Cranio-cervical-mandibular disorders and oro-cervical-facial pain: a classification based on anatomical units

Raúl E. Frugone-Zambra[1]
Edgar Molina Gómez[1]
Alfredo Gary Bufadel[2]
Diego A. Valenzuela Sepúlveda[3]
Daniel Correa Dufey[1]
Andrea Berzaghi[4]
Sergio Bortolini[4]

[1] Health and Dentistry Faculty, University Diego Portales, Santiago, Chile.
[2] Faculty of Science, University of La Serena, La Serena, Chile.
[3] Department of Dentistry, Community Health Service Villa Alegre Family Health Center, Villa Alegre, Chile.
[4] Department of Surgery, Medicine, Dentistry and Morphological Sciences with Interest in Transplant, Oncology and Regenerative Medicine, University of Modena and Reggio Emilia (UNIMORE), Modena, Italy.

Corresponding author: Andrea Berzaghi
email: andrea.berzaghi@unimore.it

Abstract

Objective: To develop an integrated classification of cranio-cervical-mandibular disorders (CCMD) and oro-cervical-facial pain (OCFP), based on anatomical units, to allow for a more precise and useful clinical approach.
Method: A multicenter study was conducted involving 8 clinical specialists from Chilean and Italian universities, as well as 14 students serving as collaborators. The methodology involved organizing the participants into subgroups and holding periodic discussion sessions. Successive drafts were produced until a final classification was agreed upon. Once completed, each included pathology was defined.
Results: The classification is organized into four hierarchical levels: (1) anatomical structure/origin (muscular, osteoarticular, nervous/neurovascular, and associated structures), (2) pathogenetic mechanism (pain/inflammation/degeneration, dysfunction/mobility, growth/development/genetics), (3) pathophysiology, and (4) associated local or systemic pathologies. A list of specific conditions that can affect the suprascapular unit was also included.
Conclusion: The proposal addresses shortcomings in previous organizations by establishing a clear, logical, and hierarchical structure. This first integrative classification favors a more precise, inferential organization of cranio-cervical disorders (CCD) and temporomandibular disorders (TMD), including OCFP. It offers an initial framework that is susceptible to future expansion and adjustments. It also represents a step towards standardizing diagnoses and optimizing treatments in this area.

Key words: Temporomandibular disorders; Cranio-cervical disorders; Orofacial pain; Cervical pain; Articular pain.

Introduction

The temporomandibular and cranio-cervical units are integrated anatomically and neurophysiologically (1,2). Indeed, pathology in the area above the shoulder belt often affects both units (3). As temporomandibular disorders (TMD) and cranio-cervical

How to Cite

Raúl E. Frugone-Zambra, Edgar Molina Gómez, Alfredo Gary Bufadel, Diego A. Valenzuela Sepúlveda.
Cranio-cervical-mandibular disorders and oro-cervical-facial pain: a classification based on anatomical units.
Annali Di Stomatologia, 17(1),120-147. https://doi.org/10.59987/ads/2026.1.120-147

10.59987/ads/2026.1.120-147

disorders (CCD) are related and various pathologies converge, specific diagnoses are required to manage the problem optimally. In relation to diagnosis, the first concept to understand when analyzing or generating a classification is 'taxonomy'. Taxonomy is the science of classification. It is based on organizing groups into a specific, exclusive hierarchy, meaning that each classified element cannot be integrated into two different hierarchies. Thus, taxonomy is a systematic organization of categories based on common characteristics. Therefore, each element in a group will have similarities with the others (4). Diagnosis is an inferential process based on the analysis of signs and symptoms (neither a sign nor a symptom should be considered a diagnosis). It relies on the clinical presentation of a disease. It determines the disease, which must be defined within a biological, psychosocial, and environmental context (5,6).

The International Classification of Diseases (ICD) is the classification that encompasses nearly all existing pathologies. It has several versions (7 and 8). The ICD provides an index of all pathologies and is divided into 22 chapters (9). Chapter 12 classifies diseases of the skin and subcutaneous tissues, while Chapter 13 classifies diseases of the musculoskeletal system and connective tissue. Diseases of the skin and subcutaneous tissues include diseases of the oral cavity and salivary glands, as well as cheilitis, abscesses, cellulitis, and lesions associated with autoimmune diseases. Diseases of the musculoskeletal system and connective tissue include arthropathies, dentofacial abnormalities, systemic connective tissue disorders, dorsopathies, soft tissue disorders (including muscles and synovium), osteopathies and chondropathies, other musculoskeletal and connective tissue disorders, intraoperative complications and disorders, periprosthetic fractures, and biomechanical injuries that are not classified under this heading. Among the classifications that include temporomandibular disorders (TMD), there are two groups: a) temporomandibular disorders and orofacial pain (TMD/OFP) classifications and b) TMD classifications (Table 1).

The International Classification of Orofacial Pain (ICOP) includes seven domains, two of which are directly associated with temporomandibular disorder-related pain (10). The first domain, 'Orofacial Pain', refers to dentoalveolar, mucosal, salivary gland, and mandibular bone pain. The fourth domain addresses diagnoses related to pain attributed to lesions of the trigeminal and glossopharyngeal nerves. The fifth domain refers to primary headaches, including orofacial migraine, tension-type headache, trigeminal autonomic orofacial pain (including cluster headaches and hemifacial paroxysmal headaches), and neurovascular orofacial pain. The sixth domain addresses pathologies involving idiopathic orofacial pain, such as burning mouth syndrome, persistent idiopathic facial pain, and persistent unilateral facial pain with additional attacks. In terms of specific temporomandibular pathology, this classification system recognizes two distinct categories: one focusing on orofacial myofascial pain, and the other addressing TMJ pain. Both of these are subdivided into primary and secondary pain. Primary pain is considered a disease in its own right, with multifactorial causes. Idiopathic pain, on the other hand, is a symptom for which no definitive cause can be identified. Secondary pain is defined as pain resulting from or caused by another condition. In the case of muscle pathology, secondary pain arises from myofascial structures and tendons. It is indicated that myofascial pain may be attributed to tendinitis, myositis, or spasms. These data allow the determination of

Table 1. The most commonly used classifications involve TMD/OFP.

ICOP 2020	IC -11	BELL
Orofacial pain	Secondary musculoskeletal pain	Muscle disorders
Myofascial pain	Neuropathic pain	Tmj disorders
Tmj pain	Pain secondary to headache	Disorders with hypo-mobility
Pain of neurological origin		Growth disorders
Pain due to primary headache		
Idiopathic orofacial pain		
Pain associated with psychosocial factors		
AAOP	**PECK**	**OKESON**
Temporomandibular disorders	Tmj disorders	Muscle disorders
Neuropathic pain	Muscle disorders	Tmj disorders
Pain secondary to headache	Secondary headache	Disorders with hypo-mobility
Cervicogenic pain in headache	Disorders of accessory structures	Growth disorders

whether the etiology is inflammatory due to overload, infectious, or a central response to a noxious agent, such as spasm. Regarding joint pain, the pathology is characterized by inflammation from various causes, tissue sensitization, and structural changes. This domain includes pain associated with arthritis (i.e., joint inflammation), whether systemic or non-systemic. Non-systemic pain does not reveal possible non-systemic causes; therefore, the diagnosis is non-specific. The following section presents the pathology associated with disc displacement, degenerative disease, and subluxation. The ICD-11 is the most recent version of the International Classification of Diseases (ICD), and it is highly accurate for coding primary and secondary chronic pain. This version includes diagnostic codes for chronic pain conditions, but, according to Treede et al., it is not systematically categorized (10). The relevant clinical disorders are classified into seven groups: primary chronic pain; chronic cancer-associated pain; chronic post-traumatic and post-operative pain; chronic neuropathic pain; chronic headaches and orofacial pain; chronic visceral pain; and chronic musculoskeletal pain. In relation to the TMD/OFP specialist area, the conditions involved are chronic neuropathic pain, chronic headaches, orofacial pain, and chronic musculoskeletal pain. However, TMD/ODP are most prevalent in the chronic headache and orofacial pain group. This group includes chronic headaches and orofacial pain caused by vascular and non-vascular cranial or cervical disorders, substances such as medications, and disorders of the skull, neck, eyes, ears, nose, sinuses, teeth, mouth, and other facial or cervical structures. Referred chronic pain to the teeth is also described, as are chronic neuropathic orofacial pain and chronic pain secondary to temporomandibular disorders. Bell's pain classification considers two inputs: one somatosensory and the other psychosocial. These inputs generate a condition based on two levels, or axes: one representing the factors responsible for the nociceptive stimulus (axis I), and the other representing the psychological factors that influence the pain experience (axis II) (11). Axis I identifies: 1. Cutaneous and mucogingival pain. 2. Pharyngeal, nasal, and paranasal mucosal pain. 3. Dental pain. 4. Pain in the musculoskeletal structures of the mouth and face. 5. Pain in the visceral structures of the mouth and face. 6. Pain in the neural structures of the mouth and face. Axis II identifies: 1. Anxiety disorders. 2. Mood disorders. 3. Somatoform disorders. 4. Other conditions, such as psychological factors that affect medical conditions. The American Academy of Orofacial Pain (AAOP) classification of orofacial pain (12,13) includes a subclassification of musculoskeletal disorders of the temporomandibular joint (TMD). The full classification considers: 1. Vascular and nonvascular intracranial pain disorders. 2. Primary headaches. 3. Neuropathic pain. 4. Intraoral pain disorders. 5. Temporomandibular disorders. 6. Cervical pain disorders. 7. Orofacial pain of extracranial and systemic causes. As we can see, item 5 covers temporomandibular disorders and uses the classifications provided by the DC/TMD and the expanded TMD taxonomy published by Peck C. (14). This subclassification is no longer exclusively related to pain, providing an approach to integrating painful and non-painful temporomandibular disorders into a single structure. Additionally, the AAOP classification incorporates diagnoses involving TMD/OFP specialists, such as headaches, neuropathic pain, and neck pain disorders. Peck's expansive taxonomy comprises four domains that lack a specific hierarchy (15). These include temporomandibular joint disorders, masticatory muscle disorders, TMD-associated headaches, and associated structures. J. Okeson's classification, adapted from his work "Orofacial Pain: Guidelines for Assessment, Diagnosis and Management (third edition, 1996)", is used to diagnose TMD and includes pathologies not addressed in other classifications (15). It presents four domains: masticatory muscle disorders; temporomandibular joint disorders; chronic mandibular hypomobility; and growth disorders. The International Classification of Diseases (ICD) also includes pathologies of the cranio-cervical unit in chapter 13. This chapter covers diseases of the musculoskeletal system and connective tissue, including arthropathies, systemic connective tissue disorders, dorsopathies, soft tissue disorders, osteopathies, chondropathies, and other unclassified biomechanical disorders, as well as complications, fractures, and injuries. In parallel, there are some more specific classifications. These include the 2021 World Health Organization (WHO) classification, the International Statistical Classification of Diseases and Related Health Problems (ICD), and the International Classification of Functioning, Disability and Health (ICF). The WHO proposes a first-level classification based on anatomical structures, including joints, bones, muscles, the spine, and various body systems or regions. The ICD proposes five pathologies: 1. Cervical pain. 2. Thoracic spine pain. 3. Headaches. 4. Spondylosis with radiculopathy. 5. Cervical disc disorders with radiculopathy. The ICF proposes a simple classification for the cervical region that includes four pain-related lesions: 1. Neck pain with movement disturbance. 2. Neck pain with headaches. 3. Neck pain with coordination disturbance. 4. Neck pain with a referred pattern. Our study aims to develop an integrated classification of cranio-cervical-mandibular disorders and oro-cervical-facial pain (CCMD/OCFP) based on anatomical units. This will allow for a more precise and useful clinical approach.

Methods

A multicenter group study was conducted by seven professionals (five dentists and two physiotherapists) from Universidad Diego Portales (Chile), the University of Modena e Reggio Emilia (Italy), and Universidad de La Serena (Chile). The study aimed to develop a classification of cranio-cervical-mandibular disorders and oro-cervical-facial pain based on anatomical units, following a precise hierarchy. The participants were three temporomandibular disorder specialists, two physiology and occlusion experts, two maxillofacial kinesiology and cranio-cervical-mandibular disorder experts, two public health experts, and 14 students from Diego Portales University in Chile specializing in temporomandibular disorders. The working group

10.59987/ads/2026.1. 120-147

met periodically in a hybrid format (in person and via streaming). Before the first meeting to develop this integrated classification, a session was held to define concepts and operational definitions and to address the study objectives. Following this, the participants met in affinity groups and spent a week developing the classification. Each group presented its draft classification for discussion, after which the final draft was written. This draft was distributed to all participants for evaluation and, once the necessary changes had been defined, the final classification was produced. Once completed, each pathology was briefly defined (Annex 1, inserted at the end of the article).

Results

According to the applicable taxonomic guidelines, the classification was organized into four hierarchies: affected anatomical structure; general mechanism involved in pathogenesis (with or without a genetic component); pathophysiology associated with each mechanism; and local pathologies (which were further subdivided as necessary). The following were defined according to the affected anatomical structure: 1) Muscular. 2) Osteoarticular. 2A) Temporomandibular joint. 2B) Vertebral/articular component. 3) Nervous and neurovascular component. 4) Associated structures. Three categories were defined for each anatomical group, based on mechanisms: 1) Pain/inflammation/degenerative diseases. 2) Dysfunction/mobility. 3) Growth/development/metabolism. Pathophysiology focuses on defining the processes involved in each mechanism to enable the diagnosis of specific pathologies. This exercise aimed to determine an appropriate management strategy for each pathology. The classification developed is shown in Table 2. Each pathology is then defined separately and presented in alphabetical order. Systemic pathologies that may manifest in structures above the shoulder girdle are listed in Table 3.

Table 2. Cranio-cervical-mandibular Disorders and Oro-Cervical-Facial Pain Classification

ANATOMICAL STRUCTURE / ORIGIN	MECHANISM (WITH OR WITHOUT GENETIC COMPONENT)	PHYSIOPATHOLOGY	LOCAL PATHOLOGIES	SPECIFIC PATHOLOGIES
1) MUSCULAR	1.1 Pain/inflammation/degenerative disesases	1.1.1 Miositis by overloading	1.1.1.1 Local myalgia	1A1.1.1.a Pain induced by extreme exercise
				1A1.1.1.b Delayed onset muscle soreness
				1A1.1.1.c Pain associated to injuries
			1.1.1.2 Myofascial pain	
			1.1.1.3 Myofasical pain with referral	
		1.1.2 Miositis by infection	1.1.2.1 Bacterial myositis	
		1.1.3 Miositis by other external agents	1.1.3.1 Viral myositis	
			1.1.3.3 Parasitic Myositis	
	1.2 Dysfunction/ mobility	1.2.1 Stiffnes/rigidity	1.2.1.1 Myostatic contracture	
			1.2.1.2 Myofribrotic contracture	
			1.2.1.3 Myospasmodic contracture	
	1.3 Growing/ development	1.3.1 Under development	1.3.1.1 Atrophy	
			1.3.1.2 Sarcopenia	
		1.3.2 Over development	1.3.2.1 Hypertrophy	
			1.3.2.2 Neoplasia	

To be continued

2) OSTEOARTICULAR				
2A) TMJ	2A.1 Pain/inflammation/degenerative disesases	2A1.1 Arthritis	2A.1.1.1 Synovitis	
			2A.1.1.2 Capsulitis	
			2A.1.1.3 Retrodiscitis	
			2A.1.1.4 Of other intracapsular ligaments	
		2A.1.2 Degenerative disease (DD)	2A.1.2.1 ED - osteoarthritis/osteoarthrosis	
			2A.1.2.2 Idiopathic condylolysis	
			2A.1.2.3 Avascular necrosis	
			2A.1.2.4 Osteochondritis dissecans	
			2A.1.2.5 Synovial chondromatosis	
	2A.2 Dysfunction/ mobility	2A.2.1 By interference	2A.2.1.1 Disc displacement with reduction	
			2A.2.1.2 Disc displacement without reduction with limited opening	
			2A.2.1.3 Disc displacement without reduction without limited opening	
			2A.2.1.4 Disc displacement with intermittent locking reduction	
			2A.2.1.5 Opening lock	
		2A.2.2 By fixation	2A.2.2.1 Disc adhesion	
			2A.2.2.2 Sticky or static disc	
			2A.2.2.3 Capsular fibrosis	
			2A.2.2.4 Ankylosis	2A.2.2.4.a Fibrous
				2A.2.2.4.b Osseous
		2A.2.3 Due to hypermobility	2A.2.3.1 Hypertranslation	
			2A.2.3.2 Dislocation	2A.2.3.2.a Subluxation
				2A.2.3.2.b Luxation
		2A.2.4 Due to morphological alteration	2A.2.4.1 Disc deformation	
			2A.2.4.2 Disc perforation	
		2A.2.5 Due to postural alteration	2A.2.5.1 Condylar positional asymmetry	

To be continued

10.59987/ads/2026.1. 120-147

	2A.3 Growing/development	2A.3.1Lack of development	2A.3.1.1 Condylar agenesis	
			2A.3.1.2 Condylar aplasia	
			2A.3.1.3 Condylar hypoplasia	
		2A.3.2 Overdevelopment	2A.3.2.1 Condylar hyperplasia	
			2A.3.2.2 Joint neoplasia	
		2A.3.3 Fractures	2A.3.3.1 Mandibular	
			2A.3.3.2 Condylar	
2B) VERTEBRAL/ ARTICULAR	2B.1 Pain/inflammation/degenerative disesases	2B.1.1 Arthritis	2B.1 1.1Facet synovitis (cervical facet syndrome)	
		2B.1.2 Degenerative disease (DD)	2B.1.2.1 Spondylosis	
			2B.1.2.2 Disc disruption	
			2B.1.2.3 Herniated disc	
	2B.2 Dysfunction/ mobility	2B.2.1 Displacements	2B.2.1.1Atlas rotation	
			2B.2.1.2 Axis rotation	
			2B.2.1.3 Spondylolisthesis	
		2B.2.2 Fixings	2B.2.2.1 Atlas-occipital	
		2B.2.3 Postural alteration	2B.2.3.1 Of cervical curvature	2B.2.3.1.a Cervical spine rectification
				2B.2.3.1.b Inversion of the cervical spine
				2B.2.3.1.c Hyperlordosis of the cervical spine
			2B.2.3.2 Of the hyoid system	2B.2.3.2.a Hyoid bone elevation
	2B3. Growing/development	2B.3.1 Lack of development	2B.3.1.1 Vertebral agenesis	
			2B.3.1.2 Vertebral aplasia	
			2B.3.1.3 Vertebral hypoplasia	
		2B.3.2 Overdevelopment	2B.3.2.1 Vertebral hyperplasia	
			2B.3.2.2 Neoplasms of the cervical spine	
		2B.3.3 Fractures	2B.3.3.1Fractures of the vertebral bodies	
			2B.3.3.2 Fractures of the vertebral arches	

To be continued

			2B.3.3.3 Fractures of the vertebral processes	
3) NEURAL AND NEUROVASCULAR COMPONENT	3.1 Pain/inflammation/degenerative disesases	3.1.1 Episodic neuropathic pain	3.1.1.2 Neuralgia	3.1.1.2.a Trigeminal neuralgia
				3.1.1.2.b Glossopharyngeal neuralgia
		3.1.2 Continuous neuropathic pain	3.1.2.1 Peripherally mediated neuropathy	
			3.1.2.2 Deafferentation neuropathy	3.1.2.2.a Neuropathy of dento-alveolar origin
				3.1.2.2.b Disestesia oclusal
				3.1.2.2.c Burning mouth syndrome
			3.1.2.3 Centrally mediated neuropathy	3.1.2.3.a Complex regional pain syndrome
				3.1.2.3.b Post-herpetic neuralgia
			3.1.2.4 Primary headaches	3.1.2.4.a Migraine
				3.1.2.4.b Tension-type headache
				3.1.2.4.c Trigeminal-autonomic headaches - cluster headache
				3.1.2.4.d Trigeminal autonomic headache - paroxysmal
				3.1.2.4.e SUNCT Unilateral neuralgiform headache with watering and redness
				3.1.2.4.f SUNA Unilateral neuralgiform headache with autonomic symptoms
	3.2 Dysfunction/ mobility	3.2.1 Central defense responses	3.2.1.1 Myospasm	
			3.2.1.2 Protective co-contraction	
	3.3 Growing/development	3.3.1 Structural malformations	3.3.1.1 Neoplasms	
4) ASSOCIATED STRUCTURES	4.1 Pain/inflammation/degenerative disesases	4.1.1 Inflammation of the temporomandibular ligament		
		4.1.2 Temporal tendonitis		
		4.1.3 Sialadenitis		
		4.1.4 Parotitis		

To be continued

10.59987/ads/2026.1. 120-147

	4.2 Dysfunction/ mobility	4.2.1Cervical injuries associated with the hyoid		
		4.2.2Elevation of the hyoid		
	4.3 Growing/develop-ment	4.3.1 Eagle syndrome		
		4.3.2 Ernest Syndrome		

Table 3. Cranio-cervical-mandibular Disorders and Oro-Cervical-Facial Pain associated with systemic diseases.

ANATOMICAL STRUCTURE / ORIGIN	MECHANISM (WITH OR WITHOUT GENETIC COMPONENT)	PHYSIOPATHOLOGY	LOCAL PATHOLOGIES
1) MUSCULAR	1.1 Pain/inflammation/degene-rative diseases	1.1.1 Due to myositis caused by systemic factors	1.1.1.1 Dermatomyositis
			1.1.1.2 Polymyalgia rheu-matica
	1.2 Dysfunction/mobility	1.2.1 Due to weakness caused by systemic factors	1.2.1.1 Myotonic muscular dystrophy type 1
			1.2.1.2 Facioscapulohume-ral muscular dystrophy
			1.2.1.3 Oculopharyngeal muscular dystrophy
			1.2.1.4 Mitochondrial myo-pathy
			1.2.1.5 Mitochondrial ence-phalomyopathy
		1.2.2 Motor disorders	1.2.2.1 Oromandibular dy-stonia
			1.2.2.2 Orofacial dyskinesia
	1.3 Growing/development	1.3.1 Due to endocrine myopathies	1.3.1.1 Hyperthyroidism
			1.3.1.2 Hypothyroidism
			1.3.1.3 Hyperparathyroidism
			1.3.1.4 Hypoparathyroidism
			1.3.1.5 Cushing's syndrome
			1.3.1.6 Addison's disease
2) OSTEOARTICULAR	2A.1 Pain/inflammation/dege-nerative diseases	2A.1.1 For underlying rheumatoid arthritis	
2A) TMJ		2A.1.2 Due to underlying psoriatic arthritis	
		2A.1.3For underlying Lyme disease	
		2A.1.4 Per gout of base	

To be continued

		2A.1.5 Due to underlying ankylosing spondylitis	
	2A.2 Dysfunction/mobility	2A.2.1 Due to underlying systemic hypermobility	
	2A.3 Growing/development	2A.3.1 For systemic rheumatic diseases	
2B) VERTEBRAL/ARTICULAR	2B.1 Pain/inflammation/degenerative diseases	2B.1.1 For rheumatoid arthritis	
		2B.1.2 Due to spondyloarthritis	
		2B.1.3 For systemic lupus erythematosus	
		2B.1.4 Due to Sjoegren's syndrome	
		2B.1.5 Due to scleroderma	
		2B.1.6 For Behcet's disease - vasculitis	
		2B.1.7 For relapsing polychondritis	
	2B.2 Dysfunction/mobility	2B.2.1 Due to underlying systemic hypermobility	
	2B3. Growing/development	2B.3.1 For ankylosing spondylitis	
		2B.3.2 Due to Scheuermann's disease	
		2B.3.3 For Jarcho-Levin Syndrome	
		2B.3.4 In association with VACTERL	
		2B.3.5 Due to Paget's disease	
		2B.3.6 For metabolic diseases	
		2B.3.7 Due to systemic sclerosis	
3) NEURAL AND NEUROVASCULAR COMPONENT	3.1 Pain/inflammation/degenerative diseases	3.1.1 Fibromyalgia	
		3.1.2 Myofascial pain syndrome	
	3.2 Dysfunction/mobility	3.2.1 Congenital torticollis	
	3.3 Growing/development	3.3.1 Due to congenital anomalies	3.3.1.1 Hydrocephalus
		3.3.2 For genetic disorders	3.3.2.1 Down syndrome
			3.3.2.2 Rett syndrome
			3.3.2.3 Fragile X chromosome syndrome

To be continued

10.59987/ads/2026.1. 120-147

		3.3.3 For metabolic disorders	3.3.3.1 Enzyme deficiencies
		3.3.4 Due to infections	3.3.4.1 Meningitis during pregnancy or childhood
			3.3.4.2 Encephalitis during pregnancy or childhood
		3.3.5 For neoplasias	
4) ASSOCIATED STRUCTURES	4.1 Pain/inflammation/degenerative diseases	4.1.1 For rheumatoid arthritis	
		4.1.2 For lupus erythematosus	
		4.1.3 Due to scleroderma	
		4.1.4 For connective tissue diseases	
	4.2 Dysfunction/mobility	4.2.1 For Duchenne muscular dystrophy	
		4.2.2Due to Pierre Robin syndrome	
		4.2.3 Due to obstructive apnea	
	4.3 Growing/development	4.3.1 For ankylosing spondylitis	
		4.3.2 Due to Forestier's disease	

Discussion

This classification is derived from the rules of taxonomy, which essentially organize groups into a hierarchical structure (4,5,6). Diagnosis is an inferential process based on clinical symptoms, aimed at defining a disease. These signs and symptoms must be specific and defined in the context of the environment. Based on this definition, it is impossible to include signs or symptoms as a diagnosis. Examples of this include bruxism, secondary pain (e.g., headaches resulting from muscle disorders), painful conditions that are not properly considered diseases (e.g., migraines and cluster headaches), arthralgia, and myalgia. A clear diagnosis should inform the clinician's choice of treatment or management, so diagnostics must consider the disease's etiology. For example, in the case of arthralgia derived from an inflammatory process (e.g., arthritis), if information about the arthralgia is lacking, a clear diagnosis will not be possible, and an effective treatment will not be defined. Conversely, if the diagnosis is capsulitis rather than arthralgia or arthritis, the clinician has more resources to infer causality: capsule pain on stretching. Therefore, the clinician should identify the cause of the stretching, which may be microtrauma within the stomatognathic system or external trauma. Once the cause is found, the treatment can be correctly applied. Classifications based on pain include some painful temporomandibular disorders, but do not classify these diseases themselves (10, 11). TMD may or may not be associated with pain. If they are associated, they are generally part of nociceptive or nociplastic pain (17). Therefore, TMD and CCD should be classified together as local diseases because they share many signs and symptoms, including muscle pain. In this respect, a new classification has been created that includes TMD, CCD, and pain.

Regarding the two major and useful classifications of TMD in the literature, several issues need to be addressed. Peck's taxonomy includes four domains that do not follow an explicit hierarchy (15). The articular, muscular, and other structures form an anatomical division, but secondary headache is neither an anatomical structure nor a pathology. Additionally, there are inconsistencies in the definition of a diagnosis, as arthralgia and myalgia are symptoms. Consequently, it is impossible to make a differential diagnosis or propose a treatment. What is the result? Treatments are advocated for the symptomatic management of pain rather than addressing the cause. This problem is solved by the Okeson classification (12), which includes capsulitis, synovitis, and retrodiscitis as inflammatory diseases that cause articular pain (arthralgia), rather than arthralgia or arthritis. Although the expanded TMD taxonomy is derived from reliable diagnostic criteria with proven criterion-related validity, many diseases have not been included, even though they exist. Also, some general diseases or pathophysiological conditions that may affect the temporomandibular structures are included; they cannot be placed within a hierarchy.

On the other hand, the Okeson classification, adapted from Orofacial Pain: Guidelines for Assessment, Diagnosis and Management (3rd ed., 1996) (12), integrates many pathologies into a hierarchy based on a common characteristic. However, it suffers from the same problem: two anatomical domains, one concerning hypomobility disorders and the other including growth and developmental disorders. This classification's greatest contribution is that it includes some prevalent disorders that are unclassified in other classification systems, such as protective co-contraction, synovitis, capsulitis, and retrodiscitis, as well as disorders classified under hypomobility. As a first approach, integrating and applying both classification systems, alongside those based on pain, in a hierarchical, criterion-based manner can enable a structured classification that includes all pathologies. This study aimed to achieve this, as well as to integrate CCD to generate a universal classification of the suprascapular unit. Information about classifications of cranio-cervical units is scarce and limited, but fortunately, they share all characteristics of TMD. Muscles and joints are common structures with common disorders. There may be some anatomical differences, but the structures, mechanisms, and physiopathology are similar. For this reason, the authors decided to create a common classification of cranio-cervical-mandibular disorders and oro-cervical-facial pain.

Conclusion

This classification of cranio-cervical-mandibular disorders and orocervical-facial pain (CCMD/OCFP) categorizes various local disorders affecting defined anatomical units, including a sub-classification of systemic disorders that impact them. As this is the first integrative classification, it can be modified according to other criteria while maintaining the structure that a diagnostic classification must have, a hierarchical development.

Acknowledgments

The authors want to appreciate our students of the Specialty Program on Temporomandibular Disorders and Orofacial Pain for their excellent contribution. Daniela Aravena DDS, Raúl Acosta. DDS, Daniela Coronado DDS, María Fernanda Durán. DDS, Paulina Espinoza. DDS, Mónica Farías. DDS, Ljubica Malinarich DDS, Viviana Moya DDS, Miguel Perez DDS, Diego Salas DDS, Francisca Urrizola DDS, Nicolás Varela DDS, Ignacio Vásquez DDS, Laura Wistuba DDS.

Funding

This research received no external funding.

Conflicts of Interest

The authors declare no conflicts of interest.

Annex 1.

Pathology	Definition
Ankylosis (fribrous and osseous)	Limited condylar movement, which can result in impaired mouth opening, may be caused by fibrous or bony ankylosis of the temporomandibular joint. This injury can result from trauma, infection or autoimmune diseases (19). However, condylar trauma is widely regarded as one of the primary causes of ankylosis (20). Regarding bony ankylosis, osteogenesis has been observed to occur at the junction between the ankylosed cartilage tissue and the bone segment, primarily through endochondral ossification (21).
Atlas rotation	The atlas, the first cervical vertebra, allows flexion and extension of the skull. Through its articulation with the axis, it also enables cranial rotation. The kinematics of the cervical spine and vertebral morphology are closely related (22,23). Cranial flexion and extension occur because the superior articular facets of the atlas are concave in both directions, accommodating the occipital condyles. The atlantoaxial joint, located between the odontoid process of the axis and the anterior arch of the atlas, enables 50–60% of cervical rotation. This joint has three axes of movement with three degrees of freedom, and its wide range of rotational movement is notable. There are no intervertebral discs between the occipital bone and the atlas or between the atlas and the axis. When the odontoid process is normal, the movement of the second cervical vertebra (C2) anteriorly is restricted by the anterior arch of the first cervical vertebra (C1). Posterior movement is limited by the cruciate ligament, primarily the transverse ligament. This unit can accommodate alterations in the position of the atlas, which manifests as a rotation that is quite benign compared to a rare subluxation of the atlas (24). Loss of stability between the atlas and the axis can result from macrotrauma, such as whiplash, or microtrauma associated with poor posture and asymmetric muscle activity. This misalignment can lead to upper cervical pain and altered cranial rotation. If the atlas rotates and misaligns to one side, the alar ligaments will tighten on the side opposite the rotation, thereby impeding proper ipsilateral rotation of the skull. Furthermore, propulsion of the atlas can lead to relative distalisation of the axis, causing the odontoid process to press on the spinal cord and result in pain and/or headaches. However, the consequences of a subluxation can be more complex as it involves the vertebral artery.
Atlas-occipital fixing	This injury does not necessarily correspond to ankylosis, a condition involving the fusion of joints. Rather, it represents an instability characterized by a significant reduction in the range of motion between the two vertebrae. This can be caused by trauma, degenerative disease or congenital anomalies, resulting in pain and impaired mobility in the craniocervical region. A rare condition called traumatic atlantoaxial rotatory fixation can also present as atraumatic and may be present alongside congenital torticollis (26,27).

To be continued

10.59987/ads/2026.1. 120-147

Avascular necrosis	Firstly, we believe it is important to clarify the concept of bone marrow edema, given its association with avascular necrosis. This term is used to describe changes in imaging signal intensity that could be attributed to various underlying pathologies. Bone marrow edema is associated with pain, dysfunction and progressive cartilage damage. Detecting it is essential as its presence is associated with pain, decreased function, cartilage damage, structural deterioration, accelerated osteoarthritis and rapid progression to total joint arthroplasty (28). The most common causes of bone marrow edema are reactive bone marrow edema in response to processes such as infections, osteomyelitis, inflammatory arthritis or tumors; ischemic bone marrow edema, such as osteonecrosis and osteochondritis dissecans; and mechanical bone marrow oedema, including joint misalignment, meniscal or ligament injury, contusions or fractures (29). Osteoarthritis is often associated with bone marrow edema, increased fluid levels and pain (30). Avascular necrosis is a condylar lesion resulting from impaired blood flow to the condyle due to factors such as trauma, sickle cell anemia or systemic lupus erythematosus. On a CT scan, the mandibular condyle appears deformed with subchondral sclerosis. On an MRI scan, the bone marrow appears dark on T1-weighted images and shows a mixed signal on T2-weighted images (31).
Axis rotation	The axis may also undergo positional changes and become unstable, resulting in neck pain, headaches and vestibular symptoms. Rotation of the axis can result in the complete rotation of the atlas-axis complex, which restricts ipsilateral cranial rotation.
Bacterial myositis	It is an inflammatory pathology of infectious origin that occurs following events such as injury, surgery or ischemia. As it can spread from neighboring sites of infection or via the bloodstream, it is categorized as a systemic muscle pathology.
Burning mouth syndrome	Burning mouth syndrome is defined as a recurring, daily burning or dysesthetic sensation in the mouth, lasting more than two hours per day for at least three months, with no clinically evident underlying cause (32). Various mechanisms have been proposed to explain the condition, including those involving neural pathways, immunological factors, stress, hormones and neuropsychiatric factors, among others (33). Notably, it has a dual etiology involving both peripheral nerve dysfunction and central sensitization. Although it involves the principles of nociplastic pain, this term should be applied with caution to avoid oversimplification (34).
Capsular fibrosis	Capsular fibrosis of a joint is characterized by the excessive differentiation and proliferation of myofibroblasts, and the abnormal secretion and accumulation of the extracellular matrix. Following trauma, the joint capsule contracts (35).
Capsulitis	The term ‘capsulitis’ is usually used alongside ‘synovitis’, perhaps because the inner layer of the joint capsule is the synovial membrane. However, unlike synovitis, capsulitis responds to capsule elongation, suggesting that their etiology is somewhat different. Indeed, capsulitis is more frequently associated with occlusal contacts that result in excessive stretching of the capsule, whereas synovitis is associated with compressive overload of the synovial tissue caused by the articular condyle (36).
Cervical injuries associated with the hyoid	Cervical injuries associated with the hyoid bone refer to traumatic damage involving both the anterior mid-neck skeleton (notably the hyoid bone) and the cervical spine and its adjacent soft tissue structures. Such injuries frequently result from high-force mechanisms (e.g. blunt anterior neck impact, hyperextension of the neck, strangulation or hanging), which transmit force through the hyoid or cervical segments. As the hyoid bone is located at approximately the level of the C3 vertebra and is positioned between the mandible at the front and the cervical spine at the back, injury to one of these structures can spread to the other. Clinically, suspicion of hyoid injury should prompt evaluation of cervical spine injury (and vice versa), since hyoid fracture or displacement may indicate high-risk cervical trauma. Furthermore, cervical instability may coincide with airway and soft-tissue compromise in the hyoid/laryngeal region (37,38).
Cervical lordosis loss	Poor craniocervical posture is a common cause of pain and movement disorders in this area (39). In growing individuals, it can also affect the normal development of bone structures. Loss of cervical stability is associated with altered cervical lordosis, altered muscle function and impaired movement. A high prevalence of disc degeneration has been observed in cases of loss of cervical lordosis, along with pronounced weakness in both the cervical flexors and extensors and an imbalance in the relative cross-sectional areas of the flexors and extensors, which has been attributed to extensor weakness (40). Furthermore, comorbidity between temporomandibular disorders and cervical spine abnormalities has been observed, and a similar connection has been suggested between the temporomandibular joint and the cervical spine (41-43). This may not be a pathological cause-and-effect relationship, but rather a morphofunctional-postural one. However, both rectification and inversion of the cervical spine occur independently of temporomandibular disorders (44). Nevertheless, due to neuroanatomical conditions (e.g. fascia, innervation and biomechanics), it is impossible to ignore the postural relationship between the two regions.
Complex regional pain syndrome	This syndrome is a chronic, painful condition that can develop in response to a variety of traumas. It can be classified as type I or type II according to whether or not it has a known cause. The condition involves both peripheral and central (autonomic nervous system) mechanisms. Reduced cortical representation of the affected organ appears to be related to hyperreactive responses or spontaneous sensorimotor activity, suggesting cortical disinhibition (45-48).

To be continued

Condylar agenesis, aplasia, hyperplasia, hipoplasia and neoplasm	Condylar agenesis refers to the complete absence of an organ or tissue, which is associated with an absence of embryonic structure. Aplasia refers to the absence of a structure that shows a rudimentary embryonic outline. Hypoplasia describes incomplete development resulting in smaller-than-normal size, while hyperplasia corresponds to an increase in the number of normal cells. In contrast, neoplasia refers to abnormal cell growth, whether benign or malignant. Congenital deformities of the temporomandibular joint (TMJ) complex typically present as a diverse range of growth disorders affecting the mandibular condyle, articular eminence and temporal bone. Among these, condylar agenesis is extremely rare (49). Condylar hypoplasia can be congenital or acquired, and is sometimes associated with head and neck syndromes (50). Acquired condylar hypoplasia is due to alterations in the condylar growth centre during development. Other morphological alterations of growth and development include bifid and trifid condyles (51).
Condylar fractures	Fractures of the mandibular condyle are relatively common among all mandibular fractures, accounting for 19–52% of cases, and can be caused by direct or indirect trauma (52). Various factors can cause the detached bone portion to be displaced, including the direction, magnitude and point of application of the force, the condition of the dentition and the occlusal position (539. A condylar process fracture is defined as a fracture located above the mandibular foramen, extending from the angle of the mandible to the sigmoid notch or condylar head. The condylar process extends upwards as a continuation of the posterior border of the ascending ramus, carrying the condylar head at its upper end and the lower portion of the temporomandibular joint. Three subregions are identified for the classification of fractures: the condylar head, the condylar neck, and the base of the condylar process (54). These subregions are further subclassified according to location (medial or lateral to the head), fragmentation (of the head, neck, or base), vertical apposition (of the head), lateral displacement (of the neck or base), angulation (of the neck or base), displacement of the fragment/fossa towards the cephalic or caudal end (of the entire process), distortion of the condylar head (of the entire process), and general loss of branch height (of the entire process).
Condylar positional asymmetry	Mandibular asymmetry is a morphological variation indicating disproportionate size, shape or position between the right and left sides of the jaw. These asymmetries can occur in the vertical, transverse and sagittal planes (55).
Degenerative disesase - osteoarthritis/osteoarthrosis	Degenerative TMJ disease is a general term that encompasses degenerative processes in the structure and function of the TMJ (56). These processes range from destructive lesions without active inflammation or pain, but with inflammatory precursors, to active inflammatory lesions characterized by pain and impaired function. The most common clinical sign is crepitus, provided there is no soft tissue between the bony surfaces due to disc perforations, displacement, or rupture of the upper posterior disc tissues. TMJ-DJD is a major public health problem affecting approximately 5% to 12% of the general population, and is the leading cause of chronic non-dental pain in the oral and facial regions. Advanced age is a risk factor for the development of degenerative TMJ disease. Its incidence progressively increases in middle-aged and older groups (20). Regarding occlusion, the literature is inconsistent, but loss of support in the occlusal relationship can lead to degenerative changes in TMJ components, including changes in the morphology and bone density of the condyle, articular fossa and articular eminence (57). In addition to these risk factors, sex and genetic makeup are also important, with genetic polymorphisms being associated with the inflammatory response, sex hormones, oxidative stress and bone metabolism. Lifestyle factors and chewing habits, such as unilateral chewing, have also been studied.
Delayed onset muscle soreness	Delayed-onset muscle soreness (DOMS) arises from unusual or repeated muscle contractions involving an eccentric component. These contractions generate high levels of tension and cause structural damage to muscle fibres. DOMS is associated with the release of algesic substances, muscle spasms and inflammation. This involves a combination of mechanical, thermal and chemical nociceptive stimuli that produce micro-injuries which attempt to heal as exercise continues. In this context, DOMS would be associated with bruxism, particularly during waking hours when a greater number of isometric muscle contractions occur (58). However, bruxism-related pain would be short-lived and self-limiting, similar to the pain experienced when people exercise without prior preparation. Therefore, we could say that, when associated with waking bruxism, DOMS corresponds to specific situations rather than periodic overload. In this case, the painful response would be more akin to myofascial pain associated with bruxism. Although data on this topic are limited, the pathophysiological relationship is clear. When recovery is impaired, neuroplastic changes can begin to occur, leading to chronic pain that would correspond to nociplastic pain affecting the myofascial system according to the mechanism.
Dental-alveolar pain by deafferentation	Persistent tooth pain, also known as phantom dentoalveolar pain, can occur following damage to the associated nerve component following trauma caused by the removal of the neurovascular bundle, either through tooth extraction or endodontics, or even by damage to the nerve root, whether traumatic or surgical. Dentoalveolar deafferentation is defined as the reduction or loss of peripheral afferent neuronal input related to the dental and masticatory apparatus. This situation involves cortical sensory reorganization and altered functional brain stimulation between sensory centers. These changes, which are related to phantom dentoalveolar pain, also produce alterations in auditory, gustatory and olfactory perception. Memory and cognitive impairment may also occur, as well as impairment of stomatognathic system functions, such as mastication. While a young person may adapt to deafferentation, brain plasticity decreases with age, causing these alterations (59).

To be continued

10.59987/ads/2026.1. 120-147

Descent of the hyoid bone	The descent of the hyoid bone is defined as an abnormally low position relative to the mandible and cervical vertebrae. This condition may be associated with obstructive sleep apnoea, swallowing disorders or dysfunction of the suprahyoid and infrahyoid muscles (60).
Disc adhesion	Adherence and adhesion both involve the immobilisation or stagnation of the articular disc, typically beneath the glenoid cavity or the temporal eminence. There are some differences between the two. Adherence refers to the resistive force that prevents one surface from displacing another, i.e. the force that prevents the disc from moving freely beneath the glenoid cavity and eminence. It is suggested that the disc can move normally thanks to phospholipids and hyaluronic acid, which lubricate the joint. However, overload can cause the degradation of hyaluronic acid, exposing the phospholipids and resulting in the smooth articular surfaces becoming highly adherent upon close physical contact. This leaves only a minimal film of fluid between them and creates an adhesion similar to that produced between two glasses with a film of water between them (61). Conversely, adhesion refers to the physical union of two structures. In this regard, closed temporomandibular joint block resulting from disc displacement without reduction, and the tethered disc phenomenon, are among the possible intracapsular aetiologies described in the literature for restricted mouth opening (62,63). It is believed that the tethered disc phenomenon may be erroneously included in blocked closure or disc displacement with restricted opening conditions. This concept arose from the perception that, among patients with unreduced disc displacement, there are individuals with sudden, severe and persistent limited mouth opening. This is too severe to be caused by a deformed and irreducible disc, but rather by total inhibition of disc sliding due to an adhesive phenomenon that can be easily reversed through routine irrigation of the upper joint space (29). Fibrous adhesions are not considered a sustainable assumption for the tethered disc phenomenon because, in general, adhesions are resistant to simple lavage, and strong adhesions cannot explain its sudden onset (27). Thus, while the concept of adhesion associated with static disc syndrome may be true, its classification within the scope of TMJ disorders as a distinct and independent entity from the condition of blockage attributed to unreduced disc displacement with limited opening has not yet been agreed upon (64). In this regard, it has been observed that the presence of adhesions is significantly greater in joints with disc displacement without reduction than in joints with well-positioned discs or discs with displacement with reduction. This leads to the hypothesis that disc hypomobility is an important factor in the development of adhered or adhesive discs (65).
Disc deformation	A classification system has been developed to define the morphology of articular discs, particularly displaced discs. In this context, a biconcave shape is considered normal. Biconvex, biplanar and inverted biconcave shapes are defined as disc deformation, which positively correlates with the type of clicking and even the prognosis of injury (66). The temporomandibular joint disc is primarily composed of collagen, and its arrangement facilitates efficient stress distribution. This explains why a non-displaced disc can vary in morphology according to areas of overload. Pathological conditions have been observed to cause a concentration of stress near the injury site due to interfibrillar collagen trafficking patterns, resulting in the early manifestation of damage (67). Therefore, if a disc is located over the condyle and has not yet been displaced, it may exhibit morphological changes that could generate a clicking noise depending on its relationship with the articular condyle during movement.
Disc displacement with reduction	For various reasons, including trauma, the articular disc loses its shape. The morphology of the mandibular and temporal bones, along with the activity of the muscles and ligaments, is then unable to hold the disc in position. The disc then shifts from its 'comfort zone' and settles in a more anterior position, either laterally, medially or ventrally. Each time the mandibular condyle moves during opening and/or movement in the horizontal plane, the injury is reduced, producing an audible clicking noise. When the mandibular condyle returns, the disc dislocates or shifts from its current location and moves forward, producing a second clicking noise. Regarding the position of the mandibular condyle, the anterior joint space increases in cases of mild to moderate anterior disc displacement but not in more advanced cases. In very rare cases where the articular disc is displaced posteriorly, the posterior joint space increases (68).
Disc displacement without reduction with/without limited opening	As in the previous case, the disc shifts from its 'comfort' position, but this situation is not resolved by any mandibular movement. This can lead to altered mouth opening and presents as two conditions: disc displacement without reduction with limited mandibular opening and disc displacement without reduction with no limited mandibular opening. When opening is limited, the condyle involved in the injury will be unable to continue moving due to the blockage caused by the displaced disc. Therefore, mandibular opening will show an uncorrected deviation towards the side of the injury. This condition, where the disc blocks the condyle from moving so that a functional opening can occur, is also known as closure blockage. If the condition persists for various reasons and the system begins to adapt, the mandibular condyle will push the displaced disc forward and the range of mandibular opening will progressively increase, potentially reaching normal levels (i.e. disc displacement without reduction or restriction of opening). In this situation, joint blockage no longer occurs during opening. As expected, there are no joint sounds associated with the mechanics of movement in either situation. Literature on this topic has shown that condylar degeneration is often associated with this lesion, which is also linked to severe chronic pain and deformation of the articular disc (69). However, it is important to note that disc displacement without reduction or restriction of opening can also be identified through imaging.

To be continued

Disc displacement with reduction with intermittent locking	This situation corresponds to an overlap of the previous two situations, typically occurring between a displaced disc with reduction and a displaced disc without reduction, resulting in restricted opening. In other words, there are times when the patient experiences no discomfort and only reports a clicking noise, and other times when they report a sensation of blockage when closing their mouth. This blockage may also be observed during clinical examination or imaging.
Disc disruption	Disc disruption refers to damage to the intervertebral discs, which can manifest as fissures or tears in the annulus fibrosus. At this stage, the disc does not appear to be herniated, but the patient may experience pain due to the release of inflammatory substances affecting the free ends of the area. Due to its morphological characteristics, this pathology is classified as a degenerative disease of the vertebral disc. The intervertebral disc is primarily composed of extracellular matrix components, such as collagen and proteoglycans. For various reasons, not all of which are understood, the disc undergoes structural and biochemical alterations resulting in functional impairment, degeneration and pain. Oxidative stress, immune system abnormalities, imbalances in mechanical loading, and metabolic alterations are currently believed to play a role in the initiation and progression of disc disruption. Oxidative stress occurs when there is an overproduction of reactive oxygen species and an inability to eliminate them, which alters redox homeostasis within the intervertebral disc. This imbalance in the redox system causes the degradation of the extracellular matrix, inducing cell apoptosis (70).
Disc perforation	A displaced articular disc is associated with altered joint biomechanics. This leads to increased compression and shear forces, predisposing specific regions of the disc, particularly the posterior region, to thinning and, ultimately, perforation (71,72). Once perforation has occurred, the disc loses its ability to effectively separate the articular surfaces. This leads to joint inflammation and bone degeneration, resulting in degenerative changes accompanied by symptoms such as crepitus.
Dislocation (luxation and subluxation)	Hypermobility disorders include dislocation, which can be divided into two types. The first type involves anterior and superior displacement of the condyle beyond the eminence, preventing the joint from returning to the closed position without a specific manoeuvre by the patient (subluxation). The second type requires professional assistance to return the joint to its normal state (luxation) (14).
Eagle syndrome	This corresponds to the mineralization of the stylohyoid ligament or the elongation of the styloid process, but with a variety of concurrent signs and symptoms due to different factors, such as irritation of the carotid artery, pain when rotating the head, dizziness, dysphagia, otalgia, headaches and vague pain (73,74).
Elevation of the hyoid	The hyoid bone reflects the tension in the muscles, ligaments and fascia to which it is attached. In a normal cervical lordosis, the hyoid bone is positioned below a control line drawn between the retrognathism and the anteroinferior border of C3. When the cervical spine is straightened, the hyoid bone tends to elevate until it reaches this line. When the spine is inverted, generating a cervical kyphosis, the hyoid bone elevates above the control line (75). During swallowing, elevation of the hyolaryngeal complex is essential to enable the tongue and associated structures to perform the necessary movements to transfer a bolus from the oral cavity through the hypopharynx to the esophagus. During this process, the suprahyoid and longitudinal pharyngeal muscles elevate the hyolaryngeal complex (76). However, when the hyoid bone and the tongue, which is supported by the hyoid bone, are both elevated, swallowing becomes difficult.
Ernest Syndrome	This corresponds to the mineralization of the stylohyoid ligament or the elongation of the stylomandibular process, but with a variety of concurrent signs and symptoms due to different factors. For the diagnosis of this syndrome, we can only refer to the clinical presentation: painful digital palpation, persistent pain, pain in the preauricular region and at the angle of the mandible, and limited range of motion are symptoms and signs frequently found in literature reviews (75,77).
Facet synovitis (cervical facet syndrome)	As with TMJ, synovitis is defined as an inflammatory process involving the synovial tissue of the intervertebral joints, characterised by arthralgia. Facet synovitis of the cervical vertebral joints can be caused by overload, wear and tear, or various injuries that alter joint function and craniocervical movement. Hyperaemia is observed, as well as an increase in fibroblast- and macrophage-type synoviocytes, and changes in cytokines. Therefore, synovitis progression could lead to osteocartilaginous damage.
Herniated disc	A herniated disc occurs when part of the nucleus pulposus of an intervertebral disc moves into the spinal canal, putting pressure on the spinal cord and nerve roots. Herniated discs are uncommon in the cervical spine, and patients may experience intense local radicular pain, as well as neurological changes in the affected areas of the skin (78).

To be continued

10.59987/ads/2026.1. 120-147

Hyperlordosis/Kyphosis of the cervical spine	Poor craniocervical posture is one of the causes of pain and movement disorders in this area. In growing individuals, it can affect the normal development of bone structures (39). Loss of cervical stability is associated with altered cervical lordosis, altered muscle function and impaired movement. A high prevalence of disc degeneration has been observed in cases of loss of cervical lordosis, along with pronounced weakness in both the cervical flexors and extensors and an imbalance in the relative cross-sectional areas of the flexors and extensors, which has been attributed to extensor weakness (40). Furthermore, comorbidity between temporomandibular disorders and cervical spine abnormalities has been observed, and a similar connection has been suggested between the temporomandibular joint and the cervical spine (41-42) perhaps not as a pathological cause-and-effect relationship, but rather as a morphofunctional-postural one. However, both rectification and inversion of the cervical spine can occur independently of temporomandibular disorders (44), although due to neuroanatomical conditions (e.g. fascia, innervation and biomechanics), it is impossible to ignore the postural relationship between the two.
Hypertranslation	Joint hypermobility refers to excessive movement of a joint beyond its normal range. TMJ hypermobility, also known as TMJ hypertranslation, is characterised by joint movement beyond the normal range during condylar translation. Joint hypertranslation is one of the conditions classified under hypermobility. TMJ hypertranslation occurs when the disc and condyle protrude beyond the articular eminence, which can lead to sudden, irregular movement accompanied by a clicking sound. The condyles appear to protrude laterally. Additionally, at both the end of maximum opening and the beginning of mouth closure, one condyle may move before the other, causing a sudden lateral oscillation of the mandible before translation occurs (79,80).
Idiopathic condylolysis	Condylar resorption is a multifactorial condition that affects the mandibular condyle. It is characterized by changes in shape and a decrease in condylar mass. These changes can lead to malocclusion, temporomandibular joint (TMJ) dysfunction and pain (81). Progressive idiopathic condylar resorption, which has no obvious cause, has been identified as a clinical joint problem that is sometimes associated with orthognathic surgery (82). This aggressive form of the disease is most frequently observed in adolescents and young women, and is attributed to an exaggerated response to mild traumatic injuries induced by an excess of estrogen receptors. Although little is known about this condition, isolated cases have been reported and two forms of condylar resorption have been theorized: (1) in adults, the mandible regresses once growth is complete, and (2) in young people, the potential rate of mandibular growth decreases (83).
Inflammation of the temporomandibular ligament	Inflammation of the temporomandibular ligament is an inflammatory process affecting the connective tissue that stabilizes the temporomandibular joint (TMJ), leading to pain and restricted movement. This is often caused by trauma or overuse (84).
Inflammation of lateral colateral ligament	The lateral collateral ligament is one of the intracapsular ligaments that can be examined clinically. As with all ligaments, it can become inflamed if overstretched. This occurs in cases of disc displacement, particularly with a lateral component, as the displaced disc elongates the ligament, which is firmly attached to the lateral pole of the condyle.
Intervertebral fusion	It refers to a congenital condition that particularly affects the cervical region and is known as Klippel-Feil syndrome. During pregnancy, two or more vertebrae develop incompletely and fuse together, resulting in this condition. Symptoms include a short neck, impaired neck mobility and a low hairline (85).
Inversion of the cervical spine	Inversion of the cervical spine is a pathological change in the sagittal alignment of the cervical vertebral column, whereby the normal lordotic curve is reversed or substantially flattened, resulting in a kyphotic (forward-bending) or straightened configuration of the cervical spine. This abnormality in alignment can lead to altered biomechanical loading, potential compression of the spinal cord or nerve roots, pain, a reduced range of motion, and impaired horizontal gaze (86).
Joint neoplasia	A temporomandibular joint neoplasia refers to a benign or malignant tumor arising from the bone, cartilage, synovium or soft tissues within or around the joint. This can lead to pain, swelling and impaired function (87).
Mandibular fractures	The severity of mandibular fractures varies depending on the number of sites involved, displacement and comminution (88). Examples include condylar, coronoid, body (involving the ramus and angle of the mandible), alveolar process, symphysis, and parasymphysis fractures (89).

To be continued

Mandibular instability	Mandibular function involves several passive structures, such as bones, and active structures, such as muscles. The masticatory process is complex and depends on multiple reflex arcs. Occlusion plays an important role in this process, maintaining harmonious and precise movement, or stable dynamics. Therefore, mandibular biomechanics is the study of biological forms from a functional perspective. As humans are biological units that function according to physiological laws, such as occlusion, and physical laws, such as levers, inclined planes and force transmission, we will also refer to biophysics. This concept encompasses two ideas: stability and equilibrium (90). Stability signifies security in space, both physically and biologically, but it is a static concept in that it refers to a specific time, place or position. Equilibrium, on the other hand, is the state of a system in which the acting forces neutralize each other in such a way that the conditions of rest or movement that characterize the system remain unchanged (91). Therefore, stability is the resistance a body offers to being overturned, whereas balance is a more dynamic concept relating to changes in movement. Combining the concepts of stability and balance provides a dynamic view of mandibular stability. Mandibular stability is defined as the ability of the mandible to maintain balance at all times — whether at rest or in motion — as a result of the interaction between the components of the stomatognathic system, provided there are no symptoms present. At rest, the mandible is in stable balance thanks to the myotatic or stretch reflex. When displaced or in eccentric positions, however, the balance is indifferent since, thanks to the neuromuscular mechanism and awareness, the mandible can remain in a new location for a time. According to the authors of this text, mandibular instability is defined as a state in which the mandible has lost its spatial centricity. This manifests as a discrepancy between its actual centricity and maximum intercuspation, caused by interfering occlusal contacts during mandibular closure. An opening with a slight tendency to deviate towards the lost midline is observed alongside a double closing arc. Additionally, neuromuscular system functionality is imbalanced, resulting in unstable positions and possible fatigue of the associated musculature.
Migraine	Migraine is a primary headache disorder and a highly prevalent neurological condition, particularly among women in their twenties and thirties. The main symptom is a moderate to severe headache, which occurs alongside other symptoms such as photophobia, phonophobia, allodynia, nausea, vertigo and dizziness (92,93). Overall, the symptomatology observed is quite heterogeneous and includes attack frequency, the presence of auras, comorbidities and treatment responses. This heterogeneity may have a genetic and molecular basis. Variants that primarily manifest in neurovascular activity and the activity of neuromodulators and neurotransmitters, such as serotonin, calcitonin gene-related peptide, amylin, pituitary adenylate cyclase-activating polypeptide and nitrous oxide, have been identified (94).
Muscle Atrophy	The loss of muscle mass is called muscle atrophy. It can be congenital or genetic, or it can be caused by an identifiable factor. Hence its categorization as primary or secondary. The mechanism involves an imbalance between protein synthesis and degradation, resulting in myofiber contraction, changes in fiber types or myosin isoforms, and net losses of cytoplasm, organelles and total protein. As a significant reduction in muscle mass, muscle atrophy can reduce patients' quality of life and increase morbidity and mortality. It involves an imbalance in protein synthesis and degradation induced by various factors such as ageing, oxidative stress and inflammation. Signal transduction mechanisms include pathways that control protein degradation, such as the ubiquitin protein degradation system, autophagy, caspases and calpains, as well as pathways that control protein synthesis, such as the mammalian target of rapamycin (mTOR) pathway (96). Atrophy is similar to sarcopenia, but it can occur at any age for reasons other than ageing. At the oral level, sarcopenia indicates a decrease in masticatory muscle activity. Masticatory muscle atrophy can be caused by occlusal conditions, TMD, trauma, systemic diseases such as myasthenia gravis, rheumatoid arthritis and polymyositis, masticatory habits and trigeminal motor neuropathy (97,98).
Muscle hypertrophy	Masseter and temporalis hypertrophy is a benign condition with various potential causes, including bruxism, temporomandibular disorders and malocclusion. However, the cause is unclear in most cases, which is why it is often classified as idiopathic (99). Theories have been described in the literature, as well as progressive hypertrophy caused by anxiety in romantic relationships. It can be associated with facial pain and asymmetry. Unilateral or bilateral masticatory muscle hypertrophy is a benign condition that most commonly affects the masseter muscles and is rare in the temporalis muscles. Bruxism, temporomandibular disorders and malocclusion have been described as potential causes, but the cause is often unclear (100,101). The average masseter muscle depth has been observed to be 10.8 ± 1.6 mm in men and 8.1 ± 1.4 mm in women. Regarding the normal range of masseter muscle depth, it has been observed that it varies depending on sex-specific facial indices. Euryprosopic individuals have masseter muscle depths averaging 11.5 ± 2.08 mm for males and 8.8 ± 1.4 mm for females. Mesoprosopic individuals have depths of 11.4 ± 1.6 mm for males and 7.8 ± 1.6 mm for females. Leptoprosopic individuals have average depths of 10.08 ± 1.2 mm for males and 7.7 ± 1.4 mm for females (102,103). A diagnosis of masseteric hypertrophy could be considered in male individuals with a masseter muscle depth greater than 12.4 mm and in female individuals with a depth greater than 9.5 mm (26). Other studies have suggested that hypertrophy can be considered in muscles with a depth greater than 13.5 mm, regardless of sex or facial type (104).

To be continued

10.59987/ads/2026.1. 120-147

Muscle neoplasia	Many organs in the body are susceptible to cancer. However, primary cancers rarely affect striated muscles, which include skeletal and cardiac muscles (105). Sarcomas can develop in muscle, fat, or connective tissue, accounting for around 1% of all malignant tumours in adults (106). Sarcomas in the masticatory muscles are particularly rare and may present with symptoms such as trismus, swelling and/or pain.
Myofascial pain	Myofascial pain is a multifactorial pathology involving the fascia, with comorbidities and a chronic component. It is therefore associated with nociplastic pain involving peripheral and central sensitization (107). Myofascial pain undoubtedly originates from continuous overload, which generates changes that induce responses in the nervous system. These changes cause the nervous system to 'learn pain' and produce sensitization. Therefore, patients with myofascial pain present with muscle pain. Due to the mechanisms underlying neuronal functional changes, the pain becomes more continuous and is expressed over a larger area than the site of the injury. This is why myofascial pain is evident when the patient performs functional and parafunctional movements, or when the physician stimulates sensitive areas. The resulting pain is usually radiated and may be due to palpation of a specific point, known as a trigger point. However, trigger points have primarily been associated with myofascial pain presenting with a referred pattern.
Myofasical pain with referral	The only difference between this situation and the previous one is that a trigger point has been clearly identified. When this trigger point is stimulated for 5 seconds with a load of 1 kg/cm², it generates clear and often specific referred pain in another region. In the case of the masticatory muscles, referred pain is not uncommon. It can originate from the muscles in the teeth or cephalic regions, as well as from the cervical muscles in the head, face and/or fundus. Although common, referred pain derives from activity occurring in different spinal and brainstem structures (108). Most theories accept the phenomenon of convergence, whereby nociceptive afferents converge with other afferents in a second-order neuron. It is also thought that referred pain may be a form of central sensitization of the neurons in question. This involves both a synaptic relationship and a change in the environment of the segmental area. A classic example of this is the convergence of spinal nerves C1–C3, which generate impulses in the trigeminal nerves and produce secondary headaches. Another theory proposes the existence of afferent fibers that branch and distribute to regions of primary dysfunction and other referred areas. Primary lesions stimulate afferent fibers in deep regions, which then trigger the activation of a reflex arc towards the muscle via somatic efferent fibers.
Myofribrotic contracture	There is little clear information on this topic. Fibrotic contracture of skeletal muscle is defined as excessive shortening that causes stiffness and deformity. It has been linked to ischemic damage resulting from compartment syndrome. The pathogenesis of this syndrome includes necrosis, fibroblastic proliferation and the formation of a shrinking scar and myotendinous adhesion following prolonged ischemia. Other causes of fibrotic contracture, such as trauma or infection, have also been documented (79,110,111).
Myositis	Myositis is defined as painful inflammation of a muscle or muscle tissue resulting from a specific localized cause. It is typically linked to infection or trauma, and is characterized by swelling, restricted movement, redness, increased muscle temperature, and persistent, widespread pain in a specific area that worsens with movement. Ossification can occur as a result of inflammation, leading to myositis ossificans. Based on the definition of myositis as 'inflammation of a muscle', we cannot ignore painful muscle pathology caused by inflammation due to overload (microtrauma), even though it is not as extensive as the aforementioned situation. Before continuing with the analysis of each pathology, it should be noted that the term 'local myalgia' indicates local muscle pain, i.e. a symptom, and therefore should not be included as a diagnosis. According to the literature, myalgia has been defined as a disorder caused by an inflammatory process rather than a symptom synonymous with myositis. In this context, it has been proposed that to be diagnosed as 'myalgia', muscle pain must be affected by movement or function and must be recognized by the patient and confirmed by the physician (in the case of TMD, it must also be associated with mandibular dysfunction) (69). Muscle pain, or myalgia, is generally caused by an inflammatory process associated with overload. As this persists, the pathogenesis changes. Therefore, three pathologies indicating a cause-related diagnosis are classified under this mechanism: pain induced by extreme exercise, delayed-onset muscle soreness and pain associated with injuries.
Myospasm	Myospasm is a response of the central nervous system to injury. In the oral area, it results in motor disturbance of the trigeminal nerve, which is defined as trismus. Trismus is not a pathology, but rather limited jaw mobility due to various causes such as trauma, oral inflammation and infections, temporomandibular disorders, coronoid hyperplasia, neoplasia, surgery and autoimmune connective tissue disorders such as lupus erythematosus. It corresponds to electromyographic activity that is not under voluntary control. Trismus is defined as the inability to open the mouth widely. It can be caused by a central motor disturbance or other factors, including joint ankylosis, infection, trauma, toothache, TMD, tumors and drugs (114-118). In other words, trismus is a symptom, not a pathology, and for this reason it is not included among TMDs.
Myospasmodic stiffness or contracture	When analyzing this topic, it is important to clearly understand the difference between stiffness and contracture. As we have seen previously, the term 'contracture' refers to changes in muscle fibers, both dimensional and structural. However, 'stiffness' implies greater electrical activity in the muscles and, consequently, greater muscle contraction due to central activity.

To be continued

Myostatic contracture	This contracture, such as myostatic contracture, can be caused by muscle disuse. It occurs in the rectus femoris muscles due to shortening caused by cranial extension. This situation is quite similar to an analgesic contracture, which is a compensatory phenomenon involving multiple reflexes. Disuse contractures of this type are not as severe as those seen in cerebral palsy. Cerebral palsy is an encephalopathy that causes myopathic contractures due to a lack of descending information, resulting in complete muscle inactivity.
Neoplasms in neural tissues	Neural tissue neoplasms are abnormal proliferations of cells originating from neurons or glial components of the peripheral or central nervous system. These neoplasms can be either benign, such as schwannomas, or malignant, such as neuroblastomas (119).
Neoplasms of the cervical spine	These are tumors that originate in the vertebrae, cartilage or soft tissues of the cervical spine. They can be benign or malignant and can potentially cause pain, neurological deficits and spinal instability (120).
Neuralgia (trigeminal and glossopharyngeal)	Trigeminal neuralgia is a painful condition characterized by chronic neuropathic pain, which causes sudden, recurrent and intense pain, usually on one side of the face. It affects one or more branches of the trigeminal nerve and patients often describe the sensation as similar to an electric shock (10,121). Glossopharyngeal neuralgia is a similar condition, but the pain is distributed along the auricular and pharyngeal branches of the glossopharyngeal and vagus nerves (122).
Neuropathy of dento-alveolar origin by deafferentation	This is a nerve condition that develops following the loss of sensory input from teeth or oral tissues. It is usually the result of tooth extractions, apicoectomies, pulp removal, periodontal surgery or injury to the inferior alveolar nerve. It presents as persistent pain or paresthesia in the affected area, and has similarities with other deafferentation pain syndromes (123).
Occlusal dysesthesia by deafferentation	Occlusal dysesthesia (OD), or phantom bite syndrome (124), refers to the persistent sensation of an uncomfortable bite without any obvious occlusal discrepancy. This rare condition is usually associated with emotional distress and is often triggered by dental treatment (125). OD can occur after any dental procedure, even in the absence of treatment. Therefore, rather than being an occlusal problem, it is considered to be a psychosomatic condition involving an alteration or maladjustment in signal processing (126).
Opening lock	Before analyzing this topic, we must remember that there is a condition called 'blocking in closure'. This condition involves the articular disc, which prevents the articular condyle from moving and thus impairs mandibular opening. If a blockage is subclassified as 'closing', then by nomenclature there must be a pathological situation involving the opposite movement. The authors of this classification therefore propose the condition known as 'blocking in opening', which indicates a type of dislocation caused not by bone interference, but by disk interference. In other words, the mandible would be unable to close due to interference from an adherent disc. It is important to clarify this, since the diagnosis should guide the clinician towards treatment. In this case, despite being similar to blocking in opening, a dislocation requires diametrically opposite treatment.
Oro-facial dyskinesia	Unlike orofacial dystonia, dyskinesia involves the involuntary, repetitive and excessive movement of the jaw, tongue and lips. Rather than irregular tone, it reveals kinetic disturbances. It can be spontaneous or associated with certain medications, such as L-dopa and anticholinergics (127,128).
Oro-mandibular dystonia	Dystonia is a movement disorder involving muscle tone with no apparent cause. It is characterised by involuntary muscle contractions that cause abnormal movements and/or postures. It primarily affects individuals between 45 and 70 years of age and women more frequently than men. Oromandibular dystonia manifests in the jaw, face and tongue. Patients with oromandibular dystonia may present with symptoms associated with temporomandibular disorder (TMD)/functional disorder (FOD), such as pain in the masticatory or facial muscles, bruxism, and so on. It may also be associated with TMD, such as dislocations. The involuntary twisting of the lips and tongue has a constant movement pattern in the same direction and is generally exacerbated by voluntary movements (129).
Osteochondritis dissecans	This joint pathology is a type of injury in which a section of bone, along with its associated cartilage, separates from the joint surface. It primarily affects children and adolescents and is caused by a lack of blood supply. While imaging findings can vary widely, the most common include the presence of loose intra-articular calcified bodies (single or multiple), signs of degenerative bone changes, disc displacement, widening of the joint space, and alterations in condylar morphology (130).
Other intracapsular ligaments pain	The intracapsular ligaments of the TMJ, such as the collateral ligaments, stabilise the articular disc within the joint capsule. Pathology or inflammation of these ligaments may lead to internal derangement and pain (131).
Pain associated to lesions	Pain associated with lesions is either nociceptive or neuropathic, and originates from structural or inflammatory damage caused by factors such as trauma, infection, or neoplastic processes. This involves the activation of peripheral and central pathways that transmit pain signals from the injured tissue (132).

To be continued

10.59987/ads/2026.1. 120-147

Pain induced by extreme exercise	It corresponds to short, intense activity that causes immediate fatigue and pain. The acute, transient and non-threatening local muscle pain caused by short, extreme, high-intensity activity differs significantly from other types of local muscle pain, such as delayed-onset muscle soreness, injury-related pain or chronic pain (133). This type of exercise-induced pain has the same mechanism as experimentally induced pain, for example when a patient is asked to clench their teeth at maximum force or to remain in a specific position that overloads a defined muscle group. This type of exercise-induced pain is caused by harmful chemical nociceptive stimuli. These are metabolites resulting from anaerobic energy, such as potassium and hydrogen ions, substance P, histamines, prostaglandins, serotonin, bradykinin and adenosine. These metabolites accumulate in the intramuscular space and stimulate the free nerve endings that supply unmyelinated type C afferent nerve fibers. The pain increases linearly with effort, remaining at an extreme intensity and requiring predominantly anaerobic metabolic input. Clinically, the pain originates in the active muscles involved in the exercise. It is acute, tonic and transient, occurring during exercise and being harmless. It is also a potential indicator of the metabolic and/or cardiorespiratory demands generated by exercise. When this activity is repeated, the experience of induced pain becomes associated with a change in motor behavior aimed at minimizing further tissue damage or injury as the body attempts to alleviate the load on sore tissue and reduce the perception of pain. This involves changes in behavioral coping strategies (e.g. exercise intensity) or other psychophysiological strategies. If exercise is continued without prior preparation and accompanied by a change in strategy, another type of situation begins to arise: exercise-induced injuries associated with delayed-onset muscle soreness.
Parasitic Myositis	This is a type of infectious myositis in which muscle inflammation is typically caused by trichinosis, cysticercosis or toxoplasmosis, although other parasites may also be involved (134).
Parotitis	Parotitis is defined as the acute or chronic inflammation of the parotid gland. Triggering factors include microbial agents (bacterial or viral), allergies, and autoimmune diseases such as Sjögren's syndrome. Genetic/hereditary factors may also play a role (135,136).
Peripherally mediated neuropathy	Peripheral neuropathies are diseases of the peripheral nervous system. They can be categorised as mononeuropathies, multifocal neuropathies or polyneuropathies. Symptoms often include numbness and paresthesia (137). Chronic polyneuropathy has an estimated worldwide prevalence of 1%, which increases with age. Mononeuropathy affects a specific area; carpal tunnel syndrome is an example of this. There are also immune neuropathies, such as Guillain-Barré syndrome, which are less common (139-141).
Post-herpetic neuralgia	Postherpetic neuralgia is the most common and severe chronic complication of the herpes zoster virus. It is characterized by a burning, electric-like pain or a sensation of tearing that persists for more than three months after the lesion has healed (141). There is an association between age and immune dysfunction (IL-10), which increases the risk of developing the disease. This suggests that an imbalance in the immune system plays a key role in its development. T-cell dysfunction and inflammatory imbalance, in particular, can lead to delayed viral clearance and persistent nerve damage (142,143).
Protective co-contraction	Protective co-contraction involves the co-activation of muscles to prevent potential muscle damage, rather than producing or regulating coordinated movements as co-contraction does. Protective co-contraction demonstrates a limitation of mandibular kinematics in the frontal plane (145). Conversely, an occlusal condition that generates instability, or acute changes to the occlusal condition, can affect muscle function via a central stimulus-dependent mechanism. These situations can generate a neuromuscular response called protective co-contraction, which causes muscle pain and is frequently associated with the development of new muscle engrams. The patient adapts to these engrams with little effect; one consequence is difficulty moving the mandible during manipulation (146). It should be noted that skeletal muscle contractures represent the permanent shortening of a muscle-tendon unit, which occurs when the soft tissue loses elasticity and is unable to stretch either passively or under the influence of antagonist muscles. Contractures are probably the most clinically significant consequence of altered passive mechanical properties of muscle. Contractures can be caused by an upper motor neuron injury, such as a stroke, head injury or cerebral palsy, or by a muscle disease such as spinal muscular atrophy or muscular dystrophy, as well as by other neuromuscular disorders and analgesic reflex responses (147,148). Furthermore, active cellular contractility can increase the stiffness of fascial tissues, thereby contributing to musculoskeletal dynamics. Various studies indicate that the presence of myofibroblasts within fascia may enable these tissues to alter their stiffness. This contractile tissue behaviour has been observed not only in various pathological fibrotic contractures, but also in normal fascia. In our field, we have observed muscle shortening or contractures associated with muscle disuse, infection or central responses.
Retrodiscitis	Retrodiscitis is an inflammatory joint condition that occurs in the posterior region of the disc. It includes the area from the posterior edge of the disc and extends to encompass the entire posterior synovium. It can therefore be caused by macro-trauma producing acute overload in the posterior synovial area (149), such as the trauma generated by the condyle on the posterior edge of the disc when displaced (150). As this area is so extensive, the authors of this text prefer to use the term 'posterior synovitis' when an overload of the posterior synovium is generated by a macro- or microtraumatic event, and 'retrodiscitis' when inflammation of the specific area of the retrodisc is caused by micro-trauma generated by the condyle on the posterior edge of the articular disc when it is displaced.

To be continued

Sarcopenia	Sarcopenia is a disease characterised by muscle weakness and loss of muscle mass that accumulates throughout life. It is common among older adults, but can also occur at a younger age (151). With regard to the masticatory muscles, there is some suggestion that tooth loss is associated with an increased risk, but the data concerning oral muscles and indices of oral hypofunction are inconclusive (152). Furthermore, low muscle mass has been shown to increase the risk of sarcopenia, perhaps also being associated with the loss of molar support, especially in men. Body mass index and age may also influence the mass of skeletal muscles distant from the axial skeleton. Notably, insulin-like growth factor 1 (IGF-1) levels are positively associated with masseter muscle volume, and the alpha-actinin-3 (ACTN3) genotype has been associated with its reduction only in men (153). Similar trends are indicated for oral function and skeletal muscle in cases of sarcopenia, which may show low oral function (154).
Sialadenitis	Sialadenitis is the term used to describe inflammation or infection of the salivary glands. This condition can be caused by various processes, including infectious, obstructive and autoimmune ones (155).
Spondylolisthesis	This injury involves one vertebra being displaced ventrally over another, which can result in pain and impaired mobility. At the cervical level, it can result in neck pain and stiffness, as well as heterotopic pain in areas corresponding to the affected dermatomes. Maintaining an upright posture and balance requires proper spinal alignment, which refers to the arrangement of the vertebrae and the curvature of the spine (156). When cervical spondylolisthesis occurs, it is generally associated with mild degenerative changes due to the interaction of anatomical structures, biomechanical effects and neurosensory factors, particularly when treating elderly patients who require balance and stable posture (157).
Spondylosis	Spondylosis is a degenerative condition affecting the bones and intervertebral discs of the spine. When it occurs at the suprascapular level, it is known as cervical spondylosis. There are different pathophysiological mechanisms. As well as stress and overload, which are associated with ageing, there are a number of related factors. These include an imbalance in the nervous, immune and endocrine systems, which generate various biological signals that act on different receptors in the cervical region (158). Biomechanical alterations affect the vertebral artery and the surrounding muscles and connective tissues, generating inflammatory cascades associated with cervical spondylosis. These cascades promote the release of various hormones that affect the central nervous system through the neuroendocrine-immune system, inducing or exacerbating negative emotional feedback and establishing a vicious 'central-local-central' cycle. One of the most common symptoms of cervical spondylosis is cervical pain, which originates in the facet joints, intervertebral discs and cervical muscles. It is also one of the most important factors affecting patients' quality of life. Magnetic resonance imaging offers good resolution of the spinal cord, intervertebral discs, muscles and other soft tissues for diagnostic imaging. Computed tomography, on the other hand, provides excellent contrast for evaluating bony structures such as the vertebrae. In both cases, sagittal views clearly demonstrate the intervertebral discs and cervical sagittal balance, while axial views provide an accurate assessment of nerve root compression and muscle fatty infiltration (159).
Sticky or static disc	The 'sticky or static disc' phenomenon refers to the limited mobility of the articular disc in the TMJ due to adhesions or loss of lubrication. This can result in disc anchorage, clicking, locking, or restricted movement (61).
SUNA Unilateral neuralgiform headache with autonomic symptoms	SUNA syndrome (unilateral neuralgiform headache of short duration with cranial autonomic symptoms) is on the same spectrum as SUNCT. It is characterised by brief, repeated attacks of unilateral pain accompanied by one or more cranial autonomic symptoms, but not conjunctival redness and tearing (160).
SUNCT Unilateral neuralgiform headache with watering and redness	SUNCT syndrome (short-term unilateral neuralgiform headache with autonomic symptoms) is a rare type of autonomic trigeminal headache. It is characterised by brief, recurrent and intense attacks of unilateral pain in the orbital or temporal region. These attacks are necessarily accompanied by ipsilateral conjunctival injection and tearing (160).
Synovial chondromatosis	It is also known as synovial osteochondromatosis or synovial chondrometaplasia. It is a joint disorder in which nodules of cartilage form and detach from the synovial membrane of the joints. These nodules then remain floating within the capsular space. This can lead to oedema, pain and disorders affecting joint movement. This benign, locally aggressive tumour rarely affects the TMJ (161). Differential diagnoses for this condition include pigmented villonodular synovitis, tenosynovial giant cell tumour and chondrosarcoma. Synovial chondromatosis frequently occurs in large joints, such as the knee, hip and temporomandibular joint. It is most commonly diagnosed in the fifth decade of life. However, the disorder can also occur in children (162).
Synovitis	Synovitis is defined as an inflammatory process or arthritis involving the synovial tissue and characterized by arthralgia. Synovitis is an initial process that alters joint function and mandibular movement. Hyperemia, an increase in fibroblast- and macrophage-type synoviocytes, and changes in cytokines are observed. Therefore, the progression of synovitis could lead to osteocartilaginous damage. It is also known that persistent inflammation in the TMJ causes neoangiogenesis, which could affect the soft tissues of the capsule, including the retrodiscal tissue and the articular disc, as well as the glenoid fossa and the mandibular condyle (163).

To be continued

10.59987/ads/2026.1. 120-147

Temporal tendinitis	Temporal tendinitis is an inflammatory and degenerative disorder affecting the fibrous insertion of the temporalis muscle tendons into the coronoid process of the mandible (164).
Tension-type headache	Tension-type headache is the most common primary headache disorder, affecting around 2.1 billion people worldwide. It has a multifactorial pathophysiology. Consistent findings from neuroimaging and neurophysiological studies point to alterations in brain regions involved in pain modulation, particularly the anterior cingulate cortex and insula. Peripheral factors, such as myofascial pain and muscle tenderness, are believed to play a role in the onset and persistence of symptoms. Central sensitisation and impaired descending pain inhibition are also believed to play a central role. In turn, sensitisation may be confined to the brainstem rather than involving cortical structures. Calcitonin gene-related peptide and vascular mechanisms appear to be involved to a limited extent, except in subgroups of mixed headaches (165).
Trigeminal autonomic headaches	This type of headache belongs to a group of primary headaches characterised by short-term, intense, unilateral pain in the dermatomes associated with the trigeminal nerve. These headaches are accompanied by parasympathetic autonomic symptoms, including conjunctival injection and/or tearing, nasal congestion and/or rhinorrhoea, oedema of the eyelids, sweating of the forehead and face, miosis and/or ptosis, and restlessness or agitation. Examples include cluster headaches, paroxysmal hemicrania, short-term unilateral neuralgiform headache attacks, hemicrania continua and probable trigeminal autonomic headaches (118,119).
Vertebral agenesis	It is a congenital condition in which one or more vertebrae fail to form properly. It is rare in the cervical spine, but more commonly observed in the sacral region. It may be associated with syndromes involving cardiorespiratory problems and premature death, but little information is available on this subject (166).
Vertebral aplasia and hypoplasia	Underdevelopment of the vertebral column manifests as hypoplasia or aplasia of the vertebral centrum specifically, without any corresponding alterations to the dorsal vertebral arch, as this occurs in the final stage of gestation. Images reveal a partial or complete absence of the vertebral centrum, with intact pedicles and a posterior body extending up to the neurocentral synchondrosis. This spectrum also includes dorsal hemivertebrae with an isolated absence or wedging of the ventral portion of the centrum (167).
Vertebral fractures	Vertebral fractures are caused by axial loads, either with or without a rotational or dislocation component. These loads may be abnormal, such as those generated by trauma, or normal loads acting on weakened bone (for example, due to osteoporosis or neoplasia) (168). According to the AO Foundation classification (169), trauma is subdivided into four regions. One region is the upper cervical spine, which extends from the occipital condyle to the axis. The other corresponds to the subaxial cervical spine between C3 and C7. Each region has a subclassification. Fractures of the upper cervical spine can involve the occipital condyle, the atlas ring or the axis. The severity of each fracture varies, ranging from an isolated injury without displacement or ligament injury without displacement to instability and/or translation of the fractured body. At the subaxial level, fractures can be compression fractures of the processes or body ranging from minor to burst fractures, or involve injuries to the tendon band that are unilateral or bilateral, vertebral body displacement, or facet injuries that are either without displacement or lead to pathological subluxation.
Vertebral hyperplasia	Vertebral hyperplasia primarily affects adults and older adults. It is increasingly associated with prolonged sitting, which can lead to inactivity and poor posture (170-172). There have also been reports of cases of partial congenital hyperplasia of some vertebral bone structures (173,174).
Vertebral neoplasm	Osteosarcoma is a rare malignant tumour that can affect the vertebrae, including those in the neck. Vertebrae can be affected by neoplastic tumours that, when confined to the spongy nucleus, do not cause spinal instability. However, if they extend to the cortex, pathological compression fractures can occur (175).
Viral myositis	Viral myositis is a rare and acute disease of the skeletal muscles. It is characterised by inflammation, pain and muscle weakness that occur during or after a viral infection. It is most commonly caused by influenza A and B viruses (176).

References

1. Levangie PK, Norkin CC. Joint Structure and Function. 5th ed. F.A. Davis Company; Philadelphia, PA, USA. 2011. Pgg. 501–537.
2. Omar MM, Westersund C, Savastano F, Anton JS. Relationship between occlusion and posture. Int. J. Health Sci. 2023;7:2768–2785. doi: 10.53730/ijhs.v7nS1.14575.
3. Hong SW, Lee JK, Kang JH. Relationship among Cervical Spine Degeneration, Head and Neck postures, and Myofascial Pain in Masticatory and Cervical Muscles in Elderly with Temporomandibular Disorder. Arch Gerontol Geriatr. 2019 Mar-Apr;81:119-128. doi: 10.1016/j.archger.2018.12.004. Epub 2018 Dec 7. PMID: 30554035.
4. Elledge ROC. Classifications for the temporomandibular joint (TMJ): A systematic review of the literature. J Craniomaxillofac Surg. 2024 Aug;52(8):890-894. doi: 10.1016/j.jcms.2024.04.010. Epub 2024 Apr 30. PMID: 39030114.
5. Joseph GM, Patel VL. Domain knowledge and hypothesis generation in diagnostic reasoning. Med Decis Making. 1990 Jan-Mar;10(1):31-46. doi: 10.1177/0272989X9001000107. PMID: 2182962.
6. Lawson AE, Daniel ES. Inferences of clinical diagnostic reasoning and diagnostic error. J Biomed Inform. 2011 Jun;44(3):402-12. doi: 10.1016/j.jbi.2010.01.003. Epub 2010 Jan 18. PMID: 20093196.

7. Harrison JE, Weber S, Jakob R, Chute CG. ICD-11: an international classification of diseases for the twenty-first century. BMC Med Inform Decis Mak. 2021 Nov 9;21(Suppl 6):206. doi: 10.1186/s12911-021-01534-6. PMID: 34753471; PMCID: PMC8577172.
8. Chute CG, Çelik C. Overview of ICD-11 architecture and structure. BMC Med Inform Decis Mak. 2022 May 16;21(Suppl 6):378. doi: 10.1186/s12911-021-01539-1. PMID: 35578335; PMCID: PMC9109286.
9. The National Center for Health Statistics (NHCS). The International Classification of Diseases, 10th revision, Clinical Modification. https://www.cdc.gov/nchs/icd/icd-10-cm/
10. International Classification of Orofacial Pain, 1st edition (ICOP). Cephalalgia. 2020 Feb;40(2):129-221. doi: 10.1177/0333102419893823. PMID: 32103673.
11. Treede RD, Rief W, Barke A, Aziz Q, Bennett MI, Benoliel R, Cohen M, Evers S, Finnerup NB, First MB, Giamberardino MA, Kaasa S, Kosek E, Lavand'homme P, Nicholas M, Perrot S, Scholz J, Schug S, Smith BH, Svensson P, Vlaeyen JWS, Wang SJ. A classification of chronic pain for ICD-11. Pain. 2015 Jun;156(6):1003-1007. doi: 10.1097/j.pain.0000000000000160. PMID: 25844555; PMCID: PMC4450869.
12. Okeson J. Bell´s Oral adn Facial Pain. Quintessence Publishing Co. ISBN: 9781647240639.
13. de Leeuw R, Klasser GD. The American Academy of Orofacial Pain. Orofacial Pain. Guideliness for Assessment, Diagnosis, and Management. Quintessence Publishing, 2018. ISBN: 9780867157680.
14. Schiffman E, Ohrbach R, Truelove E, Look J, Anderson G, Goulet JP, List T, Svensson P, Gonzalez Y, Lobbezoo F, Michelotti A, Brooks SL, Ceusters W, Drangsholt M, Ettlin D, Gaul C, Goldberg LJ, Haythornthwaite JA, Hollender L, Jensen R, John MT, De Laat A, de Leeuw R, Maixner W, van der Meulen M, Murray GM, Nixdorf DR, Palla S, Petersson A, Pionchon P, Smith B, Visscher CM, Zakrzewska J, Dworkin SF; International RDC/TMD Consortium Network, International association for Dental Research; Orofacial Pain Special Interest Group, International Association for the Study of Pain. Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) for Clinical and Research Applications: recommendations of the International RDC/TMD Consortium Network* and Orofacial Pain Special Interest Group†. J Oral Facial Pain Headache. 2014 Winter;28(1):6-27. doi: 10.11607/jop.1151. PMID: 24482784; PMCID: PMC4478082.
15. Peck CC, Goulet JP, Lobbezoo F, Schiffman EL, Alstergren P, Anderson GC, de Leeuw R, Jensen R, Michelotti A, Ohrbach R, Petersson A, List T. Expanding the taxonomy of the diagnostic criteria for temporomandibular disorders. J Oral Rehabil. 2014 Jan;41(1):2-23. doi: 10.1111/joor.12132. PMID: 24443898; PMCID: PMC4520529.
16. Okeson J. Management of Temporomandibular Disorders and Occlusion. 8th EditionElsevier Mosby. 2019. ISBN:9780323676748.
17. Capurro D, Rada G. El proceso diagnóstico [The diagnostic process]. Rev Med Chil. 2007 Apr;135(4):534-8. Spanish. doi: 10.4067/s0034-98872007000400018. Epub 2007 May 16. PMID: 17554465.
18. Bułdyś K, Górnicki T, Kałka D, Szuster E, Biernikiewicz M, Markuszewski L, Sobieszczańska M. What Do We Know about Nociplastic Pain? Healthcare (Basel). 2023 Jun 17;11(12):1794. doi: 10.3390/healthcare11121794. PMID: 37372912; PMCID: PMC10298569.
19. Mishra N, Sharma NK, Dhiman NK, Jaiswara C, Tiwari P, Singh AK. Temporomandibular joint ankylosis: A tertiary center-based epidemiological study. Natl J Maxillofac Surg. 2021 Sep-Dec;12(3):392-396. doi: 10.4103/njms.NJMS_57_20. Epub 2021 Dec 13. PMID: 35153437; PMCID: PMC8820309.
20. Bi R, Jiang N, Yin Q, Chen H, Liu J, Zhu S. A new clinical classification and treatment strategies for temporomandibular joint ankylosis. Int J Oral Maxillofac Surg. 2020 Nov;49(11):1449-1458. doi: 10.1016/j.ijom.2020.02.020. Epub 2020 Apr 1. PMID: 32247547.
21. Li JM, An JG, Wang X, Yan YB, Xiao E, He Y, Zhang Y. Imaging and histologic features of traumatic temporomandibular joint ankylosis. Oral Surg Oral Med Oral Pathol Oral Radiol. 2014 Sep;118(3):330-7. doi: 10.1016/j.oooo.2014.05.007. Epub 2014 May 20. PMID: 25151587.
22. Siccardi D, Buzzatti L, Marini M, Cattrysse E. Analysis of three-dimensional facet joint displacement during two passive upper cervical mobilizations. Musculoskelet Sci Pract. 2020 Dec;50:102218. doi: 10.1016/j.msksp.2020.102218. Epub 2020 Jul 30. PMID: 32882623.
23. Cattrysse E, Provyn S, Kool P, Clarys JP, Van Roy P. Morphology and kinematics of the atlanto-axial joints and their interaction during manual cervical rotation mobilization. Man Ther. 2011 Oct;16(5):481-6. doi: 10.1016/j.math.2011.03.002. Epub 2011 Apr 20. PMID: 21511513.
24. González DCN, Ardura Aragón F, Sanjuan JC, Maniega SS, Andrino AL, García Fraile R, Labrador Hernández G, Calabia-Campo J, Caballero-García A, Córdova-Martínez A. C1-C2 Rotatory Subluxation in Adults "A Narrative Review". Diagnostics (Basel). 2022 Jul 2;12(7):1615. doi: 10.3390/diagnostics12071615. PMID: 35885520; PMCID: PMC9316247.
25. Eghbal K, Jangiaghdam H, Tayebi Khorami S, Shabani Mayani A, Seif B, Zoghi S. Long-term outcome of adult traumatic atlantoaxial rotatory fixation: a case series. J Med Case Rep. 2024 Oct 11;18(1):469. doi: 10.1186/s13256-024-04748-0. PMID: 39390610; PMCID: PMC11468385.
26. Kamakura F, Shimizu S, Shigenobu K, Yasuda G, Ishigaki Y, Goto S. Unusual Upper Cervical Spine Disorder and Non-traumatic Atlantoaxial Rotatory Fixation in an Adult: A Case Report. Cureus. 2024 Dec 3;16(12):e75051. doi: 10.7759/cureus.75051. PMID: 39749052; PMCID: PMC11695073.
27. Kempeneers MA, Buis DR, Feller RE, Roosendaal SD, Slot KM, Wolf NI, Vandertop WP. Torticollis with Atlantoaxial Rotatory Subluxation in Children: A Clinical Review. Neuropediatrics. 2024 Dec;55(6):349-355. doi: 10.1055/a-2312-9994. Epub 2024 Apr 24. PMID: 38657679.
28. Roemer FW, Guermazi A, Javaid MK, Lynch JA, Niu J, Zhang Y, Felson DT, Lewis CE, Torner J, Nevitt MC; MOST Study investigators. Change in MRI-detected subchondral bone marrow lesions is associated with cartilage loss: the MOST Study. A longitudinal multicentre study of knee osteoarthritis. Ann Rheum Dis. 2009 Sep;68(9):1461-5. doi: 10.1136/ard.2008.096834. Epub 2008 Oct 1. PMID: 18829615; PMCID: PMC2905622.
29. Akhavan S, Martinkovich SC, Kasik C, DeMeo PJ. Bone Marrow Edema, Clinical Significance, and Treatment Options: A Review. J Am Acad Orthop Surg. 2020 Oct 15;28(20):e888-e899. doi: 10.5435/JAAOS-D-20-00142. PMID: 32701688.
30. Wahaj A, Hafeez K, Zafar MS. Association of bone marrow edema with temporomandibular joint (TMJ) osteoarthritis and internal derangements. Cranio. 2017 Jan;35(1):4-9. doi: 10.1080/08869634.2016.1156282. Epub 2016 Apr 8. PMID: 27077262.
31. Gharavi SM, Qiao Y, Faghihimehr A, Vossen J. Imaging of the Temporomandibular Joint. Diagnostics (Basel). 2022 Apr 16;12(4):1006. doi: 10.3390/diagnostics12041006. PMID: 35454054; PMCID: PMC9031630.
32. H. Gobel, "13.11 Burning mouth syndrome (BMS). ICHD-3," (n.d.), accessed October 24, 2023, https://ichd-3.org/13-painful-cranial-neuropathies-and-other-facial-pains/13-11-persistent-idiopathic-facial-pain-pifp/.
33. Lebel A, Lescaille G, Alajbeg I, Alajbeg I, Boucher Y. The Role of Stress in Burning Mouth Syndrome Triggered by Dental Treatments: A Two-Step Hypothesis. J Oral Rehabil. 2025 Jul;52(7):1001-1014. doi: 10.1111/joor.13964. Epub 2025 Mar 26. PMID: 40143520; PMCID: PMC12162416.
34. Suga T, Toyofuku A. Psychosomatic concepts in nociplastic pain: The case of burning mouth syndrome. J Dent Sci. 2025 Jul;20(3):1972-1973. doi: 10.1016/j.jds.2025.04.020. Epub 2025 Apr 30. PMID: 40654485; PMCID: PMC12254759.
35. Zheng H, Zhong ZJ, Wang YC, Sun YB, Li FF. Downregulation of interleukin 11 regulates the transforming growth factor-β/ERK1/2 signaling pathway to inhibit articular capsule fibrosis and alleviate post-traumatic articular capsule contracture. J Shoulder Elbow Surg. 2025 Feb;34(2):584-

10.59987/ads/2026.1. 120-147

594. doi: 10.1016/j.jse.2024.05.057. Epub 2024 Jul 30. PMID: 39089417.
36. Curl DD, Stanwood G. Chiropractic management of capsulitis and synovitis of the temporomandibular joint. J Orofac Pain. 1993 Summer;7(3):283-93. PMID: 9116628.
37. Keerthi R, Quadri A. Hyoid Bone Fracture: Associated With Head and Neck Trauma-A Rare Case Report. J Maxillofac Oral Surg. 2016 Jul;15(Suppl 2):249-52. doi: 10.1007/s12663-015-0761-x. Epub 2015 Apr 21. PMID: 27408446; PMCID: PMC4925585.
38. Marcon RM, Cristante AF, Teixeira WJ, Narasaki DK, Oliveira RP, de Barros Filho TE. Fractures of the cervical spine. Clinics (Sao Paulo). 2013 Nov;68(11):1455-61. doi: 10.6061/clinics/2013(11)12. PMID: 24270959; PMCID: PMC3812556.
39. Huggare J. Association between morphology of the first cervical vertebra, head posture, and craniofacial structures. Eur J Orthod. 1991 Dec;13(6):435-40. doi: 10.1093/ejo/13.6.435. PMID: 1817068.
40. Gül G, Tekin A, Çakir Ö. Is There Cervical Muscle Weakness in Patients with Loss of Cervical Lordosis? Morphometric Evaluation of Muscle Cross-Sectional Areas on MRI. International Journal of Morphology. 2025;43:373-378. doi: 10.4067/S0717-95022025000200373.
41. Stiesch-Scholz M, Fink M, Tschernitschek H. Comorbidity of internal derangement of the temporomandibular joint and silent dysfunction of the cervical spine. J Oral Rehabil. 2003 Apr;30(4):386-91. doi: 10.1046/j.1365-2842.2003.01034.x. PMID: 12631162.
42. Mannheimer JS, Rosenthal RM. Acute and chronic postural abnormalities as related to craniofacial pain and temporomandibular disorders. Dent Clin North Am. 1991 Jan;35(1):185-208. PMID: 1997352.
43. Olivo SA, Fuentes J, Major PW, Warren S, Thie NM, Magee DJ. The association between neck disability and jaw disability. J Oral Rehabil. 2010 Sep;37(9):670-9. doi: 10.1111/j.1365-2842.2010.02098.x. Epub 2010 May 27. PMID: 20524969.
44. Coskun Benlidayi I MD, Guzel R MD, Tatli U PhD, Salimov F PhD, Keceli O PhD. The relationship between neck pain and cervical alignment in patients with temporomandibular disorders. Cranio. 2020 May;38(3):174-179. doi: 10.1080/08869634.2018.1498181. Epub 2018 Jul 26. PMID: 30048225.
45. Merskey H, Bodguk N. Classification of chronic pain: descriptions of chronic pain syndromes and definitions of pain terms: 2nd ed. Seattle, Wash. IASP Press, 1994.
46. Birklein F, Dimova V. Complex regional pain syndrome-up-to-date. Pain Rep. 2017 Oct 5;2(6):e624. doi: 10.1097/PR9.0000000000000624. PMID: 29392238; PMCID: PMC5741324.
47. Di Pietro F, McAuley JH, Parkitny L, Lotze M, Wand BM, Moseley GL, Stanton TR. Primary somatosensory cortex function in complex regional pain syndrome: a systematic review and meta-analysis. J Pain. 2013 Oct;14(10):1001-18. doi: 10.1016/j.jpain.2013.04.001. Epub 2013 May 29. PMID: 23726046.
48. Kirveskari E, Vartiainen NV, Gockel M, Forss N. Motor cortex dysfunction in complex regional pain síndrome. Clin. Neurophysiol. 2010;121(7):1085-1091. doi: 10.1016/j.clinph.2010.01.032.
49. Galea CJ, Dashow JE, Woerner JE. Congenital Abnormalities of the Temporomandibular Joint. Oral Maxillofac Surg Clin North Am. 2018 Feb;30(1):71-82. doi: 10.1016/j.coms.2017.09.003. PMID: 29153239.
50. Hegde S, Praveen BN, Shetty SR. Morphological and radiological variations of mandibular condyles in health and diseases: a systematic review. Dentistry 2013;3(1):154. doi:10.4172/2161-1122.1000154.
51. González-Garrido L, Gómez-González S, Gonzalo-Orden JM, Wasterlain SN. Multi-headed (bifid and trifid) mandibular condyles in archaeological contexts: Two posttraumatic cases. Arch Oral Biol. 2022 Feb;134:105326. doi: 10.1016/j.archoralbio.2021.105326. Epub 2021 Nov 29. PMID: 34871890.
52. Lim J, Jo W, Jeon H, Song SI, Lee JK. Condylar position changes and prognosis in patients with unilateral mandibular condylar fracture treated non-surgically. Maxillofac Plast Reconstr Surg. 2024 Dec 27;46(1):42. doi: 10.1186/s40902-024-00454-5. PMID: 39725784; PMCID: PMC11671456.
53. Zachariades N, Mezitis M, Mourouzis C, Papadakis D, Spanou A. Fractures of the mandibular condyle: a review of 466 cases. Literature review, reflections on treatment and proposals. J Craniomaxillofac Surg. 2006 Oct;34(7):421-32. doi: 10.1016/j.jcms.2006.07.854. Epub 2006 Oct 19. PMID: 17055280.
54. Neff A, Cornelius CP, Rasse M, Torre DD, Audigé L. The Comprehensive AOCMF Classification System: Condylar Process Fractures - Level 3 Tutorial. Craniomaxillofac Trauma Reconstr. 2014 Dec;7(Suppl 1):S044-58. doi: 10.1055/s-0034-1389559. PMID: 25489390; PMCID: PMC4251730.
55. Narváez JTA., Ochoa KAA, Cajilima SMM, Calderón MEB. Vertical asymmetry of the condyle and mandibular ramus measured by cone beam computed tomography (CBCT). World Journal of Advanced Research and Reviews 2023; 17(01), 1146-1152. doi: 10.30574/wjarr.2023.17.1.0143.
56. Wu Z, Lin Y, Lin Y. Impact of age on degenerative joint disease of the temporomandibular joint: A systematic review and meta-analysis. Medicine (Baltimore). 2025 Apr 25;104(17):e41915. doi: 10.1097/MD.0000000000041915. PMID: 40295246; PMCID: PMC12040012.
57. Zheng H, Shi L, Lu H, Liu Z, Yu M, Wang Y, Wang H. Influence of edentulism on the structure and function of temporomandibular joint. Heliyon. 2023 Sep 23;9(10):e20307. doi: 10.1016/j.heliyon.2023.e20307. PMID: 37829806; PMCID: PMC10565695.
58. Farella M, Soneda K, Vilmann A, Thomsen CE, Bakke M. Jaw muscle soreness after tooth-clenching depends on force level. J Dent Res. 2010 Jul;89(7):717-21. doi: 10.1177/0022034510365448. Epub 2010 May 3. PMID: 20439931.
59. Jou YT. Dental deafferentation and brain damage: A review and a hypothesis. Kaohsiung J Med Sci. 2018 Apr;34(4):231-237. doi: 10.1016/j.kjms.2018.01.013.
60. Lyberg T, Krogstad O, Djupesland G. Cephalometric analysis in patients with obstructive sleep apnoea syndrome: II. Soft tissue morphology. J Laryngol Otol. 1989 Mar;103(3):293-7. doi: 10.1017/s0022215100108746. PMID: 2703771.
61. Nitzan DW, Etsion I. Adhesive force: the underlying cause of the disc anchorage to the fossa and/or eminence in the temporomandibular joint--a new concept. Int J Oral Maxillofac Surg. 2002 Feb;31(1):94-9. doi: 10.1054/ijom.2001.0153. PMID: 11936408.
62. Okeson JP. Joint intracapsular disorders: diagnostic and nonsurgical management considerations. Dent Clin North Am. 2007 Jan;51(1):85-103, vi. doi: 10.1016/j.cden.2006.09.009. PMID: 17185061.
63. Nitzan DW, Marmary Y. The "anchored disc phenomenon": a proposed etiology for sudden-onset, severe, and persistent closed lock of the temporomandibular joint. J Oral Maxillofac Surg. 1997 Aug;55(8):797-802; discussion 802-3. doi: 10.1016/s0278-2391(97)90335-9. PMID: 9251606.
64. Al-Belasy FA, Salem AS. Is the Anchored Disc Phenomenon a Truly Distinct Entity? A Systematic Review. J Oral Maxillofac Surg. 2018 Sep;76(9):1883.e1-1883.e10. doi: 10.1016/j.joms.2018.04.033. Epub 2018 May 9. PMID: 29856938.
65. Millon-Cruz A, Martín-Granizo R, Encinas A, Berguer A. Relationship between intra-articular adhesions and disc position in temporomandibular joints: Magnetic resonance and arthroscopic findings and clinical results. J Craniomaxillofac Surg. 2015 May;43(4):497-502. doi: 10.1016/j.jcms.2015.02.010. Epub 2015 Feb 24. PMID: 25797387.
66. Özel Ş, Tunç S, Şenol AU. Association between clinical findings and 3T MRI features in temporomandibular joint disorders. BMC Oral Health. 2025 Jun 5;25(1):921. doi: 10.1186/s12903-025-06283-w.
67. Jiang N, Tan P, Sun Y, Zhou J, Ren R, Li Z, Zhu S. Microstructural, Micromechanical Atlas of the Temporomandibular Joint Disc. J Dent Res. 2024 May;103(5):555-564. doi: 10.1177/00220345241227822. Epub 2024 Apr 9. PMID:

38594786.
68. Zhou L, Tao K, Ma J, Pan X, Zhang K, Feng J. Relationship between temporomandibular joint space and articular disc displacement. BMC Oral Health. 2025 Apr 21;25(1):611. doi: 10.1186/s12903-025-05991-7. PMID: 40254585; PMCID: PMC12010630.
69. Dias GM, Grossmann E, Carvalho ACP, Devito KL, Dos Santos MF, Ferreira LA. MRI changes and clinical characteristics in temporomandibular joints with displacement of the articular disk without reduction - a cross-sectional observational study. Cranio. 2025 May;43(3):521-530. doi: 10.1080/08869634.2023.2203039. Epub 2023 Apr 25. PMID: 37097122.
70. Zhang C, Li CM, Jiang Q. Role of oxidative stress in intervertebral disc degeneration: mechanisms, pathogenesis, and therapeutic strategies. Mol Biol Rep. 2025 May 2;52(1):444. doi: 10.1007/s11033-025-10538-8.
71. Kang H, Bao GJ, Qi SN. Biomechanical responses of human temporomandibular joint disc under tension and compression. Int J Oral Maxillofac Surg. 2006 Sep;35(9):817-21. doi: 10.1016/j.ijom.2006.03.005. Epub 2006 May 11. PMID: 16697140.
72. Liu XM, Zhang SY, Yang C, Chen MJ, Y Cai X, Haddad MS, Yun B, Chen ZZ. Correlation between disc displacements and locations of disc perforation in the temporomandibular joint. Dentomaxillofac Radiol. 2010 Mar;39(3):149-56. doi: 10.1259/dmfr/72395946. PMID: 20203276; PMCID: PMC3520220.
73. Marisio LG, Santamaría CA, Alzérreca AJ. Síndrome de Eagle: Revisión de la literatura. Revista de otorrinolaringología y cirugía de cabeza y cuello. 2016;76(1), 121-126. doi: 10.4067/S0718-48162016000100017.
74. Frugone-Zambra R, Berzaghi A, Gómez-Álvarez J, Bianchi A, Bortolini S. Eagle and Ernest syndromes: a scoping review. Minerva Dent Oral Sci. 2024 Apr;73(2):119-130. doi: 10.23736/S2724-6329.22.04724-6. Epub 2022 Oct 5. PMID: 36197280.
75. Rocabado M. Biomechanical relationship of the cranial, cervical, and hyoid regions. J Craniomandibular Pract. 1983 Jun-Aug;1(3):61-6. doi: 10.1080/07345410.1983.11677834. PMID: 6586872.
76. Pearson WG Jr, Hindson DF, Langmore SE, Zumwalt AC. Evaluating swallowing muscles essential for hyolaryngeal elevation by using muscle functional magnetic resonance imaging. Int J Radiat Oncol Biol Phys. 2013 Mar 1;85(3):735-40. doi: 10.1016/j.ijrobp.2012.07.2370. Epub 2012 Sep 18. PMID: 22995662; PMCID: PMC3563921.
77. Peñarrocha-Oltra D, Ata-Ali J, Ata-Ali F, Peñarrocha-Diago MA, Peñarrocha M. Treatment of orofacial pain in patients with stylomandibular ligament syndrome (Ernest Syndrome). Neurologia. 2013 Jun;28(5):294-8. English, Spanish. doi: 10.1016/j.nrl.2012.06.009. Epub 2012 Aug 14. PMID: 22898469.
78. Daentzer D, Welke B, Baseem Ismail A, Daentzer J, Plagge J. Zervikaler Bandscheibenvorfall : Symptomatik, Diagnostik, Therapie [Cervical disc herniation : Symptomatology, diagnostics, therapy]. Orthopadie (Heidelb). 2025 Jan;54(1):26-39. German. doi: 10.1007/s00132-024-04582-8. Epub 2024 Nov 19. PMID: 39560707.
79. Pertes RA, Gross SG. Clinical management of temporomandibular disorders and orofacial pain. 1st ed. Chicago: Quintessence; 1995. p. 79-86.
80. Braga SP, Cunha CO, Michelotti A, Bonjardim LR, Conti PCR. Association between generalized joint hypermobility, temporomandibular joint hypertranslation and temporomandibular disorders: a scoping review. J Appl Oral Sci. 2025 Mar 21;33:e20240302. doi: 10.1590/1678-7757-2024-0302. PMID: 40136156; PMCID: PMC12005711.
81. Wolford LM, Cardenas L. Idiopathic condylar resorption: diagnosis, treatment protocol, and outcomes. Am J Orthod Dentofacial Orthop. 1999 Dec;116(6):667-77. doi: 10.1016/s0889-5406(99)70203-9. PMID: 10587602.
82. Catherine Z, Breton P, Bouletreau P. Condylar resorption after orthognathic surgery: A systematic review. Rev Stomatol Chir Maxillofac Chir Orale. 2016 Feb;117(1):3-10. doi: 10.1016/j.revsto.2015.11.002. Epub 2015 Dec 10. PMID: 26682945.
83. Arnett GW, Milam SB, Gottesman L. Progressive mandibular retrusion--idiopathic condylar resorption. Part I. Am J Orthod Dentofacial Orthop. 1996 Jul;110(1):8-15. doi: 10.1016/s0889-5406(96)70081-1. PMID: 8686682.
84. Gauer RL, Semidey MJ. Diagnosis and treatment of temporomandibular disorders. Am Fam Physician. 2015 Mar 15;91(6):378-86. PMID: 25822556.
85. Guapi Nauñay VH, Martínez Carvajal IA. Síndrome de Klippel-Feil autosómico dominante: Una malformación de segmentación vertebral. Revista chilena de pediatría 2019; 90(2): 194-201. doi: 10.32641/rchped.v90i2.779.
86. Zhang Z, Wang J, Ge R, Guo C, Liang Y, Liu H, Xu S. A novel classification that defines the normal cervical spine: an analysis based on 632 asymptomatic Chinese volunteers. Eur Spine J. 2024 Jan;33(1):155-165. doi: 10.1007/s00586-023-07997-7. Epub 2023 Oct 25. PMID: 37880410.
87. Poveda-Roda R, Bagán JV, Sanchis JM, Margaix M. Pseudotumors and tumors of the temporomandibular joint. A review. Med Oral Patol Oral Cir Bucal. 2013 May 1;18(3):e392-402. doi: 10.4317/medoral.18799. PMID: 23385515; PMCID: PMC3668863.
88. Panesar K, Susarla SM. Mandibular Fractures: Diagnosis and Management. Semin Plast Surg. 2021 Oct 11;35(4):238-249. doi: 10.1055/s-0041-1735818. PMID: 34819805; PMCID: PMC8604616.
89. Avery LL, Susarla SM, Novelline RA. Multidetector and three-dimensional CT evaluation of the patient with maxillofacial injury. Radiol Clin North Am. 2011 Jan;49(1):183-203. doi: 10.1016/j.rcl.2010.07.014. PMID: 21111135.
90. Frugone-Zambra R, Angulo M, Doñas P. Atlas y manual práctico de preclínico para prótesis fija. Editorial Texidó 2002.
91. Camani LA. Estudio mecánico del aparato dentario. Ed. Adiar 1962. Cap 1.
92. GBD 2021 Nervous System Disorders Collaborators. Global, regional, and national burden of disorders affecting the nervous system, 1990-2021: a systematic analysis for the Global Burden of Disease Study 2021. Lancet Neurol. 2024 Apr;23(4):344-381. doi: 10.1016/S1474-4422(24)00038-3. Epub 2024 Mar 14. Erratum in: Lancet Neurol. 2024 May;23(5):e9. doi: 10.1016/S1474-4422(24)00114-5. Erratum in: Lancet Neurol. 2024 Jul;23(7):e11. doi: 10.1016/S1474-4422(24)00231-X. PMID: 38493795; PMCID: PMC10949203.
93. Dodick DW. Migraine. Lancet. 2018 Mar 31;391(10127):1315-1330. doi: 10.1016/S0140-6736(18)30478-1. Epub 2018 Mar 6. PMID: 29523342.
94. Raggi A, Leonardi M, Arruda M, Caponnetto V, Castaldo M, Coppola G, Della Pietra A, Fan X, Garcia-Azorin D, Gazerani P, Grangeon L, Grazzi L, Hsiao FJ, Ihara K, Labastida-Ramirez A, Lange KS, Lisicki M, Marcassoli A, Montisano DA, Onan D, Onofri A, Pellesi L, Peres M, Petrušić I, Raffaelli B, Rubio-Beltran E, Straube A, Straube S, Takizawa T, Tana C, Tinelli M, Valeriani M, Vigneri S, Vuralli D, Waliszewska-Prosół M, Wang W, Wang Y, Wells-Gatnik W, Wijeratne T, Martelletti P. Hallmarks of primary headache: part 1 - migraine. J Headache Pain. 2024 Oct 31;25(1):189. doi: 10.1186/s10194-024-01889-x. PMID: 39482575; PMCID: PMC11529271.
95. Chemello F, Bean C, Cancellara P, Laveder P, Reggiani C, Lanfranchi G. Microgenomic analysis in skeletal muscle: expression signatures of individual fast and slow myofibers. PLoS One. 2011 Feb 22;6(2):e16807. doi: 10.1371/journal.pone.0016807. PMID: 21364935; PMCID: PMC3043066.
96. Yin L, Li N, Jia W, Wang N, Liang M, Yang X, Du G. Skeletal muscle atrophy: From mechanisms to treatments. Pharmacol Res. 2021 Oct;172:105807. doi: 10.1016/j.phrs.2021.105807. Epub 2021 Aug 10. PMID: 34389456.
97. Imauchi Y, Sakamoto T, Abe K. A case of atrophy of the masticatory muscles due to a masticatory habit. Eur Arch Otorhinolaryngol. 2002 Nov;259(10):551-3. doi: 10.1007/s00405-002-0499-1. Epub 2002 Jul 4. PMID: 12434192.
98. Wilson MH, Hodgson EJ, Felstead AM. Focal atrophy of the masticatory muscles caused by pure trigeminal motor neuropathy: case report. Br J Oral Maxillofac Surg. 2016

Jan;54(1):e13-4. doi: 10.1016/j.bjoms.2015.08.265. Epub 2015 Sep 18. PMID: 26388069.
99. Rispoli DZ, Camargo PM, Pires JL Jr, Fonseca VR, Mandelli KK, Pereira MAC. Benign masseter muscle hypertrophy. Braz J Otorhinolaryngol. 2008 Sep-Oct;74(5):790-793. doi: 10.1016/S1808-8694(15)31393-8. Erratum in: Braz J Otorhinolaryngol. 2008 Nov-Dec;74(6):949. PMID: 19082365; PMCID: PMC9445953.
100. Arzul L, Corre P, Khonsari RH, Mercier JM, Piot B. Hypertrophie asymétrique des muscles masticateurs [Asymmetric hypertrophy of the masticatory muscles]. Ann Chir Plast Esthet. 2012 Jun;57(3):286-91. French. doi: 10.1016/j.anplas.2012.02.014. Epub 2012 Mar 26. PMID: 22455902.
101. Graziano P, Dell'Aversana Orabona G, Astarita F, Ponzo LM, Nunziata R, Salzano G, Maglitto F, Solari D, Santella A, Cappabianca M, Iaconetta G, Califano L. Bilateral hypertrophy of masseteric and temporalis muscles, our fifteen patients and review of literature. Eur Rev Med Pharmacol Sci. 2016;20(1):7-11. PMID: 26813447.
102. Frugone R, Hernández A, Valenzuela H, Retamal V. Dimensiones del músculo masetero medido mediante ultrasonido, según sexo. Rev Fac Odontol Univ Antioq 2010; 22(1): 7-11. <http://www.scielo.org.co/scielo.php?script=sci_arttext&pid=S0121-246X2010000200002&lng=en&nrm=iso>. ISSN 0121-246X.
103. Hernandez C, Frugone-Zambra R, Valenzuela H, Retamal V. Profundidad del musculo masetero medido mediante ultrasonido, según indice facial en relacion al sexo. Int. j. morphol. 2012; 30(3):964-969. ISSN 0717-9502. doi: 10.4067/S0717-95022012000300033.
104. Morse MH, Brown EF. Ultrasonic diagnosis of masseteric hypertrophy. Dentomaxillofac Radiol. 1990 Feb;19(1):18-20. doi: 10.1259/dmfr.19.1.2201578.
105. Saunders AAE, Thomson RE, Goodman CA, Anderson RL, Gregorevic P. Striated muscle: an inadequate soil for cancers. Cancer Metastasis Rev. 2024 Dec;43(4):1511-1527. doi: 10.1007/s10555-024-10199-2. Epub 2024 Jul 12. PMID: 38995522; PMCID: PMC11554797.
106. Burningham Z, Hashibe M, Spector L, Schiffman JD. The epidemiology of sarcoma. Clin Sarcoma Res. 2012 Oct 4;2(1):14. doi: 10.1186/2045-3329-2-14. PMID: 23036164; PMCID: PMC3564705.
107. Hernández-Nuño de la Rosa MF, Guerrero P, Alturki SA, Scrivani SJ. Masticatory Myofascial Pain Disorders. Dent Clin North Am. 2023 Jan;67(1):1-11. doi: 10.1016/j.cden.2022.07.001. Epub 2022 Oct 27. PMID: 36404071.
108. Jin Q, Chang Y, Lu C, Chen L, Wang Y. Referred pain: characteristics, possible mechanisms, and clinical management. Front Neurol. 2023 Jun 28;14:1104817. doi: 10.3389/fneur.2023.1104817. PMID: 37448749; PMCID: PMC10338069.
109. Santi MD, Botte MJ. Volkmann's ischemic contracture of the foot and ankle: evaluation and treatment of established deformity. Foot Ankle Int. 1995 Jun;16(6):368-77. doi: 10.1177/107110079501600610. PMID: 7550946.
110. Okeson JP, de Leeuw R. Differential diagnosis of temporomandibular disorders and other orofacial pain disorders. Dent Clin North Am. 2011 Jan;55(1):105-20. doi: 10.1016/j.cden.2010.08.007. PMID: 21094721.
111. Yamaguchi T, Satoh K, Komatsu K, Inoue N, Minowa K, Totsuka Y. Electromyographic activity of the jaw-closing muscles during jaw opening in patients with masseter muscle contracture. Cranio. 2002 Jan;20(1):48-54. doi: 10.1080/08869634.2002.11746190. PMID: 11831344.
112. Dhanrajani PJ, Jonaidel O. Trismus: aetiology, differential diagnosis and treatment. Dent Update. 2002 Mar;29(2):88-92, 94. doi: 10.12968/denu.2002.29.2.88. PMID: 11928347.
113. Hanci, D., Onaran, Ö.İ. (2025). Trismus: Definition, Causes, and Treatment. In: Ünlü, Z., Bayar Muluk, N., Cagliyan Turk, A., Cingi, C. (eds) Physical Therapy and Rehabilitation in Otorhinolaryngology. Springer, Cham. doi: 10.1007/978-3-031-90347-2_76-1.
114. Marien M Jr. Trismus: causes, differential diagnosis, and treatment. Gen Dent. 1997 Jul-Aug;45(4):350-5. PMID: 9515441.
115. Leonard M. Trismus: what is it, what causes it, and how to treat it. Dent Today. 1999 Jun;18(6):74-7. PMID: 10765845.
116. Gundlach KK. Ankylosis of the temporomandibular joint. J Craniomaxillofac Surg. 2010 Mar;38(2):122-30. doi: 10.1016/j.jcms.2009.04.006. Epub 2009 Jun 4. PMID: 19500996.
117. Luyk NH, Steinberg B. Aetiology and diagnosis of clinically evident jaw trismus. Aust Dent J. 1990 Dec;35(6):523-9. doi: 10.1111/j.1834-7819.1990.tb04684.x. PMID: 2090084.
118. Nitzan DW, Shteyer A. Acute facial cellulitis and trismus originating in the external auditory meatus. Oral Surg Oral Med Oral Pathol. 1986 Sep;62(3):262-3. doi: 10.1016/0030-4220(86)90004-6. PMID: 3462629.
119. Louis DN, Perry A, Wesseling P, Brat DJ, Cree IA, Figarella-Branger D, Hawkins C, Ng HK, Pfister SM, Reifenberger G, Soffietti R, von Deimling A, Ellison DW. The 2021 WHO Classification of Tumors of the Central Nervous System: a summary. Neuro Oncol. 2021 Aug 2;23(8):1231-1251. doi: 10.1093/neuonc/noab106. PMID: 34185076; PMCID: PMC8328013.
120. Lange N, Jörger AK, Ryang YM, Liesche-Starnecker F, Gempt J, Meyer B. Primary Bone Tumors of the Spine-Proposal for Treatment Based on a Single Centre Experience. Diagnostics (Basel). 2022 Sep 19;12(9):2264. doi: 10.3390/diagnostics12092264. PMID: 36140664; PMCID: PMC9498005.
121. Headache Classification Committee of the International Headache Society (IHS) The International Classification of Headache Disorders, 3rd edition. Cephalalgia. 2018 Jan;38(1):1-211. doi: 10.1177/0333102417738202. PMID: 29368949.
122. Han A, Montgomery C, Zamora A, Winder E, Kaye A, Carroll C, Aquino A, Kakazu J, Kaye AD. Glossopharyngeal Neuralgia: Epidemiology, Risk factors, Pathophysiology, Differential diagnosis, and Treatment Options. Health Psychol Res. 2022 Jun 28;10(5):36042. doi: 10.52965/001c.36042. PMID: 35774913; PMCID: PMC9239365.
123. Marbach JJ. Is phantom tooth pain a deafferentation (neuropathic) syndrome? Part I: Evidence derived from pathophysiology and treatment. Oral Surg Oral Med Oral Pathol. 1993 Jan;75(1):95-105. doi: 10.1016/0030-4220(93)90413-x. PMID: 8419881.
124. Marbach JJ. Phantom bite. Am J Orthod. 1976 Aug;70(2):190-9. doi: 10.1016/s0002-9416(76)90319-5. PMID: 1066055.
125. Cao Y, Xie QF. [Advance in research of occlusal dysesthesia]. Zhonghua Kou Qiang Yi Xue Za Zhi. 2021 Jan 9;56(1):109-112. Chinese. doi: 10.3760/cma.j.cn112144-20200328-00175. PMID: 34645246.
126. Imhoff B, Ahlers MO, Hugger A, Lange M, Schmitter M, Ottl P, Wolowski A, Türp JC. Occlusal dysesthesia-A clinical guideline. J Oral Rehabil. 2020 May;47(5):651-658. doi: 10.1111/joor.12950. Epub 2020 Mar 18. PMID: 32080883; PMCID: PMC7317831.
127. Blanchet PJ, Abdillahi O, Beauvais C, Rompré PH, Lavigne GJ. Prevalence of spontaneous oral dyskinesia in the elderly: a reappraisal. Mov Disord. 2004 Aug;19(8):892-6. doi: 10.1002/mds.20130. PMID: 15300653.
128. Clark GT, Ram S. Four oral motor disorders: bruxism, dystonia, dyskinesia and drug-induced dystonic extrapyramidal reactions. Dent Clin North Am. 2007 Jan;51(1):225-43, viii-ix. doi: 10.1016/j.cden.2006.09.002. PMID: 17185068.
129. Britton D, Alty JE, Mannion CJ. Oromandibular dystonia: a diagnosis not to miss. Br J Oral Maxillofac Surg. 2020 Jun;58(5):520-524. doi: 10.1016/j.bjoms.2020.02.018. Epub 2020 Mar 3. PMID: 32143935.
130. De Nordenflycht D, Matus G, Aravena JP, Toloza H. Osteochondritis dissecans of the temporomandibular joint: a scoping review. Oral Maxillofac Surg. 2024 Mar;28(1):29-38. doi: 10.1007/s10006-022-01134-2. Epub 2023 Jan 12. PMID: 36631710.
131. Willard VP, Arzi B, Athanasiou KA. The attachments of the temporomandibular joint disc: a biochemical and histological investigation. Arch Oral Biol. 2012 Jun;57(6):599-606. doi: 10.1016/j.archoralbio.2011.10.004. Epub 2011 Nov 30. PMID: 22129470; PMCID: PMC3294030.
132. Treede RD, Rief W, Barke A, Aziz Q, Bennett MI, Benoliel R, Cohen M, Evers S, Finnerup NB, First MB, Giamberar-

dino MA, Kaasa S, Korwisi B, Kosek E, Lavand'homme P, Nicholas M, Perrot S, Scholz J, Schug S, Smith BH, Svensson P, Vlaeyen JWS, Wang SJ. Chronic pain as a symptom or a disease: the IASP Classification of Chronic Pain for the International Classification of Diseases (ICD-11). Pain. 2019 Jan;160(1):19-27. doi: 10.1097/j.pain.0000000000001384. PMID: 30586067.
133. O'Malley CA, Smith SA, Mauger AR, Norbury R. Exercise-induced pain within endurance exercise settings: Definitions, measurement, mechanisms and potential interventions. Exp Physiol. 2024 Sep;109(9):1446-1460. doi: 10.1113/EP091687. Epub 2024 Jul 10. PMID: 38985528; PMCID: PMC11363130.
134. El-Beshbishi SN, Ahmed NN, Mostafa SH, El-Ganainy GA. Parasitic infections and myositis. Parasitol Res. 2012 Jan;110(1):1-18. doi: 10.1007/s00436-011-2609-8. Epub 2011 Sep 1. PMID: 21881948.
135. Bugshan A, Farag AM, Desai B. Oral Complications of Systemic Bacterial and Fungal Infections. Atlas Oral Maxillofac Surg Clin North Am. 2017 Sep;25(2):209-220. doi: 10.1016/j.cxom.2017.04.012. PMID: 28778309.
136. Carlson ER. Diagnosis and management of salivary gland infections. Oral Maxillofac Surg Clin North Am. 2009 Aug;21(3):293-312. doi: 10.1016/j.coms.2009.04.004. PMID: 19608046.
137. Hanewinckel R, Ikram MA, Van Doorn PA. Peripheral neuropathies. Handb Clin Neurol. 2016;138:263-82. doi: 10.1016/B978-0-12-802973-2.00015-X. PMID: 27637963.
138. Elafros MA, Kvalsund MP, Callaghan BC. The Global Burden of Polyneuropathy-In Need of an Accurate Assessment. JAMA Neurol. 2022 Jun 1;79(6):537-538. doi: 10.1001/jamaneurol.2022.0565. PMID: 35404377; PMCID: PMC9197927.
139. Wipperman J, Penny ML. Carpal Tunnel Syndrome: Rapid Evidence Review. Am Fam Physician. 2024 Jul;110(1):52-57. PMID: 39028782.
140. Sejvar JJ, Baughman AL, Wise M, Morgan OW. Population incidence of Guillain-Barré syndrome: a systematic review and meta-analysis. Neuroepidemiology. 2011;36(2):123-33. doi: 10.1159/000324710. Epub 2011 Mar 21. PMID: 21422765; PMCID: PMC5703046.
141. Shahrizaila N, Lehmann HC, Kuwabara S. Guillain-Barré syndrome. Lancet. 2021 Mar 27;397(10280):1214-1228. doi: 10.1016/S0140-6736(21)00517-1. Epub 2021 Feb 26. PMID: 33647239.
142. Huerta MÁ, Garcia MM, García-Parra B, Serrano-Afonso A, Paniagua N. Investigational Drugs for the Treatment of Postherpetic Neuralgia: Systematic Review of Randomized Controlled Trials. Int J Mol Sci. 2023 Aug 20;24(16):12987. doi: 10.3390/ijms241612987. PMID: 37629168; PMCID: PMC10455720.
143. de Berranger E, Derache AF, Ramdane N, Labreuche J, Navarin P, Gonzales F, Abou-Chahla W, Nelken B, Bruno B. VZV Prophylaxis After Allogeneic Hematopoietic Stem Cell Transplantation in Children: When to Stop? Cancer Rep (Hoboken). 2024 Nov;7(11):e70015. doi: 10.1002/cnr2.70015. PMID: 39506838; PMCID: PMC11541057.
144. Liu L, Chen S. Construction of influencing factors and nomogram prediction model for post-herpetic neuralgia based on T cell function and inflammatory factors. Front Med (Lausanne). 2025 Jun 18;12:1619157. doi: 10.3389/fmed.2025.1619157. PMID: 40606470; PMCID: PMC12213386.
145. Maulén-Yáñez M, Meeder-Bella W, Videla-Jiménez PJ, González-Arriagada WA. Assessment of association between muscular diagnosis in temporomandibular disorders with mandibular kinematics. Cranio. 2019 Nov;37(6):365-373. doi: 10.1080/08869634.2018.1465513. Epub 2018 May 7. PMID: 29734882.
146. Okeson, JP. Orthodontic therapy and temporomandibular disorders: should the orthodontist even care? In: McNamara JA Jr, Kapila SD, editors. Temporomandibular disorders and orofacial pain: separating controversy from consensus. Monograph 46. Craniofacial Growth Series. Ann Arbor: Center for Human Growth and Development; University of Michigan; 2009. p. 15-29.
147. Lieber RL, Fridén J. Muscle contracture and passive mechanics in cerebral palsy. J Appl Physiol (1985). 2019 May 1;126(5):1492-1501. doi: 10.1152/japplphysiol.00278.2018. Epub 2018 Dec 20. PMID: 30571285; PMCID: PMC6589815.
148. Serratrice G, Rowland LP. Les contractures musculaires. Essai d'approche physiopathologique pour une clarification de la nomenclature [Muscle contractures. Essay on a physiopathological approach to clarify the nomenclature]. Presse Med. 1999 Sep 25;28(28):1519-21. French. PMID: 10526559.
149. Leissner O, Maulén-Yáñez M, Meeder-Bella W, León-Morales C, Vergara-Bruna E, González-Arriagada WA. Assessment of mandibular kinematics values and its relevance for the diagnosis of temporomandibular joint disorders. J Dent Sci. 2021 Jan;16(1):241-248. doi: 10.1016/j.jds.2020.05.015. Epub 2020 Jun 6. PMID: 33384804; PMCID: PMC7770294.
150. Segù M, Politi L, Galioto S, Collesano V. Histological and functional changes in retrodiscal tissue following anterior articular disc displacement in the rabbit: review of the literature. Minerva Stomatol. 2011 Jul-Aug;60(7-8):349-58. English, Italian. PMID: 21709650.
151. Cruz-Jentoft AJ, Bahat G, Bauer J, Boirie Y, Bruyère O, Cederholm T, Cooper C, Landi F, Rolland Y, Sayer AA, Schneider SM, Sieber CC, Topinkova E, Vandewoude M, Visser M, Zamboni M; Writing Group for the European Working Group on Sarcopenia in Older People 2 (EWGSOP2), and the Extended Group for EWGSOP2. Sarcopenia: revised European consensus on definition and diagnosis. Age Ageing. 2019 Jan 1;48(1):16-31. doi: 10.1093/ageing/afy169. Erratum in: Age Ageing. 2019 Jul 1;48(4):601. doi: 10.1093/ageing/afz046. PMID: 30312372; PMCID: PMC6322506.
152. Moynihan PJ, Teo JL. Exploring Oral Function, Protein Intake, and Risk of Sarcopenia: A Scoping Review. JDR Clin Trans Res. 2024 Jan;9(1):4-20. doi: 10.1177/23800844231157259. Epub 2023 Mar 8. PMID: 36883673; PMCID: PMC10725128.
153. Abudurezake A, Kakehi S, Umemura F, Kaga H, Someya Y, Tabata H, Yoshizawa Y, Naito H, Tajima T, Ito N, Otsuka H, Shi H, Sugimoto M, Sakamoto S, Muroga Y, Wakabayashi H, Kawamori R, Watada H, Tamura Y. Masseter Muscle Volume, Sarcopenia, and Muscle Determinants: Insights from ACTN3 Polymorphism in Elderly Japanese in the Bunkyo Health Study. Arch Med Res. 2025 Jan;56(1):103095. doi: 10.1016/j.arcmed.2024.103095. Epub 2024 Oct 14. PMID: 39405919.
154. Suzuki M, Koyama S, Kimura Y, Ishiyama D, Otobe Y, Nishio N, Ichikawa T, Kunieda Y, Ohji S, Ito D, Yamada M. Relationship between characteristics of skeletal muscle and oral function in community-dwelling older women. Arch Gerontol Geriatr. 2018 Nov-Dec;79:171-175. doi: 10.1016/j.archger.2018.09.003. Epub 2018 Sep 14. PMID: 30265912.
155. Moore J, Simpson MTW, Cohen N, Beyea JA, Phillips T. Approach to sialadenitis. Can Fam Physician. 2023 Aug;69(8):531-536. doi: 10.46747/cfp.6908531. PMID: 37582587; PMCID: PMC10426371.
156. Hasegawa K, Dubousset JF. *Cone of Economy with the Chain of Balance*-Historical Perspective and Proof of Concept. Spine Surg Relat Res. 2022 Apr 20;6(4):337-349. doi: 10.22603/ssrr.2022-0038. PMID: 36051675; PMCID: PMC9381078.
157. Challier V, Nassar JE, Castelain JE, Campana M, Jacquemin C, Ghailane S. Alignment considerations in degenerative spinal conditions: A narrative review. N Am Spine Soc J. 2024 Oct 1;20:100562. doi: 10.1016/j.xnsj.2024.100562. PMID: 39554214; PMCID: PMC11565030.
158. Hu K, Dong P, Wu H, Wang Y, Hou R, Yao G. [Pathophysiological mechanisms linking chronic stress and cervical spondylosis of vertebral artery type: A theoretical framework of the neuroendocrine-immune network]. Xi Bao Yu Fen Zi Mian Yi Xue Za Zhi. 2025 Jul;41(7):655-660. Chinese. PMID: 40620124.
159. Yu QS, Shan JY, Ma J, Gao G, Tao BZ, Qiao GY, Zhang JN, Wang T, Zhao YF, Qin XL, Yin YH. Multi-modal and Multi-view Cervical Spondylosis Imaging Dataset. Sci

Data. 2025 Jul 1;12(1):1080. doi: 10.1038/s41597-025-05403-z. PMID: 40593874; PMCID: PMC12219229.
160. Duggal AK, Chowdhury D. SUNCT and SUNA: An Update. Neurol India. 2021 Mar-Apr;69(Supplement):S144-S159. doi: 10.4103/0028-3886.315990. PMID: 34003160.
161. Moraes RM, Lescura CM, Amstalden EMI, Anbinder AL. Temporomandibular joint synovial chondromatosis with a giant cell component. Int J Oral Maxillofac Surg. 2025 Dec;54(12):1208-1213. doi: 10.1016/j.ijom.2025.05.003. Epub 2025 May 27. PMID: 40436718.
162. Kriegsmann S, Krenn V, Liebisch M. Die Synoviale Chondromatose : Ergebnisse aus dem histopathologischen Arthritisregister der Deutschen Gesellschaft für Orthopädische Rheumatologie (DGORh) [Synovial chondromatosis : Results from the histopathological arthritis register of the German Society for Orthopedic Rheumatology]. Z Rheumatol. 2024 Dec;83(10):866-871. German. doi: 10.1007/s00393-024-01569-w. Epub 2024 Sep 26. PMID: 39327325.
163. Bouloux GF, Chou J, DiFabio V, Ness G, Perez D, Mercuri L, Chung W, Crago CA. The Contemporary Management of Temporomandibular Joint Intra-Articular Pain and Dysfunction. J Oral Maxillofac Surg. 2024 Jun;82(6):623-631. doi: 10.1016/j.joms.2024.01.003. Epub 2024 Jan 6. PMID: 38280727.
164. Dupont JS Jr, Brown CE. The concurrency of temporal tendinitis with TMD. Cranio. 2012 Apr;30(2):131-5. doi: 10.1179/crn.2012.019. PMID: 22606857.
165. Pan LH, Ling YH, Wang SJ, Al-Hassany L, Chen WT, Chiang CC, Cho SJ, Chu MK, Coppola G, Pietra AD, Dong Z, Ekizoglu E, Els C, Farham F, Garcia-Azorin D, Ha WS, Hsiao FJ, Ishii R, Kim BK, Kissani N, Labastida-Ramirez A, Lange KS, Lytvyak E, Onan D, Ozge A, Papetti L, Pellesi L, Raffaelli B, Raggi A, Straube S, Takizawa T, Tanprawate S, Uludüz DU, Vongvaivanich K, Waliszewska-Prosół M, Wang Y, Wijeratne T, Wu JW, Yener SM, Martelletti P. Hallmarks of primary headache: part 2- Tension-type headache. J Headache Pain. 2025 Jul 17;26(1):164. doi: 10.1186/s10194-025-02098-w. PMID: 40676501; PMCID: PMC12273213.
166. Carlo WA, Kliegman RM, Dixon MS Jr, Fletcher BD, Fanaroff AA. Vertebral agenesis: comprehensive care and outcome. Am J Dis Child. 1982 Jun;136(6):533-7. PMID: 7091067.
167. Dias MS. Normal and abnormal development of the spine. Neurosurg Clin N Am. 2007 Jul;18(3):415-29. doi: 10.1016/j.nec.2007.05.003. PMID: 17678747.
168. Whitney E, Alastra AJ. Vertebral Fracture. 2023 Apr 3. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan–. PMID: 31613453.
169. Schnake KJ, Schroeder GD, Vaccaro AR, Oner C. AOSpine Classification Systems (Subaxial, Thoracolumbar). J Orthop Trauma. 2017 Sep;31 Suppl 4:S14-S23. doi: 10.1097/BOT.0000000000000947. PMID: 28816871.
170. Zhang A, Yin S, Xia J. X-ray analysis of cervical spine in 334 patients. Chin J Convalescent Med 2007; 3: 172–174.
171. Badve SA, Bhojraj S, Nene A, Raut A, Ramakanthan R. Occipito-atlanto-axial osteoarthritis: a cross sectional clinico-radiological prevalence study in high risk and general population. Spine (Phila Pa 1976). 2010 Feb 15;35(4):434-8. doi: 10.1097/BRS.0b013e3181b13320. PMID: 20110845.
172. Betsch MW, Blizzard SR, Shinseki MS, Yoo JU. Prevalence of degenerative changes of the atlanto-axial joints. Spine J. 2015 Feb 1;15(2):275-80. doi: 10.1016/j.spinee.2014.09.011. Epub 2014 Sep 30. PMID: 25277533.
173. Farooqi RR, Mehmood M, Kotwal HA. Hyperplasia of Lamina and Spinous Process of C5 Vertebrae and Associated Hemivertebra at C4 Level. J Orthop Case Rep. 2017 Jan-Feb;7(1):79-81. doi: 10.13107/jocr.2250-0685.698. PMID: 28630847; PMCID: PMC5458705.
174. Kazanci B, Tehli O, Adilay U, Guclu B. Unilateral hyperplasia of lamina and spinous process of C6 vertebra: case report. Surg Radiol Anat. 2012 Nov;34(9):875-8. doi: 10.1007/s00276-012-0934-y. Epub 2012 Jan 19. PMID: 22258285.
175. Fourney DR, Gokaslan ZL. Spinal instability and deformity due to neoplastic conditions. Neurosurg Focus. 2003 Jan 15;14(1):e8. doi: 10.3171/foc.2003.14.1.9. PMID: 15766225.
176. Narayanappa G, Nandeesh BN. Infective myositis. Brain Pathol. 2021 May;31(3):e12950. doi: 10.1111/bpa.12950. PMID: 34043257; PMCID: PMC8412094.