sedation, death* Topiramate 25 mg + 25 mg every 2 wk < 100–400 mg/d	Gabapentin 100 mg qd + 100 mg/d, < 1,800 mg/d Amitriptyline 10–35 mg qhs (*Risk: cardio, diabetics, seizure, UT disorders*; *BB: suicide, < 25 y*) LLLT	NSAIDs	NSAIDs/corticosteroids Gabapentin 100 mg qd + 100 mg/d, < 1,800 mg/d Amitriptyline 10–35 mg qhs *(Risk: cardio, diabetes, seizure, UT disorders*; *BB: suicide, < 25 y)* Muscle relaxants

NECK PAIN				
	Cervicalgia (M54.2)	Sprain and strain of cervical spine (S13.4)	Cervical osteoarthritis (M47.8)	Radiculopathy (M54.1)
Differential diagnosis	Oromandibular dystonia Spasm	Migraine Cervicogenic headache Cluster headache Hemicrania continua/paroxysmal hemicrania. Lupus erythematosus Tension-type headache Giant cell arteritis of occipital artery	Carotidynia Tension-type headache Migraine Neuralgia	Migraine Tension-type headache Vertebral artery syndrome Lupus erythematosus

BB = FDA Black Box warning; CBT = cognitive behavioral therapy; LLLT = low-level laser therapy; NSAIDs = nonsteroidal anti-inflammatory drugs; PSR = physical self-regulation; qd = every day; qhs = before bed; tid/qid = three/four times a day; ROM = range of motion; SCM = sternocleidomastoid.

SYSTEMIC DISEASE–RELATED PAIN

	Multiple sclerosis	Lyme disease	Systemic lupus erythematosus	Sjögren syndrome	Systemic sclerosis	Giant cell arteritis
Clinical characteristics	Autoimmune disease Demyelinating lesions and plaques within the CNS Multifactorial cause: genetic predisposition + vitamin deficiency, infectious agents, and smoking Onset age 30–40 y (trigeminal neuralgia onset 50–70 y) 20 times greater chance of trigeminal neuralgia than general population: ▪ Lesions within the pons and root entry zone ▪ 31% of cases are bilateral	Infection from tick bite: *Borrelia burgdorferi* Characteristic rash: erythema migrans History of outdoor activities in prone geographic regions Attacks three systems: ▪ Heart: conduction block ▪ Joints: arthralgia ▪ Nervous system: cranial neuropathy, lymphocytic meningitis, radiculopathy Facial nerve palsy (similar to Bell's) in early stage, can be bilateral May also cause diplopia, hypoesthesia, headaches, hearing loss, and/or vertigo Chronic fatigue and muscle aches can last for 6 mo or longer after treatment	Autoimmune disease Abnormal production of autoantibodies, multisystem inflammation, and vasculopathy Butterfly rash, oral ulcers, arthralgia may be present TMJ pain, locking, and crepitus may be present Trigeminal neuropathy may be initial presentation	Chronic inflammation of exocrine glands, primarily salivary and lacrimal Keratoconjunctivitis sicca and hyposalivation (< 0.1 mL/min) Trigeminal neuropathy with facial numbness and paresthesia TMD signs more common in Sjögren patients 78% have headaches, including migraines and tension-type headaches.	Abnormal fibrosis and dysfunction of the skin, vasculature, and organs Microstomia due to fibrosis-induced limited mouth opening TMJ arthralgia and arthritis, myalgia, headache, and limited ROM may be present Trigeminal neuralgia symptoms and trigeminal neuropathy may be present GCA may also be present	Temporal arteritis, occipital arteritis Granulomatous inflammation of a branch of the aorta Age > 50 y Associated with polymyalgia rheumatica Swollen, tender superficial temporal artery, new onset temporal headache, hip and shoulder pain Jaw or tongue claudication: aching cramp in the masseters, temporalis, or tongue after chewing Scalp tenderness Morning stiffness/soreness in the neck and shoulders Most serious risk: blindness
Tests	MRI with and without contrast through CP angle; vascular loop protocol CBC with differential and platelets, liver and kidney functions, sodium level (< 136 mEq/L), and HLAb*1502 genetic testing in Asian and Indian populations	CBC with differential, arthritis panel, ANA, enzyme immunoassay, then Western Blot if no response in 30 d CBCT	CBC with differential, arthritis panel, ANA, enzyme	CBC with differential, arthritis panel, CRP, ANA	Panoramic or CBCT: erosion of coronoid process, ramus, or condyle may be present	CBC with differential, ESR, CRP Temporal artery biopsy or high-resolution ultrasound ESR > 50 mm/h OR elevated CRP ≥ 10 mg/L
Treatment	Patient education and awareness training Antiepileptic medication Amitriptyline if constant pain Percutaneous balloon microdecompression (best), glycerol rhizotomy, thermocoagulation Gamma knife Trigeminal ganglion–level interventions (balloon, heat, glycerol)	Patient education and awareness training Self-care: restrict function to within pain-free limits and minimize tooth contact ("lips together, teeth apart"); soft diet; stress reduction; avoid overuse; moist heat Oral antibiotic therapy	Patient education and awareness training Manage arthralgia Manage neuropathy	Patient education and awareness training Palliative care Pilocarpine 5 mg qid	Patient education and awareness training Palliative care	Patient education and awareness training Immediate referral to ER due to risk of blindness

SYSTEMIC DISEASE–RELATED PAIN

	Multiple sclerosis	Lyme disease	Systemic lupus erythematosus	Sjögren syndrome	Systemic sclerosis	Giant cell arteritis
Medications	Carbamazepine 100 mg/d + 100 mg every 2 d, < 1,200 mg/d Oxcarbazepine 300 mg + 300–600 mg/d, < 2,400 mg/d Add or alone: Baclofen 5–15 mg + 5 mg every 3d, < 30–60 mg Pregabalin 150 mg + 50 mg every 2 d, < 300–600 mg/d Gabapentin 300 mg, titrate to 3/d at 3-day intervals; usual dosage 1,800–2,400 mg	Doxycycline 100 mg bid for 21 d Amoxicillin 500 mg tid for 21 d	Long-term prednisone	Pilocarpine Topical fluoride	Topical fluoride Physical therapy	Prednisolone 60–80 mg qd for 4–6 wk and then tapered gradually over 12–24 mo
Differential diagnosis	Classical/idiopathic trigeminal neuralgia	Bell's palsy Myofascial pain Lupus erythematosus Fibromyalgia Degenerative joint disease	Aphthous stomatitis Trigeminal neuralgia Lichen planus Neuropathy TMD	Systemic sclerosis Migraine Tension-type headache	Sjögren syndrome SCCa Migraine Trigeminal neuralgia Tension-type headache Neuropathy	Migraine Tension-type headache Trigeminal autonomic cephalgia Other primary headache

ANA = anti-nuclear antibodies; bid = twice a day; CBC = complete blood count; CNS = central nervous system; CP = cerebropontine; CRP = C-reactive protein; ESR = erythrocyte sedimentation rate; GCA = giant cell arteritis; tid = three times a day; SCCa = squamous cell carcinoma.

NEUROPATHIC PAIN

	Trigeminal neuralgia (G50.0)	Glossopharyngeal neuralgia (G52.1)	Nervus intermedius neuralgia (G51.9)	Painful posttraumatic trigeminal neuropathy (S04.30XA)	Painful trigeminal neuropathy attributed to herpes zoster	Trigeminal postherpetic neuralgia (G51.9)
Clinical characteristics	Paroxysmal, severe, shooting, electric-like pain that lasts for a few seconds followed by a refractory period; "worst pain ever"; sometimes aching in the affected zone starts several hours before attack (pre–trigeminal neuralgia); unilateral Classic, purely paroxysmal: at least three attacks, 1–120 s, innocuous stimuli, no neurologic deficit Classic with concomitant continuous pain: persistent pain of moderate intensity between attacks (previously known as atypical or type 2) Secondary (usually multiple sclerosis, arteriovenous malformation or tumor). Idiopathic, purely paroxysmal No evidence of neurovascular compression Idiopathic with concomitant continuous pain No evidence of neurovascular compression (Fig 2)	Glossopharyngeal distribution = posterior mandible, oropharynx, tonsillary fossa, and ear Severe, shooting, electric-like pain that lasts for a few seconds followed by a refractory period; "worst pain ever" Less tooth pain than trigeminal neuralgia; pain elicited by swallowing, chewing, or talking Also appears as: ▪ Secondary glossopharyngeal neuralgia ▪ Idiopathic glossopharyngeal neuralgia	Unilateral paroxysmal pain in depth of the ear lasting seconds or minutes Geniculate neuralgia Trigger zone in posterior wall of exterior auditory canal Taste, lacrimation, and salivation disorders may be present Ramsay Hunt Syndrome: secondary to herpes zoster infection; requires history of pain < 1 wk prior to blister formation in ear canal or mouth and facial palsy–like symptoms Also appears as: ▪ Secondary nervus ▪ Intermedius neuralgia ▪ Idiopathic nervus ▪ Intermedius neuralgia	Anesthesia dolorosa, "phantom" pain Following damage to CNV (eg, rhizotomy, surgical nerve injury, implant compression, etc) Decreased sensitivity to pain and temperature in one or more divisions Persistent pain in defined area for > 3 mo Dull, aching or burning, worsens with barometric change, prickly or itchy Maxillary anterior teeth most common	VZV Itching, numbness, tingling in specific dermatome followed by blisters and pain Unilateral facial pain in the distribution(s) of a trigeminal nerve branch or branches lasting < 3 mo Herpetic eruption in the same trigeminal distribution Most people heal within 3–4 wk	Unilateral pain recurring for > 3 mo associated with previous herpes zoster of the same trigeminal nerve branch or branches Pain developed in temporal relation to the herpes zoster infection Develops in 50%–75% of acute herpes zoster infections affecting > 1 branch of CN V lasting 3 mo or more Burning with superimposed brief, stabbing exacerbations of pain May be accompanied by hyperalgesia, allodynia, or sensory loss with anesthesia dolorosa Risk factors: female, older age, prodrome, severe rash, severe pain during infection
Tests	LA of trigger zone completely eliminates sharp pain and associated toothache but likely would not eliminate background pain MRI with or without contrast through CP angle; vascular loop protocol CBC with differential and platelets, urea/electrolytes, liver function, sodium level (< 136 mEq/L), and HLAb*1502 genetic testing in Asian and Indian populations; CBC, urea/electrolytes every 2–4 wk for 3 mo and then every 6 mo; and liver function every 6 wk for 2 normal intervals	Inferior alveolar block does not affect pain but may stop the trigger for the pain Topical anesthetic to the lateral pharyngeal wall may stop pain MRI with or without contrast through CP angle; vascular loop protocol CBC with differential and platelets, urea/electrolytes, liver function, sodium level (< 136 mEq/L), and HLAb*1502 genetic testing in Asian and Indian populations; CBC, urea/electrolytes every 2–4 wk for 3 mo and then every 6 mo; liver function every 6 wk for 2 normal intervals	MRI with or without contrast through CP angle; vascular loop protocol CBC with differential and platelets, urea/electrolytes, liver function, sodium level (< 136 mEq/L), and HLAb*1502 genetic testing in Asian and Indian populations; CBC, urea/electrolytes every 2–4 wk for 3 mo and then every 6 mo; liver function every 6 wk for 2 normal intervals	Percussion and vitality testing Radiograph CBCT Cold test to gingiva: exacerbates Sharp and light touch test Topical anesthetic with 20% benzocaine: no change LA infiltration: no change MRI with or without contrast through CP angle; vascular loop protocol CBC with differential, thyroid function, CRP, ANA, urine function, CMP, HbA1c	CSF tap: VZV has been detected by PCR Direct immunofluorescence assay for VZV antigen or PCR assay for VZV DNA is positive in cells obtained from the base of lesions	

NEUROPATHIC PAIN

	Trigeminal neuralgia (G50.0)	Glossopharyngeal neuralgia (G52.1)	Nervus intermedius neuralgia (G51.9)	Painful posttraumatic trigeminal neuropathy (S04.30XA)	Painful trigeminal neuropathy attributed to herpes zoster	Trigeminal postherpetic neuralgia (G51.9)
Treatment	Patient education and awareness training Antiepileptics Percutaneous microvascular decompression (best), glycerol rhizotomy, thermocoagulation Gamma knife Alcohol injections (short term)	Patient education and awareness training Referral to neurology Antiepileptics Microvascular decompression surgery, glycerol rhizotomy, or gamma knife surgery (the earlier, the better)	Patient education and awareness training Referral to ENT to rule out other causes of otalgia Antiepileptics Surgical resection of the nervus intermedius or chorda tympani	Patient education and awareness training Stress reduction techniques Surgery within 30 h to 3 mo of iatrogenic injury; remove implant within 24 h; IAN injury repair < 4 wk; lingual nerve repair < 3 mo Consider drug combination therapy: SNRI or TCA/GBP or PGB	Patient education and awareness training	Patient education and awareness training
Medications	Carbamazepine 100 mg/d + 100 mg every 2 d, < 1,200 mg/d Oxcarbazepine 300 mg + 300–600 mg/d, < 2,400 mg/d Add-on or alone: Baclofen 5–15 mg + 5 mg every 3 d, < 30–60 mg; similarly, lamotrigine as add-on Pregabalin 150 mg + 50 mg every 2 d, < 300–600 mg/d Gabapentin 300 mg, titrate to 3/d at 3-day intervals; usual dosage 1,800–2,400 mg	Carbamazepine 100 mg/d + 100 mg every 2 d, < 1,200 mg/d Oxcarbazepine 300 mg + 300–600 mg/d, < 2,400 mg/d Add-on or alone: Baclofen 5–15 mg + 5 mg every 3 d < 30–60 mg Pregabalin 150 mg + 50 mg every 2 d, < 300–600 mg/d Gabapentin 300 mg, titrate to 3/d at 3-day intervals; usual dosage 1,800–2,400 mg	Carbamazepine 100 mg/d + 100 mg every 2 d, < 1,200 mg/d Oxcarbazepine 300 mg + 300–600 mg/d, < 2,400 mg/d Add-on or alone: Baclofen 5–15 mg + 5 mg every 3 d < 30–60 mg Pregabalin 150 mg + 50 mg every 2 d, < 300–600 mg/d Gabapentin 300 mg, titrate to 3/d at 3-day intervals; usual dosage 1,800–2,400 mg	Time of injury: methylprednisolone (Medrol), then NSAIDs for 3 wk Amitriptyline 10–35 mg qhs *(Risk: Cardio, Diabetics, Seizure, UT disorders); BB: suicide, < 25 y* Duloxetine 60 mg qd; *BB: Suicide* Gabapentin 300 mg qd + 300 mg/d 1,800–2,400 mg/d in three daily doses Pregabalin 150 mg + 50 mg every 2 d, < 300–600 mg/d Lidocaine 5% topical 12 h on/off	Acyclovir 800 mg 5x/d for 7 d; *(Risk: kidney function)* Famciclovir 500 mg tid for 7 d *(Risk: kidney function)* Amitriptyline 10–35 mg qhs *(Risk: cardio, diabetes, seizure, urinary tract disorders); BB: suicide, < 25 y* Analgesics Avoid corticosteroids because they are immunosuppressive	Gabapentin 100 mg qd + 100 mg/d, < 1,800 mg/d Pregabalin 150 mg + 50 mg every 2 d, < 300–600 mg/d Amitriptyline 10–35 mg qhs *(Risk: cardio, diabetes, seizure, urinary tract disorders); BB: suicide, < 25 y* Lidocaine 5% topical 12 h on/off Capsaicin 8% patch for appropriate extraoral areas Other medications, including opioids, uncertain
Differential diagnosis	Paroxysmal hemicrania Multiple sclerosis Cluster headache Lupus	Paroxysmal hemicrania Cardiomyopathy Cluster headache Lupus Multiple sclerosis	Otitis media Trigeminal neuralgia/geniculate neuralgia Bell's palsy Multiple sclerosis Cluster headache Lupus	Pretrigeminal neuralgia Trigeminal neuralgia Multiple sclerosis Periodontal pain Migraine Lyme disease Lupus		Pretrigeminal neuralgia Hemicrania continua/paroxysmal continua Trigeminal neuralgia Lupus Multiple sclerosis Cluster headache Lyme disease

NEUROPATHIC PAIN				
	Painful neuropathy: multiple sclerosis	Central poststroke pain (G89.0)	Tolosa-Hunt syndrome (H51.9)	Complex regional pain syndrome (G90.50)
Clinical characteristics	Migraine-type headaches due to multiple sclerosis or treatment (interferons) Episodic or constant; constant more typical Common associated conditions: ▪ Optic neuritis ▪ Painful tonic spasms ▪ Presence of neurologic deficits in extremities ▪ Trigeminal neuralgia: age < 40 y, may be bilateral	Unilateral facial or head pain, dysesthesia, and impaired sensation to pinprick and temperature that occurs within 6 mo of a stroke, not due to a lesion of the trigeminal nerve Imaging must confirm stroke site is spinothalamic tract Not limited to craniofacial region, possibly entire half of the body Side contralateral to the lesion	Episodic orbital pain accompanied by paralysis of 1 or more cranial nerves III, IV, or VI Granulomatous inflammation of superior orbital fissure, cavernous sinus, or orbit Episodes last 8 wk if untreated	CRPS 1: Reflex sympathetic dystrophy (G90.59): ▪ After mild injury ▪ Disproportionate to the initiating event CRPS 2: Causalgia (G90.58.9), evidence of nerve injury preceding pain: ▪ Persistent, burning pain accompanied by allodynia and hyperalgesia, swelling, changes in blood flow, and/or abnormal sudomotor activity ▪ Not generally occurring in the head/neck; usually extremities ▪ Stress and stimulation increase pain: sympathetically maintained pain
Tests	LA of trigger zone completely eliminates pain and toothache MRI with or without contrast through CP angle; vascular loop protocol CBC with differential and platelets, urea/electrolytes, liver function, sodium level (< 136 mEq/L), and HLAb*1502 genetic testing in Asian and Indian populations; CBC, urea/electrolytes every 2–4 wk for 3 mo and then every 6 mo; liver function every 6 wk for 2 normal intervals	Confirm MRI evidence of stroke	MRI Biopsy	
Treatment	Patient education and awareness training Antiepileptics Percutaneous balloon microdecompression (best), glycerol rhizotomy, thermocoagulation, or gamma knife Alcohol injections (short term)	Patient education and awareness training Referral to neurology Deep brain and cortical stimulation may be helpful	Patient education and awareness training Referral to ophthalmologist	Patient education and awareness training Stress reduction techniques CBT Physical therapy Sympathetic blocks

NEUROPATHIC PAIN				
	Painful neuropathy: multiple sclerosis	Central poststroke pain (G89.0)	Tolosa-Hunt syndrome (H51.9)	Complex regional pain syndrome (G90.50)
Medications	Carbamazepine 100 mg/d, including 100 mg every 2 wk up to 1,200 mg/d Oxcarbazepine 150 mg, then to 300–600 mg/d, up to 2,400 mg/d Add-on or alone: Baclofen 5–15 mg + 5 mg every 3 d, < 30–60 mg Pregabalin 150 mg + 50 mg every 2 d, < 300–600 mg/d Topiramate 25 mg + 25 mg every 2 wk, < 100–400 mg/d	Amitriptyline 25–150 mg qhs *(Risk: cardio, diabetes, seizure, urinary tract disorders); BB: suicide, < 25 y* Lamotrigine 25 mg/d; *BB: serious rash, SJS* Gabapentin promising, but was not studied sufficiently in 2006 systematic review; carbamazepine was ineffective	Methylprednisolone (Medrol)	Carbamazepine 100 mg/d, including 100 mg every 2 wk up to 1,200 mg/d Oxcarbazepine 150 mg, then to 300–600 mg/d, up to 2,400 mg/d Add-on or alone: Baclofen 5–15 mg + 5 mg every 3 d, < 30–60 mg Pregabalin 150 mg + 50 mg every 2 d, < 300–600 mg/d Topiramate 25 mg + 25 mg every 2 wk < 100–400 mg/d
Differential diagnosis	Trigeminal neuralgia Paroxysmal hemicrania Cluster headache Lupus erythematosus	Cardio Multiple sclerosis Lyme disease	Vasculitis Giant cell arteritis Opthalmoplegic migraine	

ANA = anti-nuclear antibodies; CBC = complete blood count; CBT = cognitive behavioral therapy; CMP = comprehensive metabolic panel; CP = cerebropontine; CSF = cerebrospinal fluid; CRP = C-reactive protein; ENT = ear, nose, throat; GBP = gabapentin; GCA = giant cell arteritis; HbA1c = hemoglobin A1c; IAN = infraorbital nerve; LA = local anesthetic; PGB = pregabalin; SJS = Stevens-Johnson syndrome; SNRI = serotonin noradrenaline reuptake inhibitor; TCA = tricyclic antidepressant; VZV = varicella zoster virus.

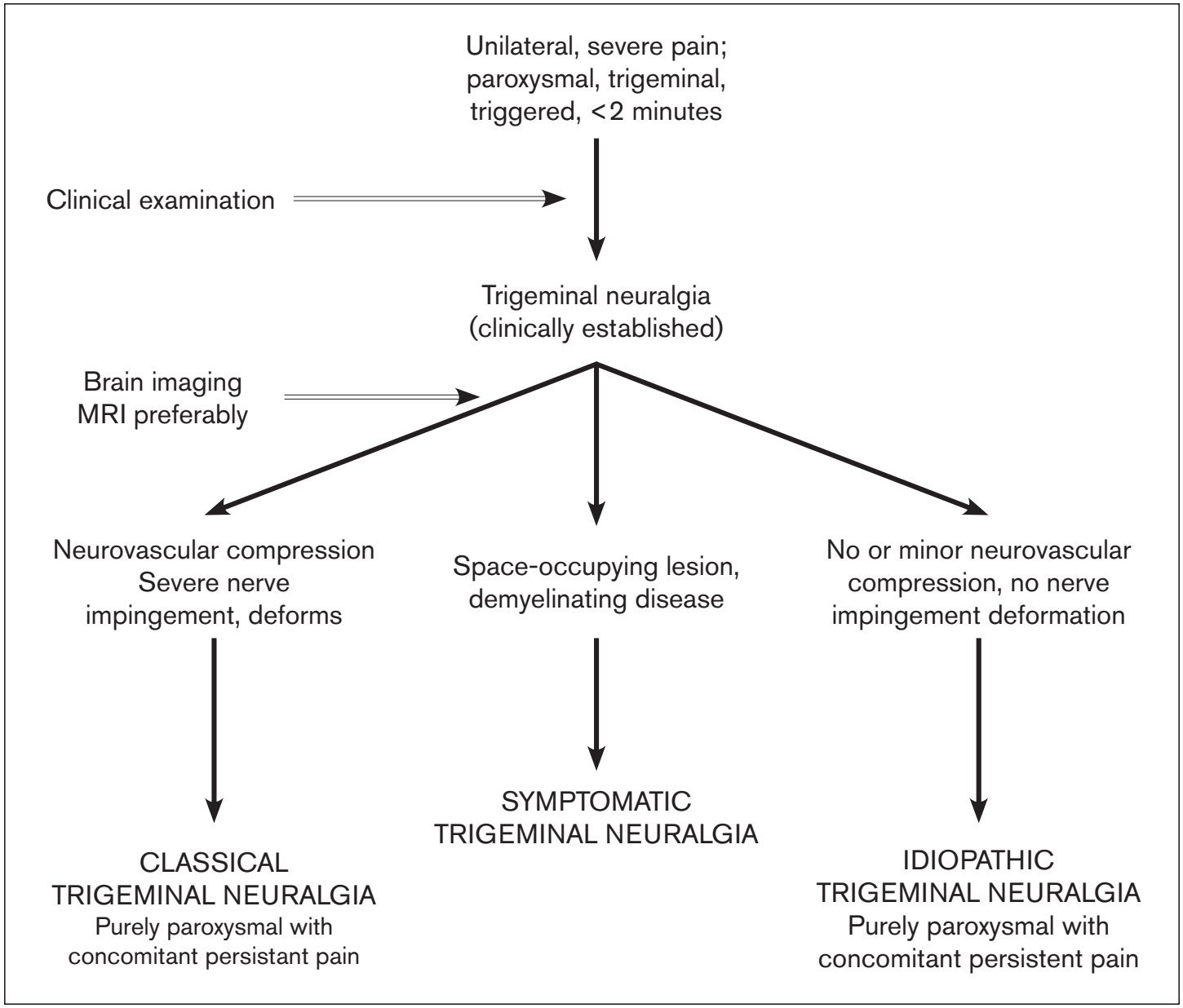

Fig 2 Flow diagram for the diagnosis of trigeminal neuralgia according to the ICHD-3 2018 classification subtypes. Trigeminal neuralgia as a diagnosis may be established based on clinical findings. Rural areas and many underdeveloped countries may not have easy access to imaging modalities. Treatment may be initiated based on this diagnosis. Once imaging is performed, the presence of a neurovascular conflict would establish classical trigeminal neuralgia, any causative pathology would establish a diagnosis of symptomatic trigeminal neuralgia, and the absence of both would establish a diagnosis of idiopathic trigeminal neuralgia. Reprinted from Maarjberg and Benoliel with permission.[1]

1. Maarbjerg S, Benoliel R. The changing face of trigeminal neuralgia—A narrative review. Headache 2021;61:817–837.

PRIMARY HEADACHES

	Migraine (G43.xxx) With aura (G43.1) Without aura (G43.0)	Tension-type headache (G42.xx)	Cluster headache Episodic (G44.01X) Chronic (G44.02x)	Paroxysmal hemicrania Episodic (G44.03) Chronic (G44.04)	Short-lasting unilateral neuralgiform headache attacks (G44.05x)	Hemicrania continua (G44.51)
Clinical characteristics: Migraine. TTH TACs Other: ▪ Primary cough headache ▪ Primary exercise headache ▪ Primary headache associated with sexual activity ▪ Primary thunderclap headache ▪ Cold-stimulus headache ▪ External-pressure headache ▪ Primary stabbing headache ▪ Nummular headache ▪ Hypnic headache ▪ NDPH	History of five headaches lasting between 4 and 72 h Must have 2 of 4: Pulsating, unilateral, moderate-severe, aggravation with exertion Must have at least one of two: Photophobia AND phonophobia, and/or nausea or vomiting Chronic = 15 or more per mo for more than 3 mo and has the features of migraine on at least 8 d per mo If less than five attacks, "probable" Pathophysiology: ▪ Migraine has three phases: premonitory, headache, and postdrome. Additionally, the interictal period has been characterized in migraine sufferers ▪ Premonitory phase begins around 1–3 d before headache and involves a complex interplay between various cortical and subcortical brain regions, including the hypothalamus and brainstem nuclei, that modulate nociceptive signaling. The headache phase involves activation of the trigeminovascular system. ▪ In one third of patients, an aura phase may occur during some attacks and likely correlates with a cortical spreading depression-like event; a slowly propagating wave of neuronal and glial cell depolarization and hyperpolarization.	Temporalis and masseters may be involved with pain on chewing At least 10 episodes occurring on < 1 d per mo on average (< 12 d per y) Lasts from 30 min to 7 d No nausea or vomiting (anorexia may occur) No more than one: photophobia, phonophobia Headache has at least two of the following characteristics: ▪ Bilateral location ▪ Band-like pressure or tightness, nonpulsating quality ▪ Mild or moderate intensity ▪ Not aggravated by routine physical activity such as walking or climbing stairs	At least five attacks of severe, strictly unilateral pain (hot, stabbing) that is orbital, supraorbital, temporal, or in any combination Lasting 15–180 min and occurring from once every other day to 8 times/d Pain is associated with: ▪ Ipsilateral conjunctival injection ▪ Lacrimation ▪ Nasal congestion ▪ Rhinorrhea ▪ Forehead and facial sweating ▪ Miosis, ptosis, and/or restlessness or agitation Commonly wakes 90 min after falling asleep: REM-locked Smoking and EtoH-related Episodic: attacks occurring in periods lasting from 7 d to 1 y separated by pain-free periods lasting ≥ 3 mo. These "clusters" are usually 6–8 wk Chronic: attacks occurring for ≥ 1 y without remission, or with remission periods lasting < 3 mo	At least 20 attacks of severe, strictly unilateral pain—orbital, supraorbital, temporal, or any combination—lasting 2–30 min and occurring several or many times a day Attacks are usually associated with ipsilateral conjunctival injection, lacrimation, nasal congestion, rhinorrhoea, forehead and facial sweating, miosis, ptosis, and/or eyelid oedema Episodic: attacks of pain occurring in periods lasting from 7 d to 1 y, separated by pain-free periods ≥ 3 mo Chronic: attacks of pain occurring for > 1 y without remission, or with remission periods lasting < 3 mo Background pain may be present May wake from sleep Absolute response to indomethacin	Attacks of moderate or severe, strictly unilateral head pain lasting 1–600 s Occurring ≥ 1/d and usually associated with prominent lacrimation and redness of the ipsilateral eye SUNCT ▪ Includes both conjunctival injection and lacrimation ipsilateral to the pain ▪ Can get up to 200 attacks/d ▪ Episodic and chronic forms with same criteria as paroxysmal hemicrania SUNA ▪ Only one: conjunctival injection or lacrimation (tearing) ▪ Episodic and chronic forms with same criteria as paroxysmal hemicrania Not responsive to indomethacin Not relieved by oxygen Not relieved by subcutaneous sumatriptan	Persistent, strictly unilateral headache associated with: ▪ Ipsilateral conjunctival injection ▪ Lacrimation ▪ Nasal congestion ▪ Rhinorrhea ▪ Forehead and facial sweating ▪ Miosis, ptosis, and/or eyelid oedema ▪ Restlessness or agitation Can be remitting (pain-free episodes of ≥ 24 h) or unremitting (no pain-free periods for ≥ 1 y) May have photophobia, phonophobia, and nausea, as in migraine Absolute response to indomethacin
Tests			Sleep study (closely associated with OSA) MRI with or without contrast of CP angle and pituitary views	MRI with or without contrast CP angle and pituitary views	MRI with or without contrast CP angle; vascular and pituitary views	MRI with or without contrast CP angle; vascular and pituitary views

PRIMARY HEADACHES

	Migraine (G43.xxx) With aura (G43.1) Without aura (G43.0)	Tension-type headache (G42.xx)	Cluster headache Episodic (G44.01X) Chronic (G44.02x)	Paroxysmal hemicrania Episodic (G44.03) Chronic (G44.04)	Short-lasting unilateral neuralgiform headache attacks (G44.05x)	Hemicrania continua (G44.51)
Treatment	Pain diary to identify and avoid triggers Patient education and awareness training Maintain routine schedule Regular exercise PSR CBT	Patient education and awareness training Headache diary Caffeine reduction Stress reduction/PSR CBT with biofeedback	Patient education and awareness training Headache diary; begin prophylactic medications if predictable times Psych referral if suicidal	Patient education and awareness training Headache diary Indomethacin; wean off during remission periods Occipital nerve blocks	Patient education and awareness training Headache diary CBT Stress reduction	Patient education and awareness training Headache diary Indomethacin; wean off during remission periods Greater occipital nerve block
Medications	Abortive: ▪ Nonspecific: NSAIDs, acetaminophen ▪ Specific: sumatriptan 6 mg injectable; zolmitriptan; rizatriptan; frovatriptan (menstrual) ▪ Ditans ▪ Gepants (CGRP and CGRPr) antagonists ▪ Greater occipital nerve block with local anesthetic and/or steroid Preventive: ▪ Gepants ▪ Anti-CGRP and -CGRPr monoclonal antibodies Beta-blocker: ▪ Primary: - Propranol 20–40 mg qid + 20 mg/wk to 160 mg/d - Timolol 10–15 mg bid ▪ Secondary: - Metoprolol succinate 50 mg qd - Metoprolol tartrate 25–100 mg bid - Atenolol 50–150 mg qd - Nadolol 40–240 mg qd Anticonvulsants: ▪ Topiramate 25 mg bid ▪ Divalproex sodium 250–500 mg bid Tricyclic antidepressants: ▪ Amitriptyline 25–50 mg qd ▪ Nortriptyline 10–50 mg qd ▪ Doxepin 10–50 mg qd Botulinum toxin (chronic migraine)	NSAIDs Acetaminophen Amitriptyline 10–35 mg qhs; *Risk: cardio, diabetes, seizure, urinary tract disorders; BB: suicide, < 25 y* Venlafaxine extended release 37.5 mg qd + 37.5 mg every 3 d < 150 mg: *Risk: bleeding, glaucoma, liver, cardio; BB: suicide*	Abortive: ▪ 100% oxygen 12–15 mL/min in nonrebreather mask ▪ Sumatriptan 6 mg subcutaneous ▪ Zolmitriptan Transitional: ▪ Prednisone Prophylactic: ▪ Greater occipital nerve block with local anesthetic/steroid combination. If effective, may be repeated as needed ▪ Verapamil ▪ Lithium ▪ Gepants ▪ Anti-CGRP and -CGRPr MABs	Indomethacin 50 mg tid up to 250 mg/d; *Risk: cardio, bleeding, HTN, asthma, smoking, EtOH; BB: cardio, GI* Add: omeprazole 40 mg qd for GI protection Topiramate 25 mg bid + 25 mg/d < 50 mg/d: may reduce weight; *Risks: ketogenic diet, bleeding, depression/suicidal* Nerve blocks with dexamethasone 4 mg/mL or triamcinolone 10 mg/mL and 1% lidocaine or 3% mepivacaine without vasoconstrictor	Lamotrigine 25 mg/d: *BB: Serious rash, SJS* Topiramate 25 mg bid + 25 mg/d < 50 mg/d: may reduce weight; *Risks: ketogenic diet, bleeding, depression/suicidal* Gabapentin 100 mg qd + 100 mg/d < 1,800 mg/d Very difficult; no meds have proven highly effective	Indomethacin 50 mg tid up to 250 mg/d; *Risk: cardio, bleeding, HTN, asthma, smoking, EtOH; BB: cardio, GI* Add: omeprazole 40 mg qd for GI protection Topiramate 25 mg bid + 25 mg/d < 50 mg/d: may reduce weight; *Risk: ketogenic diet, bleeding, depression/suicidal* Nerve blocks with dexamethasone 4 mg/mL or triamcinolone 10 mg/mL and 1% lidocaine or 3% mepivacaine without vasoconstrictor

PRIMARY HEADACHES						
	Migraine (G43.xxx) With aura (G43.1) Without aura (G43.0)	Tension-type headache (G42.xx)	Cluster headache Episodic (G44.01X) Chronic (G44.02x)	Paroxysmal hemicrania Episodic (G44.03) Chronic (G44.04)	Short-lasting unilateral neuralgiform headache attacks (G44.05x)	Hemicrania continua (G44.51)
Differential diagnosis	Hemicrania continua TTH Cervicogenic headache NDPH Myofascial pain CPSP MS	Myofascial pain Cervicogenic headache Migraine NDPH External-pressure headache	Pulpitis CP angle tumor SUNCT/SUNA Pituitary tumor Migraine Hypneic headache Trigeminal neuralgia Primary stabbing headache	SUNCT CP angle tumor Cluster headache Pituitary tumor Trigeminal neuralgia Migraine	Cluster headache Pituitary tumor Paroxysmal hemicrania Trigeminal neuralgia CP angle tumor	Pre–trigeminal neuralgia CP angle tumor Cluster headache Pituitary tumor Migraine NDPH Paroxysmal hemicrania

TTH = tension-type headache; PH = paroxysmal hemicrania; HC = hemicrania continua; TAC = trigeminal autonomic cephalgia; SUNCT = short-lasting unilateral neuralgiform headache with conjunctival injection; SUNA = short-lasting unilateral neuralgiform headache with autonomic symptoms; CH = cluster headache; CPSP = central post stroke pain; MS = multiple sclerosis; NDPH = new daily persistent headache; CP= cerebellopontin.

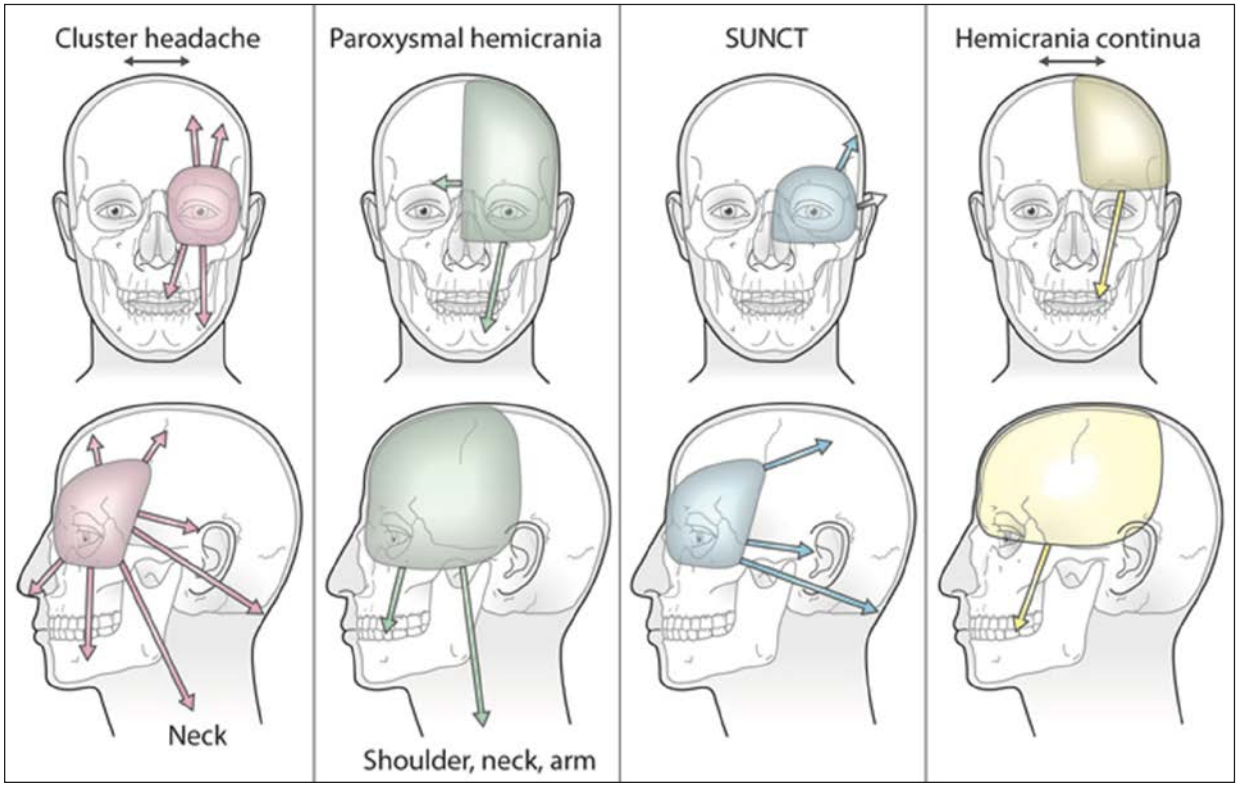

Fig 3 TACs occur in unique patterns and are categorized by the temporal (time) aspects of attacks. These are common pain patterns of TACs. Figure reprinted with permission from Sharav and Benoliel.[1]

1. Sharav Y, Benoliel R. Orofacial Pain and Headache, ed 2. Quintessence, 2015.

PRIMARY HEADACHES: FACIAL PRESENTATIONS (from ICOP)

	Orofacial migraine (G44.00)	Orofacial cluster attacks	Paroxysmal hemifacial pain	Short-lasting unilateral neuralgiform facial pain with cranial autonomic signs	Neurovascular orofacial pain (short-lasting/long-lasting)
Clinical characteristics	At least five attacks of pain exclusively in the orofacial region, without head pain, with the characteristics and associated features of migraine Typical characteristics of the pain: ▪ Unilateral location ▪ Pulsating quality, moderate or severe intensity ▪ Aggravation by routine physical activity ▪ Association with nausea and/or photophobia and phonophobia Chronic facial and/or oral pain occurring on ≥ 15 d per mo for > 3 mo that has the features of migraine on ≥ 8 d per mo	At least five attacks of severe, strictly unilateral facial and/or oral pain, without head pain Lasting 15–180 min and occurring from once every other day to 8 times/d The pain is associated with: ▪ Ipsilateral conjunctival injection ▪ Lacrimation ▪ Nasal congestion ▪ Rhinorrhea ▪ Forehead and facial sweating ▪ Miosis, ptosis, and/or eyelid oedema ▪ Restlessness or agitation Episodic: occurring in periods lasting from 7 d to 1 y, separated by pain-free periods lasting ≥ 3 mo Chronic: attacks occurring for > 1 y without remission or with remission periods lasting < 3 mo	At least 20 attacks of severe, strictly hemifacial pain without head pain Typical characteristics of the pain: ▪ Lasting 2–30 min ▪ Occurring many times a day Attacks may be associated with: ▪ Ipsilateral conjunctival injection ▪ Lacrimation ▪ Nasal congestion ▪ Rhinorrhea ▪ Forehead and facial sweating ▪ Miosis, ptosis, and/or eyelid oedema ▪ Absolute response to indomethacin Episodic: attacks of pain occurring in periods lasting from 7 d to 1 y, separated by pain-free periods lasting ≥ 3 mo Chronic: attacks of pain occurring for > 1 y without remission or with remission periods lasting < 3 mo	At least 20 attacks of moderate or severe, strictly unilateral oral and/or facial pain without head pain Lasting 1–600 s Occurring at least once a day Usually associated with prominent lacrimation Redness of ipsilateral eye and/or other local autonomic symptoms and/or signs Episodic: attacks occurring in periods lasting from 7 d to 1 y, separated by pain-free periods lasting ≥ 3 mo Chronic: attacks occurring for > 1 y without remission, or with remission periods lasting < 3 mo	At least five attacks of moderate or severe intraoral pain, without head pain, of variable duration Often accompanied by toothache–like symptoms, with mild autonomic and/or migrainous symptoms Possibly an isolated intraoral form of migraine Two subforms are represented by patients with relatively short attacks (1–4 h) and those with longer attacks (> 4 h) Although essentially an intraoral pain, there may be referral and/or radiation to adjacent sites, particularly when pain is severe
Tests		Response to O_2 MRI with and without contrast of CP angle and pituitary views	MRI with and without contrast through CP angle; pituitary views	MRI with and without contrast through CP angle and pituitary views	Full-mouth periapical imaging
Treatment	Pain diary to identify and avoid triggers Patient education and awareness training Maintain routine schedule Regular exercise PSR CBT	Patient education and awareness training Headache diary; begin transitional/prophylactic medications if high frequency Monitor closely if refractory for suicidal ideation	Patient education and awareness training Headache diary Indomethacin; wean off during remission periods Occipital nerve blocks	Patient education and awareness training Headache diary CBT Stress reduction	Responds to antimigraine therapy No data on gepants or MABs

PRIMARY HEADACHES: FACIAL PRESENTATIONS (from ICOP)

	Orofacial migraine (G44.00)	Orofacial cluster attacks	Paroxysmal hemifacial pain	Short-lasting unilateral neuralgiform facial pain with cranial autonomic signs	Neurovascular orofacial pain (short-lasting/long-lasting)
Medications	Abortive: ▪ Nonspecific: NSAIDs, acetaminophen ▪ Specific: sumatriptan 6 mg injectable; zolmitriptan; rizatriptan; frovatriptan (menstrual) ▪ Ditans Gepants (CGRP and CGRPr) antagonists Preventive: ▪ Gepants ▪ Anti-CGRP and CGRPr MABs Propranol 2040 mg qid + 20 mg/wk to 160 mg/d Divalproex sodium 250–500 mg bid Topiramate 25 mg bid Amitriptyline 25–50 mg qhs Botulinum toxin (chronic migraine) Greater occipital nerve block with lidocaine/dexamethasone injections 4 mg/mL	Abortive: ▪ 100% oxygen 12–15 mL/min in nonrebreather mask ▪ Sumatriptan 6 mg subcutaneous ▪ Zolmatriptan Transitional: ▪ Prednisone Prophylactic: ▪ Greater occipital nerve block with local anesthetic and/or steroid injections. Repeat weekly for 4 wk and reassess. If effective, may be repeated as needed Verapamil Lithium Gepants Monoclonal antibodies: anti-CGRP and -CGRPr	Indomethacin 50 mg tid up to 250 mg/d; *Risk: cardio, bleeding, HTN, asthma, smoking, EtOH; BB: Cardio, GI* Add: omeprazole 40 mg qd for GI protection Topiramate 25 mg bid + 25 mg/d < 50 mg/d: may reduce weight; *Risks: ketogenic diet, bleeding, depression/suicidal*	Lamotrigine 25 mg/d: *BB: Serious rash, SJS—must titrate slowly* Topiramate 25 mg bid + 25 mg/d < 50 mg/d: may reduce weight; *Risks: ketogenic diet, bleeding, depression/suicidal* Gabapentin 100 mg qd + 100 mg/d < 1,800 mg/d Very difficult; no meds have proven highly effective Drug of choice: lamotrigine	
Differential diagnosis	Hemicrania continua TTH Cervicogenic headache NDPH Myofascial pain CPSP TMD MS	Pulpitis CP angle Tumor SUNCT/SUNA Pituitary tumor Migraine Hypneic headache Trigeminal neuralgia Primary stabbing Headache	SUNCT CP angle tumor Cluster headache Pituitary tumor Trigeminal neuralgia Migraine	Cluster headache Pituitary tumor Paroxysmal hemicrania Trigeminal neuralgia CP angle tumor	

CBT = cognitive behavioral therapy; CGRP(r) = calcitonin gene-related peptide (receptor); CP = cerebropontine; CPSP = chronic postsurgical pain; EtOH = alcohol; GI = gastrointestinal; HTN = hypertension; MABs = monoclonal antibodies; MS = multiple sclerosis; NDPH = new daily persistent headache; OSA = obstructive sleep apnea; PSR = physical self-regulation; REM = rapid eye movement; SJS = Stevens-Johnson syndrome; SUNA =short-lasting unilateral neuralgiform headache attacks with cranial autonomic symptoms; SUNCT = short-lasting unilateral neuralgiform headache with conjunctival injection and tearing; SUNFA = short-lasting unilateral neuralgiform facial pain with cranial autonomic signs; TAC = trigeminal autonomic cephalalgias; tid = three times a day; TTH = tension-type headache.

COMMENTARY: HEADACHES

- There are two headaches that are "absolutely responsive" to indomethacin: *(1)* paroxysmal hemicrania and *(2)* hemicrania continua. However, there are cases of these headaches that do not respond to indomethacin. Indomethacin is a unique NSAID because it crosses the blood-brain barrier. As with all NSAIDs, it can cause GI issues via direct and indirect actions. It is wise to recommend concomitant use of omeprazole or famotidine, each having different adverse drug event profiles. Indomethacin is also a teratogen and must be stopped during pregnancy.
- Indomethacin is a reasonable trial medication to abort many of the other primary headaches. For example, anecdotal evidence suggests that primary sex headaches can be prevented by taking 25 mg of indomethacin prior to sexual intercourse.
- Migraines and TTHs do not typically require MRI/CT; however, imaging is indicated for all TACs to rule out intracranial pathology.
- Headaches considered to be primary are migraines, TTHs, TACs, NDPHs, and those considered "other primary headaches" (see ICHD-3).
- Secondary headaches require advanced imaging and serology to rule out life-threatening etiology.

The following is a mnemonic system for recognizing secondary headaches that may be life-threatening[1]:

SNOOP$_5$ Red Flag System for Secondary Headaches

- **S**ystemic symptoms or diseases
  - Fever, chills, unexplained weight loss, nuchal rigidity
  - NEED TO RULE OUT: malignancy, HIV, infection
- **N**eurologic symptoms or signs
  - Precipitous onset with change in mental status: confusion, impaired alertness, or consciousness
  - NEED TO RULE OUT: stroke, mass, encephalitis
- **O**nset sudden (acute or thunderclap)
  - URGENT NEED TO RULE OUT: brain bleed
- **O**nset after age 50 y
  - NEED TO RULE OUT: giant cell arteritis, mass, glaucoma
- **P$_5$**
  - **P**revious headache history
    - New
    - Different (change in frequency, severity, or clinical features)
    - Headache in late night or early morning
  - **P**rogressive and/or pattern change
  - **P**recipitated by Valsalva, bending, straining
  - **P**ostural
  - **P**regnancy

1. Dodick D. Pearls: Headache. Semin Neurol 2010;30:74–81.

COMMON MEDICATIONS USED IN THE MANAGEMENT OF ORAL, FACIAL, AND HEAD PAIN

NSAIDs	Steroids and non-NSAID analgesics	Muscle relaxants	Psychiatric antidepressants	Antiepileptics/ anticonvulsants	Antihypertensives	Triptans/ditans	MABs	Gepants
All NSAIDs carry a degree of CV risk. COX-2–specific drugs probably have a higher CV risk with a lower GI risk.	Non-NSAID analgesics include opioids, but considering the addiction potential, we do not advise using them. Moreover, they have little if any efficacy in neuropathic pain, myalgia, migraine, and TACs.	To actually obtain significant muscle relaxation, these drugs would need very high doses that are not clinically relevant. Nevertheless, this is their classification grouping.	These are TCAs or SNRIs and are, as a group, effective central analgesics across multiple pain disorders (eg, myalgia, neuropathic pain, and migraine). SSRIs are generally not effective central analgesics. Consider that, once initiated, SNRIs are difficult to cease. In general, effect on pain will start at ≥ 2 wk	Depending on the specific drug, this group offers effective management of multiple pain disorders (eg, myalgia, neuropathic pain, and migraine).	Used in the prophylactic management of neurovascular pain, migraine, and cluster headache	Until recently, triptans were considered the best choice for migraine. They are also effective in episodic cluster headache. Their main limitation has been their CV side effects. The ditans act on serotonin receptor 5HT1F and circumvent this adverse event. Gepants also offer an excellent alternative and are currently in wide usage.	Used for the prophylactic management of migraine and cluster headache. Produced from human antibodies that target the CGRP molecule or the binding site of the CGRP receptor	A group of drugs that target the CGRP receptor binding site. Used as abortive[a] and prophylactic[b] agents for migraine
Paracetamol (acetaminophen) 350–500 mg, by mouth, 3/d, < 3,000 mg/d; *Risk: liver toxicity*	Tramadol available as drops or tablets, 50 to 100 mg, 2/d	Cyclobenzaprine 10–60 mg/d Structurally similar to AMI ➔Myalgia/fibromyalgia	Amitriptyline 10–35 mg by mouth, 1/d nocte Warn of weight gain Avoid in elderly and CV patients; ECG warranted	Carbamazepine 400 mg, 3/d Start at 200 mg and titrate to above. SR = 2 doses/d Monitor sodium, liver enzymes. Risk of SJS in Asian patients with HLAb*1502 ➔Trigeminal neuralgia	Propranolol/SR 80–240 mg/d by mouth Start 40–80 mg/d in 2–3 doses Consider transfer to SR ➔Migraine	Sumatriptan 50–100 mg Sumatriptan NS 5–22 mg/dose, by mouth Sumatriptan SC 6 mg/dose	Erenumab 70–140 mg/mo SC	Zavegepant[a] 10 NS
Ibuprofen 200–400 mg by mouth, 3/d; moderate GI side effects	Tramadol/paracetamol 37.5 mg/325 mg 2 tabs, 3/d Use for short-term therapy (≤ 5 d)	Baclofen 5–15 mg, 3/d Acts on upper motor neurons ➔Trigeminal neuralgia	Nortriptyline 25–50 mg by mouth, 1/d nocte Fewer side effects than amitriptyline. Warn of weight gain Avoid in elderly and CV patients	Oxcarbazepine 300–600 mg, 3/d Monitor sodium, liver enzymes Less CNS side effects than carbamazepine When switching patients from carbamazepine, increase dose by ~50%. ➔Trigeminal neuralgia	Verapamil/SR 480–720 mg/d by mouth Start with baseline ECG. Repeat with any dose increase ➔Cluster headache	Eletriptan 40 mg/dose by mouth	Migraine: Galcanezumab 120 mg/mo SC Cluster headache: 300 mg/mo	Rimegepant[a] 75 mg ODT

Arrows represent common indications.

COMMON MEDICATIONS USED IN THE MANAGEMENT OF ORAL, FACIAL, AND HEAD PAIN

NSAIDs	Steroids and non-NSAID analgesics	Muscle relaxants	Psychiatric antidepressants	Antiepileptics/ anticonvulsants	Antihypertensives	Triptans/ditans	MABs	Gepants
Naproxen sodium 225–450 by mouth, 2/d Considered safest NSAID from a CV risk angle; more prominent GI side effects	Steroids are excellent for transitional prophylaxis of cluster headache. Allows for prophylactic therapy to fully control headaches. Oral prednisone at 60–100 mg daily in the morning for 5–7 d, then tapered every 2–3 d by 10 mg		Venlafaxine 150–225 mg/d (in two doses) SR = 150–225 mg/d (1 dose) Desvenlafaxine 50–100 mg/d	Valproic acid 300–1,000 mg by mouth, 2/d ➔Migraine		Frovatriptan 2.5 mg/dose by mouth	Fremanezumab 225 mg/mo, 675 mg/quarter SC	Ubrogepant[a] 50–100 mg
Meloxicam 7.5–15 mg/d; similar GI side effects to ibuprofen	Dipyrone 500 mg, 3/d (by mouth) Not available in the USA		Duloxetine 30–120 mg by mouth, 1/d Monitor BP/HR	Gabapentin 200–600 mg by mouth, 3/d Initial target 900 mg/d Titrate further if needed until 2,400 mg max		Rizatriptan 10 mg/dose by mouth Available as oral film	Eptinezumab 100–300 mg infusion/quarter	Atogepant[a,b] 10–60 mg
Ibuprofen 200 mg with paracetamol 500 mg, 3/d			Psychiatric bipolar disorder drugs	Pregabalin 25–150 mg × 2/d (PO) Leg swelling May be further titrated with care to 600 mg		Zolmitriptan 2.5 mg/dose by mouth Zolmitriptan NS 2.5 mg/dose		
			Lithium 300–900 mg by mouth Prophylaxis of chronic cluster headache Monitor blood levels	Topiramate 100–200 mg/d by mouth Weight loss, dysgeusia, memory loss ➔Migraine		Almotriptan 6.25–12.5/dose by mouth		
				Lamotrigine 25–200 mg, 2/d May cause SJS: slow titration Trigeminal neuralgia SUNA		Lasmitidan 50–200 mg/d by mouth		

Arrows represent common indications.

COMMON MEDICATIONS USED IN THE MANAGEMENT OF ORAL, FACIAL, AND HEAD PAIN

NSAIDs	Steroids and non-NSAID analgesics	Muscle relaxants	Psychiatric antidepressants	Antiepileptics/ anticonvulsants	Antihypertensives	Triptans/ditans	MABs	Gepants
				Clonazepam 0.25–2 mg, 3/d No evidence other than for BMS				
Other NSAIDs not listed may be clinically effective. Clinicians with knowledge of and experience with other drugs (eg, meloxicam, ketorolac, etodolac), including side effects and drug interactions, may choose to use those medications					Other beta blockers not listed may be clinically effective in the prophylaxis of migraine. Clinicians with knowledge of and experience with other drugs (eg, metoprolol, atenolol), including side effects and drug interactions, may choose to use those medications.	When one triptan fails, it is worth trying a different triptan that may help. The advent of gepants and ditans challenge the monopoly that triptans have enjoyed.	In most countries these are reserved as second line, but will likely eventually be the drug of choice for many patients due to their excellent side effect profile.	

[a]Episodic migraine. [b]Chronic migraine.
Note: Corticosteroids are effective in about 70% to 80% of patients and may induce remission of a cluster period in about one-quarter of cases.
BMS = burning mouth syndrome; BP/HR = blood pressure/heart rate; CGRP = calcitonin gene-related peptide; CV = cardiovascular; CNS = central nervous system; ECG = electrocardiogram; GI = gastrointestinal; NS = normal saline; NSAIDs = nonsteroidal anti-inflammatory drugs; ODT = orally dissolving tablet; SC = subcutaneous; SNRI = serotonin and norepinephrine reuptake inhibitors; SR = sustained release; SUNA = short-lasting unilateral neuralgiform headache attacks with cranial autonomic symptoms; TACs = trigeminal autonomic cephalalgias.

COMMON INJECTIONS USED IN THE MANAGEMENT OF ORAL, FACIAL, AND HEAD PAIN

Injections				
Technique	Components	Indications	Frequency	Dosages and comments
Greater occipital nerve block	Local anesthetic and/or steroids	Migraine and cluster headache prophylaxis	One session (uni- or bilateral injections) every wk for 1 mo and reassess	Large total volumes are recommended; eg, 5 mL per side Local anesthetics used: lidocaine 2%, bupivacaine 0.5%, prilocaine 1% Steroids used: triamcinolone 10–80 mg, methylprednisone 20–160 mg, betamethasone 2–21 mg
Botulinum toxin	Botulinum toxin	Chronic migraine	Inject once and assess effect. May be repeated. A subcutaneous approach is advised.	A total of 155 units are administered as 5-unit injections per site (31 sites) using a sterile 30-gauge, short (0.5-inch) needle to the corrugator, procerus, frontalis, temporalis, occipital, cervical paraspinal group, and trapezius muscles, bilaterally.
		Trigeminal neuralgia	Effect may last months. Injection may be repeated if efficacy shown. Shortage of evidence regarding the optimal dose, route, depth of injection, onset of action, and period of effectiveness	A total of 20–50 units injected into the trigger zones. Lower (5–9 U) and higher doses (75 U) have been successfully employed. Effect appears after 1–2 wk
Sphenopalatine ganglion block	Local anesthetic and/or steroids	Cluster headache	3 injections and 3- to 6-da intervals. Assess effect. Usually performed with fluoroscopy to locate the ganglion accurately An intranasal approach has been described with no fluoroscopy.	Triamcinolone acetonide (40 mg) with bupivacaine 1% (4 mL) and mepivacaine 2% with adrenaline 1/100,000 (4 mL) Via intranasal approach: 1.5 mL each nostril of 2% lidocaine (viscous or liquid)
Trigger point injection	Lidocaine 2%, mepivacaine 3%	Muscle pain with taut bands of hypersensitive tissue	Four to six injections every wk. Assess. May be repeated if successful	Research indicates efficacy that is not inferior to Botulinum toxin.

BoNT-A = onabotulinumtoxin A.

APPROPRIATE SEROLOGIC TESTS FOR THE DIAGNOSIS AND MANAGEMENT OF ORAL, FACIAL, AND HEAD PAIN

Test name	Description	Interpretation	Possible indications
Hematology	Differential CBC	Composed of a number of measurements of blood components—some are measured directly and others are calculated. A general test used for screening of disease: infection, malignancy, anemia, etc.	General test for health screening; perform whenever requesting other blood work.
	Erythrocyte count	↑ Secondary polycythemia, decreased tissue oxygenation, increased erythropoietin, iron deficiency ↓ Anemia, drug-induced aplastic anemia, hemolysis (eg, G6PD deficiency)	
	Hematrocit	↑ Polycythemia ↓ Anemia	
	Hemoglobin	↑ Polycythemia ↓ Anemia	
	Leukocyte differential count	Measures levels of specific white cells that react to infectious diseases, malignancies, and allergies as a group: neutrophils, lymphocytes, eosinophils, basophils, monocytes	
Early markers	ESR	Used as a general marker for disease groups as below Correlates with plasma fibrinogen levels ↑ Infections, inflammatory disease, tissue damage, conditions that increase fibrinogen or globulins (eg, malignancy)	Suspicion of inflammation, autoimmune disease, or malignancy
	CRP	↑ Acute phase reactant; very rapid increase. Rapid, marked increases in inflammation, infection, trauma, tissue necrosis, malignancy, autoimmune disease. Not affected by hormones	
Complete metabolic panel	Calcium	↑ Primary hyperparathyroidism, PTH-producing tumors, excess vitamin D intake ↓ Primary hypoparathyroidism, vitamin D deficiency	Bone diseases, parathyroid diseases
	Sodium	↑ Associated with water loss; sweating, hyperapnea, vomiting/diarrhea, polyuria ↓ Low sodium intake, sodium loss via diuretics, nephropathy, drug-induced (carbamazepine, oxcarbazepine)	Antiepileptic medication
	Potassium	↑ Primary hyperparathyroidism, PTH-producing tumors, excess vitamin D intake ↓ Primary hypoparathyroidism	Thyroid disease
	Carbon dioxide	↑ In respiratory acidosis, caused by poor gas exchange due to lung disease.	
	Glucose	↑ Diabetes (types 1 and 2), strenuous exercise, infection (inconsistent), thyrotoxicosis, acromegaly, pancreatitis, pancreatic neoplasm	Diabetic control, BMS
	HbA1c	↑ Level reflects mean glucose levels during the lifespan of erythrocytes (120 d) ↑ Diabetes (types 1 and 2)	
	Creatinine kinase	↑ Trauma, surgery, MI, muscle ischemia, myopathies (eg, polymyositis, dermatomyositis)	

APPROPRIATE SEROLOGIC TESTS FOR THE DIAGNOSIS AND MANAGEMENT OF ORAL, FACIAL, AND HEAD PAIN

Test name	Description	Interpretation	Possible indications
Complete metabolic panel *(cont.)*	Creatinine	↑ All cause acute and chronic renal disorders, acromegaly, hyperthyroidism.	Renal disease
	e/mGFR (estimated/measured glomerular filtration rate)	↑ Increased cardiac output, high-protein diet ↓ Shock, bleeding, congestive heart failure, renal disease, glomerulonephritis, multiple myeloma	
	Urea	↑ Renal dysfunction, dehydration; insufficient intake, increased fluid output	
	Protein	↑ Dehydration, cancer (eg, multiple myeloma) ↓ Liver or kidney disease, malabsorption of protein, problems with protein digestion	
	Albumin	↑ Dehydration ↓ Acute phase reaction and chronic inflammation: infection, surgery, trauma, malignancy	
	Bilirubin	↑ Hepatocellular damage (inflammatory, toxic, neoplastic), biliary tree obstruction	
	Liver enzymes		
	ALT	Found in liver and heart ↑🕐↑ All cause acute liver necrosis ↑ Cirrhosis, obstructive jaundice, liver tumor. ↑ Drug-induced heart disease	Diagnosis and follow-up of liver function. Detect alcohol abuse. Monitor drug-induced liver injury
	AST	↑🕐↑ Fulminant forms of acute hepatitis, especially viral ↑ All cause liver injury or necrosis ↑ Cholestasis, drug-induced injury, alcohol, viral ↑ Trauma to heart or skeletal muscle	
	GGT	↑ Liver disease, fatty liver, bile duct disease, drug-induced	
	Alkaline phosphatase alpha	↑ Liver disease, bone disorders	
Other enzymes	Amylase (diastase)	Found in pancreas and parotid salivary glands: ↑ Pancreatitis (very sensitive early marker, wanes over 5 d) ↑ Parotitis, intestinal obstruction	
	LDH	↑ General marker for organ or tissue damage	
Lipid profile	Cholesterol (total)	↑ Familial, coronary heart disease, obstructive liver disease, type 2 diabetes, hypothyroidism, obesity	Increased cardiovascular disease risk
	LDL	↑ Familial hypercholesterolemia, secondary to hypothyroidism, nephrotic syndrome, obstructive liver disease ↓ Hyperthyroidism, hepatocellular dysfunction	
	VLDL	Carries triglycerides to tissues	
	HDL	↑ Antiatherogenic, probably anti-inflammatory. Inverse relation between HDL levels and coronary heart disease	Decreased cardiovascular disease risk
	Triglycerides	↑ Familial hypertriglyceremia, pancreatitis, obesity, type 2 diabetes, alcoholism	Increased cardiovascular disease risk

APPROPRIATE SEROLOGIC TESTS FOR THE DIAGNOSIS AND MANAGEMENT OF ORAL, FACIAL, AND HEAD PAIN

Test name	Description	Interpretation	Possible indications
Endocrinology	TSH	↑ Primary hypothyroidism, Hashimoto's thyroiditis ↓ Primary/secondary hyperthyroidism	Thyroid disease
	Free thyroxine	↑ Hyperthyroidism, hypothyroidism treated with thyroxine ↓ Hypothyroidism, triiodothyronine treatment	
	Cortisol	↑↑ Ectopic ACTH syndrome ↑ Cushing's syndrome, adrenal adenoma, carcinoma ↓ Addison's disease, hypopituitarism Diurnal variation in normal states and highest around 8 am	Cushing syndrome
	GH	↑ Pituitary gigantism, acromegaly, renal failure; ectopic GH secretion from stomach and lung neoplasms ↓ Pituitary dwarfism, hypopituitarism, adrenocortical hyperfunction	Acromegaly
	Prolactin	↑ Secretion from pituitary tumors, amenorrhea, galactorrhea; hypothyroid ↓ Pituitary apoplexy	Rule out suspicion of pituitary tumors. Pituitary adenomas are a common cause of symptomatic TACs, in particular cluster headache
Vitamins	Vitamin B6	↑ Chronic alcoholism, malnutrition, malabsorption, smoking ↓ Hypophosphatasia	Include B group in BMS work-up
	Vitamin B12	↑ Chronic renal failure, congestive heart failure, diabetes (types 1 and 2), myelogenous leukemia, liver disease ↓ Untreated deficiency, megaloblastic anemia, malabsorption, antibodies to intrinsic factor	
	Vitamin C	↓ Scurvy, hemodialysis, anemia, alcoholism, hyperthyroid, cancer	Suspicion of malnutrition
	Vitamin D3	Essential for calcium and bone metabolism ↓ Azotemic renal failure, hypoparathyroidism, postmenopausal osteoporosis, type 1 diabetes in adolescents ↓ Tumoral calcinosis, primary hyperthyroidism	Associated with rickets and a number of health problems. Has recently been associated with a number of health disorders. Possible association with generalized muscle pain; data inconclusive
	Folic acid	↑ Vegetarians ↓ Alcoholism, enzyme deficiency, liver disease	Work-up when diet is a concern BMS
Iron metabolism	Iron	↑ Pernicious, aplastic, hemolytic anemia ↑ Iron deficiency, anemia, chronic infection, hypothyroidism, carcinoma	BMS
	Ferritin	Marker of iron stores ↑↑ Iron overload (eg, liver disease) ↑ Acute leukemia, inflammatory disease ↓ Iron deficiency	BMS
	Transferrin		BMS
	Total iron binding capacity		

APPROPRIATE SEROLOGIC TESTS FOR THE DIAGNOSIS AND MANAGEMENT OF ORAL, FACIAL, AND HEAD PAIN

Test name	Description	Interpretation	Possible indications
Coagulation	PT	INR allows comparison of values across laboratories and assesses function of factors II, V, VII, and X Problems in coagulation may be hereditary or due to underlying liver disease	Liver disease, anticoagulant therapy
	PTT	Assesses function of factors VIII, IX, XI, and XII Problems in coagulation may be hereditary or due to underlying liver disease	
	Fibrinogen	↑ Sensitive acute phase reactant Investigated together with PT/PTT for DIC	
Tumor markers	PSA	Prostate disease, cancer	Tumor screening and follow-up
	AFP	Most widely tested biomarker in HCC. Overexpression of AFP considered reflective of aggressive tumors. ~40% of patients with unresectable HCC have very high baseline AFP	
	CEA	Colorectal or bowel cancer, prostate, ovary, lung, thyroid, liver, pancreas, breast	
	CA 19-9	Consider pancreatic cancer, gallstones, and cirrhosis of the liver	
	CA 15-3	↑ ~80% in breast cancer; useful to predict recurrence	
	CA 125	↑ Serous, endometrial, and other ovarian cancers	
	CA 72-4	Highly sensitive for gastric and GI metastatic cancer	
Immune profile	ANA generic	May be positive in ≤ 20% of healthy women > 40 y. Screening, nonspecific test for CTD	Diagnosis and follow-up of autoimmune disease
	RhF	False positives are common	
	Antismooth muscle antibodies	Appears in patients with lupus erythematosus	
	Antiparietal antibodies	Targets gastric parietal cells; 90% of pernicious anemia patients test positive	
	Antimitochondrial antibodies	PBC	
	Anti-Scl-70	Positive in ~60% of systemic sclerosis (scleroderma) patients	
	Anti-CCP	Rheumatoid arthritis	
	Intrinsic factor antibody	↑ In 50% of patients with pernicious anemia	
	Total IgM	↑ Polyclonal—selective increase in response to infection, chronic inflammatory conditions, exposure to viral infection ↑ Monoclonal—Waldenstrom's macroglobulinemia, lymphoma, chronic lymphocytic leukemia	
	Total IgG	↑ Polyclonal–chronic and recurring infections; rheumatoid arthritis and other autoimmune disease ↑ Monoclonal—multiple myeloma, plasmocytoma, lymphoma	
	Total IgA	↑ Polyclonal—found in chronic inflammatory conditions, infections; rheumatoid arthritis, MCTD ↑ Monoclonal—multiple myeloma, plasmocytoma, lymphoma, chronic lymphocytic leukemia	

APPROPRIATE SEROLOGIC TESTS FOR THE DIAGNOSIS AND MANAGEMENT OF ORAL, FACIAL, AND HEAD PAIN

Test name	Description	Interpretation	Possible indications
Immune profile *(cont.)*	Total IgE	↑ Reaction to allergens may lead to an allergic reaction. Parasitic infection, some immune system conditions	Diagnosis and follow-up of autoimmune disease
	P-ANCA	Consider inflammatory bowel disease, particularly ulcerative colitis	
	C-ANCA	Autoimmune vasculitis, Wegener's granulomatosis	
	ASCA	Consider Crohn's disease	
	Tissue transglutaminase IgA (tTG-IgA)	Celiac disease	
	Anti-Jo 1	Myositis (20%)	
	Anti-SSA/Ro	Connective tissue disease: Sjögren syndrome, lupus erythematosus, rheumatoid arthritis	
	Anti-SSB/La	Connective tissue disease: Sjögren syndrome, lupus erythematosus; less commonly positive than Ro	
	Complement	↓ Complement consumption occurs in immune complex diseases, infection, malignancy, autoimmune disease	
HLA tissue typing	HLA-DQ2 and HLA-DQ8	Celiac disease	
	HLA-DQB*06:02	Narcolepsy	
	HLA-B*57:01 and HLA-B*15:02	Adverse drug reaction	To abacavir
	HLA-B*1502	Drug-induced SJS	Carbamazepine-induced
	HLA class II DRB1	Multiple sclerosis	Often associated with generalized pain. A small percentage of individuals develop symptomatic trigeminal neuralgia.
	HLA-B27	Autoimmune disease	
Relevant viral evaluation	Anti-HIV antibodies	Infection with HIV will lead to an increasing antibody titer—normally no antibody is detected. Antibodies are detectable within 2 mo. However, in the seronegative early stage, the patient is already infected with HIV.	Testing for HIV in appropriate conditions that may indicate the patient is immunocompromised.
	CMV IgM	Positive results indicate recent infection (primary, reactivation, or reinfection). IgM in secondary (reactivation) CMV infections has been shown in some CMV mononucleosis patients, pregnant women, and kidney and cardiac transplant patient.s	CMV is a Herpes virus. It is usually a subclinical infection but remains latent within bone marrow cells. It may manifest as a mononucleosis-type syndrome with fever, malaise, and lymphadenopathy.
	CMV IgG	Positive CMV IgG indicates past or recent CMV infection. Patients may transmit CMV to susceptible individuals through blood and tissue products	Past infection
	EBV VCA IgM	Three components: VCA IgG, VCA IgM, and EBNA. Presence of VCA IgM antibodies indicates recent primary infection with EBV. The presence of VCA IgG antibodies indicates infection sometime in the past.	EBV infection status
	EBV VCA IgG		
	EBV EBNA IgG		

APPROPRIATE SEROLOGIC TESTS FOR THE DIAGNOSIS AND MANAGEMENT OF ORAL, FACIAL, AND HEAD PAIN

Test name	Description	Interpretation	Possible indications
Relevant viral evaluation *(continued)*	Hepatitis B DNA	Presence indicates active hepatitis B infection	Assessment of viral hepatic disease
	Anti-HBs	Appears some weeks after infection in naturally occurring infections, after HBsAg disappears	
	HBsAg	Detection of presence is usually first marker	
Drug levels	Carbamazepine	Levels correlate with antiepileptic effect but no data on antineuralgic effects	Carbamazepine therapy; compliance and absorption
	Lithium	Narrow therapeutic window; testing is needed	Cluster headache prophylaxis
Lyme serology	First tier	Lyme testing is now recommended as a two-stage (or tier) process that has been shown to have higher accuracy.	In patients with suspected exposure to animal vector; symptoms may be vague.
	IgG/IgM ELISA using a whole cell lysate		
	Second tier		
	IgG/IgM ELISA targeting specifically VlsE1 and pepC10 antigens		
	Borrelia burgdorferi (North American), *B burgdorferi, B afzelii and B garinii* (European)		
	ELISA testing (PCR in CSF, synovial fluids)		
Pharmacogenomic testing	Available for antidepressants	eg, the GeneSight test examines the transporter and receptor gene profile and has the ability to guide drug choice. Some other medications include carbamazepine, warfarin, tamoxifen, and abacavir.	Slightly in the future but approaching fast—pharmacogenomic testing will be available to assist clinicians in choosing medications for pain management (antidepressants already available). Examine risk and metabolism of carbamazepine

AFP = alfa fetoprotein; ALT = alanine aminotransferase; Anti-HB = hepatitis B surface antibody; ASCA = antisaccharomyces cerevisiae antibody test; AST = aspartate aminotransferase; BMS = burning mouth syndrome; C-ANCA = C-antineutrophil cytoplasmic antibodies; CBC = complete blood count; CA = carbohydrate antigen; CCP = cyclic citrullinated peptide antibody; CEA = carcinoembryonic antigen; CMV = cytomegalovirus; CRP = C-reactive protein; CSF = cerebrospinal fluid; CTD = connective tissue disease; DIC = disseminated intravascular coagulation; e/mGFR = estimated/measured glomerular filtration rate; EBNA = Epstein-Barr nuclear antigen; EBV = Epstein-Barr virus; ELISA = enzyme-linked immunosorbent assay; ESR = erythrocyte sedimentation rate; GGT = gamma-glutamyl transferase; GH = growth hormone; GI = gastrointestinal; HbA1c = glycated hemoglobin; HBSAg = hepatitis B surface antigen; HCC = hepatocellular carcinoma; HLA = human leukocyte antigen; Ig = immunoglobulin; INR = international normalized ratio (for PT); LDH = lactate dehydrogenase; LDL = low-density lipoproteins; MCTD = mixed connective tissue disease; MI = myocardial infarction; P-ANCA = P-antineutrophil cytoplasmic antibodies; PBC = primary biliary cholangitis; PSA = prostate-specific antigen; PT(T) = prothrombin (time); RhF = rheumatoid factor; Scl = scleroderma; SSB = anti-Sjögren syndrome type B; TSH = thyroid stimulating hormone; TACs = trigeminal autonomic cephalalgias; VCA = viral capsid antigen; VLDL = very low–density lipoproteins.

↑ High levels

↓ Low levels