Chronic pain is a treatable problem affecting an estimated 50% of community-dwelling older adults and over 75% of long-term care facility residents. Because of its numerous consequences, including impaired physical function, depression, anxiety, disrupted sleep and appetite, and excessive use of health care resources, chronic pain should be treated aggressively. Chronic pain may accompany both malignant and nonmalignant disorders. This article focuses on the evaluation and management of chronic nonmalignant pain.

DEFINITION AND PATHOGENESIS OF CHRONIC NONMALIGNANT PAIN

Chronic pain is defined as pain that persists beyond the expected time of healing, or more than 3 to 6 months. While a number of disorders may cause chronic pain, generally speaking there are 2 types of conditions that underlie its pathogenesis—nociceptive and neuropathic. The nervous system is responsible for the perception of pain. Nociceptive pain is associated with tissue damage and a normal nervous system (eg, pain associated with osteoarthritis), while neuropathic pain is associated with physiological nervous system dysfunction (eg, diabetic neuropathy, postherpetic neuralgia). Not infrequently, these 2 types of pain coexist. The discussion that follows focuses on the most common chronic nonmalignant pain disorders—myofascial pain, osteoarthritis, chronic low back pain, fibromyalgia syndrome, and peripheral neuropathy.

GENERAL APPROACH TO ASSESSMENT AND TREATMENT

To afford an optimal response to therapy, the practitioner must keep 3 general principles in mind, and communicate them to their older patients in order to establish reasonable treatment expectations:
• Chronic pain is a syndrome with many potential contrib-
utors, all of which require treatment to afford an optimal
clinical outcome.
• Chronic pain is treatable but not curable; improvement is
the rule, not the exception.
• It is often possible to improve functional ability to a
greater extent than the severity of pain is reduced.

EVALUATION AND TREATMENT OF SPECIFIC CHRONIC PAIN DISORDERS

Myofascial Pain

Definition and Pathogenesis. Because myofascial pain
(MP) occurs in the majority of patients with chronic pain
disorders, learning how to recognize, diagnose, and treat
this condition is essential. Broadly defined, myofascial pain
is pain in physiologically abnormal muscles. The pathogno-
monic features of this disorder are taut bands and trigger
points elicited by firm palpation.1 Patients may present with
a range of pain symptoms such as aching, soreness, stab-
bining, or burning. The descriptors used by patients suffering
from myofascial pain may be similar to those of neuropathic
pain, and sometimes these disorders occur together; thus,
hands-on assessment is critical to accurate diagnosis. Myo-
fascial pain is commonly associated with degenerative
changes of the axial skeleton and other factors (eg, spinal
deformity, such as scoliosis or kyphosis) that may irritate
nerves. As a result, the musculature supplied by these
nerves does not receive adequate trophic factors and it, in
turn, becomes irritable.2

Evaluation. Careful physical examination is required to
diagnose myofascial pain. Historical clues include: relief of
pain by exerting mild pressure over the area (eg, massaging
the affected area), engaging in low level activity, or applica-
tion of heat or vapocoolant spray; and worsening of pain
with excessive activity. Physical examination performed by
firmly palpating involved muscles may reveal both latent
and active trigger points.1 Palpation of latent trigger points
may cause tenderness, but may not reproduce the radiation
of pain that occurs when active trigger points are palpated.
Taut bands can be identified by firmly palpating the muscle
along the same plane but in the axis that runs perpendicular
to the direction of the muscle fibers. The examiner may need
to apply pressure in this area for 10-15 seconds before the
characteristic pain pattern is reproduced.3 Local twitch re-
sponses, due to sharp contractions of the taut band (not the
entire muscle) initiated by an intense physical stimulus such
as needle insertion or plucking of the band may also be
noted.1 Sensory abnormalities include hypersensitivity and
allodynia (ie, pain caused by a stimulus that does not nor-
mally provoke pain) of the trigger points. Autonomic phe-
nomena, such as temperature change, piloerection, and
sweating, may be present in the affected area but are not
required for diagnosis.

Treatment. The first step in the treatment of MP is identi-
fication and modification of perpetuating factors, such as
posture, sleeping position, and body mechanics during ac-
tivity. Early involvement of a physical therapist skilled in
the treatment of myofascial pain disorders is crucial. Phys-
ical therapy techniques commonly employed include gentle
sustained stretching of involved muscles to reduce focal
contractions and inactivate trigger points, sometimes in
combination with a vapocoolant spray; exercise programs
that include graded stretching and strengthening; and teach-
ing of flare self-management techniques.4 In order to facil-
itate physical therapy-directed rehabilitation, trigger point
deactivation may be useful. This can be accomplished with
either “dry needling” (ie, deactivating the trigger point with
a needle alone) or injection of the trigger point with an
anesthetic or steroid. Either technique may be effective as
long as a local twitch response (ie, transient contraction of
a group of tense muscle fibers [taut band] that traverse a
trigger point) is achieved.5 This author also has had success
with a variety of acupuncture techniques. In refractory
cases, referral to a multidisciplinary pain center may be
helpful.

Osteoarthritis and Chronic Low Back Pain

Definition and Pathogenesis. Osteoarthritis (OA) is com-
monly referred to as degenerative arthritis, but this is some-
what of a misnomer. Accumulating evidence indicates that
inflammation plays an important role in the pathogenesis of
OA. While virtually all older adults have radiographic evi-
dence of OA, most of these individuals are asymptomatic,
and the severity of OA on radiograph correlates poorly with
symptom severity. In the case of chronic low back pain
(CLBP), degenerative disease of the discs and facets is only
one factor that contributes to pain; thus, its evaluation and
treatment should be approached comprehensively.

Evaluation. Proper treatment of appendicular OA (eg,
knees, hips) and CLBP requires a detailed evaluation. Sev-
eral rheumatologic disorders can cause generalized pain and
may be confused with OA. Table 1 summarizes clinical
features of common rheumatologic conditions.

The first step in evaluating low back pain is the identifi-
cation of “red flags” (eg, fever, unintentional weight loss,
sudden change in pain quality) indicative of a serious un-
derlying disorder such as malignancy or spinal infection
(Figure 1). These should be screened with a targeted history
and physical examination. If a serious condition is sus-
pected, diagnostic imaging should be pursued promptly.
Conversely, in the absence of red flags, imaging is rarely
indicated.6 Practitioners receive little training in musculo-
skeletal assessment, thus, basic and advanced imaging (ie,
radiographs and magnetic resonance imaging [MRI/com-
puterized tomography [CT], respectively) is often relied
upon as an initial diagnostic test. This approach, however, is
likely to lead to incorrect management because degenerative
abnormalities are a nonspecific finding in older adults with
<table>
<thead>
<tr>
<th>Disorder</th>
<th>History</th>
<th>Location of Pain</th>
<th>Physical Examination</th>
<th>Extrasynovial Disease</th>
<th>Other Diagnostic Features/Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Osteoarthritis</td>
<td>Generally short-lived, eg, &lt;30 minutes</td>
<td>Weight-bearing appendicular joints, cervical and lumbar spine, DIPs, PIPs, 1st CMC, MCP and wrist involvement go against OA.</td>
<td>Absent or mild</td>
<td>None related to arthritis itself</td>
<td>Because OA is ubiquitous in older adults, radiographs should be used to rule out other disorders, not to diagnose OA. Chondrocalcinosis may be asymptomatic. Identification of intracellular CPPD crystals offers a definitive diagnosis in acute flares. Acute and chronic forms occur.</td>
</tr>
<tr>
<td>Pseudogout</td>
<td>Pseudo-rheumatoid pattern may be associated with prolonged AM stiffness</td>
<td>Knee and wrist are most common locations; disease is often symmetrical</td>
<td>Acute flares are intensely inflammatory</td>
<td>Chondrocalcinosis on radiographs; eye deposits, bursitis, tendonitis, carpal and cubital tunnel syndromes may occur. Tophaceous soft tissue deposits uncommon.</td>
<td>Hyperuricemia may be asymptomatic. Serum uric acid cannot diagnose gout. Identification of intracellular monosodium urate monohydrate crystals offers a definitive diagnosis in acute flares. Patients may be seronegative. If disease is suspected, patient should promptly be referred to a rheumatologist to retard disease progression.</td>
</tr>
<tr>
<td>Gout</td>
<td>Pseudo-rheumatoid pattern may be associated with prolonged AM stiffness</td>
<td>Joints of the lower extremities are most often involved, especially 1st MTP; disease is typically asymmetrical</td>
<td>Acute flares are intensely inflammatory</td>
<td>Tophi may deposit in soft tissues.</td>
<td></td>
</tr>
<tr>
<td>Rheumatoid Arthritis</td>
<td>Prolonged, eg, &gt;30 minutes. Duration of stiffness is used as one parameter of disease activity</td>
<td>Any synovial joint. The lumbar spine is typically spared.</td>
<td>Present.</td>
<td>Not uncommon; rheumatoid nodules can develop in soft tissues. Many other possible manifestations including anemia, vasculitis (skin lesions, peripheral neuropathy, pericarditis, visceral arteritis, palpable purpura), pulmonary disease, etc.</td>
<td></td>
</tr>
<tr>
<td>Systemic Lupus Erythematosus</td>
<td>Not a prominent feature.</td>
<td>Depends upon tissues involved—may or may not be limited to joints. Comorbid fibromyalgia is not uncommon.</td>
<td>Generally absent; arthralgias are more common than arthritis.</td>
<td>Common—eg, anemia, skin rash, pleuritis, peritonitis, pericarditis, nephritis, meningitis, etc.</td>
<td>Anyone with suspected SLE should promptly be referred to a rheumatologist.</td>
</tr>
<tr>
<td>Fibromyalgia Syndrome</td>
<td>Generally short-lived, eg, &lt;30 minutes</td>
<td>Typically diffuse. Worst symptoms often involve the axial skeleton.</td>
<td>Absent. Joints themselves are not involved, although patients experience pain in joints and soft tissues.</td>
<td>Many other disorders may coexist (Table 4)</td>
<td>Fibromyalgia syndrome is not a diagnosis of exclusion, but one based upon careful history and physical examination (see text).</td>
</tr>
<tr>
<td>Polymyalgia Rheumatica</td>
<td>May be prolonged, lasting several hours</td>
<td>Typically proximal—eg, shoulder girdle, hip girdle, neck. If headaches, jaw claudication, or prominent systemic symptoms (eg, fever), consider temporal arteritis.</td>
<td>May occur, especially in small joints of hands.</td>
<td>Occurs if comorbid temporal arteritis and relates to involvement of arteries (eg, Raynaud’s phenomenon, bruises, claudication).</td>
<td>Because the erythrocyte sedimentation is very nonspecific, this test should be used to assist with confirmation of a suspected diagnosis. Note that cases of PMR and TA with a normal ESR have been reported.</td>
</tr>
</tbody>
</table>

CMC = carpo-metacarpal joint; CPPD = calcium pyrophosphate dihydrate; DIP = distal interphalangeal joint; ESR = erythrocyte sedimentation rate; MCP = metacarpophalangeal joint; MTP = metatarsophalangeal joint; OA = osteoarthritis; PIP = proximal interphalangeal joint; PMR = polymyalgia rheumatica; SLE = systemic lupus erythematosus; TA = temporal arteritis.
back pain. Indeed, virtually 100% of older adults have degenerative pathology, whether or not they have pain. When used as a screening tool, imaging should be thought of as a way to demonstrate the absence of disease (e.g., compression fractures, metastatic bone disease, disk space infection) rather than as a way to diagnose the cause of pain.

Moderate to severe central spinal canal stenosis identified by MRI is also as common in older adults with pain as

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**Table 2** Essential Clinical History Questions for Older Adults with Chronic Low Back Pain

<table>
<thead>
<tr>
<th>Question</th>
<th>Potential Diagnostic Clue(s) Obtained</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Can you show me where your back hurts?</td>
<td>If patient places hand to right or left of midline, over sacrum rather than lumbar spine, this suggests sacroiliac joint syndrome (look for associated scoliosis, hip or knee disease, leg length discrepancy), inflammatory disorder, or sacral insufficiency fracture.</td>
</tr>
<tr>
<td>2. Does the pain get better or worse when you curl up in bed?</td>
<td>Improvement in fetal position suggests spinal stenosis. Worsening in fetal position suggests sacroiliac disease because of joint compression in this position.</td>
</tr>
<tr>
<td>3. Does the pain go into your buttocks? If “yes”: Is the pain sharp or dull?</td>
<td>Buttocks involvement can be associated with hip disease, piriformis myofascial pain (often sharp or burning), or spinal stenosis and requires contextual evaluation.</td>
</tr>
<tr>
<td>4. Do you have pain in your groin?</td>
<td>Groin pain can be associated with intrinsic hip disease, local myofascial pathology, sacroiliac joint syndrome, or an insufficiency fracture.</td>
</tr>
<tr>
<td>5. Does the pain shoot down your leg(s)? If “yes”: In what part of your leg do you feel the pain? Is the pain sharp or dull?</td>
<td>Posterior radiation is consistent with sciatica (sharp) or spinal stenosis (dull). Lateral thigh radiation suggests tensor fascia lata/iliotibial band pain (not past the knee) or gluteus minimus (past the knee “pseudo-sciatica”) myofascial pain. Lateral leg pain with paresthesias or numbness suggests L5 radiculopathy. Anterior thigh pain suggests hip disease, meralgia paresthetica, quadriceps strain with knee osteoarthritis, or L2/3/4 radiculopathy.</td>
</tr>
<tr>
<td>6. Is the pain made better or worse with walking?</td>
<td>Worsening with walking suggests spinal stenosis or vasogenic claudication. Improvement with walking suggests myofascial pathology or neuropathic pain. Prolonged walking may worsen myofascial pain. Degenerative disease may be associated with initial pain/stiffness, then improvement and worsening with excessive use.</td>
</tr>
<tr>
<td>7. Do you sometimes feel that you have pain all over?</td>
<td>Patients with fibromyalgia syndrome often have prominent axial pain, and may present with a chief complaint of severe low back pain, but in fact low back pain is just one of many sites of pain.</td>
</tr>
</tbody>
</table>

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### Table 3  Physical Examination Abnormalities in Older Adults with Low Back Pain

<table>
<thead>
<tr>
<th>Finding</th>
<th>Operational Definition</th>
<th>Examination Technique</th>
</tr>
</thead>
</table>
| Fibromyalgia tender points    | Presence of pain when approximately 4 kg of force is applied to defined tender points. | Have patient sit comfortably on examination table, arms resting in lap. Tell patient that you are going to apply pressure at several points on the body, and that you want to know if pressure on any point causes pain. Examine the following points bilaterally, using enough pressure to blanch thumbnail:  
  (1) Occiput at suboccipital muscle insertions  
  (2) Low cervical at the anterior aspects of the intertransverse spaces at C5-C7  
  (3) Trapezius, midpoint of upper border  
  (4) Supraspinatus at origins, above the scapular spine near the medial border  
  (5) 2nd rib at the 2nd costochondral junction, just lateral to the junction on the upper surfaces  
  (6) Lateral epicondyle 2 cm distal to the epicondyle  
  (7) Medial fat pad of the knee, proximal to joint line  
  (8) Greater trochanter, just posterior to the trochanteric prominence  
  (9) Gluteal at upper outer quadrant of buttocks in anterior fold of muscle |
| Functional leg length discrepancy | Pelvic asymmetry                                           | Have patient stand with both feet on floor, shoes removed. Ask him to stand with feet together, and as erect as possible. Kneel behind patient. With palms parallel to floor, and fingers extended, place lateral surface of index finger of both hands atop pelvic brim bilaterally. Eyes should be level with hands. Determine if right and left thumbs are at different heights. |
| Scoliosis (lateral/rotational) | Lateral/rotational curvature of thoracolumbar spine       | Have patient stand on floor with shoes removed. Stand behind patient. Run index finger along spinous processes (do not lift hand between vertebrae) a series of 3 times. If you do not detect scoliosis, then: Ask patient to bend forward. Determine if there is asymmetry in height of paraspinal musculature. |
| Sacroiliac joint pain         | Pain with direct palpation of sacroiliac joint or with Patrick’s test | Direct Palpation: Have patient stand on floor with shoes removed. Stand behind patient. Exert firm pressure over sacroiliac joint, first on one side, then the other. Palpate right joint with right thumb, standing to left side of patient; palpate left joint with left thumb, standing to right of patient.  
  Patrick's (FABER) Test—Have the patient lie supine on the examining table and place the foot of involved side on opposite knee. Then slowly lower the test leg in abduction toward the examining table. If patient reports pain in back (not groin, buttocks or leg), then test is positive. |
| Myofascial pain, piriformis   | Presence of pain on deep palpation of piriformis.          | Have patient lay supine on examination table. Have patient flex right hip and knee, keeping sole of foot on table. Cross bent leg over opposite leg; again place sole on table and exert mild medially directed pressure on lateral aspect of knee to put piriformis in stretch. Exert firm pressure (4 kg) over middle extent of piriformis. Repeat examination on opposite side. |
| Myofascial pain, tensor fascia lata (TFL) ± iliotibial (IT) band pain | Presence of pain on deep palpation of TFL or IT band.      | Have patient lying supine on examination table. Using thumbs of both hands, exert firm pressure (4 kg) over full extent of TFL and IT band. Repeat examination on opposite side. |
| Kyphosis                      | Deformity of thoracic spine creating forward flexed posture | Have patient stand on floor with shoes removed. Ask him to stand fully erect. Inspect posture from the side. |
| Myofascial pain of paralumbar musculature | Presence of pain on deep palpation of paralumbar musculature. | Have patient stand on floor with shoes removed. Stand behind and to left of patient and brace patient in front with left arm. Palpate full extent of right paravertebral musculature with right thumb. Exert approximately 4 kg force. Repeat, palpatating the left paravertebral musculature. |
| Vertebral body pain           | Presence of pain on firm palpation of lumbar spinal processes | Position yourself behind patient, as for examination of paravertebral musculature above. Using dominant thumb, firmly palpate spinous processes L1-L5. |
| Hip disease                   | Pain and restricted motion of hip                           | Hip Internal Rotation—Have patient lie supine on examining table with hip and knee bent to 90°. Put the hip into maximum internal rotation (should be >15°) and ask patient if he experiences pain.  
  Patrick’s Test—As above, but pain may be in groin or leg. |

in those without pain. In part for this reason, failed back surgery syndrome occurs in up to 40% of patients when treatment decisions are based on the results of advanced imaging alone. Factors that increase the risk of failed back surgery include scoliosis, hip disease, and osteoporotic compression fractures, all of which are common in older adults. Because these factors can alter gait or spinal biomechanics, their close relationship with low back pain is not surprising.

Essential elements of the history and physical examination for the older adult with CLBP are shown in Tables 2 and 3. Because low back pain is a syndrome, typically more than one abnormality will be uncovered.

**Treatment.** Treatment of appendicular OA and CLBP should be directed toward each of its individual contributors. In addition to treating the physical causes, careful attention should be paid to the many psychosocial factors that can impact pain and function, such as anxiety, depression, and fear. A stepped care approach to the treatment of appendicular OA and CLBP is shown in Figure 2. Note on the right side of Figure 2, “topical preparations, cognitive-behavioral therapy, interdisciplinary pain treatment, and complementary and alternative modalities (CAM).” These treatments should be considered alone or in combination with any of the individual step components described below. Discussion of CAM is beyond the scope of this article. Interested readers are referred to a recent review of CAM for chronic pain in older adults.

*Step 1* includes patient education, exercise, weight loss, and prescription of assistive devices (eg, canes, walkers).

*Step 2* focuses on injection therapies. Even though injections are invasive, the risk of these procedures when performed by experienced clinicians is less than that of systemic analgesics. Injections should be considered for the older adult with pain in 1 or 2 joints, eg, bilateral knee osteoarthritis. The value of corticosteroid injections for the patient with CLBP is less clear. The strongest evidence for effectiveness is in the setting of a herniated disk associated with radiculopathy. A critical review of minimally invasive procedures for the patient with low back pain is provided elsewhere. In general, injection therapies should be viewed as a tool to enhance compliance with rehabilitation efforts, which represent the mainstay of CLBP and generalized OA treatment.

*Steps 3 through 6* include a range of pharmacologic options. If regularly scheduled acetaminophen fails, nonacetylated salicylates (eg, salicylate, choline magnesium trisalicylate) should be considered because of their superior side-effect profile as compared with traditional nonsteroidal anti-inflammatory drugs (NSAIDs). Medications in Step 5 (other NSAIDs and weak opioids) include propoxyphene, codeine, hydrocodone, and tramadol. Propoxyphene is generally contraindicated in older adults because it is no more effective than placebo and retains many of the toxicities of more potent and effective opioids. Codeine is an effective antitussive but a weak analgesic. Before prescribing either weak or strong opioids, patients must be counseled about their potential side effects, and those that are preventable should be addressed. For example, opioids increase the risk of falls; therefore, physical therapy to enhance the patient’s mobility and stability is recommended before opioids are prescribed. In addition, a stimulant laxative (eg, senna) should be taken at the first sign of constipation. Detailed recommendations regarding dosing of nonopioid and opioid analgesics, precautions, and potential drug-drug interactions have been published by the American Geriatrics Society.

*Step 7* includes surgical procedures. In general, total joint replacement is considered after noninvasive or minimally invasive strategies have failed to control pain. Guidelines regarding when to pursue surgical treatment for refractory low back pain are less clear. Back surgery is elective in the vast majority of cases and rates of failed back surgery syndrome are substantial, with estimates ranging from 5% to 20%

**Figure 2** Stepped care approach to the treatment of axial and appendicular osteoarthritis.
<table>
<thead>
<tr>
<th>Medication Class</th>
<th>Medication</th>
<th>Pharmacokinetics</th>
<th>Key Drug-Drug Interactions</th>
<th>Key Drug-Disease Interactions</th>
<th>Important Adverse Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tricyclic Antidepressants (TCAs)</td>
<td>Nortriptyline Desipramine (Amitriptyline)</td>
<td>Hepatic metabolism; higher levels of active metabolites in the elderly</td>
<td>Antipsychotics, anticholinergics, selective serotonin reuptake inhibitors (SSRI), sedative-hypnotics, antiarrhythmics, MAO inhibitors, clonidine, antiretrovirals</td>
<td>Myocardial infarction, QT prolongation, AV block, bundle branch block, ileus, prostatic hypertrophy, glaucoma, seizure disorder, dementia</td>
<td>Dementia, ataxia; Arrhythmia, cardiac conduction block, orthostatic hypotension, urinary retention, constipation, cognitive impairment; adverse withdrawal events after abrupt discontinuation; death if torsades de pointes</td>
</tr>
<tr>
<td>Anticonvulsants</td>
<td>Gabapentin</td>
<td>Renal elimination; prolonged half-life with renal impairment</td>
<td>Opioids</td>
<td>Dementia, ataxia</td>
<td>Somnolence, dizziness, peripheral edema, increased appetite and weight gain, adverse withdrawal events after abrupt discontinuation</td>
</tr>
<tr>
<td>Opioids and opioid-like drugs</td>
<td>Oxycodone Morphine Tramadol</td>
<td>Hepatic metabolism and renal elimination; plasma levels may be higher in the elderly</td>
<td>Anticholinergics, sedative-hypnotics, anxiolytics, CYP2D6 inhibitors, SSRIs, TCAs, muscle relaxants</td>
<td>Ileus, chronic obstructive pulmonary disease, dementia, prostatic hypertrophy</td>
<td>Constipation, sedation, nausea/vomiting, respiratory depression, nervous system symptoms, pruritis, adverse withdrawal events after abrupt discontinuation</td>
</tr>
<tr>
<td>Topical anesthetics</td>
<td>Topical lidocaine patch 5%</td>
<td>Very little systemic absorption</td>
<td>Class I antiarrhythmics</td>
<td></td>
<td>Skin rash</td>
</tr>
</tbody>
</table>

to 40%. Thus, back surgery should be considered for clear-cut cases, such as deterioration of neurological function or a herniated disc with anatomical location correlating precisely with symptom location and failure to respond to nonsurgical management. Otherwise, back surgery should be considered cautiously. The value of minimally invasive surgical procedures for the treatment of low back pain is currently under investigation.

**Fibromyalgia Syndrome**

**Definition and Pathogenesis.** Fibromyalgia syndrome (FMS) affects 7% of women between the ages of 60 and 79 years. FMS is characterized by generalized pain and characteristic tender points on physical examination (Table 3). Most patients with FMS also suffer from morning stiffness, fatigue, and nonrestorative sleep. Once thought of as an illness based in psychopathology, it is now recognized that FMS is associated with dysregulation of the central nervous system. That is, the nervous system of the FMS patient does not process pain normally.

**Evaluation.** A careful history and physical examination is the key to FMS diagnosis. It is not a diagnosis of exclusion. A number of other disorders may coexist in patients with FMS, such as migraine headaches, depression, and sleep apnea. Table 4 lists these disorders as well as other symptoms from which patients with FMS suffer. Often, patients report pain for decades, even during childhood (eg, “growing pains”). There is increasing evidence that FMS is a heritable disorder, thus practitioners should obtain a thorough family history in the older adult with suspected FMS.

A combination of characteristic symptoms along with palpation-elicited tenderness supports a diagnosis of FMS. The presence of ≥11 of 18 characteristic tender points has been used for classification purposes (ie, distinguishing patients with FMS from those with other rheumatologic disorders), but this exact number is not required to make a diagnosis. Typically, patients with FMS have widespread tenderness (ie, pain elicited by palpation when the examiner uses enough pressure to blanch the thumbnail bed).

**Treatment.** Aerobic exercise is an important component of treatment. The symptoms that most interfere with patients’ quality of life include pain, fatigue, limited activity tolerance, and sleep disturbance. A variety of medications, such as tricyclic antidepressants, selective serotonin reuptake inhibitors, and cyclobenzaprine, may be effective in targeting these symptoms. For detailed recommendations on the treatment of fibromyalgia, the reader is referred to guidelines recently published by the American Pain Society. Patients

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**Table 6 Dosage of First-Line Medications for Neuropathic Pain**

<table>
<thead>
<tr>
<th>Medication</th>
<th>Beginning Dosage</th>
<th>Titration</th>
<th>Maximum Dosage</th>
<th>Duration of Adequate Trial</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gabapentin</td>
<td>100-300 mg qhs</td>
<td>Increase by 100-300 mg a day every 1-7 days as tolerated in divided doses up to tid None needed</td>
<td>3600 mg daily (1200 mg tid); reduce if creatinine clearance less than 60 mL/min Maximum of 3 patches daily for a maximum of 12 hours</td>
<td>3-8 weeks for titration plus 1-2 weeks at maximum tolerated dosage 2 weeks</td>
</tr>
<tr>
<td>Lidocaine patch 5%</td>
<td>Maximum of 3 patches daily for a maximum of 12 hours 2.5-15 mg every 4 hours as needed</td>
<td>After 1-2 weeks, convert total daily dosage to long-acting opioid analgesic and continue short-acting medication as needed</td>
<td>No maximum with careful titration; consider evaluation by pain specialist at dosages exceeding 120-180 mg daily</td>
<td>4-6 weeks</td>
</tr>
<tr>
<td>Opioid analgesics (dosages given are for morphine)</td>
<td>25 mg once daily</td>
<td>Increase by 25-50 mg daily in divided doses every 3-7 days as tolerated</td>
<td>400 mg daily (100 mg qid); in patients over 75 years of age, 300 mg daily in divided doses</td>
<td>4 weeks</td>
</tr>
<tr>
<td>Tramadol</td>
<td>10-25 mg qhs</td>
<td>Increase by 10-25 mg daily every 3-7 days as tolerated</td>
<td>75-150 mg daily; if blood level of active drug and its metabolite is below 100 ng/ml, continue titration with caution</td>
<td>6-8 weeks with at least 1-2 weeks at maximum tolerated dosage</td>
</tr>
</tbody>
</table>

with refractory symptoms should be referred for interdisciplinary treatment that may include cognitive-behavioral therapy, instruction in flare self-management techniques and how to pace engagement in aerobic exercise, and methods to enhance sleep hygiene. Recently pregabalin also has been approved.

Peripheral Neuropathy

Definition and Pathogenesis. Neuropathic pain is defined as pain “initiated or caused by a primary lesion or dysfunction in the nervous system.”16 The most common causes in older adults are diabetes mellitus and reactivation of herpes zoster (ie, postherpetic neuralgia). Axial arthritis (eg, cervical and lumbar spondylosis) associated with radiculopathy is also considered by many experts to be a form of neuropathic pain. Other causes of peripheral neuropathy in older adults include alcoholic polyneuropathy, chemotherapy-induced polynuropathy, enrapment neuropathies, postmastectomy pain, post-thoracotomy pain, nerve compression or infiltration by tumor, phantom limb pain, postradiation plexopathy, and trigeminal neuralgia.17 Central poststroke pain may also present with symptoms that mimic peripheral neuropathy.

Evaluation. Although several specialized tests are available for the assessment of neuropathic pain, such as quantitative sensory testing and electromyography/nerve conduction, the history and physical examination remain the cornerstone of the evaluation. Patients with peripheral neuropathy often complain of constant or intermittent pain (typically worse at night and with inactivity), stimulus evoked-pain such as allodynia or hyperalgesia, pruritis or tingling, and sensory loss. A comprehensive neurological examination should be performed on all older adults with neuropathic pain. When the cause is not obvious (eg, postmastectomy pain, post-thoracotomy pain), a thorough general physical examination should be performed and any signs of malignancy should be pursued diagnostically. If no cause can be found, the following laboratory studies should be obtained: vitamin B12 and folate levels, fasting blood glucose, and serum/urine electrophoresis. If a cause still cannot be identified, the patient should be referred to a neurologist.

Treatment. Neuropathic pain is typically more refractory to treatment efforts than nociceptive pain. A number of pharmacologic options are available. Tables 5 and 6 summarize medications and dosing guidelines for the treatment of neuropathic pain.

Table 8  Treatment Outcome Parameters for the Older Adult with Chronic Pain

<table>
<thead>
<tr>
<th>Pain interference with performance of:</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Basic activities of daily living</td>
</tr>
<tr>
<td>• Instrumental activities of daily living</td>
</tr>
<tr>
<td>• Discretionary activities</td>
</tr>
<tr>
<td>Mobility/activity level</td>
</tr>
<tr>
<td>Energy level</td>
</tr>
<tr>
<td>Appetite</td>
</tr>
<tr>
<td>Sleep</td>
</tr>
<tr>
<td>Mood – eg, irritability, depression</td>
</tr>
<tr>
<td>Interpersonal interactions</td>
</tr>
<tr>
<td>Attention and concentration</td>
</tr>
<tr>
<td>Frequency of prn analgesic ingestion</td>
</tr>
<tr>
<td>Pain severity</td>
</tr>
</tbody>
</table>

THE ROLE OF DEPRESSION AND DEMENTIA IN PAIN MANAGEMENT

In 1965, the Gate Control Theory of Pain was born, and its principles continue to guide pain management practice today. This theory emphasizes the central role of the brain in pain processing and accounts for the effectiveness of cognitive behavioral therapy in management of chronic pain. When the brain functions abnormally, as in the case of depression or dementia, pain control becomes more challenging. Thus, practitioners that care for older adults with chronic pain must be as aggressive about recognizing and treating these disorders as they are about the pain itself.

While depression is eminently treatable, the presence of dementia in the older adult with chronic pain poses a considerable challenge. Often, those with depression are both in pain and with inactivity, stimulus evoked-pain such as allodynia or hyperalgesia, pruritis or tingling, and sensory loss. A comprehensive neurological examination should be performed on all older adults with neuropathic pain. When the cause is not obvious (eg, postmastectomy pain, post-thoracotomy pain), a thorough general physical examination should be performed and any signs of malignancy should be pursued diagnostically. If no cause can be found, the following laboratory studies should be obtained: vitamin B12 and folate levels, fasting blood glucose, and serum/urine electrophoresis. If a cause still cannot be identified, the patient should be referred to a neurologist.

While depression is eminently treatable, the presence of dementia in the older adult with chronic pain poses a considerable challenge. Often, those with depression have exaggerated fear responses to pain which, in turn, can intensify the pain experience. The pharmacologic and nonpharmacologic modalities that have already been discussed should also be offered to the older adult with dementia. Education should be emphasized, especially education of caregivers. Components of care-giver education are provided in Table 7.
HOW TO DETERMINE WHETHER PAIN TREATMENT IS EFFECTIVE

Pain intensity is only one of many parameters that may improve when chronic pain is managed successfully. Patients with chronic nonmalignant pain should anticipate, on average, no more than 30%-50% reduction in pain intensity. Physical function, mood, endurance, sleep, appetite, and interpersonal interactions may respond even more substantially to treatment, as may overall quality of life. As part of the initial assessment, therefore, the practitioner should clearly identify treatment goals in order to determine the effectiveness of therapy. Although treatment goals must be individualized, a list of potential outcome parameters is provided in Table 8.

HOW AND WHEN TO REFER TO A PAIN SPECIALIST

The decision regarding when and to whom to refer the older adult with refractory pain depends upon the patient’s goals. In general, referral to a pain specialist should be considered when the patient continues to experience disabling pain despite efforts to control symptoms with medications and other therapeutic modalities (eg, injections, physical therapy). Pain specialists also vary widely in their training and approach to treatment. Some specialists focus exclusively on injection procedures, while others work with an interdisciplinary team that utilizes a rehabilitative approach. Because of the multifactorial nature of chronic pain syndromes in older adults, the latter strategy is favored and appears to be most effective.

References