



## **Orofacial Pain**

Guidelines for Assessment, Diagnosis, and Management, Seventh Edition



# American Academy of Orofacial Pain

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# Orofacial Pain

## Guidelines for Assessment, Diagnosis, and Management

SEVENTH EDITION

The American Academy of Orofacial Pain

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# Preface



The American Academy of Orofacial Pain (AAOP), a membership organization, was founded in 1975 with the goal to improve the understanding and quality of education in temporomandibular disorders (TMDs) and orofacial pain. The stated mission of the AAOP is to be an organization of health care professionals dedicated to alleviating pain and suffering through the promotion of excellence in education, research, and patient care in the field of orofacial pain and associated disorders. The AAOP has also been the sponsoring organization for orofacial pain to be recognized as a specialty, which was achieved in March 2020 as it is now considered the 12th dental specialty by the American Dental Association.

There have been six previous publications prior to this current edition of what commonly is referred to as the AAOP Guidelines. Dr Charles McNeill spearheaded the first two editions called *Cranio-mandibular Disorders: Guidelines for Evaluation, Diagnosis, and Management* (Quintessence, 1990) and *Temporomandibular Disorders: Guidelines for Classification, Assessment, and Management* (Quintessence, 1993). These publications focused predominantly on TMDs. As health care professionals and researchers became more conscious of the relationship between TMDs and other disorders of the head and neck, there was a need to expand the Guidelines to include disorders presenting as or related to TMDs. These disorders included not only headaches and neck disorders but several neuropathic pain conditions as well as biobehavioral factors. In 1996, under the editorship of Dr Jeffrey Okeson, the third version of the AAOP Guidelines was published, titled *Orofacial Pain: Guidelines for Assessment, Diagnosis, and Management*. That edition used the term

*orofacial pain* to echo the rapidly changing and expanding field of orofacial pain and to reflect the fact that TMDs and orofacial pain should not be regarded as separate conditions, but that TMDs should be considered part of the disorders that fall under the umbrella of orofacial pain. Under the editorship of Dr Reny de Leeuw, the fourth edition of the Guidelines was published, which started to express evidence-based concepts. This edition included a separate chapter on cervical disorders to emphasize the close relationships between some orofacial pain disorders and cervical pain disorders, and—more importantly—to call attention to the differences and similarities associated with these disorders. The fifth edition, published in 2013, adopted the Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD) and the expanded TMD taxonomy based on the work of the International RDC-TMD Consortium now known as the International Network of Orofacial Pain and Related Disorders Methodology (INFORM) Consortium. An updated definition of *bruxism* based on another international consensus work group was also adopted. Moreover, a new chapter was added in the fifth edition dedicated to the relationship between pain disorders and sleep. The sixth edition, published in 2018, employed the *International Classification of Diseases, Tenth Revision (ICD-10)* diagnostic codes, and several chapters referenced the International Classification of Headache Disorders, third edition (beta version; ICHD).

While the structure of the present work resembles previous editions, significant changes have been implemented in this current edition. All chapters contain essential updates, and some have undergone more changes than others. References have been updated throughout to



reflect the most current literature. When available, evidence-based material has been included to provide the reader with scientifically sound and effective diagnostic procedures and treatment options. *ICD-10* diagnostic codes and the new *ICD-11* diagnostic codes have been used throughout the text. Furthermore, the International Classification of Headache Disorders, third edition (ICHD) codes have been used, where appropriate. Chapter 1 has been updated with discussions related to the influence of gut-brain interactions and dysbiosis, nutrition, and stem cell therapies in relation to chronic pain. In chapter 2, the future of potential biomarkers and the need for personalized/precision care are explored. Chapter 3 introduces the Analgesic, Anesthetic, and Addiction Clinical Trial Translations, Innovations, Opportunities, and Networks (ACTTION) and the American Pain Society (APS) pain taxonomy (AAPT) along with the International Classification of Orofacial Pain (ICOP) system. Moreover, the chapter reviews the newly implemented *ICD-11* classification system. Chapter 4 contains general updates in patient assessment, and chapter 5 contains various general updates with greater emphasis placed on headache pathophysiology as well updates to management including new pharmacologic agents. Chapter 6 was updated and expanded in terms of pharmacotherapeutics and recognition of red ear and first bite syndromes. Chapter 7 contains general updates to the management strategies for several disorders in addition to a section on “newer trends” related to electronic cigarettes/vaping and the SARS-CoV-2 global pandemic. In chapter 8, general updates to content and references have been provided with mapping of *ICD-10* to *ICD-11* classification coding changes. Chapters 9 and 10 have been updated

to reflect more contemporary knowledge gained in relationship to cervical spinal disorders and associated headaches and extracranial and systemic causes of orofacial pain. Chapter 11 provides expanded information related to bruxism with brief discussions on the topics of pediatric obstructive sleep apnea, myofunctional therapy, and the lymphatic system. Chapter 12 provides updates on the implementation of various screening tools for biobehavioral factors following the recommendations from the Diagnostic Criteria for Temporomandibular Disorders (DC/TMD). The chapter also incorporates updates in the description of several psychiatric diseases in line with the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5)*. Lastly, the glossary has undergone updates to reflect the emerging and expanding field of orofacial pain. New terms have been added, and obsolete and superfluous terms have been removed.

Finally, despite the various chapters contained within the Guidelines and the depth of discussion among the included topics, it must be remembered that research is continuing and rapidly evolving, thus advancing and updating our knowledge and understanding of various conditions and management protocols. This is not intended to be an all-encompassing textbook exploring complete details regarding all aspects of orofacial pain. Instead, it is meant to provide an overview and insights to assist the reader with the procedures of evidence-based assessment, diagnosis, and management of orofacial pain conditions, based on the most current scientific knowledge.

Gary D. Klasser and Marcela Romero Reyes  
Co-chairs, AAOP Guidelines Committee

# Acknowledgments



Over the years, numerous AAOP members and nonmembers have participated in the evolution of the AAOP Guidelines, resulting in the seventh edition of this publication. The contributors to the current edition of the AAOP Guidelines are listed on a separate page. Each new edition has reflected the emerging and expanding field of orofacial pain. Based on these developments, new and evidence-based materials have been added. However, this ever-evolving work has built on and edited the work others have done in the past. As such, some parts of previous contributions may still be intact. We therefore want

to extend our sincere appreciation to all of you who have contributed to any of the past editions, and especially to those of you who laid the foundation of this publication. We also would like to offer much gratitude to the publishers for providing us with timely advice and guidance so that deadlines could be met. The staff support at Quintessence has been incredibly accommodating and meticulous in their efforts and should be applauded. We truly hope that you will get great enjoyment and practical help from this new edition to the benefit of the patients we are privileged to assist.





# 1

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## Introduction to Orofacial Pain

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### Key Points

- Orofacial pain remains a prevalent and debilitating condition that exerts a significant social and economic impact on patients and the health care system.
- Many of the risk factors associated with temporomandibular disorders (TMDs) involve mechanical, chemical, or environmental stressors that increase the likelihood of developing and maintaining a chronic pathologic state.
- Sensitization and activation of trigeminal nerves and the subsequent development of peripheral and central sensitization are key pathophysiologic events leading to allodynia and hyperalgesia.
- Glial cells play an important role in the transition of acute to chronic pain by modulating the excitability state of nociceptive neurons in the trigeminal ganglion and spinal cord.
- Epigenetic influences on gene expression, mediated by our lifestyle and environment, significantly impact the progression of TMD and migraine pathology, necessitating comprehensive therapy.
- In March 2020, the National Commission on Recognition of Dental Specialties and Certifying Boards officially recognized Orofacial Pain as the dental profession's 12th specialty.
- Discoveries from the Orofacial Pain Prospective Evaluation and Risk Assessment (OPPERA) study have helped to clarify specific risk factors and genes implicated in the development of TMDs.
- Given the complex multifactorial etiology of orofacial pain conditions, management may require multiple interventions, such as—but not limited to—pharmaceuticals, physical therapy, oral appliances, behavioral modifications, diet modifications, and forms of exercise that emphasize proper breathing and increasing flexibility.
- The COVID-19 pandemic has had a major impact on the healthcare community and has driven advances in digital technology such as the use of telemedicine, phone apps, and other electronic aids that will forever change the medical landscape.



## The Spectrum of Orofacial Pain

*Orofacial pain* refers to pain disorders of the jaw, mouth, face, head, and neck. These anatomical regions comprise an array of widely diverse structures and tissues ranging from dental pulp to the meninges of the brain. Pain conditions associated with these structures may derive from local factors or involve systemic, autoimmune, infectious, traumatic, or neoplastic pathologies. These conditions include odontogenic and periodontal pains, musculoskeletal disorders such as temporomandibular disorders (TMDs), headache and neurovascular pains, vascular disorders, and neuropathic pains.

Underlying this kaleidoscope of pain possibilities is a unifying system, the trigeminal sensory complex, in which impulses from the head and neck are conveyed by the branches of the trigeminal and upper cervical nerves to the trigeminal sensory nucleus in the brain stem. These impulses are modified by input originating within the trigeminal system and from higher regions of the central nervous system (CNS). Multiple areas of the brain process and interpret this input, giving rise to the sensation of pain and facilitating physiologic/adaptive responses, including behavioral changes.<sup>1</sup>

The diversity and complexity of orofacial pain conditions have led to recognition of the need for a specialized field of dentistry and for collaboration among multiple fields of medicine to improve care for patients afflicted with these disorders.

## The Specialty of Orofacial Pain

Orofacial pain as a specialty has made significant strides in recent years. In 2009, the Commission on Dental Accreditation (CODA) approved orofacial pain as an area of advanced education, and since 2011, multiple residency and fellowship programs have been accredited in the United States. The International Association

for the Study of Pain (IASP) has recognized the need for orofacial pain education by developing a core curriculum on this subject for health care professionals.<sup>2</sup> In March 2020, the National Academies of Sciences, Engineering, and Medicine released a consensus study report on TMD entitled *Priorities for Research and Care*.<sup>3</sup> This study, which was supported by the National Institute of Dental and Craniofacial Research, recommended improvements to education and training on TMDs for health care professionals, stating that “Deans of health professional schools ... should ensure that their schools’ curricula include attention to TMDs...” and “The Commission on Dental Accreditation (CODA) should amend the accreditation standards for predoctoral dental programs to include screening, risk assessment, and appropriate evidence-based interventions for TMDs.” In addition to these educational initiatives, the discipline of orofacial pain has been bolstered by the establishment of board certification processes and by increasing cooperation among advocacy groups, universities, professional organizations, and federal agencies. A landmark event occurred in March 2020 when the National Commission on Recognition of Dental Specialties and Certifying Boards officially recognized Orofacial Pain as the dental profession’s 12th specialty.

The material covered in this chapter represents a broad overview of findings reported in peer-reviewed publications spanning the spectrum of conditions at the root of orofacial pain and current management practices. It is intended for health care professionals who evaluate and treat patients with orofacial pain and face the daunting task of keeping up with the literature in the ever-evolving arena of pain management.

## Pain

Understanding of orofacial pain conditions must be grounded in principles and concepts of pain in general. The IASP offers the following defi-



CLASSIFICATION SCHEME			TYPICAL BEHAVIORS
Anatomical	Physiologic	Functional/ Chronologic	
Somatic - Superficial - Deep	Nociceptive	Acute	Defense/Withdrawal
	Inflammatory		Rest/Guarding
Visceral	Nociplastic	Chronic	Illness
Neuropathic			

**Fig 1-1** Pain classification schemes and typical behaviors.

nition of *pain* (revised in 2020): “An unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage.”<sup>4</sup> The IASP expands upon this definition with these six key notes:

1. Pain is always a personal experience influenced to varying degrees by biologic, psychological, and social factors.
2. Through their life experiences, individuals learn the concept of pain.
3. A person’s report of an experience as pain should be respected.
4. Although pain usually serves an adaptive role, it may have adverse effects on function and social and psychological well-being.
5. Verbal description is only one of several behaviors to express pain; inability to communicate does not negate the possibility that a human or a nonhuman animal experiences pain.
6. Pain and nociception are different phenomena; pain cannot be inferred solely from activity in sensory neurons.<sup>4</sup>

This final key note highlights an important distinction between the concepts of nocicep-

tion and pain. *Nociception* has been defined as “information processing triggered by noxious stimuli ... (which) may lead to withdrawal or vegetative responses and/or to the sensation of pain”<sup>5</sup> In simple terms, nociception refers to basic signaling in the nervous system, while pain involves the interpretation and perception associated with those signals.

### Classifications of pain

Pain may be viewed through the lenses of different classification schemes, including what may be termed physiologic and anatomical classifications (Fig 1-1). A physiologic classification includes categories of nociceptive and inflammatory pain, as well as a third mechanistic descriptor recently adopted by the IASP and referred to as *nociplastic pain*. Nociceptive pain is momentary, nonpersistent pain that matches its stimulus (does not display an exaggerated response). It acts as a vital defense mechanism, stimulating behavioral and physiologic actions to prevent tissue damage in the face of a noxious stimulus. *Inflammatory pain* occurs in the setting of tissue damage (eg, due to mechanical trauma, heat, or infection).<sup>6</sup> Tissue injury

## 1 Introduction to Orofacial Pain



prompts release of a host of inflammatory mediators that interact with sensory neurons to increase the intensity and duration of nociceptive signaling, leading to persistent pain that, in contrast to the momentary experience of nociceptive pain, outlasts its stimulus. This persistence of inflammatory pain encourages protective behaviors (“vegetative responses” such as resting an inflamed body part) intended to limit further injury while healing occurs.<sup>7</sup> *Nociplastic pain* is defined by the IASP as pain that arises from altered nociception despite no clear evidence of actual or threatened tissue damage causing the activation of peripheral nociceptors. The IASP differentiates nociplastic from neuropathic pain (discussed later) with the clarification that nociplastic pain displays no evidence for disease or lesion of the somatosensory system.<sup>4</sup> This type of pain is characteristic of a group of disorders known as chronic overlapping pain conditions (COPCs) that includes, for example, fibromyalgia and irritable bowel syndrome (IBS).

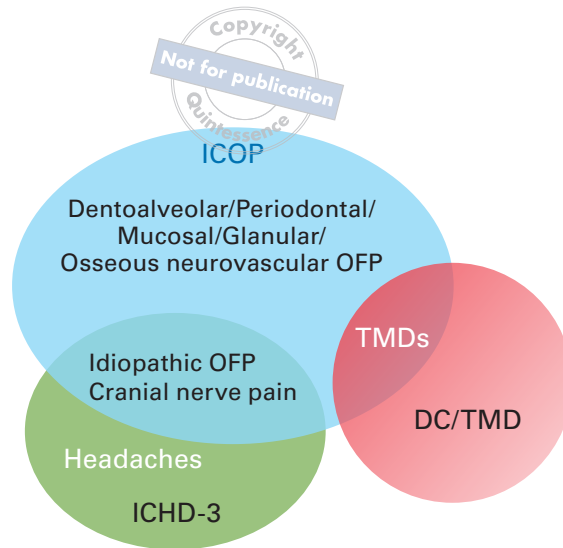
Anatomical classifications include categories of somatic pain and visceral pain (see Fig 1-1). *Somatic pain* encompasses both superficial (eg, cutaneous, mucosal) and deep (musculoskeletal) pain. A site of noxious stimulation in superficial somatic tissues is usually easy for an individual to precisely locate, due at least partly to a relatively high density of free nerve endings in dermal tissue relative to deeper tissues. This ability to localize pain serves as an early warning system to protect against injury to the integrity of the body’s outermost protective layers.<sup>8</sup> In contrast, pain arising from musculoskeletal structures is prone to spread or refer beyond the locus of noxious stimulation. *Visceral pain* arises from internal organs and is often diffuse and difficult to localize. Such pain is also prone to refer to areas remote from the source of noxious stimulation, often to superficial somatic areas, as in the classic example of myocardial infarction being perceived in areas such as the chest wall.

The category of neuropathic pain traverses both physiologic and anatomical classification schemes. The IASP defines *neuropathic pain* as “pain caused by a lesion or disease of the somatosensory nervous system.”<sup>4</sup> Such pain may be thought of as a faulty or hyperactive alert system, like a smoke alarm (the nervous system) going off even though fire (nonneural tissue damage) is absent.

Probably the most common distinction drawn in classifying pain is between acute and chronic.<sup>9</sup> This distinction is often framed in terms of time, with pain of less than 3 to 6 months duration labeled acute and pain persisting beyond this timeframe as chronic. These labels may be viewed in a slightly different light as functional rather than merely chronologic. In a functional sense, *acute pain* is pain that serves an essential protective function, alerting an organism to potential tissue damage and stimulating behavior to avoid or mitigate that damage. *Chronic pain*, on the other hand, is pain that persists despite the lack of any biologically useful role, potentially becoming a disorder unto itself.

Chronic pain has profound societal effects. The 2016 National Health Interview Survey found a point prevalence of chronic pain in the U.S. adult population of over 20% (50 million people), with 8% (approximately 20 million) having high-impact chronic pain causing frequent limitations to life or work activities. Female sex, lack of current employment, residence in a rural area, low socioeconomic status, and non-Hispanic white ethnicity were associated with higher prevalence of chronic pain after adjusting for age.<sup>10</sup> These findings highlight the need for a better appreciation of the multifactorial nature of pain and the challenge of personalizing treatment to the needs of each individual patient.

Chronic pain is associated with high economic costs. In the United States, billions of dollars are lost annually due to decreased productivity, loss of work, and disability compensation. Chronic pain increases expenses for emergency room



**Fig 1-2** Diagnostic criteria for specific pain disorders.

visits, medication costs, and psychologic treatment.<sup>8</sup> The cost of pain is estimated to be similar to the combined annual costs of heart disease, cancer, and diabetes.<sup>11</sup>

Orofacial pain conditions may fit simultaneously within more than one of the pain classification schemes described above. For example, momentary pain elicited in a carious tooth by a cold beverage (reversible pulpitis) is an acute pain from a functional perspective, a nociceptive pain from a physiologic perspective, and a visceral pain from an anatomical perspective. Temporomandibular joint (TMJ) pain of several months' duration that is stimulated by chewing and lasts for several hours after cessation of the stimulus represents a deep somatic pain that is both chronic and inflammatory.

In addition to the general categories of pain outlined above, criteria have been established to guide the diagnosis of specific pain disorders. Three sets of diagnostic criteria focus specifically on orofacial pain:

- The diagnostic criteria for temporomandibular disorders (DC/TMD), developed by the International RDC/TMD Consor-

tium Network (now known as InFORM, the International Network for Orofacial Pain and Related Disorders Methodology) of the International Association for Dental Research and the Orofacial Pain Special Interest Group of the International Association for Dental Research and published in 2014<sup>12</sup>

- The International Classification of Headache Disorders (ICHD-3), published by the International Headache Society and published in its third edition in 2018<sup>13</sup>
- The International Classification of Orofacial Pain (ICOP), also by the International Headache Society and published in its first edition in 2020<sup>14</sup>

Each of these sets of diagnostic criteria includes a taxonomy that arranges pain conditions hierarchically from broad categories of disorders to specific diagnoses based on patient history and examination findings. Specific conditions described by these criteria are more fully elucidated in subsequent chapters. As seen in Fig 1-2, there is overlap among these sets of criteria, and some differences in definitions and terminology exist with respect to specific



diagnoses. Hopefully, these differences will be reconciled in the future as the field of orofacial pain continues to evolve to include data from a diverse array of disciplines.

## Pathways of Nociception

### Peripheral innervation

The progression of a nociceptive impulse from its initiation in peripheral tissues to its transformation in the cerebral cortex into the experience of pain may be visualized as occurring along a simplified pathway of successive sensory neurons (primary, secondary, tertiary). A more accurate picture might be that of a dense urban highway system encompassing a vast number of roads of varying sizes, from multi-lane freeways to one-way back alleys, with a dizzying network of on-ramps, off-ramps and intersections, and a plethora of signs and signals regulating traffic flow.

Nociception from peripheral tissues is initiated by sensory receptors called *nociceptors*, which are free nerve endings located at the peripheral terminals of specialized primary sensory neurons. Nociceptors detect potentially harmful mechanical, thermal, or chemical stimuli. Many nociceptors respond to more than one of these forms of stimuli. This polymodal receptivity is a feature unique to nociceptors; other sensory receptors of the body are specialized to a single modality (eg, innocuous mechanical or thermal stimuli). A sufficiently intense noxious stimulus triggers nociceptors to initiate action potentials that travel centrally along the axons of primary sensory neurons. The frequency of nociceptive action potentials increases as a function of increasing stimulus intensity, causing a summation in signal strength at the neuron's central connection with a secondary neuron.<sup>15</sup>

The vast majority of nociceptive primary neurons have thin axonal fibers that conduct impulses more slowly than the larger-diameter

fibers that are responsible for innocuous sensory impulses and initiation of skeletal muscle activity. Classification of peripheral neurons is based on axon diameter, degree of myelination, and the resulting conduction velocity (Table 1-1). Nociceptive primary neurons generally fall within the categories of A $\delta$  fibers (thinly myelinated) and C fibers (unmyelinated), although the borders between categories associating fiber types with types of sensory impulses are not well delineated: some unmyelinated fibers convey innocuous sensory information (eg, C fibers that conduct pleasant stroking sensations<sup>16</sup>), and under conditions of sensitization or demyelination, large-diameter fibers (A $\beta$  fibers) that normally respond to innocuous stimuli convey information that is interpreted as nociceptive (see discussion below).

The cell bodies of primary sensory neurons that carry input from the head and neck reside in the trigeminal ganglion (also referred to as the *Gasserian* or *semilunar ganglion*) at the convergence of the three branches of the trigeminal nerve. Axons travel centrally from the trigeminal ganglion via a single large sensory root to enter the pons. Cell bodies of sensory neurons from the trunk and limbs reside in the dorsal root ganglia, and the axons of these neurons join with the spinal cord via the dorsal roots. Axons of primary sensory neurons synapse with secondary neurons in the trigeminal sensory nucleus (for impulses from the head and neck) or dorsal horn (for impulses from the trunk and limbs).

### Trigeminal branches

The ophthalmic nerve branch (V1) transmits only sensory information from its branches in the scalp, forehead, upper eyelid, conjunctiva, cornea, nose (including the tip of the nose), nasal mucosa, frontal sinuses and deep structures in these regions, as well as parts of the meninges (the dura and blood vessels). These branches merge to enter the skull through the superior orbital fissure. The ophthalmic branch

**Table 1-1** Sensory fiber type and known stimulus

Sensory fiber	Stimulus
A $\alpha$ and A $\beta$	Proprioception and light touch.
A $\delta$	Nociception (noxious mechanical, thermal, chemical). Fast, pricking quality of pain.
C	Nociception (noxious mechanical, thermal, chemical). Slower, burning quality of pain.

also carries postganglionic parasympathetic motor fibers to the glands and sympathetic fibers to the pupillary dilator muscles.

The maxillary nerve branch (V2) carries only sensation from the lower eyelid and cheek, the nares and upper lip, the maxillary teeth and gingiva, the nasal mucosa, the palate roof of the pharynx, the uvula, the maxillary and ethmoid and sphenoid sinuses, and parts of the meninges. Peripheral branches converge to enter the skull through the foramen rotundum. The nerve is then joined by the middle meningeal nerve carrying sensation from the middle meningeal artery and part of the dura.

The mandibular nerve branch (V3) transmits both sensory and motor impulses. The sensory component carries information from the lower lip, mandibular teeth and gingiva, floor of the mouth, anterior two-thirds of the tongue, chin and jaw (except the angle of the jaw, which is supplied by C2 and C3), parts of the external ear, parts of the meninges, and deep structures. The auriculotemporal nerve innervates most of the

TMJ. Sensory branches merge to enter the skull via the foramen ovale. Motor axons transmit impulses from the trigeminal motor nuclei in the mid-pons to the muscles of mastication (ie, masseter, temporalis, medial pterygoid, lateral pterygoid, anterior digastric, and mylohyoid); the tensor veli palatini, involved with eustachian tube function; and the tensor tympani, which attaches to the malleus bone in the eardrum.

The CNS structures affected by trigeminal nociceptive input are also contacted by secondary neurons from the dorsal horn of the spinal cord. Therefore, potential pain input from regions outside trigeminal receptive fields may excite CNS structures that communicate with trigeminal nuclei and modulate their functions. The trigeminal system is directly or indirectly associated with the other cranial nerves, which are extensions of the brain that innervate tissues of the head and face.<sup>8</sup> The specialized neurons of the olfactory, optic, and vestibulocochlear nerves that send smell, sight, sound, and balance information to the CNS do not travel through

## 1 Introduction to Orofacial Pain



the trigeminal nuclei. However, nerves associated with the nose, eye, and ear tissues do transmit proprioceptive, pressure, and potential pain impulses into the trigeminal nuclei. These cranial nerves, along with the oculomotor, trochlear, abducens, and the hypoglossal nerves, will not be reviewed here. However, a comprehensive orofacial pain evaluation should include a basic assessment of the function of all cranial nerves (see chapter 2).

### Facial nerve

The seventh cranial nerve is a mixed nerve that has five branches (temporal, zygomatic, buccal, mandibular, and cervical) that course through the parotid gland but do not innervate the gland. Its main function is motor control of most of the muscles of facial expression and the stapedius muscle of the middle ear. The facial nerve supplies parasympathetic fibers to the sublingual and submandibular glands via the chorda tympani and to the lacrimal gland via the pterygopalatine ganglion. In addition, it conveys taste sensations from the anterior two-thirds of the tongue to the solitary tract nucleus and transmits cutaneous sensation from the skin in and around the earlobe via the nervous intermedius.

### Glossopharyngeal nerve

The ninth cranial nerve is a mixed nerve comprised of somatic, visceral, and motor fibers. It conveys sensory information from the posterior third of the tongue, tonsils, pharynx, middle ear, and carotid body. Taste sensation from the posterior third of the tongue as well as carotid body baroreceptor and chemoreceptor information is transmitted to the solitary tract nucleus. Nociceptive input from the ear is sent to the spinal trigeminal nucleus. From the inferior salivatory nucleus, the glossopharyngeal nerve delivers parasympathetic control to the parotid and mucous glands throughout the oral cavity while motor fibers from the nucleus ambiguus project to the stylopharyngeus muscle and

upper pharyngeal muscles. An altered gag reflex indicates glossopharyngeal nerve damage.

### Vagus nerve

The tenth cranial nerve originates in the brain stem and extends to the abdomen and innervates virtually all organs from the neck to the transverse colon except the adrenal glands. It supplies visceral afferent fibers to the mucous membranes of the pharynx, larynx, bronchi, lungs, heart, esophagus, stomach, intestines, and kidneys and distributes efferent or parasympathetic fibers to the heart, esophagus, stomach, trachea, bronchi, biliary tract, and most of the intestine. Also, the vagus nerve affects motor control of the voluntary muscles of the larynx, pharynx, and palate and carries somatic sensory fibers that terminate in the skin of the posterior surface of the external ear and the external acoustic meatus. Through these connections, the vagus affects activities as varied as respiration, cardiac function, sweating, digestion, peristalsis, hearing, and speech.

### Spinal accessory nerve

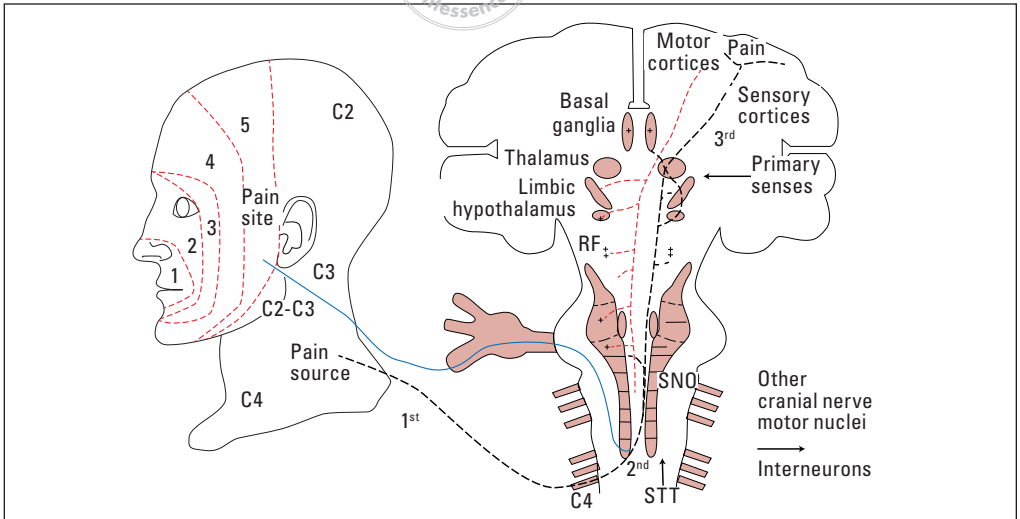
The eleventh cranial nerve innervates the cervical muscles, the sternocleidomastoid and trapezius, which are coactivated during masticatory behaviors. Like the trigeminal motor nucleus, the accessory motor nuclei are rich in norepinephrine receptors, which can facilitate vigilant behaviors. Nociceptive afferents from the cervical muscles converge onto the spinal trigeminal nucleus. It is notable that cervical myofascial pain seems to be prominent in patients with orofacial pain.

## Central connections

### Trigeminal sensory nuclei

All pressure, proprioception, chemical, and temperature sensory input from the face and all nociceptive inputs from the face, head, and neck are transmitted to the cell bodies of second-



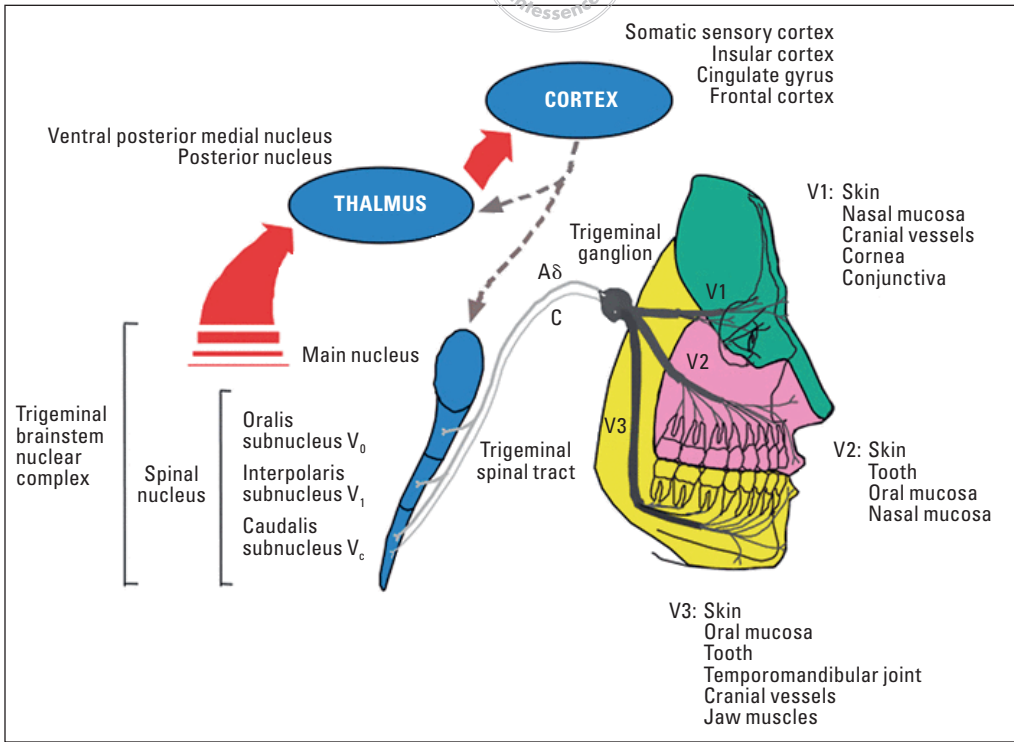


**Fig 1-3** Trigeminal sensory pathways and motor response to referred pain. The primary neurons from a pain site in facial lamina 5 and from the pain source in the C4 receptive field each converge on lamina 5 of the subnucleus caudalis and excite the same secondary neurons. As these secondary neurons ascend, they arborize with the subnucleus oralis and subnucleus interpolaris (not shown) and many reticular formation structures before synapsing with tertiary neurons in the thalamus. The tertiary neurons are thalamo-cortico-basal ganglia-limbic circuits that interpret pain and generate the descending motor and pain modulatory reactions to pain interpretation. The descending motor neurons also arborize with reticular formation locations and connect, via interneurons, to the trigeminal motor nucleus and to all cranial nerve motor nuclei. Note that trigeminal input is never analyzed in isolation, as primary sensory and spinal thalamic tract input is also always being presented to the brain for analysis. RF, reticular formation structure; SNO, subnucleus oralis; STT, spinal thalamic tract.

ary neurons in the trigeminal sensory nuclei.<sup>8</sup> These nuclei are located in bilateral columns on either side of the brain stem, stretching from the midbrain rostrally and blending caudally with the dorsal horn of the cervical spinal cord (Fig 1-3). Moving in a rostrocaudal direction, the trigeminal sensory nuclei comprise the mesencephalic nucleus (in the midbrain), which is involved in jaw reflex activity, the main sensory nucleus (in the pons), which receives proprioceptive and pressure input from orofacial structures, and the spinal trigeminal nucleus.<sup>15</sup>

The mesencephalic nucleus, which is functionally more a ganglion than a nucleus, houses the cell bodies of the proprioceptive neurons that convey input from the apical periodontal ligament and the muscle fibers that contract

during the jaw-closing reflex. These proprioceptive neurons, and possibly the blink reflex nerves, represent the only peripheral nerves with cell bodies located within the CNS.<sup>8,17</sup> The neurons are monosynaptic and pass through the mesencephalic nucleus to synapse in the trigeminal motor nuclei located medially to the much larger main sensory nucleus. The main sensory nucleus receives the facial proprioceptive and pressure input for orofacial behaviors other than the jaw-closing reflex (eg, chewing, kissing, smiling, and light touch). These neurons have their cell bodies in the trigeminal ganglion and synapse in the main sensory nucleus where input is conveyed to the motor nuclei by arrays of small interneurons.<sup>8</sup>



**Fig 1-4** The trigeminal (V) complex. (Reprinted from Hu and Wada<sup>8</sup> with permission.)

The spinal trigeminal nucleus (SpV) is located in the medulla and consists of three subnuclei.<sup>18</sup> From rostral to caudal, they are the subnucleus oralis ( $V_0$ ), subnucleus interpolaris ( $V_1$ ), and subnucleus caudalis ( $V_c$ ). Subnuclei oralis and interpolaris receive chiefly temperature information from  $A\delta$  fibers and non-nociceptive tactile input from  $A\beta$  fibers.<sup>8</sup> They also receive some nociceptive input originating from intra-oral structures. The subnucleus caudalis is sometimes referred to as the *medullary dorsal horn*<sup>1</sup> due to the similarity of its cellular organization to that of the dorsal horn of the spinal cord. It receives the bulk of the nociceptive input from trigeminal receptive fields. In response to nociceptor activation, neuropeptides and other inflammatory agents are released in the

spinal trigeminal nucleus and can cause excitation of neurons and glial cells that promotes development of central sensitization, allodynia, and hyperalgesia, which are physiologic events associated with acute and chronic pain.<sup>19,20</sup>

Receptive fields in the face are organized somatotopically in an “onion peel” configuration (Fig 1-4) such that, regardless of which trigeminal nerve branch carries their input, nociceptive primary afferent neurons from the most anterior areas of the face synapse with secondary neurons in the most rostral areas of the  $V_c$ . Moving outward and posteriorly in a ring-like fashion from the most anterior regions of the face, primary neurons terminate centrally in progressively more caudal areas of  $V_c$  (see Fig 1-4).<sup>8</sup> For instance, A and C-fiber neurons

<sup>1</sup> This term is sometimes used to refer to the spinal nucleus in its entirety, ie, all three subnuclei taken together.

**Table 1-2** Nociceptive input to spinal nucleus

Peripheral nerve or nerve branch	Structures/areas innervated
Ophthalmic (V1)	Nasal mucosa Cranial vessels Cornea Conjunctiva
Maxillary (V2)	Skin Teeth Oral mucosa Nasal mucosa
Mandibular (V3)	Skin Oral mucosa Temporomandibular joint Jaw muscles Cranial vessels
Nervus intermedius of facial nerve (VII)	Ear lobe
Glossopharyngeal (IX)	Ear Tongue (posterior third) Pharynx Larynx
Vagus (X)	Pharynx Larynx
C2–C4, possibly C5	Posterior head and neck Auricle External acoustic meatus Anterior neck Angle of mandible Shoulders Upper thoracic region (greater occipital nerve [GON] and lesser occipital nerve, greater auricular, transverse cervical, supraclavicular)

from area 5 in the face, whether they start in V1, V2, or V3, all synapse with secondary nociceptive neurons in the most caudal aspect of the subnucleus caudalis, lamina 5. Such convergence means that a dural blood vessel, masseter muscle, or a tooth or tongue nociceptive afferent could excite the same secondary neurons.

This convergence is a probable anatomical basis for referred pain and is not restricted to the trigeminal nerve branches. SpV receives peripheral input from the upper cervical sensory

nerves as far caudally as C5. Therefore, nociceptive input from cervical and upper shoulder areas converges onto the same secondary neurons that receive signals from the face, head, and oral cavity, meaning that trapezius or sternocleidomastoid nociceptive afferents can excite secondary neurons that also receive input from facial tissues.<sup>21-23</sup> Nociceptive input carried by branches of the facial, glossopharyngeal, and vagus nerves likewise converges onto SpV neurons (Table 1-2). This neuronal organi-

## 1 Introduction to Orofacial Pain



zation may help to explain the high prevalence of painful comorbidities in tissues of the head and face (ie, headache and sinusitis, headache and TMDs, neck pain and facial pain).

All of the CNS structures affected by trigeminal nociceptive input are also contacted by secondary neurons from the dorsal horn of the spinal cord.<sup>24</sup> Therefore, potential pain input from regions outside trigeminal receptive fields may excite CNS structures that communicate with trigeminal nuclei and modulate their functions.

Neurons whose cell bodies are housed within the dorsal horn include projection neurons, whose ascending axons relay sensory input to higher CNS areas, as well as a vast number of interneurons, which modify signals at the level of the brainstem. Some projection neurons are nociceptive specific, ie, they receive input only from nociceptive primary afferent fibers. Other projection neurons receive a variety of signals, including both nociceptive and non-nociceptive sensory input, and are called *wide dynamic range neurons* (WDRs).

Whether and with what frequency a secondary neuron fires action potentials in response to peripheral nociceptive input depends on both spatial and temporal summation, ie, whether incoming signal frequency at a given location within a given time is sufficient to overcome inhibitory influences (see following section, “Modulation of Pain”) and reach the threshold required to excite the secondary neuron’s membrane and evoke a response. This threshold is not “set in stone” but is dynamic and can be modified as cytokines and other sensitizing molecules induce changes in the type, number, and activity level of receptors and ion channels in the secondary neuron’s cell membrane (see discussion of sensitization below).

Axons of secondary neurons that project information for touch and proprioception follow the lemniscal pathway. This pathway ascends via the dorsal column of the spinal cord before decussating in the medulla.<sup>25</sup> Branches from the

lemniscal pathway are involved in local inhibition of pain. On the other hand, the pathway responsible for transmitting information about pain and nonpainful temperatures is called the *spinothalamic pathway*.<sup>26</sup> In this pathway, the synapse between a primary and a dorsal horn secondary sensory neuron occurs on the same side of the spinal cord. Secondary neurons have a single axon that crosses the midline to the contralateral side of the spinal cord before ascending as part of the lateral spinothalamic tract. Hence, this pathway follows the side of the body opposite to where the signal originated. The difference between the lemniscal pathway (for touch and proprioception) and the spinothalamic pathway (for pain and temperature) has important clinical implications because some injuries that only affect one side of the spinal cord may interrupt only the sense of touch with respect to one side of the body, while other injuries may only alter pain sensation. In similar fashion to the spinothalamic tract, trigeminal secondary axons originating in the main sensory nucleus and SpV decussate before ascending via the ventral trigeminal lemniscus to higher CNS centers.<sup>25</sup>

As secondary axons carrying information about pain and nonpainful temperatures ascend from the SpV toward the thalamus, they send multiple offshoots (arborizations) to various areas of the reticular formation of the brain stem. The reticular formation plays important roles in the control of baseline processes such as behavioral arousal, consciousness, and motivation and therefore represents an early opportunity in the chain of neuronal transmission for nociception to elicit subconscious physiologic responses.<sup>8,24,27,28</sup> Secondary neurons that are stimulated by glutamate-releasing A $\delta$  fibers arborize less than those receiving impulses from slower-conducting C fibers, which release a wide variety of neurotransmitters and neuropeptides.<sup>8,29,30</sup> Thus, information from A $\delta$  fibers stimulates a much faster nocifensive (ie, protective reflex) response than that elicited by C

**Table 1-3** Brain regions associated with dimensions of pain

Cerebral structure/region	Aspect of pain	Dimension of pain (per Melzack and Casey <sup>31</sup> )
1° somatosensory cortex	Sensory processing	Sensory-discriminative
Insular cortex	Emotional and arousal response; attentional processing	
Cingulate cortex	Emotional and arousal response; attentional processing	Motivational-affective
Amygdala and hippocampus	Anticipation of pain	
Prefrontal cortex	Reframing/contextual evaluation of event	Cognitive-evaluative
Thalamus	Sensory processing; modulation of input to limbic system	

fiber input, which is important in maintaining persistent pain and coordinating reparative and behavioral responses.

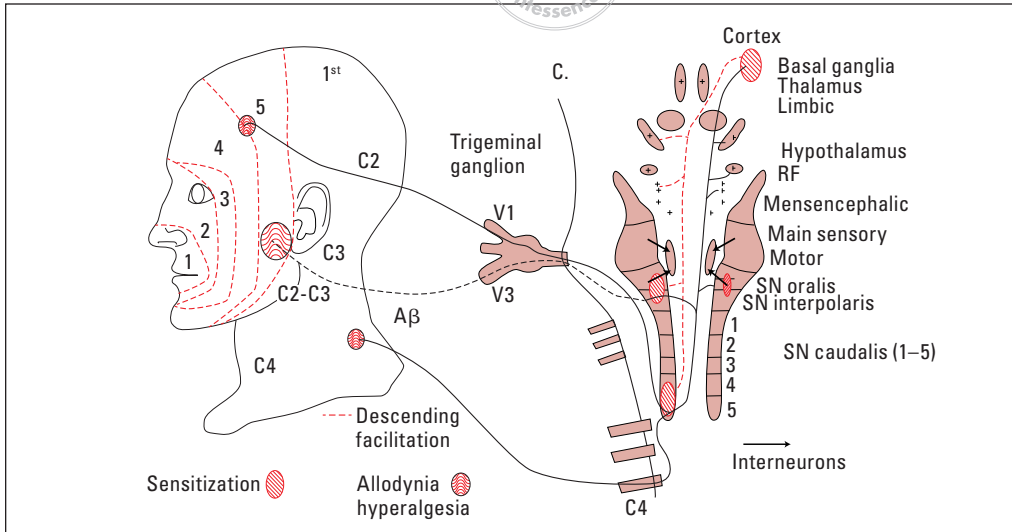
Axons of secondary SpV neurons carry nociceptive impulses to the rostral ventral medulla (RVM) and periaqueductal gray (PAG) and then on to tertiary neurons whose cell bodies reside in the thalamus. These thalamic neurons form connections with higher brain centers, including primary and secondary somatosensory cortices, prefrontal cortex, anterior cortex, amygdala, and nucleus accumbens. The brain contains no single “pain center”; rather, the interplay of multiple cerebral regions transforms the neural activity of nociception into the multidimensional experience of pain.

Three dimensions of this pain experience were described in a 1968 chapter by Melzack and Casey as sensory-discriminative, cognitive-evaluative, and motivational-affective.<sup>31</sup> The sensory-discriminative dimension includes sensation of pain characteristics such as location, intensity, duration, and quality. Motivational-affective refers to the emotion-charged urge to take action to escape the unpleas-

antness of the pain. The cognitive-evaluative encompasses appraisal of the meaning of the pain experience and may be heavily influenced by cultural factors.

Melzack and Casey<sup>31</sup> provided a useful illustration of these three pain dimensions in action: Imagine someone being given a hot beverage in a mug belonging to her friend. Upon picking up the mug, the individual immediately senses painful, unpleasant heat in the palms and fingers (sensory-discriminative) and feels a strong urge to immediately drop the mug (motivational-affective). However, the individual is simultaneously aware that allowing the mug to shatter on the floor would be socially awkward and might give offense to her friend (cognitive-evaluative) so instead she quickly but carefully sets the mug on the table.

Functional magnetic resonance imaging has provided insights into areas of the cortex that are active during pain and the roles these areas play in different dimensions of pain. Some of these insights are summarized in Table 1-3 and grouped according to the three dimensions of pain described above.



**Fig 1-5** Sensitization of trigeminal system. Primary nociceptive neurons from facial lamina 5 transmitted via V1 and C4 converge onto lamina 5 of the subnucleus caudalis. If the pain sources are not controlled, summation exceeds descending inhibition, and progressive levels of central sensitization occur, first at the subnucleus caudalis, then at the ipsilateral subnucleus oralis, where A $\beta$  fibers are carried on V3 synapse. With continued summation, sensitization occurs at higher brain sites and at the contralateral subnucleus oralis. Nonpainful thermal and tactile inputs are experienced as painful (allodynia) or a more intense pain is felt (hyperalgesia) because of the effects of central sensitization. RF, reticular formation structure; SN, subnucleus.

Nociceptive impulses generated by potential or actual tissue damage are one of many types of input that are continually assessed and evaluated throughout the various levels of the CNS. The senses (smell, sight, hearing, touch, and taste) alert the brain to stimuli through thalamic-amygdala and thalamic-cortical-amygdala circuits, and those data streams are analyzed and compared to stored information within the brain to facilitate an efficient behavioral response.<sup>32,33</sup> Ongoing proprioceptive, nociceptive, thermoreceptive, baroreceptive, chemoreceptive, and vestibular input provides sensory information to the brain about how effectively its tissues are responding and enables the brain to make ongoing behavioral

adjustments aimed at maintaining efficiency. Pain stimulates CNS-coordinated behavioral responses including motor commands and changes in muscle and glandular activity and also evokes autonomic nervous system modulated cranial nerve responses.<sup>8,34,35</sup> Hence, pain signals provide the brain an opportunity to make behavioral adjustments to avoid further, potentially damaging stimuli.<sup>36</sup> The multilevel complexity of pain processing in the nervous system suggests that in many cases, successful pain control needs to rely on more than an attempt to isolate and control peripheral tissue damage because pain is ultimately an experience generated by the brain, and brain activity must therefore be a focus of treatment.

Page numbers followed by “t” denote tables, “f” denote figures, and “b” denote boxes.

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