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Orofacial Pain and Sensory Disorders in the Elderly

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An elderly patient who attends his or her physician's or dentist's office with a complaint of pain is more likely to be taking multiple medications and to have two or more active chronic medical problems than is a patient 2 or 3 decades younger. Likewise, the diagnostic process and time needed to investigate the pain complaint will be more complex for the older patient. The prevalence of general pain in the elderly population is moderately high, with estimates of persistent pain ranging from 25% to 88%, depending on the definition used and the nature of the population [1,2]. Chronic pain is defined as pain that persists beyond the time expected for healing. Alternatively, chronic pain may be due to a long-standing persistent disease process, such as an autoimmune disease or neurologic sensitization in peripheral or central nerves. In the elderly, the cause of chronic pain is frequently neuropathy or polyarthritis. Riley et al [3] conducted telephone interviews of community-dwelling older ($\geq$65) north Floridians (N = 1636) and found that 17.4% reported some form of current or recent (ie, within the last year) orofacial pain. The pains were generally of four types: jaw joint pain, facial pain, burning mouth pain, and oral sores. In this article the authors discuss the likely disorders and diseases that produce facial pain and burning pain in the mouth. They do not cover jaw joint pain, oral sores, or ulceration-induced pain, as these conditions are better discussed in the context of arthritis and oral pathologies of the mouth. At the same time, the authors supplement their discussion with that of another disease category seen in the elderly, namely, occlusal dysesthesia.

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Facial pain related to oral motor disorders

An oral motor disorder is a "movement disorder," and they can be broadly classified into "hypokinetic" and "hyperkinetic" conditions. The hypokinetic disorders (eg, parkinsonian rigidity) are not associated with pain. By contrast, the hyperkinesias can and do produce pain. These can be subclassified into the stereotypic dyskinesias, tremors, dystonias, tics, myoclonus, and choreas. Some might even add the parasomnias (eg, sleep bruxism, periodic leg movement syndrome) and secondary spasms to this group. All movement disorders, and oral movement disorders in particular, are more common in older age. Bourgeois et al [4] examined 270 elderly subjects in a residential nursing facility for dyskinesias, both spontaneous and drug-induced. They reported that females were twice as likely to have a dyskinesia (27%) as males (12%). Among those who had dyskinesia, two thirds of the dyskinesias were related to neuroleptic medications and one third were of spontaneous onset. The good news is that, although some oral motor disorders do induce pain, the link between pain and abnormal motor function is generally not strong. The hyperkinetic disorders that produce pain are discussed later.

Dystonia presents as an involuntary, briefly sustained contraction of muscles. If the dystonic contraction is strong and frequent enough, pain may result. When the dystonia involves only one or two areas of the body, it is labeled a focal dystonia. For example, some patients exhibit an involuntary repetitive contraction of the orbicularis oculi muscles, which produce eye closure. This disorder has been called blepharospasm. If the cervical (usually sternocleidomastoid and trapezius muscles) contract, this is called a torticollis. Several focal dystonic patterns involving some combination of jaw, neck, tongue, and perioral muscles are described as focal orofacial, orolingual, oromandibular, or cervical dystonias [5]. Frequently, the patient with a significant oromandibular dystonia will have compromised mastication and be unable to function with a removable dental prosthesis (especially mandibular full dentures). Some of the severe orofacial dystonias may actually create such difficulty that patients are unable to eat and lose weight. If the dystonia strongly affects the tongue musculature, it may compromise the patient's ability to speak clearly. A combination of blepharospasm and jaw opening dystonia has been labeled Meige's syndrome [6,7].

Bruxism is another example of a frequently seen motor disorder that, if severe, produces pain and even damage to the masticatory structures, such as broken or worn teeth, derangement, temporomandibular joint (TMJ) arthritis, and jaw muscle pain. Between 6% and 20% of the population have been reported to exhibit bruxism. This disorder is more common in children (14%) and generally decreases after the age of 50 years [8]. The distinction between tooth grinding and tooth clenching is not clear-cut, but the latter is thought to occur more frequently and to be more common in women than in men. Actually, it is somewhat difficult to confirm or refute the presence of

bruxism or clenching, because patients often do not know they are grinding and certainly may not know they are clenching. Moreover, there has been no population-based study involving large numbers of patients in which polysomnography has been performed. Dental wear or attrition is not always a good indicator of current bruxism or clenching.

The causation is not clear for any of the spontaneous-onset motor disorders, whether one is discussing dystonia or bruxism. A causative agent can be identified when the movement disorder is secondary to a prescription medication or abuse drug [9]. The most commonly reported offending drug regimen is chronic exposure to neuroleptic drugs. A wide range of other drugs have been linked to involuntary movements, usually by isolated case reports in the literature, although it is often difficult to evaluate the clinical significance of such reports. One class of medications recently associated with motor side effects is the serotonin selective reuptake inhibitors (eg, paroxetine, fluoxitine). Winocur et al [10] reviewed the literature on drug-induced bruxism and concluded that, despite the widespread anecdotal case reports, there is insufficient evidence-based data to draw definite conclusions about the effects of various drugs on bruxism. Although certain substances related to the dopaminergic, serotonergic, and adrenergic systems suppress or exacerbate bruxist activity in humans and animals, the literature is still controversial. Evaluation of a patient's drug history may prevent oral motor dysfunction or identify an iatrogenic cause.

Oral motor dysfunction is best managed with a multidisciplinary approach, including medications, protective devices (bite guards), and motor-paralyzing injections (ie, botulinum toxin). The protocol for these injections has been described in a recent review [11]. One common medication used in this disorder is high-dose anticholinergic medication (trihexyphenidyl); however, older patients tolerate this drug poorly, and it works in only one third of those to whom it is prescribed. Side effects consist of substantial dryness of the mouth, jitteriness, stomatitis, blurred vision, and forgetfulness.

Facial pain related to muscle pain (myalgia, myofascial pain, and fibromyalgia)

Localized myalgia, regional myofascial pain, and the more generalized fibromyalgia syndrome (FMS) are common chronic pain problems that predominantly affect middle-aged women [12]. Although local myalgia and myofascial pain are more prevalent in the middle aged, fibromyalgia increases with age and is substantially more evident in the elderly population. FMS affects up to 2% of the population and can start at any age; it is at least seven times more common in women than in men [13]. By the time the diagnosis is made, patients have often had symptoms for many years. Patients with fibromyalgia complain of pervasive muscular and sometimes joint pain and,

by definition, have pain on both sides of the body, above and below the waist and in both the trunk and the extremities. These findings suggest (and most researchers agree) that an aberrant central pain processing mechanism produces a state of sensitized pain perception in FMS [14]. Because of the widespread muscle and joint pain, fibromyalgia patients usually have poor quality, nonrestorative sleep. They also frequently report irritable bowel syndrome and headaches. Because of the negative effect fibromyalgia has on activities of daily living, it usually induces depression and anxiety, and it often accompanies other chronic, painful disorders [15]. Specific clinical history and examination criteria must be met before a diagnosis of fibromyalgia is given. These criteria, adopted by the American College of Rheumatology [16], specify that a diagnosis of fibromyalgia is made when the patient has widespread pain for at least 3 months accompanied by tenderness at discrete locations. To be eligible for research studies, patients must have at least 11 tender points out of a possible 18, but, in practice, the diagnosis can be made in patients with fewer tender points if there is widespread pain and many of the other characteristic symptoms. Patients with fibromyalgia often are tender all over; the presence of tenderness in locations other than the classic ones does not exclude the diagnosis.

Medications often used for FMS are listed in Table 1. The most common medication used in this group of patients is a tricyclic antidepressant agent (eg, nortriptyline, amitriptyline) [17]. These medications are versatile and effective in treating many symptoms associated with FMS, but tolerability remains a problem, and this is especially true in the elderly. By contrast, serotonin selective reuptake inhibitors show improved tolerability and have demonstrated much clearer activity against depressed mood in the context of FMS than have tricyclic antidepressants. However, their activity against other symptoms appears less robust. Sedative-hypnotic compounds, such as zolpidem (Ambien) appear to be useful adjuncts for the treatment of disturbed sleep, and the use of tramadol to treat FMS pain is supported by three trials. Nonsteroidal anti-inflammatory drugs, by contrast, have not been shown to be particularly effective in FMS.

Table 1
Medications for myofascial pain and fibromyalgia

Medication class	Effect	Comments
Tricyclic anti-depressant agents (eg, nortriptyline)	+++	Moderately to mildly helpful for pain, but high side effects
Serotonin selective reuptake inhibitors (eg, citalopram)	+	Lower side effects than TCAs; more for depression than for pain
Opioid/SNRI: serotonin-norepinephrine reuptake inhibitor/narcotic (eg, tramadol)	+++	Several studies show it is moderately helpful for FMS-related pain.
NSAIDs (eg, ibuprofen)	+/−	Not particularly effective in FMS

Abbreviations: NSAIDs, nonsteroidal anti-inflammatory drugs; TCAs, tricyclic antidepressants.

Behavioral treatments entail making sure the patient has a good understanding of the disorder and engages in daily physical exercise and relaxation. The self-management program is crucial to ensuring that the patient does not have increasing feelings of anxiety and helplessness, which aggravate the disease [18]. Many patients can be helped by encouragement and reassurance, along with regular aerobic exercise. However, patients with fibromyalgia tend to remain symptomatic at unchanged levels for many years. Most, if not all, should be encouraged to continue working and to maintain regular social activities despite their symptoms. The management of fibromyalgia patients involves a complex interplay between pharmacologic management of pain and associated symptoms and nonpharmacologic modalities. Regular follow-up and modification of the initial management strategy is usually required, depending on the response pattern. FMS patients typically have a number of complaints beyond pain, and a vast majority cite fatigue as a significant cause of morbidity. The potential causes of fatigue in these patients are manifold, but recent evidence suggests that sleep disturbances play a particularly important role. Exercise interventions for these patients vary, depending on the extent and severity of symptoms as well as on factors that affect patient motivation and adherence. Secondary psychosocial effects are pervasive, including depression, reduced confidence in one's ability to manage the disease, and disruption of relationships with friends and family. Unfortunately, depression and reduced self-confidence make it particularly difficult for these patients to adhere to an exercise program.

Facial pain of vascular origin

Facial pain can result from a generalized chronic vascular inflammatory syndrome of large and middle-sized blood vessels that is characterized by the presence of giant cell accumulation inside the arteries. When giant cell arteritis predominantly affects the cranial and scalp vessels, it is called temporal arteritis, and the palpable vessels of the scalp are usually sore, tender, thickened, and pulseless [19]. Temporal arteritis commonly presents for the first time in older people; the mean age at onset is 70 years, with most cases occurring between 60 and 75 years. It is rare in people less than 50 years of age [20]. Women are affected twice as often as men. Temporal arteritis is probably a polygenic disease in which multiple environmental and genetic factors influence susceptibility and severity. Genetic polymorphisms have also been considered important candidates for factors of susceptibility to giant cell arteritis and polymyalgia rheumatica, a similar disorder. However, additional studies are required to clarify the genetic influence on susceptibility to these conditions.

Gonzalez-Gay et al [21] examined the influence of age on the clinical expression of giant cell arteritis in a population in northwest Spain. Using patients with biopsy-proven giant cell arteritis, they reported that this

disorder was more common in women (rate ratio 1.58 [females] to 1.00 [males]) and occurred in patients with an age greater than or equal to 50 years. Systemic symptoms (eg, fever) occur in about half of patients; in approximately 15% of patients, this may be the presenting clinical manifestation. In approximately two thirds of patients, headache is the most frequent presenting symptom. The onset tends to be gradual but can also be abrupt, with new headache pain, such as scalp tenderness, as a primary complaint. The pain symptoms are usually confined to the temporal and sometimes the occipital arteries, but the occipital arteries are less often involved. Intermittent claudication (fatigue or pain on function) may occur in the muscles of the jaw or even tongue. In rare cases, more marked vascular narrowing may lead to infarction of the scalp or the tongue. One serious complication of temporal arteritis is permanent partial or complete loss of vision in one or both eyes. Affected patients typically report partially obscured vision in one eye, which may progress to total blindness. If untreated, the other eye is likely to become affected within 1 to 2 weeks. Warning signals for temporal arteritis include onset of a new headache after the age of 50 and the progressive course and systemic symptoms of malaise and jaw claudication on function. The screening investigations usually ordered for clinically suspected temporal arteritis are complete blood count, erythrocyte sedimentation rate (ESR), C reactive protein, urea electrolytes, liver function, glucose, thyroid function, rheumatoid factor, electrophoresis, and chest radiography. If the ESR is elevated, a biopsy of a clinically affected scalp vessel should immediately be performed; if there is no clearly involved vessel, the superficial temporal artery ipsilateral to the headache should be sampled. If the clinical situation suggests giant cell arteritis, a biopsy should be performed even if the ESR is not elevated, because the disease is sometimes found on biopsy in patients without ESR elevation [22].

Temporal arteritis is usually treated with corticosteroids in a dose sufficient to relieve symptoms and normalize the ESR [23]. A usual starting regimen is 80 mg of prednisone daily. The dose of steroid may be regarded as balancing the risk for relapse and complications of the disease process against the risk for steroid-induced side effects. It is important to adhere to the correct dosage and regimen of steroid treatment to avoid both undertreatment (resulting in increased risk for arteritic complications) and overtreatment (resulting in increased risk for steroid-associated side effects). The National Osteoporosis Guidelines (http://www.rcplondon.ac.uk/pubs/wp_osteo_update.htm) state that individuals on more than 7.5 mg of prednisolone for more than 6 months should be given osteoporosis prophylaxis. Calcium and vitamin D supplementation should be given with corticosteroid therapy in all patients with temporal arteritis. The treatment for temporal arteritis can be divided in four parts. Part 1: the starting dose of 20 to 40 mg prednisolone can be maintained for 1 month but should be supplemented with an osteoporosis prophylaxis medication. Part 2: once the clinical symptoms have been reduced, the dose of prednisolone

can be reduced by 5 mg every 2 weeks. Once a daily dose of 5 mg per day is achieved, it is wise to hold the patient at this daily dose for 12 months. If there is no recurrence, this dosage can be stopped.

Facial pain of neurovascular origin

Approximately 66% of headache pain in the elderly is caused by migraines or tension-type headaches, and this figure is well over 90% in a younger cohort [24]. Unfortunately, when new headaches develop in the elderly, the other 33% are more likely to be due to an intracranial lesion or a systemic disease. The good news is that the overall prevalence of headaches declines with age; indeed, this prevalence has been reported to decline from 83% in individuals between 21 and 34 years to 59% in those between ages 55 and 74 [25]. One exception to this pattern is that migraines sometimes occur for the first time after age 50; about 2% of all migraines start at this late age [26].

It is prudent to diagnose systemic and intracranial diseases and other disorders that are often a cause of headaches in old age using CT or MRI of the head, just in case. Wilkinson [27] has described how auras tend to disappear with age, but one concern in the elderly is that transient ischemic attack might be mistaken as an aura and vice versa. A good general rule is that an unusual initial presentation or a change in symptomatology (other than frequency or intensity) of migraine is a ''red flag'' that calls for consideration of imaging studies.

Managing headaches in older patients presents several challenges [28]. First, diagnosis is always difficult, because of the possibility that the pain is due to a central nervous system pathology or some other organic disease process. Second, treating the elderly with migraine abortive medications (triptans) requires extra caution, because of their reduced tolerance to medications and potential for increased contraindications due to concomitant disease (eg, coronary artery disease) or polypharmacy. Third, the use of prophylaxis for the chronic daily headache sufferer (eg, beta blockers, tricyclic antidepressants, calcium channel blockers) may cause unacceptable lethargy and confusion in the elderly. These medications are usually more sedating and more apt to result in kidney failure because of the decreased renal reserve in this age group.

Facial pain related to trigeminal neuralgia

Trigeminal neuralgia (TN) presents as a sudden, usually unilateral, severe, brief, stabbing, recurrent pain in the distribution of one or more branches of the fifth cranial nerve. Although recent evidence points to vascular injury (abrasion) of the trigeminal nerve root within the cranial vault, this alteration is not usually visible using current imaging modalities.

However, as many as 15% of patients may have an underlying cause, such as a benign or malignant tumor of the posterior fossa or multiple sclerosis [29]. Some patients have many features of TN, yet aspects of their history do not agree with the typical manifestations of the condition. Many patients attribute their pain to dental causes and will seek dental therapy as a first line of treatment. Because dental pain is extremely common, this is a valid assumption; however, it is important that dentists consider possible nondental causes of pain and not attempt complex and irreversible procedures. TN may also present exclusively intraorally, a manifestation that can be confusing for patients and clinicians. The International Headache Society has suggested criteria for the diagnosis of TN (Box 1) [30].

Most drugs used in the management of TN interfere with ion channels, in line with the assumption that pain in TN is primarily caused by spontaneous ectopic neuronal firing [31]. Some drugs may also inhibit noxious transmission by reducing glutamate release and by augmenting segmental gamma-aminobutyric acid inhibition. The effects of both the drugs whose efficacy has been evidenced in controlled trials and most of those being used on the basis of uncontrolled observations seem to be sound and consistent with the proposed pathophysiologic mechanisms in TN and the basic pharmacology of the drugs. The current evidence leaves no doubt that carbamazepine should be the first-line treatment for patients with TN. Dosing according to efficacy and side effects is clinically feasible; after dose

Box 1. International Headache Society criteria for trigeminal neuralgia

- Paroxysmal attacks of facial or frontal pain that last from a few seconds to <2 minutes
- Pain has at least four of the following characteristics:
  (a) Distribution along one or more divisions of the trigeminal nerve
  (b) Sudden, intense, sharp, superficial, stabbing, or burning in quality
  (c) Severe intensity
  (d) Precipitation from trigger areas or by certain daily activities, such as eating, talking, washing the face, or cleaning the teeth
  (e) The patient is entirely asymptomatic between paroxysms.
- No neurologic deficit
- Attacks are stereotyped in the individual patient
- Exclusion of other causes of facial pain by history, physical examination findings, and special investigation (when necessary)

titration, the patients should preferably be given slow-release formulations, so that diurnal variations in serum drug levels do not influence efficacy. Carbamazepine and oxcarbazepine are similar in terms of basic pharmacology and may be interchangeable; moreover, oxcarbazepine appears to be better tolerated. However, the efficacy of oxcarbazepine needs to be evidenced in controlled trials. In some countries, despite the lack of controlled clinical trials, oxcarbazepine is the first drug of choice. The choice of treatment will, of course, also depend on patient-related factors. Elderly patients are more prone to some central nervous system side effects of carbamazepine, and some of the other drugs may be preferable for them. Carbamazepine cannot be used in patients with cardiac conduction disturbances.

Facial pain related to a chronic trigeminal neuropathy

Over the years, many different terms have been used to describe patients with dental pain of unknown origin. The most common is ''atypical odontalgia'' [32–37]; once the tooth is extracted and the pain continues, the term ''phantom tooth pain'' is used [38–41]. Although the mechanism is debated, this is most likely a neuropathic pain process, which is defined by the International Association for the Study of Pain [42] as ''pain initiated or caused by a primary lesion, or dysfunction in the nervous system.'' In a recent paper, Marbach [43] introduced some specific diagnostic criteria for what he termed ''phantom tooth pain,'' based mainly on the clinical characteristics of the pain.

Currently, the most accepted theory regarding these pain phenomena is that trauma to the orofacial structures (eg, traumatic injury, periodontal surgery, pulp extirpation, endodontic therapy, apicoectomy, tooth extraction, implant insertion) or even minor trauma (eg, crown preparation, inferior alveolar nerve block) alters the neural continuity of the tissues, creating sensitization of the peripheral nociceptive nerves. This mechanism falls into the category of neuropathic pain, in that, after the wound has healed, the neural tissue is responsible for the pain and other related symptoms (paresthesia, dysesthesia). Of course, multiple mechanisms are involved in the pathogenesis of neuropathic pain. But the bottom line is that, following a nerve injury or regional inflammation, the afferent nociceptive fibers become sensitized, showing a lower activation threshold and sometimes developing spontaneous ectopic activity as a result of increased expression or redistribution of sodium channels on the axon. This sensitization could easily explain some clinical manifestations of oral neuropathic pain, such as the clear-cut mechanical or thermal allodynia and persistent spontaneous pain.

The diagnosis of chronic trigeminal neuropathy is essentially a clinical process. The most prominent and sometimes the only evident symptom is pain. It is commonly described as a continuous and spontaneous dull ache localized in a tooth or tooth region. The location may change to an

edentulous area or entire parts of the maxilla or mandible. The pain also may be described as burning, sharp, or throbbing. It usually persists for months or years, being continuous and persistent but oscillating in intensity, with episodes when the pain is more acute and severe. For a diagnosis of trigeminal neuropathy to be made, other pathologies characterized by tooth pain need to be ruled out. Several have been listed: pulpal toothache, TN, myofascial pain, sinusitis, cracked tooth syndrome, and migrainous neuralgia. Probably the most difficult task is to distinguish between trigeminal neuropathy and toothache from pulpal origin. Five characteristics that are common in trigeminal neuropathy but not in pulpal toothache are listed in Box 2.

Once the diagnosis of trigeminal neuropathy has been made, appropriate treatment needs to be established, including avoidance of any further dental procedure that could aggravate the pain. Most medications used are formulated for the treatment of neuropathic pain and appear to be effective in most patients with trigeminal neuropathy [44]. In many of the articles reviewed, tricyclic antidepressants (TCAs) were prescribed with good results. The use of these medications is limited by the occurrence of side effects; TCAs cause dry mouth, weight gain, constipation, and urinary retention. They are contraindicated in patients with angle-closure glaucoma or high intraocular pressure and those taking other medications, such as monoamine oxidase inhibitors, central nervous system depressants (eg, alcohol, barbiturates, narcotics), anticholinergics, and sympathomimetics, because of drug-to-drug interactions. It is usually possible to avoid or minimize the side effects by adjusting the dose or switching to a different medication within the same category (eg, nortriptyline has less anticholinergic effect than amitriptyline and imipramine). The primary alternative to TCAs is the mild anticonvulsant gabapentin, or clonazepam. Opioid narcotic analgesics (eg, oxycodone, meperidine, controlled-release morphine, fentanyl, ketamine, methadone) have been tried, but they are usually only moderately effective for neuropathic pain and produce unacceptable

Box 2. Five clinical features of trigeminal neuropathy

Constant pain is experienced in the tooth with no obvious source of local pathology.
Local provocation of the tooth does not relate consistently to the pain. Hot, cold, or loading stimulation does not reliably affect the pain.
The toothache is unchanging over weeks or months, whereas pulpal pain tends to worsen or improve with time.
Repeated dental therapies fail to resolve the pain.
Response to local anesthesia is often equivocal.

side effects in the elderly. Injections of local anesthetics alone or in combination with corticosteroids (triamcinalone) can be repeated and have been found effective as a management tool for many patients. Sympathetic and parasympathetic nerve blocks, through the stellate ganglia or sphenopalatine ganglia, have been reported with similar results.

Finally, medications applied to the site of the pain sometimes give good results. Capsaicin at a concentration of 0.025% for 4 weeks, eventually associated with a topical anesthetic if the treatment is too painful, and eutectic mixture of lidocaine and prilocaine bases cream at the concentration of 5% may be helpful.

Facial pain related to postherpetic neuralgia

Herpes zoster strikes millions of older adults annually worldwide and disables a substantial number of them by means of postherpetic neuralgia. The incidence of herpes zoster increases sharply among patients aged 50 to 60 years and continues its upward course in the decades after 60 [45]. Herpes zoster is caused by renewed replication and spread of varicella-zoster virus in sensory ganglia and afferent peripheral nerves in the setting of age-related, disease-related, or drug-related decline in cellular immunity to varicella-zoster virus. When reactivated, the varicella-zoster virus overwhelms immune control and spreads in the affected ganglia and sensory nerves to the skin. As already mentioned, this event is more likely to occur in elderly people, partially because of age-related decline in specific cell-mediated immune responses to varicella-zoster virus. The disease begins with localized abnormal skin sensations, ranging from itching or tingling to severe pain, which precede the skin lesions by 1 to 5 days. Healing of the skin lesions occurs over a period of 2 to 4 weeks and often results in scarring and permanent changes in pigmentation. The cutaneous eruption is unilateral and does not cross the midline. Along with the rash, most patients experience a dermatomal pain syndrome caused by acute neuritis. The neuritis is described as burning, deep aching, tingling, itching, or stabbing pain and ranges from mild to severe. This pain continues after the rash has healed in as many as 60% to 70% of people aged over 60 years and develops into postherpetic neuralgia.

Postherpetic neuralgia is the most frequently feared complication of herpes zoster in the elderly [46]. The best-established risk factors for postherpetic neuralgia are older age, immunocompromised status, and greater severity of acute pain and rash during zoster. The patient with postherpetic neuralgia may experience constant pain (described as burning, aching, or throbbing), intermittent pain (described as stabbing or shooting), and stimulus-evoked pain, such as allodynia (described as tender). Furthermore, postherpetic neuralgia can impair the elderly patient's functional status by interfering with basic activities of daily life, such as dressing, bathing, and mobility, and instrumental activities of daily life, such

as traveling, shopping, cooking, and housework. The appearance of herpes zoster is sufficiently distinctive that a clinical diagnosis is usually accurate. A direct immunofluorescence assay, when needed, is the best way (other than culture) to distinguish herpes simplex virus infections from varicella-zoster virus infections. Polymerase-chain-reaction techniques are useful for detecting varicella-zoster virus DNA in fluid and tissues [47,48].

No single treatment has proved effective for postherpetic neuralgia [49]. Combination therapy and a consultation with a pain-management specialist are often required. No one treatment is uniformly effective in elderly patients with the disease. Patients and clinicians have employed a large number of treatments for postherpetic neuralgia, but few have been carefully evaluated. Recent clinical trials indicate that topical lidocaine, gabapentin, opiates, TCAs, and intrathecal methylprednisolone can significantly reduce pain in patients with postherpetic neuralgia. Treatment of the disease is complex, often requiring a multifaceted approach. Clinical trials have shown that opioids, TCAs, and gabapentin reduce the severity or duration of postherpetic neuralgia, either as single agents or in combination. The adverse effects of these medications can be additive, especially in elderly patients. Topical application of lidocaine patches has been a major therapeutic advance for some patients. Although multiple studies have demonstrated that antiviral therapy alone reduces the duration of pain, antiviral drugs do not reliably prevent postherpetic neuralgia. Chronic neuropathic pain will develop in a subgroup of patients despite appropriate antiviral treatment.

Burning mouth symptoms (not related to hyposalivation)

Stomatopyrosis (commonly called burning mouth syndrome) and its variant glossopyrosis (burning tongue) are chronic pain syndromes that mainly affect middle-aged and elderly subjects. The sufferers are typically within an age range from 38 to 78 years [50]. Occurrence below the age of 30 is rare, and the female-to-male ratio is about 7:1. Most (90%) of the women with these symptoms are peri- or postmenopausal women. Burning sensation is the main complaint and is usually described as constant, gradually increasing throughout the day, or intermittent, without any reliable alleviating agents. Diagnosis of burning mouth syndrome (BMS) is one of exclusion, because, as in other neurosensory disorders, there are no measurable physical signs other than pain. More than two thirds of BMS patients report a bitter, metallic taste sensation along with the burning. The BMS patient typically reports pain onset ranging from 3 years before to 12 years after menopause; approximately 50% of BMS patients complain of dry mouth (xerostomia) but do not exhibit measurable hyposalivation. The pain symptoms of BMS are invariably bilateral and are usually in multiple areas of the mouth. These symptoms often increase in intensity at the end of each day, and they seldom interfere with sleep. To be considered for BMS,

the patient should have had the pain continuously for at least 4 to 6 months. Pain levels may vary from mild to severe, but moderate pain is the most frequent presentation. The pain should be described as daily bilateral oral burning (or pain-like) sensations deep within the oral mucosa, unremitting for at least 4 to 6 months. The symptoms should generally be continuous throughout the day and should not interfere with sleep.

Like many idiopathic diseases, BMS is a diagnosis made by a careful history and a thorough process of excluding other causes or diseases. Abnormalities that must be excluded include local pathology of the mucosal tissues, nutritional deficiencies (vitamin B_1, B_2, B_6, or B_{12}, folic acid), salivary hypofunction, and diabetic neuropathy. If any of these problems is discovered or if oral lesions are present, the diagnosis is not stomatopyrosis. The frequent observation of taste changes or sensory or chemosensory dysfunctions in BMS patients has suggested that this syndrome might reflect a neuropathic disorder [51]. The notion that BMS is due to psychogenic or psychosomatic factors has generally not been supported by scientific evidence [52,53]. However, many BMS patients exhibit high levels of anxiety and depression as well as pain relief after suitable administration of psychotropic drugs, such as antidepressants or benzodiazepines.

BMS treatment is still unsatisfactory, and there is no definitive cure. BMS patients have shown a good response to long-term therapy with systemic regimens of TCAs, gabapentin, or benzodiazepine-class drugs (anxiolytics). In a recent study, Gremeau-Richard et al [54] examined the effect of topical clonazepam in stomatodynia in a randomized placebo-controlled study. Their protocol required patients to suck a tablet of 1 mg of either clonazepam or placebo and hold their saliva near the pain sites in the mouth without swallowing for 3 minutes, then spit. This procedure was repeated three times a day for 14 days. The investigators included 48 patients (4 men and 44 women, aged 65 $\pm$ 2.1 years), and 41 completed the study. The reported decrease in pain intensity scores was 2.4 out of 10 ($\pm$0.6) for clonazepam and only 0.6 out of 10 ($\pm$0.4) for placebo. Not only was this a statistically significant difference but, more importantly, the blood concentration of clonazepam was relatively low with this method.

It is important to provide patients with information and reassurance about their condition. BMS subjects are likely to have consulted numerous specialists who stated that the mucosa was healthy, and may thus be convinced that their problems are imaginary. Patients must be made aware that their pain is ''real,'' that the syndrome is common in middle-aged and elderly individuals, and that it is often linked to identified conditions. They must also be informed that the oral pain is not related to any form of cancer, that the treatment will be prolonged, and that not all the symptoms are sure to disappear. Avoidance of any triggering stimulants should be practiced (eg, smoking, spicy foods). Peri- and postmenopausal women with BMS should be referred to gynecologists to consider estrogen replacement therapy. Finally, BMS represents a disorder with a very poor prognosis in

terms of quality of life, and the patient's lifestyle may worsen when psychologic dysfunctions occur [55].

Uncomfortable bite without obvious dental cause: occlusal dysesthesia

One final condition to be discussed here is occlusal dysesthesia [56]. The three most common complaints of patients with a persistently uncomfortable bite are "My bite is not comfortable," "My bite is off," and "I don't know where my teeth belong anymore." When such complaints surface, it is necessary to distinguish between those patients with true bite discrepancies and those with no observable abnormality of the occlusion. Most dentists with a few years of experience have experienced such a patient. Severe uncomfortable bite disorder patients are often given either a diagnosis of atypical facial pain or a psychiatric diagnosis, such as that of exaggerated somatic focus. One explanation for these complaints is that some individuals develop a sensitization of the mechanoreceptors in and around selected teeth, which produces an altered or even enhanced proprioceptive signaling from the teeth. This signaling produces an enhanced awareness of all mechanical stimulation. Unfortunately, this problem is fairly frequent and more likely to occur in the elderly. Gerstner et al [57] collected data using a survey (0–4 scale) that asked, "Do you have a problem with your bite being uncomfortable?" The data involved 127 consecutive TMJ clinic patients attending a university temporomandibular disorder (TMD) clinic for diagnosis and help with their jaw pain and dysfunction problems. Nearly 30% of the respondents scored 2 or higher on the question (Fig. 1). This study did not clarify the specific age of the patients who responded yes to the question, but, as mentioned, it is not uncommon to see this problem in an elderly population.

Sometimes, clear and demonstrable abnormality of the teeth, TMJ, or maxillomandibular relationship is present in these patients, and an immediate plan of correction can be made. In some cases, the complaint of bite discomfort develops spontaneously. When no occlusal abnormality or dentoalveolar pathology is evident, the confusion begins, and there are more opportunities for misadventure. For example, a dentist may attempt to make a good occlusion even better by performing occlusal adjustment of the teeth or by replacing the new dental restoration in the suspected tooth or teeth, hoping that the problem will simply resolve with time. If this measure fails, increasingly more aggressive and often irreversible interventions may follow. Unfortunately, these subsequent interventions may aggravate the patient's initial complaint. For example, Remick et al [58] described 21 patients with atypical facial pain who had undergone 65 dental or surgical treatments. Only one patient had less pain as a result of these treatments. The authors indicated in their case series that frequently a psychologic diagnosis was comorbid with the atypical facial pain and suggested that patients with this diagnosis should receive conservative dental and medical

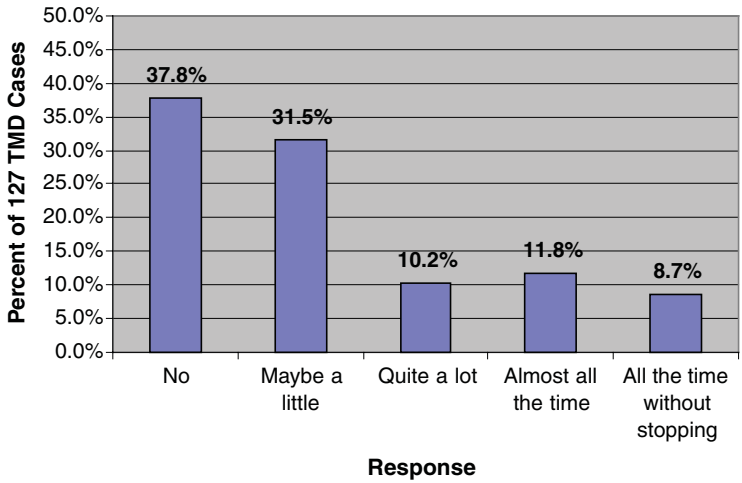

Fig. 1. Response to a question on bite comfort taken from a group of 127 consecutive clinic patients with TMD.

treatment and a behavioral assessment before dental and surgical procedures are contemplated. Although these cases were characterized as pain cases, the basic observation is equally applicable to patients with persistent uncomfortable bite without an observable occlusal abnormality.

Although many terms have been used in the past, including ''phantom bite syndrome,'' Clark et al [59] suggested the term ''occlusal dysesthesia'' (OD) as the best one to describe the complaint of a persistently uncomfortable bite when a bite discrepancy cannot be observed. Occlusion is defined as the act of closure or the state of being closed. The dental meaning of the term ''occlusion'' relates to the static intercuspal relationship of the teeth and also to the act of closing the teeth. Dysesthesia can be described as a disagreeable or impaired (abnormal) sensation, whether spontaneous or evoked. OD is therefore defined as a ''persistent uncomfortable sense of maximum intercuspation after all pulpal, periodontal, muscle and TMJ pathologies have been ruled out and a physically obvious bite discrepancy cannot be observed.''

Two explanations for the phenomenon of OD (severe bite discomfort) are that patients have (1) a seriously altered oral kinesthetic ability or (2) a diagnosable psychiatric disorder. The kinesthetic ability of the jaw is defined as the tested accuracy of subjects to discriminate the position of the mandible in the path from maximum opening to maximum intercuspation. At least one theory attributes this disorder to a general loss of peripheral sensory receptor function in the jaw and teeth and, more specifically, to effects on the function of muscle spindles in the jaw closers. This theory is based on the report by Hellsing [60] that distortion of kinesthesia is independent of the amplitude and frequency of vibration to confound

muscle spindle sensory afferents and persists during anesthesia of the TMJ and loading of the mandible. Hellsing also speculated that jaw muscle receptors may contribute to mandibular kinesthesia. Morimoto and Kawamura [61] also investigated mandibular kinesthesia and suggested that muscle spindles in jaw-closing muscles are primarily responsible for interdental thickness discrimination. Finally, van Duersen et al [62] showed that, with aging and associated peripheral neuropathy, the kinesthetic ability deteriorates. This group demonstrated that vibration had a greater effect on younger subjects, a reduced effect on the elderly, and an even smaller effect on those with peripheral neuropathy. This diminishing effect is presumably owing to the progressive dysfunction of the muscle spindles with aging and peripheral neuropathy, making them less relied on by the subjects.

Marbach [63] suggested that some subjects with phantom bite could instead have a neurotic disorder termed "dysmorphophobia." This is the belief that a body part is cosmetically defective in a person of normal appearance. In contrast, Greene and Gelb [64] reported on five subjects with what they termed proprioceptive dysfunction and symptoms enduring between 44 and 264 months. They found only one subject with a somatoform disorder; the other four "did not qualify for a psychopathological diagnosis, and certainly were not delusional or psychotic." Given this unresolved controversy, all occlusal dysesthesia cases warrant consideration for psychometric testing and psychologic evaluation. The primary psychologic disorder to be ruled out is somatoform disorder. The diagnostic condition somatoform disorder is also the most frequent diagnosis associated with a subjective sensory abnormality unconfirmed by objective signs and tests.

The treatment options for OD are (1) drug therapy, (2) psychotherapy, and (3) strategies to be used by the dentist to reduce mechanical stimulation of the sensitive area. Pimozide is one psychotomimetic drug that has been reported to be helpful in the treatment of phantom sensory phenomena. Pimozide is a nonphenothiazine neuroleptic drug similar in structure to haloperidol. It has many side effects that are problematic for the elderly, and even though positive somatic form behaviors have been noted within a week, it is still dangerous to use. Reports of up to 4.5 years of successful maintenance on pimozide are encouraging. More recently, resperidone has emerged as a preferred drug for complex problems like this one [65]. Patients with OD are reluctant to seek traditional psychotherapy, but the physician who has substantial suspicion of a somatoform disorder should obtain a psychiatric consultation and appropriate testing. No good direct treatment exists for idiopathic OD. The best symptomatic management, for those cases in which the complaint is localized to a single tooth or teeth in one region of the mouth, is to make the patient an occlusal disengagement device (eg, an anterior bite plane). Such a device allows the patient more or less to avoid contact on the offending posterior teeth. In such cases, when the disengagement splint is helpful, it is logical to reduce the thickness of the occlusal splint over several weeks or months until the posterior teeth come

back into contact. No specific medication has been found helpful, other than a partially sedating medication (eg, clonazepam), which helps reduce anxiety and increase tolerance to the symptoms. The final and crucial component of treatment for OD is education. Specifically, it should be explained to patients that their complaints of a persistent uncomfortable bite often are not a physical problem but rather a mechanosensory disorder, which may best be characterized as the outward consequence of a pathologic alteration of the mandibular proprioceptive system. In these cases, mechanical therapies (eg, occlusal adjustment, restoration replacement) are not helpful, but it is hoped that, with time and symptomatic management, the severity of the problem will fade to a level of tolerance.

Summary

The actual prevalence of general pain in the elderly population is moderately high (ranging from 25% to 88%), and research suggests that 17.4% of the elderly will report one or more current or recent orofacial pains within a single year. Several causes of orofacial pain exist, including strong contraction-based movement disorders (such as dystonia and bruxism). Oral motor dysfunction is best managed with a multidisciplinary approach, including medications, protective devices (bite guards), and motor paralyzing injections (ie, botulinum toxin). One muscle condition that is more evident in the elderly than in younger patients is fibromyalgia. This chronic pain condition increases with age, affects up to 2% of the population, and is at least seven times more common in women. Several medications are used for FMS, but efficacy is low and tolerability remains a problem in the elderly. The best medications at present are the tricyclic medications (eg, nortriptyline), the sedative-hypnotics (zolpidem), and a weak opioid analgesic (tramadol). It is probably more important in the treatment of FMS that a physical medicine-behavioral treatment program be established and that the patient engage in daily physical exercise and relaxation.

Another cause of orofacial pain is temporal arteritis, which is commonly seen in older people; the mean age of onset is 70 years. Treatment of temporal arteritis usually involves corticosteroids in a dose sufficient to relieve symptoms. Approximately two thirds of headache pain in the elderly is caused by migraines or tension-type headaches, but when a new headache develops in the elderly, the other third may have an intracranial lesion or a systemic disease. The difficulty with headaches in the elderly is that the medications used in a younger cohort are problematic for many reasons. A frequently misdiagnosed disease in the elderly is TN. Once it is fully developed, it presents as a sudden, usually unilateral, severe, brief, stabbing, recurrent pain. In the early stages of the disease, it is often mistaken for a toothache due to dental abscess.

When the oral pain problem is a sustained pain in the teeth or gingival tissues, this condition is called atypical oral pain or atypical odontalgia;

once the tooth is extracted and the pain continues, the term ''phantom tooth pain'' is used. This disorder is most likely due to a long-lasting and perhaps permanent neuropathic alteration in the trigeminal nerve. Other oral neuropathic diseases that affect the elderly are postherpetic neuralgia, BMS, and an unusual disturbance in the patient's sense of bite comfort, described as OD. Suggested treatments for these neuropathic diseases have been described.

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