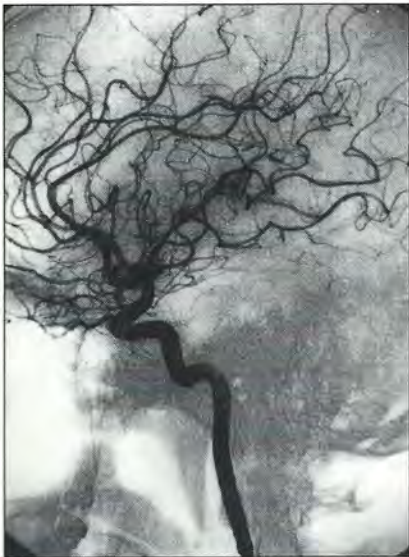


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THE
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PHYSICIANS

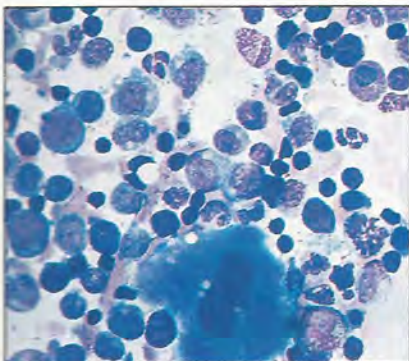
patient care



SUBARACHNOID HEMORRHAGE



KERATOSES



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Spelling relief for TMJ troubles

Pain, noise, and limited mobility of the jaw can signal a structural abnormality, inflammation—or trouble elsewhere. Today's wider diagnostic resources for this chronic problem include MRI and arthrography.

EXPRESS STOP

Focusing the history: When you suspect a temporomandibular disorder, obtain a detailed description of the pain, including its character, location, onset, severity, and duration. Pain commonly focuses in the jaw muscles, usually the masseter or temporalis. It can begin over several days, and in some patients, it can progress over weeks or even months. Pain is often associated with emotional stress or, less frequently, with jaw trauma, and often occurs with jaw or head motion.

The history is crucial in determining the likelihood of a temporomandibular disorder and the need for a more searching evaluation (see Table 1, page 160). A few key questions can help:

- *What kind of pain do you have?* Most patients have a dull, muscular ache in the jaw that worsens with use. Jaw muscles can be exquisitely sensitive to palpation. Referred pain is usually a dull ache or burning sensation that does not change on palpation of the painful area.
- *Where is your pain?* Have the patient outline the area where the pain is present, using one finger. Often, a patient indicates the base of the skull, the temporomandibular joint (TMJ), the masseter, or the temporalis (see Figure 1). Referred pain is often dispersed and affects the ear canal, outer ear,

neck, supraorbital area, temporal areas, occiput, sinuses, or teeth.

- *How did the pain begin?* Pain associated with a temporomandibular disorder can begin over several days, or progress over weeks or months. Typically, the patient gradually becomes aware of discomfort in the jaw, then of pain, and does not recall exactly when it began. Its occurrence is usually associated with insidious-onset emotional stress (see "An overview of TMJ disorders," page 161).

Less frequently, pain can result from trauma incurred by a lengthy dental procedure, a wide yawn, or an activity such as playing a wind instrument or snorkeling. Automobile accidents resulting in whiplash or a direct blow to the mandible also cause injury to the TMJ, the cervical spine, and the muscles of mastication (see "A role for physical therapy in TMJ disorders?" page 162). Sports injuries often involve direct trauma to the mandible.

When the signs and symptoms suggest muscular pain, consider the patient's occupation. Some musicians (for example, clarinetists and violinists) and people who regularly cradle a phone against their shoulder can traumatize the TMJ and the surrounding muscles. Another possible precipitating fac-

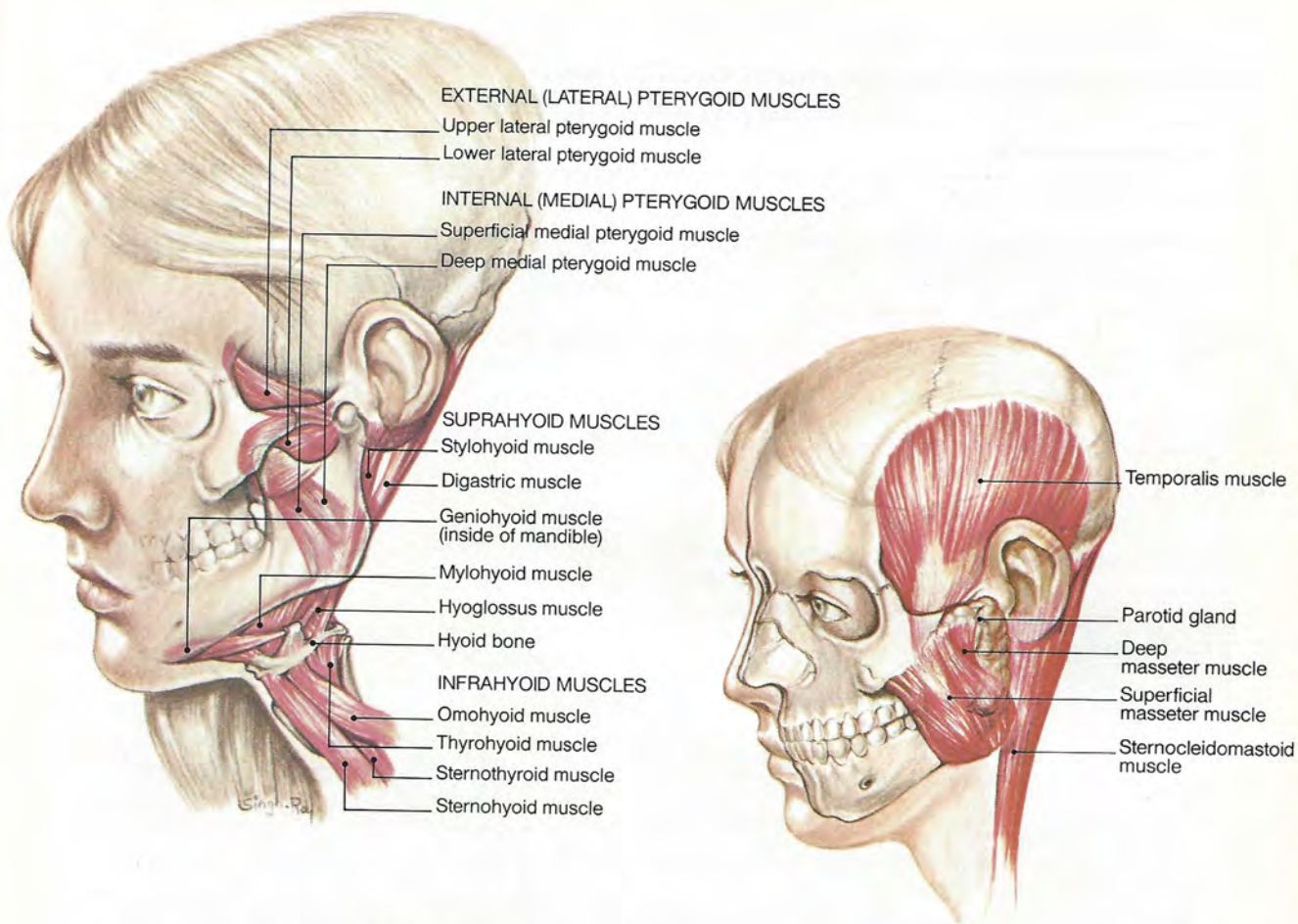


FIGURE 1: **Muscular structures of the TMJ** are often the source of temporomandibular pain. Spasm and fatigue un-

derlie many temporomandibular complaints, and the differential begins with evaluation of these muscles.

tor is a change in work habits that results in an alteration of posture.

• *When is your pain most severe?* Most patients complain of pain or difficulty when chewing, yawning, or opening the mouth wide; some have pain when turning the head from side to side or nodding up and down. The pain is also often associated with a sound

or sensation of clicking, popping, or grinding in the TMJ during opening or closing of the mouth.

Ask the patient about discomfort in the jaw or TMJ, the sensation of loose or painful teeth in the morning, or headaches. These symptoms generally indicate nocturnal teeth grinding or clenching or poor sleeping pos-

ture. In rare instances, painful mastication on arising can indicate rheumatoid arthritis.

Many patients have pain after meals and at the end of the day without showing signs of joint inflammation. For these patients, suspect involvement primarily of the masticatory muscles.

● *How long have you had this pain?* Most patients seek help after having pain for a

month. Some may have experienced pain for a year or longer, however.

Occasionally you will find little in the history to guide your diagnosis. If the patient's pain is obscure in character and location, check for signs and symptoms of atypical facial neuralgia,* keeping in mind the following:

*See "When the complaint is facial pain," *Patient Care*, April 15, 1988, page 75.

TABLE 1

What the history can tell you about TMJ pain

If the patient reports	Increase your suspicion of . . .
Jaw pain that is: <ul style="list-style-type: none"> ● Limited to the masseter, the temporalis, the base of the skull, or the temporomandibular joint (TMJ) <li style="text-align: center;">or ● Associated with insidious onset of emotional stress or trauma or with direct physical trauma <li style="text-align: center;">or ● Brought on by nodding the head or turning it from side to side <li style="text-align: center;">or ● Worse after meals and at the end of the day 	Masticatory muscle fatigue*
Jaw pain or difficulty in chewing, yawning, or opening the mouth wide	
Occupational activities that may stress the TMJ, the muscles of mastication, or the cervical spine	
Jaw pain that is related to clicking or crepitus	Internal derangement such as disk dislocation or degenerative joint disease
Any of the following symptoms, when the patient experiences them in association with arising: <ul style="list-style-type: none"> ● Discomfort in the jaw or TMJ, headaches, or a sensation of loose or painful teeth <li style="text-align: center;">or ● A dull, aching, or throbbing pain in the angle and base of the mandible, with a family history of cardiovascular disease <li style="text-align: center;">or ● Facial pain that is obscure in character or location <li style="text-align: center;">or ● Morning jaw stiffness that subsides with less than half an hour of jaw use, with evidence of rheumatoid arthritis in joints other than the TMJ 	Nocturnal teeth grinding or clenching or poor sleeping posture Referred pain from angina or cardiac infarction Atypical facial neuralgia Rheumatoid arthritis of the TMJ

*If more benign causes have been ruled out, consider osteoarthritis as an explanation for pain or stiffness that increases with jaw use.

- Pain from atypical facial neuralgia is constant; it does not change with jaw function, time of day, or palpation.
- Atypical facial neuralgia is usually diffuse and follows neuroanatomy. In contrast, pain from temporomandibular disorders generally involves specific muscles.
- Unlike temporomandibular disorders, atypical facial neuralgia commonly affects

emotionally depressed women older than 40.

- It does not present with trigger points, as does a true tic douloureux.

Also inquire about any family history of cardiovascular disease. Angina and cardiac infarction can refer dull, aching, or throbbing pain to the angle and base of the mandible. Temporal arteritis, most common in men older than 50, results in tenderness to

An overview of TMJ disorders

Disorders that cause pain in and around the temporomandibular joint (TMJ) in the past were referred to by the term temporomandibular joint syndrome or one of its variants. The more global term temporomandibular disorders is now preferred, acknowledging that dysfunction is not always limited to the joint proper.

Temporomandibular disorders fall into three general diagnostic categories:

- Joint abnormalities resulting from trauma or from conditions such as ankylosis, synovitis, arthritis, and neoplasm
- Structural defects of the articular disk (meniscus), ligaments of the disk, condyles, glenoid fossae, or articular tubercles
- Pain or restricted jaw motion in the absence of evidence of organic disease or structural defects.

Patients whose problems would fall into the last group (and presumably at least some in the first two) experience pain as part of a spasm-pain-spasm cycle affecting muscles in the area. This spasm is a sudden involuntary contraction of a muscle or group of muscles attended by pain and interference with function. Contraction is sustained even when the muscle is at rest. This cycle may subject the TMJ to chronic microtrauma.

Broadly construed, there are five common explanations for symptoms of temporomandibular disorders:

- One prevalent opinion is that emotional stress can be responsible for masticatory muscle fatigue and the spasm-pain-spasm cycle by causing jaw clenching, teeth grinding, and tensing of masticatory muscles. When dysfunction and pain are present without evidence of organic disease or structural abnormality, the disorder is commonly called myofascial pain-dysfunction syndrome.
- Trauma or disease involving the TMJ may cause pain in and dysfunction of the joint proper or masticatory muscles or both.
- Structural abnormalities of the joint may cause symptoms directly or by affecting muscle tone and initiating the spasm-pain-spasm cycle.
- Some clinicians maintain that malocclusion can give rise to the spasm-pain-spasm cycle.
- According to some orthopedic physiotherapists,

poor posture can influence mobility and positioning of the mandible, resulting in the spasm-pain-spasm cycle (see "A role for physical therapy in TMJ disorders?" page 162). Proponents of this view, which is controversial, prefer the term craniomandibular-cervical dysfunction syndrome, since they include derangement of the cervical spine in the etiology.

Typical symptoms include, in order of decreasing prevalence, pain in and around the TMJ or masticatory muscles, followed by joint noise—clicking, popping, or crepitus—and limited range of jaw motion. Referred pain may be felt in the occiput, ears, eyes, sinuses, and the angle of the mandible. Clinical evidence suggests that pain in the neck, shoulders, arms, and fingers may be related to disorders in the cervical spine that may be associated with temporomandibular disorders. Muscular pain often affects the temporalis, the masseter, and muscles of the occipital area and the neck.

Temporomandibular disorders are reported in women about 3-4 times as often as in men. Recent data suggest that the disorders may occur equally in both sexes, but that women are more apt to seek treatment than men. While older studies reported that most patients were women in their 40s and 50s, newer studies report an increasing proportion of men and women under age 20. Some 10-46 million Americans may have symptoms.

Some 70-90% of patients present with pain on jaw movement as the primary symptom, 40-60% also have unilateral joint noise, and 15-25% have a limited range of jaw motion. Pain on palpation of the masseter is present in about half of patients. Pain is almost always unilateral. Nearly 60% of patients with temporomandibular disorders habitually grind or clench their teeth, most without knowing it, and about 20% exercise other potentially deleterious oral habits such as excessive gum chewing, biting on hard objects, or biting their fingernails, cheeks, lips, or tongue.

Of patients with temporomandibular disorders, some 70-80% suffer from masticatory muscle spasm. The spasm is usually attributed to overuse from clenching or grinding of the teeth or tensing of masticatory muscles, either as a long-standing habit or as a result of acute emotional stress.

palpation over the blood vessels in the neck and face. Patients generally describe the pain as throbbing or aching.

EXPRESS STOP

Physical findings: Begin your examination of the patient with temporomandibular complaints by palpating the masticatory muscles. Investigate the condition of the temporomandibular joint (TMJ) proper by palpating both over the joint and through the auditory canal. Listen to the joint with a stethoscope as the patient's jaw moves. Also note any asymmetry in jaw motion and in the face. If treatment with a refrigerant spray improves joint mobility, suspect underlying muscle tension.

Physical examination for temporomandibular disorders begins with palpation of masticatory muscles over their bony origins. Tenderness,

which is quite common, signals muscular involvement but does not reveal information about the specific cause (see "Current thinking on TMJ disorders," page 164).

The masseter, the deep masseter, the temporalis, the internal (or medial) pterygoid, and the infrahyoid and suprahyoid muscles are within your reach. Palpating the internal pterygoids can elicit exquisite pain when a patient has a TMJ disorder.

Refrigerant sprays or vapocoolants, such as halogenated hydrocarbons, can also help diagnose muscular involvement. When a patient with a limited range of jaw motion suddenly improves after a treatment with a vapocoolant, you can usually safely suspect muscle tension as a major component of the disorder.

Continued

A role for physical therapy in TMJ disorders?

Steven L. Kraus, PT, one of the consultants for this article, explains that if jaw, facial, or head pain persists when appropriate treatment has been offered for TMJ and masticatory involvement, or if such symptoms persist in the absence of any hallmark findings for temporomandibular disorder and/or masticatory muscle involvement, you may want to consider cervical spine dysfunction in your diagnosis. Cervical spine dysfunction need not be related to disease, developmental abnormality, or trauma but originates in altered positioning and mobility of the cervical spine and surrounding muscle tissue.

The cervical spine can be a primary source of symptoms to the neck, shoulder, and arm. Dysfunction of the cervical spine can contribute to masticatory muscle hyperactivity and soft tissue tension in the jaw area. The influence of the cervical spine on mandibular muscle and soft tissue tension may reflect the dentist's evaluation and treatment program for TMJ and mandibular muscle involvement.

The trigeminal and cervical root fibers converge on the same sensory neurons in the upper cervical spinal cord; this is the anatomic and physiologic basis for the referral of pain from the cervical to trigeminal territories. Symptoms can be referred from the cervical spine to the occiput, ears, eyes, preauricular area, and the angle of the mandible—the same areas of referred pain as are attributed to TMJ disorders. Therefore, disorders of the cervical spine and those of the TMJ can appear quite similar, and can easily be mistaken for one another.

The diagnosis of cervical spine dysfunction is usually made following the exclusion of other organic disorders. A cervical spine screening evaluates the patient's posture, active range of motion,

and response to muscle palpation and manual traction of the head from the neck. Questions such as these are also asked:

- Do you have pain, tension, tightness, or other symptoms in your neck, shoulders or arms?
- Does movement of your neck or arms increase any of your symptoms?
- If your neck or shoulder symptoms increase, do you have any change in head, face, or jaw symptoms?
- Do you have difficulty in getting your neck and shoulders comfortable at night?
- Does prolonged sitting increase your symptoms?

If a patient with cervical spine dysfunction with or without an accompanying TMJ disorder is seen by a physical therapist, the treatment approach may involve therapeutic modalities such as heat, ice, ultrasound, and electric stimulation to control inflammation of the TMJ as well as masticatory and cervical spine muscle hyperactivity. Manual techniques, intended to improve the mobility of the muscles and joints in the cervical spine and shoulder girdle areas, are also employed.

Patient education—and reassurance—is the third aspect of the physical therapist's treatment program. Neuromuscular reeducation encourages a more relaxed position of the mandible. Exercises can aid in muscle relaxation, and the patient learns how to sleep, sit, and stand so as to avoid muscle strain to the cervical spine and thereby relieve symptoms stemming from that area.

Patients experiencing symptoms in the head, face, jaw, neck, and shoulder areas, with or without a TMJ disorder and masticatory muscle involvement, may therefore benefit from an evaluation for cervical spine dysfunction by a physical therapist.

Current thinking on TMJ disorders

Since the 1950s, when the "TMJ syndrome" became generally recognized, general dentists, oral and maxillofacial surgeons, orthodontists, anatomists, physiatrists, physical therapists, and orthopedists have debated the possible causes of disorders of the temporomandibular joint (TMJ). Questions frequently addressed include the following: Does the problem begin with masticatory muscle spasm or joint derangement? Does malocclusion of the teeth play a role? And what part—if any—do posture and derangement of the cervical spine play?

Laszlo Schwartz, DDS, and his coworkers at Columbia University first proposed in 1959 that psychological stress and anxiety resulted in masticatory muscle spasm. Since then, several other workers have modified Schwartz's ideas. Among them, Daniel Laskin, DDS, has become a leading spokesperson and has gone a step further. He proposes that occlusal abnormalities in most patients are *not* the cause of muscle spasm.

Meanwhile, the idea that occlusal disharmony is the primary cause of temporomandibular disorders has fallen into disfavor. Much of the evidence in its support has been interpreted as anecdotal. Many maintain that the relief occlusal therapy frequently affords is evidence for an occlusal component in the disorder, but a causal role for such a component has not been confirmed.

Another proposal concerning temporomandibular disorders, based on clinical data, is that children who have so-called dual bites may be more susceptible to temporomandibular disorders than adults. Such children can chew with their jaws in protruded, middle, or retruded positions.

Ongoing work by John Rugh, PhD, a psychologist, suggests that about 10% of patients with masticatory muscle disorders suffer from a sleep disorder that appears unrelated to anxiety. His work indicates that some people sleep well and grind their teeth, remembering neither their dreams nor bruxism. When sedated, their sleep is deeper and they grind their teeth more. In contrast, people whose teeth grinding is stress-related usually can recall their dreams after they wake, and they appear to grind their teeth less when sedated.

To evaluate the TMJ itself, begin by palpating over the outside of the joint. You are likely to elicit pain if there is synovitis, arthritis, or a structural defect such as a damaged ligament of the articular disk (meniscus).

Next, stand behind the patient, and, with your palms turned forward, gently insert your little fingers into both ear canals. The patient's mouth should initially be open; with your fingertips pressed gently forward you can feel the condyles translate on opening and pivot in the fossae as the mouth closes and opens again. A finding of tenderness at the head of the condyle suggests an organic disorder, displacement of the disk, inflammation of the disk ligaments associated with joint laxity, or inflammation of the joint capsule.

With a stethoscope, listen to the joint as the patient opens and closes his or her mouth. Joint noise, when it becomes present with pain, can indicate derangement of the disk, condyle, or both. Crepitus commonly indicates an organic disorder, such as osteoarthritis (OA) or rheumatoid arthritis (RA). Joint noise without pain or restricted motion is not a symptom of TMJ, especially in a patient who is asymptomatic and has no dysfunction.

Observe the patient opening and closing the mouth, and note any gross asymmetry in jaw motion. If you see an S-curve or C-curve deviation in movement, increase your suspicion of involvement of the TMJ proper, including even the possibility of congenital anomalies.

Also check for facial asymmetry. Pronounced asymmetry can indicate bony tumors, fractures, abscessed or impacted teeth, and disease of the parotid gland. Hypertrophy of the masseter or temporalis may be the result of teeth grinding or jaw clench-

ing, but this too may be congenital.

Given the large proportion of patients whose temporomandibular disorders are caused entirely by muscle spasm and fatigue, it is reasonable to assume provisionally that the patient with evident TMJ dysfunction and no signs or symptoms of organic or structural problems has a disorder of the masticatory muscles. If the patient does not respond to treatment for muscle spasm and fatigue, you can go on to explore the possibilities of joint disease and structural problems—diagnoses that are more difficult to establish.

EXPRESS STOP

Referred pain: The diagnosis of temporomandibular disorders can be complicated by referred pain. Check for tooth decay, pulpitis, cementitis, periapical abscess, impaction, and occlusal trauma by percussing teeth, applying heat and cold, or both. Sinusitis, masticatory muscle spasm, an ear disorder, migraine, temporal arteritis, or (rarely) glaucoma may be responsible for pain in the jaw or cheek.

Look for disorders that can mimic temporomandibular pain—and detect temporomandibular pain that presents like other disorders—before narrowing your diagnosis or consulting with a general dentist. Examine teeth, paranasal sinuses, eyes, ears, and head and neck muscles and vasculature. Keep in mind that sites of referred pain are typically marked by hyperesthesia and diffuse tenderness. Referred pain from a tooth usually occurs first in tissue innervated by the same division of the trigeminal (fifth cranial) nerve that connects to the tooth—the maxillary or mandibular division—but pain can spread to tissue innervated by other divisions. For example, referred pain from a disorder in a lower molar is likely to be felt initially along the mandibular division and subsequently along the maxillary or ophthalmic divisions.

In determining whether the source of referred pain is from a nontemporomandibular disorder, start by examining the teeth for abnormalities. If you find or suspect any of the following, the patient should be examined by a dentist:

- *Tooth decay* Direct and referred pain from dental caries is detectable by probing and percussing all surfaces of teeth you suspect are afflicted. The decaying tooth may be sensitive to pressure, heat, cold, or sweetened foods and drinks.

- *Pulpitis* In early stages of pulpitis, heat and cold applied to an afflicted tooth will elicit pain. When you can produce pain by applying heat and relieve it by applying cold, the pulp is usually moribund. Rarely, these stimuli cause discomfort at the referred pain site.

- *Cementitis* When an infection of the dental pulp spreads through the apical foramen, it may infect the cementum. The tooth is no longer sensitive to thermal change but is tender to percussion. Often, however, you will find that both types of tenderness are present.

- *Periapical abscess* Cementitis can develop into periapical abscess. The patient may present with a constant aching or throbbing pain, gingival swelling, and, sometimes, with fever. The tooth and its gingiva may be exquisitely sensitive to pressure.

- *Impaction of a tooth* Like abscess, the infection of an unerupted or impacted tooth can produce a constant pain and a sensation of pressure in a well-defined area. Look for osseous and soft tissue swelling on the afflicted side; in the mandible, swelling produces restricted or painful jaw motion. If you suspect impaction with infection or abscess, refer the patient to a dentist for X-ray examination. Most often pain and tenderness in and around the molars signal impac-

Minipress®

(prazosin HCl) Capsules 1mg, 2 mg, 5 mg

Brief Summary

MINIPRESS (prazosin hydrochloride) CAPSULES

For Oral Use

INDICATIONS AND USAGE: MINIPRESS (prazosin hydrochloride) is indicated in the treatment of hypertension. It is mild to moderate in activity and can be used as the initial agent or in a general treatment program in conjunction with a diuretic and/or other antihypertensive drugs as needed.

CONTRAINDICATIONS: None known.

WARNINGS: MINIPRESS may cause syncope with sudden loss of consciousness. In most cases this is believed to be due to an excessive postural hypotensive effect, although occasionally the syncope episode has been preceded by a bout of severe tachycardia with heart rates of 120-160 beats per minute. Syncope episodes have usually occurred within 30 to 90 minutes of the initial dose of the drug; occasionally they have been reported in association with rapid dosage increases or the introduction of another antihypertensive drug into the regimen of a patient taking high doses of MINIPRESS. The incidence of syncope episodes is approximately 1% in patients given an initial dose of 2 mg or greater. Clinical trials conducted during the investigational phase of this drug suggest that syncope episodes can be minimized by limiting the initial dose of the drug to 1 mg, by subsequently increasing the dosage slowly, and by introducing any additional antihypertensive drugs into the patient's regimen with caution (see DOSAGE AND ADMINISTRATION). Hypotension may develop in patients given MINIPRESS who are also receiving a beta-blocker such as propranolol.

If syncope occurs, the patient should be placed in the recumbent position and treated supportively as necessary. This adverse effect is self-limiting and in most cases does not recur after the initial period of therapy or during subsequent dose titration. The patient should also be cautioned to avoid situations where injury could result should syncope occur during the initiation of MINIPRESS therapy.

PRECAUTIONS: Information for Patients: Dizziness or drowsiness may occur after the first dose of this medicine. Avoid driving or performing hazardous tasks for the first 24 hours after taking this medicine or when the dose is increased. Dizziness, lightheadedness or fainting may occur, especially when rising from a lying or sitting position. Getting up slowly may help lessen the problem. These effects may also occur if you drink alcohol, stand for long periods of time, exercise, or if the weather is hot. While taking MINIPRESS, be careful in the amount of alcohol you drink. Also, use extra care during exercise or hot weather, or if standing for long periods of time. Check with your physician if you have any questions.

Drug Interactions: MINIPRESS has been administered without any adverse drug interaction in limited clinical experience to date with the following: (1) cardiac glycosides—digitalis and digoxin; (2) hypoglycemics—insulin, chlorpropamide, phenformin, tolazamide, and tolbutamide; (3) tranquilizers and sedatives—chloriazepoxide, diazepam, and phenobarbital; (4) antihypertensive agents—allopurinol, cimetidine, and probenecid; (5) antiarrhythmics—procainamide, propranolol (see WARNINGS however), and quinidine; and (6) analgesics, antipyretics and anti-inflammatories—propoxyphene, aspirin, indomethacin, and phenylbutazone.

Addition of a diuretic or other antihypertensive agent to MINIPRESS has been shown to cause an additive hypotensive effect.

Drug/Laboratory Test Interactions: False positive results may occur in screening tests for pheochromocytoma in patients who are being treated with prazosin. If an elevated VMA is found, prazosin should be discontinued and the patient retested after a month.

Laboratory Tests: In clinical studies in which lipid profiles were followed, there were generally no adverse changes noted between pre- and post-treatment lipid levels.

Carcinogenesis, Mutagenesis, Impairment of Fertility: No carcinogenic potential was demonstrated in an 18 month study in rats with MINIPRESS (prazosin hydrochloride) at dose levels more than 225 times the usual maximum recommended human dose of 20 mg per day. MINIPRESS was not mutagenic in *in vivo* genetic toxicology studies. In a fertility and general reproductive performance study in rats, both males and females, treated with 75 mg/kg (225 times the usual maximum recommended human dose), demonstrated decreased fertility while those treated with 25 mg/kg (75 times the usual maximum recommended human dose) did not.

In chronic studies (one year or more) of MINIPRESS in rats and dogs, testicular changes consisting of atrophy and necrosis occurred at 25 mg/kg/day (75 times the usual maximum recommended human dose). No testicular changes were seen in rats or dogs at 10 mg/kg/day (30 times the usual maximum recommended human dose). In view of the testicular changes observed in animals, 105 patients on long term MINIPRESS therapy were monitored for 17-ketosteroid excretion and no changes indicating a drug effect were observed. In addition, 27 males on MINIPRESS for up to 51 months did not have changes in sperm morphology suggestive of drug effect.

Usage in Pregnancy: Pregnancy Category C. There are no adequate and well controlled studies which establish the safety of MINIPRESS (prazosin HCl) in pregnant women. MINIPRESS should be used during pregnancy only if the potential benefit justifies the potential risk to the mother and fetus.

Nursing Mothers: MINIPRESS has been shown to be excreted in small amounts in human milk. Caution should be exercised when MINIPRESS is administered to a nursing woman.

Usage in Children: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS: Clinical trials were conducted on more than 900 patients. During these trials and subsequent marketing experience, the most frequent reactions associated with MINIPRESS therapy are: dizziness 10.3%, headache 7.8%, drowsiness 7.6%, lack of energy 6.9%, weakness 6.5%, palpitations 5.3%, and nausea 4.9%. In most instances side effects have disappeared with continued therapy or have been tolerated with no decrease in dose of drug.

Less frequent adverse reactions which are reported to occur in 1-4% of patients are: **Gastrointestinal:** vomiting, diarrhea, constipation; **Cardiovascular:** edema, orthostatic hypotension, dyspnea, syncope; **Central Nervous System:** vertigo, depression, nervousness; **Dermatologic:** rash; **Genitourinary:** urinary frequency; **EENT:** blurred vision, reddened sclera, epistaxis, dry mouth, nasal congestion. In addition, fewer than 1% of patients have reported the following (in some instances, exact causal relationships have not been established):

Gastrointestinal: abdominal discomfort and/or pain, liver function abnormalities, pancreatitis; **Cardiovascular:** tachycardia; **Central Nervous System:** paresthesia, hallucinations; **Dermatologic:** pruritus, alopecia, lichen planus; **Genitourinary:** incontinence, impotence, priapism; **EENT:** tinnitus; **Other:** diaphoresis, fever.

Single reports of pigmentary mottling and serous retinopathy, and a few reports of cataract development or disappearance have been reported.

OVERDOSAGE: Should overdose lead to hypotension, support of the cardiovascular system is of first importance. Restoration of blood pressure and normalization of heart rate may be accomplished by keeping the patient in the supine position. If this measure is inadequate, shock should first be treated with volume expanders. If necessary, vasopressors should then be used. Renal function should be monitored and supported as needed. Laboratory data indicate MINIPRESS is not dialyzable because it is protein bound.

DOSAGE AND ADMINISTRATION: The dose of MINIPRESS should be adjusted according to individual blood pressure response.

Initial Dose: 1 mg two or three times a day.

Maintenance Dose: Dosage may be slowly increased to a total daily dose of 20 mg given in divided doses. The therapeutic doses most commonly employed have ranged from 6 mg to 15 mg daily given in divided doses. Doses higher than 20 mg usually do not increase efficacy; however, a few patients may benefit from further increases up to a daily dose of 40 mg given in divided doses. After initial titration some patients can be maintained adequately on a twice daily dosage regimen.

Use With Other Drugs: When adding a diuretic or other antihypertensive agent, the dose of MINIPRESS should be reduced to 1 mg or 2 mg three times a day and retitration then carried out.

Revised November 1986

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tion; abscess can affect any tooth. A tooth that is impacted and not infected produces a more diffuse, radiating pain, usually with less swelling.

● **Occlusal trauma** A tooth that is continually traumatized at one point on its occlusal surface can traumatize the socket at a single, corresponding point. To find the traumatized tooth and the point of occlusion on the tooth, look for obvious signs of load-bearing. These include wear facets and areas flattened by overload, gingival recession and inflammation, jagged or fractured cusps, and molar cusps without wear (which indicate that other teeth are bearing the load). To elicit pain, percuss the traumatized tooth at the point of occlusion and at precisely the angle at which the tooth is normally stressed.

Pain from other areas of the head can also cloud the diagnosis. Referred pain may be associated with:

● **Sinusitis** Check for acute frontal sinusitis when you find a history of postnasal drip or respiratory allergies. Pain from sinusitis can be referred to the jaw, cheek, or frontal areas. Unlike pain from temporomandibular disorders, the pain of sinusitis usually increases when the barometric pressure falls, when the patient bends over, or when you press on the interior and lateral walls of the sinuses.

● **Muscle spasm** Just as pain may be referred to the temporomandibular area, the reverse may be true. Statistically significant relationships have been found between headaches and external pterygoid muscle tenderness; between aching, stiffness, or tingling in the ear and internal pterygoid muscle tenderness; and between sinus pain and tenderness in temporal and internal pterygoid muscles.

● **An ear disorder** Pain in the jaw, the back of the neck, or the temples may stem from an

ear disorder. Check the ear canals and tympanic membranes for signs of inflammation or infection.

- *Migraine* Classic migraine and cluster headaches can cause pain in the jaw and cheek, although it is usually felt in the orbital, parietal, and temporal regions. Expect a description of either throbbing pain or steady ache that lasts from a few hours to a day or two.*

- *Temporal arteritis* This disorder affects men of more than 50 almost exclusively; it is marked by the distinct onset of lateral head pain and by tenderness or a burning sensation over the lateral neck muscles and blood vessels. The pain is constant and becomes progressively more severe, and occasionally is referred to the jaw and back of the neck.

- *Glaucoma* Glaucoma and other eye disorders may produce pain in jaw, cheek, or teeth.

*See "The changing picture in migraine," *Patient Care*, March 15, 1988, page 39.

EXPRESS STOP

Joint inflammation: Synovitis, rheumatoid arthritis, and osteoarthritis are the most common inflammatory disorders affecting the TMJ. Synovitis is associated with tenderness over the joint, and—when accompanying RA—joint stiffness that abates with jaw movement. It responds to corticosteroid injections. Discomfort from OA does not lessen with movement. Imaging procedures usually reveal erosion of the TMJ structures with arthritis.

Of the common inflammatory disorders of the TMJ, synovitis tends to produce the mildest symptoms. This condition is often associated with chronic trauma to the joint and with certain systemic diseases. Most commonly, it is a direct complication of OA or of adult RA. Occasionally it accompanies juvenile RA. Mumps, measles, and infectious mononucleosis can also result in TMJ synovitis.

Although synovitis is difficult to diagnose,

suspect it when you find tenderness on palpation of the TMJ posteriorly (from the ear canal) without evidence of arthritis. You will also find tenderness on palpation of the lateral joint, just anterior to the tragus, where there is a slight depression.

If you suspect an arthritic inflammation, inquire about the time of day when the jaw dysfunction is worst. If there is accompanying synovitis and the jaw stiffness abates in less than a half hour after the patient arises, look for evidence of RA. Keep in mind, however, that RA usually affects the TMJ only after involvement of the fingers, wrists, and weight-bearing joints, and only in about half of all patients with RA.

If the patient has no TMJ stiffness on arising but has increasing stiffness with use, suspect OA in the absence of evidence for more benign sources of pain. In the early stages of OA, stiffness disappears with rest but reappears as the patient tires, and often at the end of the day. When present, the joint noise is usually grinding rather than clicking.

For synovitis that does not appear to be associated with arthritis, first try conservative therapies for several weeks. If the trouble is uncomplicated synovitis, the patient should find relief with application of moist heat to the TMJ, a soft diet, use of a bite plate or night guard, and, when masticatory muscle spasm is present, isotonic exercise to break the spasm. If these therapies are unsuccessful, consider injecting the joint with corticosteroids.

Several imaging procedures are used for diagnosis. The options include panoramic and transcranial X-rays, computed tomography (CT) scans, and magnetic resonance imaging (MRI) or arthrography to assess disk position.

In severe synovitis or arthritis, X-rays

XANAX® Tablets
(alprazolam) ©

INDICATIONS AND USAGE

Anxiety disorders, short-term relief of the symptoms of anxiety, and anxiety associated with depression. Anxiety or tension associated with the stress of everyday life usually does not require an anxiolytic. Effectiveness for more than four months has not been established; periodically reassess the usefulness of the drug for the individual patient.

CONTRAINDICATIONS

Sensitivity to XANAX or other benzodiazepines, and in acute narrow angle glaucoma.

WARNINGS

Benzodiazepines can cause fetal harm in pregnant women, hence women who may become pregnant should be warned. Avoid during the first trimester. Withdrawal seizures have been reported upon rapid dose reduction or abrupt discontinuation, thus reduce dose gradually. (See Drug Abuse and Dependence and Dosage and Administration.)

PRECAUTIONS

General: If XANAX is combined with other psychotropics or anticonvulsants, consider drug potentiation. (See Drug Interactions). Use the usual precautions in patients with renal or hepatic impairment and regarding prescription size in depressed and suicidal patients. In elderly and debilitated patients, use the lowest possible dose. (See Dosage and Administration.) Hypomania and mania has been reported in depressed patients.

Information for Patients: Alert patients about: (a) consumption of alcohol and drugs, (b) possible fetal abnormalities, (c) operating machinery or driving, (d) not increasing dose of the drug due to risk of dependence, (e) not stopping the drug abruptly.

Laboratory Tests: Not ordinarily required in otherwise healthy patients. **Drug Interactions:** Additive CNS depressant effects with other psychotropics, anticonvulsants, antihistamines, ethanol and other CNS depressants. Plasma levels of imipramine and desipramine are increased. Pharmacokinetic interactions with other drugs have been reported. Cimetidine can delay clearance of benzodiazepines. **Drug/Laboratory Test Interactions:** No consistent pattern for a drug or test. **Carcinogenesis, Mutagenesis, Impairment of Fertility:** No carcinogenic potential or impairment of fertility in rats.

Pregnancy: See Warnings. **Nonteratogenic Effects:** The child born of a mother on benzodiazepines may be at some risk for withdrawal symptoms, neonatal flaccidity and respiratory problems. **Labor and Delivery:** No established use. **Nursing Mothers:** Benzodiazepines are excreted in human milk. Women on XANAX should not nurse. **Pediatric Use:** Safety and effectiveness in children below the age of 18 have not been established.

ADVERSE REACTIONS

Side effects are generally observed at the beginning of therapy and usually disappear with continued medication. In the usual patient, the most frequent side effects are likely to be an extension of the pharmacologic activity of XANAX, e.g., drowsiness or lightheadedness.

Central nervous system: Drowsiness, lightheadedness, depression, headache, confusion, insomnia, nervousness, syncope, dizziness, akathisia, and tiredness/sleepiness. **Gastrointestinal:** Dry mouth, constipation, diarrhea, nausea/vomiting, and increased salivation. **Cardiovascular:** Tachycardia/palpitations, and hypotension. **Sensory:** Blurred vision. **Musculoskeletal:** Rigidity and tremor. **Cutaneous:** Dermatitis/allergy. **Other side effects:** Nasal congestion, weight gain, and weight loss.

Withdrawal seizures with rapid decrease or abrupt discontinuation. (See Warnings.)

The following adverse events have been reported with benzodiazepines: dystonia, irritability, concentration difficulties, anorexia, transient amnesia or memory impairment, loss of coordination, fatigue, seizures, sedation, slurred speech, jaundice, musculoskeletal weakness, pruritus, diplopia, dysarthria, changes in libido, menstrual irregularities, incontinence, and urinary retention.

Paradoxical reactions such as stimulation, agitation, rage, increased muscle spasticity, sleep disturbances, and hallucinations may occur. Should these occur, discontinue the drug.

During prolonged treatment, periodic blood counts, urinalysis, and blood chemistry analysis are advisable. Minor EEG changes, of unknown significance, have been observed.

Liver enzyme elevations, gynecomastia and galactorrhea have been reported but no causal relationship was established.

DRUG ABUSE AND DEPENDENCE

Physical and Psychological Dependence: Withdrawal symptoms including seizures have occurred following abrupt discontinuance or rapid dose reduction of benzodiazepines. (See Warnings). Dosage should be gradually tapered under close supervision. Patients with a history of seizures or epilepsy should not be abruptly withdrawn from XANAX. Addiction-prone individuals should be under careful surveillance. **Controlled Substance Class:** XANAX is a controlled substance and has been assigned to schedule IV.

OVERDOSAGE

Manifestations include somnolence, confusion, impaired coordination, diminished reflexes and coma. No delayed reactions have been reported.

DOSAGE AND ADMINISTRATION

Dosage should be individualized.

The usual starting dose is 0.25 to 0.5 mg, t.i.d. Maximum total daily dose is 4 mg. In the elderly or debilitated, the usual starting dose is 0.25 mg, two or three times daily. Reduce dosage gradually when terminating therapy, by no more than 0.5 mg every three days.

HOW SUPPLIED

XANAX Tablets are available as 0.25 mg, 0.5 mg, and 1 mg tablets.

CAUTION:

FEDERAL LAW PROHIBITS DISPENSING WITHOUT PRESCRIPTION.

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April 1988

TMJ disorders

and scans reveal some erosion of the temporal bone, the articular tubercle, and the condyle. In milder cases of synovitis, imaging procedures are not always diagnostic. A CT scan may show a narrowing of the joint space, increased radiopacity, osteophytes, and condensation of the subchondral bone. CT is the study of choice for bony abnormalities of the TMJs.

X-ray studies show the same kind of structural erosion in OA as in RA, but only after about 30% of the bone has been destroyed. In patients with more advanced RA, look for an open anterior bite and overbite as evidence of condylar and temporal bone recession.

EXPRESS STOP

Structural joint disorders: If you rule out an organic TMJ disorder, suspect a displacement of the articular disk (meniscus), the most common structural disorder. This is nearly always associated with an opening and closing click and tenderness over the TMJ. Coordinate the radiographic workup with a dentist. Indications for arthrography and magnetic resonance imaging are growing, but arthrography is the preferred technique for visualizing perforations of the disk.

If failure of noninvasive therapy for arthritis or synovitis has ruled out organic disorders but the patient still has pain, joint noise, limited range of jaw motion or a locking jaw, suspect a derangement of the articular disk.

Anterior displacement of the disk on jaw opening is the most common structural disorder (see Figure 2, page 187). Second is subluxation of the TMJ, which you can sometimes feel by palpating over the joint on opening of the mouth.

Displacement of the disk is nearly always associated with tenderness over the TMJ and clicking, crepitus, or locking of the joint. It is thought that the disk can become dis-

Text continues on page 187.

placed by one or a combination of factors that stretch its medial and lateral collateral ligaments: chronic microtrauma from masticatory muscle spasms, acute trauma (such as a blow to the jaw), malocclusion that stresses the joint, stressful oral habits, or congenital abnormality of joint structures.

To help confirm your suspicion of a structural disorder, look to radiographic studies. While such studies will not help in identifying disk problems, they can show the condition of the bony surfaces. You can coordinate the radiographic workup with—or delegate it to—a dentist. The American Dental Association guidelines suggest panoramic, tomo-

graphic, or transcranial radiographic studies initially, and CT scans for patients in whom the first procedures indicate possible TMJ abnormality. CT scanning costs about 40% more than a conventional X-ray series, but it exposes the patient to about half the radiation.

Since the mid-1970s, the use of arthrography to diagnose the condition and reveal the position of the disk has grown in popularity and is now the most reliable technique. The procedure takes about a half hour and involves an injection of local anesthetic followed by injection of water-soluble, iodinated radiographic contrast material. In all, up

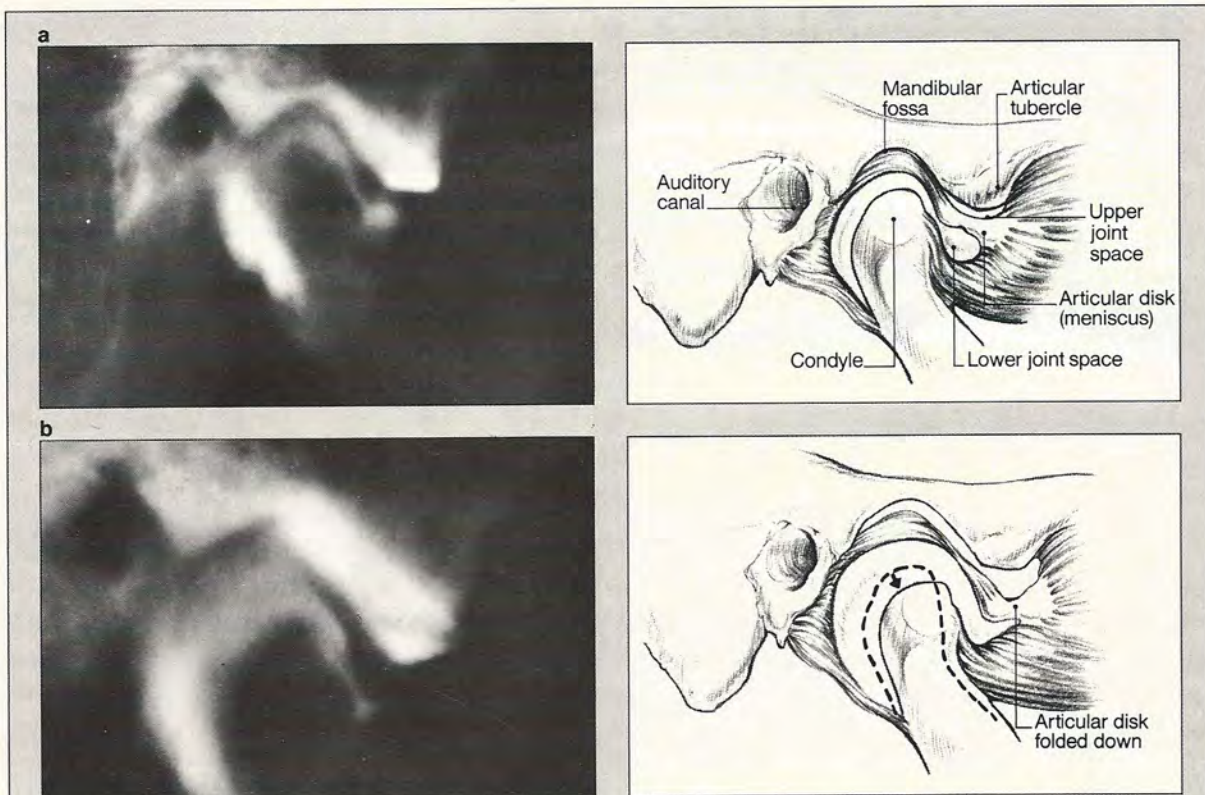


FIGURE 2: **Temporomandibular joint arthrograms** can reveal the condition and position of the joint's soft tissue. Here, arthrograms show anterior displacement of the articular disk (meniscus) with the mouth closed (a) and par-

tially open (b). The artist's renderings above depict the action of the condyle on opening, as it folds the articular disk downward and causes it to be displaced even further anteriorly.

to 1.3 mL of fluid is injected into the joint space. The procedure may alter the patient's bite for a day or two and cause mild to moderate discomfort for a few days and tenderness for up to two weeks.

Over the past several years, the development of small joint arthroscopes has enabled oral surgeons to visualize the joint directly and has increased their ability to diagnose arthrosis, dislocation, and perforation. MRI has also become increasingly valuable in de-

termining disk position, eliminating the need for dye injection. Arthrography remains the procedure of choice for disk perforations, however.

For some patients, such as the elderly, the frail, or those who have suffered recent myocardial infarctions, oral and maxillofacial surgeons often choose CT scans rather than arthrography. Generally, CT scans cost about the same as arthrography, while MRI is more expensive than either. □

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