# Pain Medicine in Older Adults: How Should It Differ?

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**Key Points**

- Degenerative skeletal disease is a normal part of aging; imaging should not be used to guide care in the majority of older adults with chronic pain.
- Pain is common, but it is not a normal part of aging; the majority of older adults are motivated to get better, and their pain and associated disability should be treated as aggressively as in younger patients.
- The older adult with chronic pain should not be treated as a chronologically older version of a younger patient with chronic pain; they should be treated as an older adult first and a patient with pain second.
- Successful pain management for the older adult requires differentiating the patient's weak link(s) from their treatment target(s).
- Non-pharmacological pain management strategies should be prioritized for older adults in an effort to limit medication-associated toxicities that are more common and dangerous than those experienced by younger patients.
- All older adults should undergo formal screening of their cognitive function; the older adult with dementia requires an approach to pain evaluation and management that is distinct from that used for cognitively intact patients.
- Primum non nocere: Opioid analgesics and pain itself can both cause harm (e.g., falls, cognitive dysfunction); the potential risks associated with treatment must be weighed against the risks of no treatment.

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## Introduction

Older adults (commonly defined as those ≥ age 65) are not simply a chronologically older version of younger patients. Homeostenosis, that is, progressive restriction of an aging organism's capacity to respond to stress because of diminution of its biological, psychological, and social reserves, underlies the distinction of old from young [1]. As pain is a stressor that commonly accompanies aging, the provision of safe, clinically effective, and cost-effective pain care to older adults requires awareness of these specific aging-related changes [2]. The main goals of this chapter are to (1) educate the pain practitioner in basic principles of aging needed to guide the evaluation and treatment of older adults, (2) provide clinical case examples to illustrate the advantages of treatment that is guided by these principles as compared with traditional pain care and why traditional pain care may actually harm these patients, and

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(3) offer specific therapeutic guidelines for the treatment of nociceptive, neuropathic, and widespread pain in older patients.

## Background

America is aging at a rapid pace. In 2000, approximately 35 million people (12.4 % of the total population) were ≥ age 65, and in 2050, this number is anticipated to rise to an estimated 86.7 million (20.6 % of the total population) [3]. Those ≥ age 85 represent the most rapidly growing segment of the population. Of all chronic health conditions that limit activity and heighten the risk of disability in older adults (e.g., dementia, diabetes mellitus, cardiovascular disease), painful musculoskeletal disorders such as arthritis and low back pain are the most common [4]. Pain practitioners are, therefore, ideally positioned to impact the lives of older adults in a profound way.

## Scientific Foundation

The purpose of this section is to present scientific data that negate commonly held beliefs about older adults that have lead to the undertreatment of pain in this vulnerable population.

**Myth #1**
Pain is a normal part of aging.

**Reality #1**
Although degenerative skeletal disease is a normal part of aging, chronic pain is not. Additionally, chronic pain can lead to serious health consequences for older adults.

*Discussion*: While pain is common in older adults, it is not normal. A key principle of aging is as follows: *Many findings that are abnormal in younger patients are common in older people and may not be responsible for a particular symptom* [5]. Using low back pain (LBP) as an example, data demonstrate clearly that degenerative disease of the lumbar spine is nearly ubiquitous in those age 65 and older [6–8] and that magnetic resonance imaging (MRI) evidence of moderate to severe lumbar spinal stenosis occurs not uncommonly in those who are asymptomatic [9]. Although large epidemiological studies that focus exclusively on older adults are lacking, existing data suggest that fewer than half of these individuals experience LBP and an estimated 14 % experience associated functional decline [10]. Degenerative disease of the appendicular skeleton also is common. For example, asymptomatic hip osteoarthritis occurs in over half of older women [11].

Further, older adults with chronic noncancer pain may experience numerous adverse consequences such as impaired physical function, depression and anxiety, social isolation, sleep and appetite disturbance, impaired neuropsychological performance, an increased burden of medical comorbidity, and excessive utilization of health care resources [10, 12–23]. Community-dwelling older adults with chronic pain also have significantly worse self-rated health (a powerful predictor of morbidity and mortality) than those without pain [24], suggesting that unrelieved pain may be associated with enhanced mortality.

**Myth #2**
Older adults do not feel pain as much as younger patients; thus, conditions associated with pain in younger patients may not be associated with pain in older adults. Thus, pain treatment does not need to be aggressive in these individuals.

**Reality #2**
Laboratory data do not support diminished ability of older adults to perceive pain. Some data point to their diminished ability to regulate (through top-down inhibition) peripheral nociceptive stimuli, and this suggests that practitioners may need to provide even more aggressive analgesia to older adults.

*Discussion*: Histopathological and biochemical studies indicate decreased density of myelinated and unmyelinated peripheral nerve fibers [25–27], and an increased number of degenerated fibers are associated with aging [28]. Selective age-related impairment of myelinated nociceptive fiber function also has been demonstrated [29, 30].

Additional evidence points to age-associated central changes in significant neurotransmitters. In the dorsal horn of the rat, progressive age-related loss of serotonergic and noradrenergic neurons has been demonstrated [31, 32]. There is a decline in the concentration and turnover of catecholamines, gamma-aminobutyric acid (GABA), and opioid receptors [33–35] in the limbic system and a lower density of serotonin receptors [36]. Aging-associated biochemical changes are also evident in the cerebral cortex in general [37–44] and in the prefrontal cortex in particular [45]. Thus, older adults may have an inadequate quantity of key pain-modulating neurochemicals.

Laboratory studies of pain threshold and tolerance have been performed exclusively on healthy individuals. The application of these data to patients in pain is unknown.

Somatosensory thresholds for non-noxious stimuli in healthy older adults increase with age, while results associated with noxious stimuli have been variable and dependent upon the type of stimulus applied [30]. One of the most carefully designed studies comparing pain threshold and tolerance to pressure, heat, and ischemic stimuli in young and old humans demonstrated no significant age-associated differences in response to heat or pressure and significantly lower tolerance and threshold to ischemic stimuli in old versus young [46].

Age differences in temporal summation (i.e., enhancement of perceived pain intensity when noxious stimuli are delivered repetitively above a critical rate), a correlate of wind up in animals, also have been examined in the laboratory, with findings summarized as follows: (1) Older adults appear to have enhanced temporal summation to heat but not pressure as compared with younger individuals [47]. (2) Older adults have enhanced temporal summation in response to electrical stimulation compared to younger adults [48]. These findings suggest that older adults may have reduced capacity to downregulate their nervous system response to pain after an initial period of sensitization [49].

**Myth #3**

As with younger chronic pain patients, treatment of psychological dysfunction (e.g., depression, anxiety, poor coping skills) is the most important aspect of chronic pain treatment for older adults.

**Reality #3**

For the majority of older adults with chronic pain, identifying and treating the numerous physical pain contributors (i.e., the appropriate treatment targets) holds the key to optimizing symptomatic relief. The law of parsimony (Occam's razor) *should not* guide treatment, and the "weakest link" may not be the treatment target.

*Discussion*: Although older adults with chronic pain tend to have more physical limitations than younger patients, in general, they are more psychologically robust, with better coping skills and mental health, less fear avoidance, and a greater sense of life control [50]. While large population-based studies have not been performed, preliminary data indicate an estimated one in three older adults with chronic nonmalignant pain seen in a tertiary referral center's interdisciplinary pain clinic has a high burden of psychological dysfunction [17]. For these individuals, the practitioner should consider prescribing interdisciplinary pain rehabilitation that includes psychological treatment. Two-thirds of the older adults in this sample did not have high levels of psychological dysfunction and would not, therefore, require such treatment.

Our research and clinical experience suggests that for the majority of older adults with chronic pain who do not have significant psychological dysfunction, ascertaining the numerous biological/physical contributors to the patient's pain syndrome and their pain-associated functional limitations holds the key to prescribing effective treatment. More often than not, the older adult with chronic pain has numerous contributors to pain, even when the patient reports pain at a single site. A related key principle of aging is as follows: *Because many homeostatic mechanisms are often compromised concurrently, there are usually multiple abnormalities amenable to treatment and small improvements in each may yield dramatic benefits overall* [5]. We recently published a case series of older adults with postherpetic pain and comorbid myofascial pain [51]. These patients had been treated with numerous neuropathic pain medications that resulted in side effects and/or suboptimal pain relief. Significant symptomatic improvement occurred only after the myofascial component of their pain was treated.

Another example of multiple pathologic contributors to single-site pain is chronic low back pain. We have demonstrated that 82% of older adults with chronic low back pain have multiple potential sources of pain including myofascial pain (95.5 %), sacroiliac joint pain (83.6 %), hip disease (24 %), and fibromyalgia syndrome (19.3 %) [52]. Further, while 25 % of these individuals reported neurogenic claudication, 50 % of them also had other spinal/leg pathology that might have accounted for their low back and leg pain.

These data should be considered in the context of studies that have demonstrated substantial rates of failed back surgery syndrome in those who undergo decompressive laminectomy for the treatment of lumbar spinal stenosis [53, 54]. The effect on low back/leg pain is unknown of addressing associated pathology (e.g., fibromyalgia, hip joint arthritis) instead of or in addition to surgically treating the degenerative lumbar disease. Until the answer to this question is ascertained, the most clinically effective and cost-effective treatment(s) for these patients will remain elusive.

A related key principle of aging is as follows: *Presentation of a new disease depends on the organ system made most vulnerable by previous changes, and because the most vulnerable organ system* ("*weakest link*") *often differs from the one newly diseased, presentation is often atypical* [5]. Consider, for example, the hospitalized older adult who develops acute confusion (i.e., delirium). The most common causes of delirium in hospitalized older adults are adverse drug reactions and infections [55]. Rational evaluation and treatment of these patients is guided by a search for potentially offensive medications and/or infections such as a urinary tract infection or pneumonia. Unless there are focal neurological findings, brain imaging is not indicated because

while the brain is the "weakest link," it is not the treatment target. Similarly, for older adults with low back pain, the lumbar spine may be the weakest link and successful treatment might lie in identifying and treating conditions outside of the spine itself. An illustrative case is presented later in this chapter (Case 1 below).

**Myth #4**
Treating pain in older adults will reduce the risk of disability.

**Reality #4**
While quality of life can be improved by treating pain in older adults, effective strategies to reduce the risk of disability are elusive. Preliminary data indicate that brain-targeted as opposed to body-targeted treatment may represent the "missing link." Additional research in this area is needed.

*Discussion*: While treating pain is essential for improving quality of life and diminishing its interference with performance of daily activities [23], treating pain as a physical symptom does not appear to reduce the risk of future dependent living status, that is, disability. Large studies examining the efficacy of physical therapy for the treatment of chronic low back pain (CLBP) in older adults have not been performed. Preliminary evidence suggests that lumbar spine-focused physical therapy for these patients does not improve pain or physical function [56, 57]. Those who undergo decompressive laminectomy for lumbar spinal stenosis experience less pain, but not significantly improved function [58].

We have gathered several sets of data that support the potential role of the brain in generating pain-related disability in older adults with CLBP. Specifically, evidence supports the following: (1) Neuropsychological performance mediates the relationship between pain and physical function [13]. We have shown in older adults with CLBP that the modest relationship between pain severity and disability is no longer significant when neuropsychological performance (NP) is statistically controlled (i.e., after NP is removed from the relationship). This implies either that NP mediates the relationship between pain and disability or that NP and disability share common pathways in the brain. (2) Older adults with CLBP, as compared with older adults who are pain-free, have structural brain changes in the middle corpus callosum, middle cingulate white matter, and gray matter of the posterior parietal cortex as well as impaired attention and mental flexibility [59]. (3) Older adults with CLBP that is self-reported as being disabling have more severe changes in brain morphology than older adults with CLBP that is not disabling, and the duration of chronic pain is associated with the severity of changes in brain morphology [60]. The exact cause of the brain changes and the extent to which these changes are reversible or modifiable is not known. (4) Mindfulness meditation, a treatment directed at altering the brain's perception of/reaction to pain, reduces pain's interference with performing daily activities [61]. Additional research in this area may be at the cutting edge of developing treatments that not only reduce pain but reduce the risk of disability for older adults with CLBP. Given the suboptimal outcomes associated with lumbar spine-focused treatments, such research is critically needed.

**Myth #5**
Opioids should be used with extreme caution if at all in older adults.

**Reality #5**
Opioids and pain itself are associated with multiple potential deleterious effects. If opioids are prescribed, the adage "start low and go slow" should guide treatment. Meticulous, ongoing follow-up is the only way to answer, "Do the benefits of opioids outweigh their risks?"

*Discussion*: As with all medications, risks and benefits must be balanced. Opioids may result in a number of deleterious side effects in older patients. As noted in the introduction to this chapter, as people age, there is progressive restriction in their physiological reserve capacity (i.e., homeostenosis). This can take many forms that include decline in neuropsychological performance [62], sarcopenia and reduced mobility [63, 64], changes in analgesic pharmacokinetics and pharmacodynamics [65], and social isolation. When opioids are used, therefore, the practitioner must be vigilant for side effects for which older adults may be at increased risk such as falls, hip fracture, and delirium. And because older adults have enhanced pharmacodynamic sensitivity to opioids [66, 67], the patient and his caregiver must be educated about these risks, even when low doses are prescribed.

That being said, risks associated with opioids must be balanced with the risks associated with pain itself. As summarized in Table 88.1, many of the deleterious effects associated with opioids are identical to those associated with pain. Older adults with chronic low back pain have more impaired balance [68] and, therefore, a greater risk of falls than those who are pain-free. While delirium is a potential side effect of opioids, it is also a potential side effect of pain,

**Table 88.1** Opioids in older adults: balancing risks and benefits

| Symptom/side effect | Associated with opioids | Associated with pain | Management/monitoring approach |
|---|---|---|---|
| Depression | X | X | Consider treating depression as first step and observe effect on pain |
| Anxiety | X | X | Consider treating anxiety as first step and observe effect on pain |
| Agitation | X | X | Consider referral to psychiatry to determine cause and most appropriate treatment of agitation |
| Mobility difficulty/ falls | X | X | Falls risk should always be screened in the older adult with chronic pain. If balance impairment is evident, an assistive device should be recommended along with referral to physical therapy for instruction in proper use. If opioids are considered, education regarding the risk of falls is essential for all older adults. If opioids are considered for the older adult with baseline mobility impairment, the practitioner must refer to physical therapy in an effort to optimize balance *prior* to prescribing opioids |
| Delirium | X | X | Patients with dementia have a heightened risk of delirium with opioids and with pain. A cognitive function screen should be considered an essential vital sign for older adults |
| Constipation | X | | Discussion about starting a stimulant laxative at the first sign of constipation should occur at the time that the opioid is prescribed |
| Urinary retention | X | | Especially important to educate the older male with benign prostatic hypertrophy and baseline voiding symptoms about this risk |
| Respiratory depression | X | | More common in high doses |
| Sleep disturbance | X | X | Although nocturnal pain may prompt prescription of an opioid at bedtime, patients should be educated about their potential deleterious impact on sleep |
| Diminished appetite | X | X | As with other symptoms, the patient should ascertain the relative risks and benefits |
| Increased utilization of health care resources | ? | X | Our clinical experience suggests that drug-seeking behavior is unusual in older adults in the absence of poorly treated pain |

especially for hospitalized older adults or those in nursing homes. A study of 541 older adults who underwent hip fracture repair demonstrated that better pain control on higher doses of intravenous morphine was associated with a lower risk of postoperative delirium [69]. Others have shown that cancer patients who require long-term opioids may experience improved neuropsychological performance as a result of more effective pain management [70, 71].

**Myth #6**

Treatment of pain in older adults with dementia should be guided by the same basic principles as for those who are cognitively intact.

**Reality #6**

Older adults with dementia are not simply a cognitively impaired version of those who are cognitively intact. An evidence base to guide treatment of pain in older adults with dementia is lacking; there is no substitute for thoughtful implementation and critical observation of empirical interventions.

*Discussion*: Just as aging is associated with extreme heterogeneity in the deterioration of biological, psychological, and social reserves as well as physical function, so too is dementia a heterogeneous process. The most common form of dementia is Alzheimer's disease (AD) and the vast majority of data regarding pain, and dementia applies to this condition.

A number of studies that have been done with pain-free older adults in the laboratory highlight that those with Alzheimer's disease have altered pain processing as compared with cognitively intact individuals. Functional brain imaging suggests that those with AD experience enhanced attention to painful stimuli as compared to those without AD [72]. Others have demonstrated that AD patients self-report pain intensity of acute stimuli (e.g., pressure, venipuncture) similar to that of cognitively intact individuals, but that their facial expressions associated with these stimuli are more exaggerated and nonspecific [73, 74]. Data also suggest that other behavioral manifestations of pain, such as guarding, bracing, and rubbing, also may be nonspecific in those with AD [75], that is, these "pain" behaviors may be an expression of the disordered movement that occurs in association with dementia, even in the absence of pain. Additional research in this area is clearly needed so that pain can be accurately detected in patients who have dementia and others with communication impairment.

Evidence also suggests that older adults with dementia may have blunted treatment expectancy [76]. It has been well-established in the pain literature that treatment expectancy is synergistic with pharmacodynamic analgesic efficacy

[77, 78]. That is, the absence of belief in treatment efficacy negatively impacts treatment outcomes, even in those who are cognitively intact. If, in fact, patients with dementia have reduced treatment expectancy, these individuals may require larger analgesic doses to achieve desirable treatment outcomes. The reader should be aware that controlled studies of this hypothesis have never been undertaken and are needed. Until scientific evidence exists, the practitioner should be aware of the differences in pain processing between older adults with and without dementia and approach treatment prescribing accordingly.

## Application to Clinical Practice

The key to optimizing treatment outcomes for older adults with chronic pain is to start with comprehensive assessment. The purpose of this assessment is threefold: (1) to identify all treatment targets, (2) to establish the patient's unique pain signature that should be used to determine the efficacy of treatment, and (3) to identify key comorbidities that could constrain various treatment options. Table 88.2 outlines the essential components of a comprehensive history [79] and physical examination for the older adult with chronic pain that is designed to address each of these three goals.

Below is a series of real cases that actualize how to integrate principles of aging into the practice of pain medicine and illustrate how to comprehensively identify treatment targets, establish the older adult's pain signature (i.e., the way(s) that the patient manifests pain such as reduced appetite, difficulty walking, and confusion) [79], and identify potentially limiting comorbidities.

### Case 1

An 82-year-old woman presented with low back pain for many years that had started insidiously and had lead to increasing functional limitations. She reported 7–8/10 sharp/burning daily pain that she experienced bilaterally, below the waist, and was worsened by standing, lifting, walking, and bending. There were no red flags. She had undergone numerous treatments without benefit including acupuncture, chiropractic, traction, physical therapy, aqua therapy, multiple epidural corticosteroid injections, and inpatient pain rehabilitation. She took prn naproxen for pain relief. Musculoskeletal examination revealed mild kyphoscoliosis, tenderness to palpation of both sacroiliac regions, and bilateral piriformis taut bands and trigger points. Neurological examination revealed symmetrical reflexes, 5/5 strength throughout, shortened stride length, and an anxious affect. The initial working diagnoses were (1) sacroiliac joint syndrome, (2) myofascial pain, and (3) anxiety for which physical therapy, sacroiliac joint injections, and gabapentin were prescribed.

One month later, she had experienced no pain reduction or functional improvement. A more detailed history uncovered the development over the past year of change in her voice (softening), handwriting (smaller), posture (increased forward flexion), and facial expression (less animated). A more detailed physical examination uncovered mild cogwheeling of her right arm. A neurology consultation was obtained to address the possibility of Parkinson's disease. The consultant felt that there were "no full-blown Parkinsonian signs or symptoms," but the presence of her masked facies, diminished blink, minimal asymmetrical cogwheeling, Myerson's sign, and tendency to retropulse prompted a trial of levodopa/carbidopa 25/100 bid.

One month later, the patient reported average 4/10 pain (~50 % reduction from baseline), improved posture, and balance as well as walking capacity and flexibility.

*Discussion*: The synthesis of this case is presented in Fig. 88.1. The treatment targets for this patient were her Parkinson's disease and her myofascial pain. Her pain signature was comprised primarily of decreased physical function. Her impaired gait was also the primary comorbidity of concern. This placed her at heightened risk of falls. Had opioids been prescribed, the practitioner would have had to be especially vigilant for worsening mobility. Prior to prescribing such medications, the patient would have had to be educated about the risk of falls and hip fracture.

This case highlights the fact that PD is not infrequently associated with pain. Forty to fifty percent of patients with PD have pain that is not explained by other obviously painful disorders [80, 81]. Fifteen percent have pain as their presenting symptom (e.g., unilateral shoulder pain) [82]. Twenty-five percent have pain that precedes motor symptoms [83]. Patients may report muscle cramps or tightness, typically in the neck, paraspinal, or calf muscles; painful dystonias; joint pain; neuropathic pain; or less commonly, generalized pain [84]. Oral and genital pain syndromes that are similar to symptoms occurring in patients with tardive dystonia and akathisia from neuroleptics also have been described in patients with PD [85, 86]. The underlying pathogenesis of pain in PD can be central, peripheral, or mixed. Sensory thresholds to experimentally delivered painful stimuli are reduced in PD [87]. Unlike peripherally generated pain, such as that experienced by our patient, central PD pain that is associated with abnormal nociceptive input processing is not affected by dopamine administration [88].

Perhaps most importantly, this case illustrates that successful treatment of the older adult with low back pain requires identifying the proper treatment targets (Parkinson's disease [PD]) rather than simply treating the weak link (axial spondylosis). Had this patient elected to go forward with spinal surgery, the likely outcome, as compared with the actual

**Table 88.2** History and physical examination for the older adult with persistent pain: the essentials

| History [79] | |
|---|---|
| Answers to the following questions will help to ascertain the older adult's pain signature and, therefore, key treatment outcomes. | |
| 1. How strong is your pain (right now, worst/average over past week)? | |
| 2. How many days over the past week have you been unable to do what you would like to do because of your pain? | |
| 3. Over the past week, how often has pain interfered with your ability to take care of yourself, for example, with bathing, eating, dressing, and going to the toilet? | |
| 4. Over the past week, how often has pain interfered with your ability to take care of your home-related chores such as going grocery shopping, preparing meals, paying bills, and driving? | |
| 5. How often do you participate in pleasurable activities such as hobbies, socializing with friends, and travel? Over the past week, how often has pain interfered with these activities? | |
| 6. How often do you do some type of exercise? Over the past week, how often has pain interfered with your ability to exercise? | |
| 7. Does pain interfere with your ability to think clearly? | |
| 8. Does pain interfere with your appetite? Have you lost weight? | |
| 9. Does pain interfere with your sleep? How often over the past week? | |
| 10. Has pain interfered with your energy, mood, personality, or relationships with other people? | |
| 11. Over the past week, how often have you taken pain medications? | |
| 12. How would you rate your health at the present time? Excellent, good, fair, poor, or bad? | |
| Past history/review of systems: This portion of the history will identify key medical, psychological, and social comorbidities that may impact treatment response. | |
| Medical comorbidities | Relationship to treatment |
| Constipation | If present at baseline, a stimulant laxative should be prescribed (e.g., senna) at the same time that an opioid is started |
| Lower extremity edema | May be exacerbated by a nonsteroidal anti-inflammatory drug |
| Hypertension | Gabapentin and pregabalin can contribute to lower extremity edema |
| Congestive heart failure | Renal insufficiency should be kept in mind when dosing various analgesics (see Tables 88.4 and 88.5) |
| Peptic ulcer disease | |
| Renal insufficiency | |
| Obesity | Some medications may contribute to weight gain, such as gabapentin, pregabalin, and tricyclic antidepressants |
| Sleep disturbance | While pain may disrupt sleep, opioids are also associated with disruption in sleep architecture |
| Difficulty walking/falls | While pain itself can contribute to weakness, difficulty walking, and falls, older adults can have mobility difficulty independent of pain. In these individuals, care must be taken to avoid medications that can themselves contribute to mobility impairment, for example, opioids, pregabalin, gabapentin, and tricyclic antidepressants |
| Memory loss | As noted in the text, pain itself can cause decrements in multiple domains of neuropsychological performance. With effective pain treatment, memory may improve. Practitioners must be aware, however, that many pain medications may contribute to confusion, for example, opioids, pregabalin, gabapentin, tricyclic antidepressants, and others (see Tables 88.4 and 88.5) |
| *Psychological factors* | |
| Depression | Untreated depression and/or anxiety can impair top-down inhibition; thus, the older adult with comorbid depression and/or anxiety must be treated for these disorders as part of pain treatment |
| Anxiety | |
| Coping skills | Poor coping skills (e.g., tendency to catastrophize) can inhibit the efficacy of pain treatment. While most cognitively intact older adults seem to cope well with chronic pain, the minority who do not should be referred for cognitive behavioral therapy as a part of pain treatment |
| Self-efficacy | Physical therapy reduces fear avoidance beliefs (i.e., fear of moving because of concerns about exacerbating pain) in older adults [57]. Older adults with a history of falls may exhibit fear of falling, may have low confidence in mobility, and may have low self-efficacy (i.e., lack of confidence in their ability to engage in certain behaviors to affect desired outcomes). For these individuals, referral to a pain psychologist and physical therapist should be part of pain treatment |
| Confidence in mobility | |
| Fear of movement | |
| Treatment expectancy | Treatment expectancy must be established at the outset of pain evaluation. Patients who believe that treatment will work will likely improve (i.e., placebo effect). Those who believe that treatment will not work will likely not improve (i.e., nocebo effect) |

(continued)

**Table 88.2** (continued)

| | |
|---|---|
| *Social factors* | |
| Social/caregiver support | Social isolation can interfere with the older adult's ability to distract themselves from their pain and, therefore, intensify their pain experience. This may be especially problematic for the older adult with dementia. |
| Financial status | The practitioner should always consider the older adult's financial resources when prescribing treatments. |
| Physical examination | |
| 1. Vital signs<br>(a) Cognitive function<br>Mini-Cog [92, 93]: Examiner gives the patient three unrelated words to remember. Then, she/he gives the patient a blank piece of paper and asks them to draw a clock with the hands pointing to a specific time. Then, the patient is asked to recall the three words. Patients who are able to recall all three words have a low likelihood of dementia. Those who recall zero words have a high likelihood of dementia. For those who recall 1–2 words, the examiner should assess the accuracy of the clock-drawing test. If there are gross errors, the patient should be referred for evaluation of possible dementia.<br>(b) Mobility<br>(c) Traditional vital signs | |
| 2. Functional performance<br>(a) Balance<br>Modified postural stress test ([94]; see Fig. 88.4): Examiner stands behind the patient with hands on sides of pelvis and states, "I am going to pull you backwards gently and try to throw you off balance…Do not let me…Are you ready?" Then, the examiner pulls the patient toward himself gently. If the patient is able to resist easily, try pulling a little more forcefully and observe response. The older adult whose balance is easily perturbed has decreased postural control and may be at heightened risk for falls.<br>(b) Basic functional tasks – chair rise, ability to pick up object from floor, ability to place hands behind neck and waist (movements needed for dressing), and manual dexterity (e.g., ability to button and unbutton clothing, tie shoes) | |
| 3. Comprehensive identification of pain comorbidities<br>(a) Knee/hip arthritis in patients with low back pain<br>(b) Shoulder disease in those with neck/upper back pain<br>(c) Myofascial pain in all patients, including those with neuropathic pain [51] | |
| 4. Comprehensive routine physical examination | |

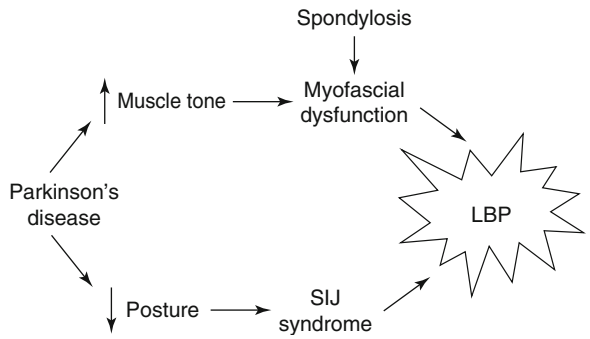


**Fig. 88.1** Synthesis of Case 1. For details, see text. *LBP* low back pain, *SIJ* sacroiliac joint

outcome, is depicted in Fig. 88.2. In this figure, the "existing approach" represents common practice and the "proposed approach" is what we recommend.

## Case 2

An 82-year-old woman presented with low back pain and right leg pain for two years with documented central canal stenosis on MRI. She had worked full time in a dress shop and was forced to retire 2 years ago because the company was downsizing. She said that her pain started at that time and had gotten progressively more severe. Her pain was made worse by prolonged standing or walking, and she was having increasing difficulty performing heavy housework. Her pain was made better with rest and heat application. She denied fever, chills, weight loss, and change in her bowels or bladder function. She reported poor balance and multiple near falls at home. She lived alone. She was becoming increasingly fearful of leaving her home. Medications at the time of presentation, all of which had been prescribed to treat her pain and pain-associated anxiety, included gabapentin, oxycodone CR, celecoxib, tramadol/acetaminophen, olanzapine, escitalopram, and lorazepam. Physical examination revealed poor balance, dementia (memory problems and very impaired clock-drawing test) [89], kyphoscoliosis, and tenderness of the right sacroiliac joint/lumbar paraspinal musculature/tensor fasciae latae/iliotibial band. Because of extreme guarding behavior, strength testing was invalid.

Because of polypharmacy, high falls risk, and social isolation, the patient was admitted to a nursing home for detoxification. All of her medications were discontinued with the exception of regularly scheduled acetaminophen and prn tramadol. She reported minimal pain and her balance improved markedly. It was recommended that her

family strongly consider placing her in an assisted living facility. They chose to seek other opinions from pain practitioners. Immediately following discharge, the patient's pain complaints escalated and multiple other pain regimens were attempted including a morphine pump trial, all of which failed. She was eventually placed in an assisted living facility where she did well.

*Discussion*: The synthesis of this case is presented in Fig. 88.3. As noted earlier in this chapter, to prescribe effective treatment, the practitioner must differentiate the weak link from the treatment target(s). In this case, chronic pain was the weak link and fear/social isolation the treatment targets. Her pain signature consisted of pain perseveration and significant utilization of health care resources. The main potentially treatment-limiting comorbidities were her dementia and balance frailty.

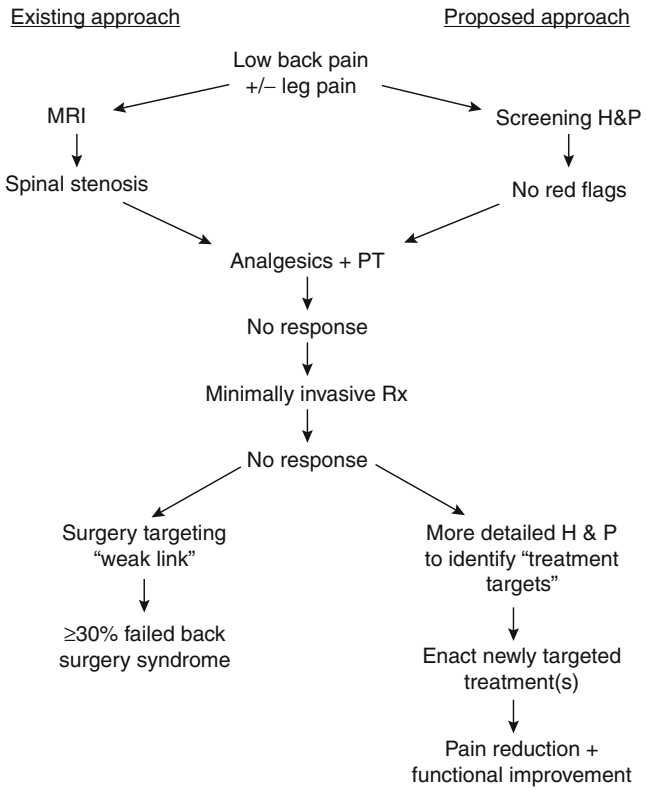


**Fig. 88.2** A comparison of two approaches for the management of older adults with low back +/– leg pain. The approach commonly used (existing approach) focuses on imaging to direct treatment. Because the predictive value of abnormal imaging has not been critically examined in older adults and because abnormalities occur commonly, with or without pain, this approach frequently results in failed treatment. The proposed approach relies on a comprehensive history and physical examination to guide treatment that often targets multiple pain contributors. *H&P* history and physical examination, *MRI* magnetic resonance imaging, *PT* physical therapy

One of the first discussions that we have with patients in chronic pain revolves around treatment expectations. Specifically, patients with chronic pain need to understand that it is realistic to expect partial but not complete pain relief. Treatment of the older adult with dementia is complicated by the fact that information provided in treatment counseling sessions may not be remembered and ongoing reinforcement may be necessary. Such reinforcement is often successful when the patient has an involved and supportive caregiver (and one who does not catastrophize about the patient's pain) and health care providers who are willing to communicate a consistent message. In this patient's case, inconsistent messages were delivered (i.e., although it was clear that the patient's fear and social isolation in the setting of dementia were primarily responsible for her suffering, the patient's family insisted that her pain was responsible and more aggressive pain treatment was sought).

While many patients with dementia can report pain reliably [90, 91], the meaning of these reports must be ascertained in order to prescribe effective treatment. Is the patient's pain reporting a manifestation of perseveration (that occurs not uncommonly in patients with dementia)? Or, is the patient's pain reporting a more general signal of distress? Or, is the patient's pain reporting an indication of pain-related suffering? If there is pain-related suffering, then pain-specific treatment must be implemented. In the case of our patient, her pain reporting appeared to be a manifestation of both perseveration and a more general signal of distress (i.e., anxiety surrounding social isolation and dementia). Thus, while treatment did involve analgesics, providing a supportive environment was the primary therapeutic element.

This case highlights the need to screen for dementia at the time of the initial history and physical examination. One of the most efficient and effective screening tools is the Mini-Cog, described in Table 88.2 [92, 93]. It takes no more than 2–3 min to perform. If this testing uncovers the possibility of dementia, the patient should be referred to a geriatrician for further evaluation. Older adults with and without dementia often have mobility difficulty and a risk of falling; thus, a

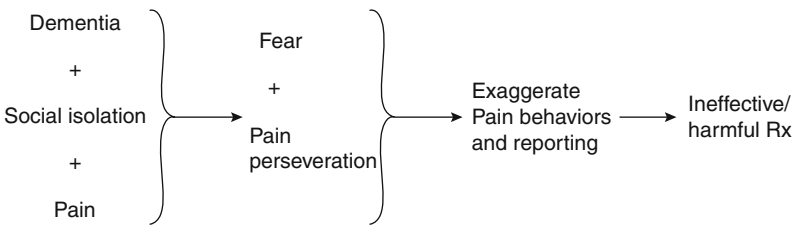


**Fig. 88.3** Synthesis of Case 2. For details, see text

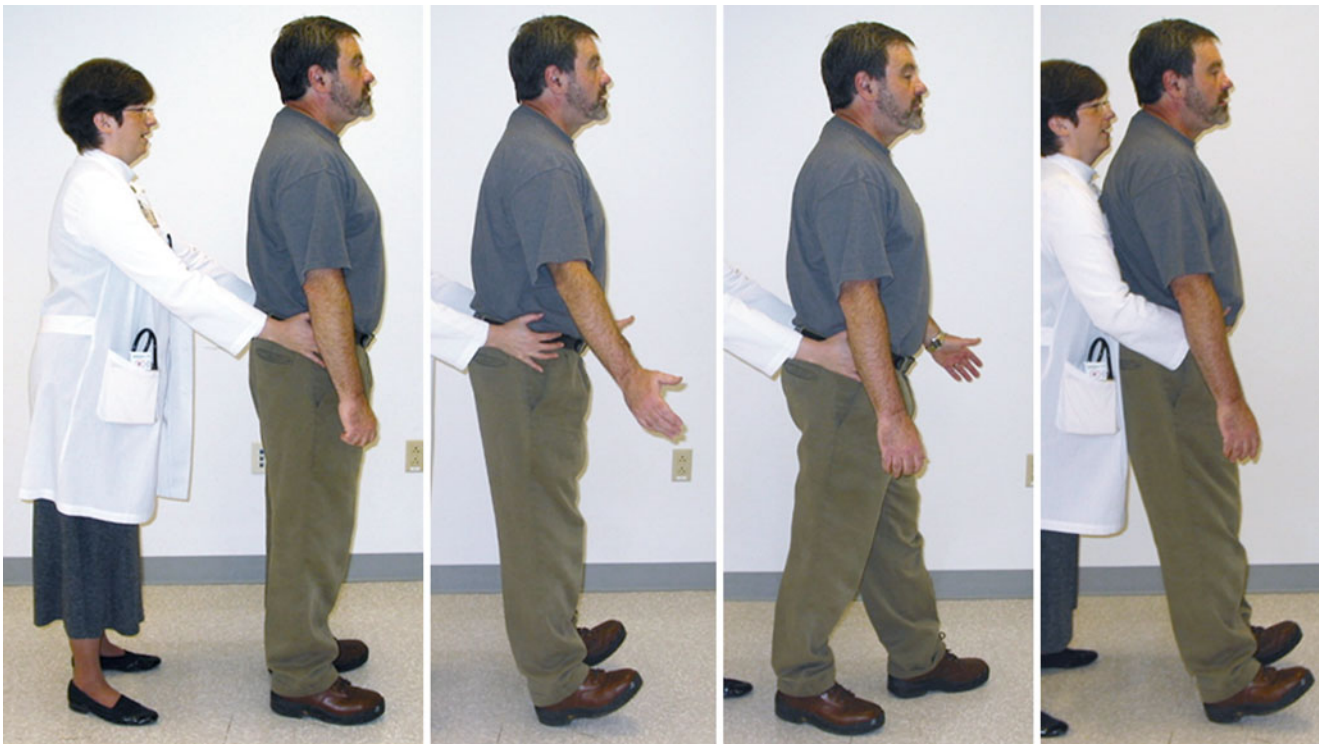

**Fig. 88.4** Modified postural stress test. The highest level postural response (i.e., associated with the best balance) is shown on the far left, where there is no obvious movement in response to attempted perturbation. The lowest level "timber response" is shown on the right, where the patient makes no effort to recover upright stance. This response is highly unusual and typically indicates severe supratentorial dysfunction. The middle two photographs depict intermediary responses.

balance screen should also be included as part of the baseline assessment. A modified postural stress test [94] can readily be done in the office and is described in Table 88.2 and shown in Fig. 88.4. If this test reveals poor balance, a referral to physical therapy should precede any intervention that could further impair balance (e.g., opioid prescription).

## Case 3

An 85-year-old man with advanced Alzheimer's disease presented, along with his wife of 60 years and their daughter, for treatment recommendations to address "persistent reporting of pain" in his lower back. His primary care provider was concerned because the patient's pain ratings had not changed despite numerous analgesic prescriptions. Most recently, he had been prescribed fentanyl that had been titrated to a dosage of 100 mcg/72 h and resulted in hospitalization because the patient became semicomatose. When the dosage was decreased to 50 mcg/72 h, his mental status returned to baseline and he continued to report pain so a pain clinic consult was requested.

At the time of the evaluation, he was sitting in a wheelchair, appeared very comfortable, smiled throughout most of the interview, and had no pain complaints. His history was unreliable because of advanced dementia. His wife reported that the patient had low back pain for many years. She was asked, "Do you think your husband is suffering from his pain, or is he just talking about it?" Without hesitation, she replied, "Oh…he's just talking about it." Together, we decided that the most appropriate treatment would include tapering him off the fentanyl and have him participate in a local day care program for socialization and distraction. His family was educated about the fact that patients with chronic low back pain cannot be made pain-free and that the main goal of treatment is preservation and/or improvement in function to the extent possible. She understood and fully supported the plan.

*Discussion*: The synthesis of this case is presented in Fig. 88.5 and reinforces the complexities of pain evaluation and management in older adults with dementia. In this patient, chronic pain was the weak link and pain perseveration the treatment target. Pain perseveration was also the major component of his pain signature. The main potentially treatment-limiting comorbidity was his dementia (i.e., increased risk of falls and/or delirium with opioids). As opposed to the patient in *Case 2* whose pain reporting reflected pain perseveration as a general signal of distress, this patient's pain reporting was a simple representation of pain perseveration that was treated with distraction. Often, this type of perseveration behavior in older adults with

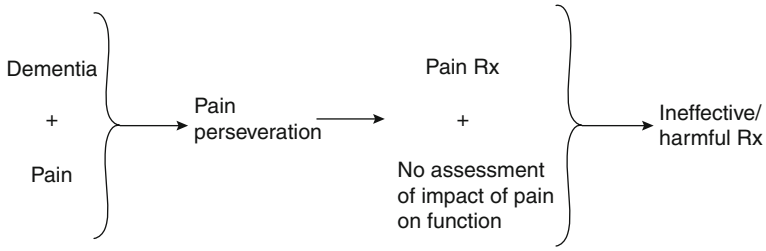


**Fig. 88.5** Synthesis of Case 3. For details, see text

dementia is more a problem for the caregiver (i.e., it is stressful to observe the perceived suffering of a loved one, contributing to caregiver burden) than for the patient, and treatment strategies should keep this in mind.

This case also highlights the importance of patient-centered or patient/caregiver-centered decision making. In busy office practices, it may be difficult to take the extra time required to engage in these discussions. Not doing so, however, may lead to unnecessary morbidity, as was the case with this patient.

## Case 4

A 67-year-old man presented with low back and left leg pain for 10 months. He had injured his back nearly 50 years earlier associated with heavy lifting. He was treated conservatively and his pain abated within 1–2 months. Ten months ago, he experienced the insidious onset of sharp/burning pain in his left lower back with occasional radiation to the left leg (lateral aspect) that was getting progressively more severe. He reported occasional weakness and numbness of the left leg and progressively more restricted walking tolerance. At the time of presentation, he ambulated with a walking stick and could go one-half block before he had to stop because of pain. He reported multiple falls because his leg "gave way." His pain was worsened by lying prone and trying to straighten his leg while lying supine. It was made better by lying on his side and assuming a fetal position. He denied fever, chills, or change in his bowels/bladder.

A lumbar MRI performed 2 months following the onset of his pain revealed diffuse lumbar spondylosis and moderate central canal stenosis. Treatment had included (1) physical therapy for lumbar spinal stenosis that resulted in no improvement in his pain or function; (2) tramadol that was ineffective; (3) gabapentin that caused nausea, vomiting, and a 15-lb weight loss; and (4) hydrocodone/acetaminophen that was associated with moderate pain relief. Spinal surgery was recommended, but he declined. His only significant medical comorbidity was hypertension.

Notable on physical examination was blood pressure 178/96, ¾ in. leg length discrepancy, mild scoliosis, mild left piriformis tenderness, and an antalgic gait with favoring of the left leg. His gait was slow but steady when performed with his walking stick. Examination of the left hip revealed <15° painful internal rotation. Right hip exam was normal. Neurological exam revealed symmetrical lower extremity reflexes and 5/5 strength throughout with the exception of the left hip flexors and left quadriceps that were 4/5. When the patient was lying supine and asked to raise his left leg, he did so by picking it up with his hands. Hip x-rays revealed marked joint space narrowing of the superior and inferior aspects on the left and no abnormalities on the right. Based on these findings, he was instructed to continue regularly scheduled hydrocodone/acetaminophen and he was referred to physical therapy specifically directed toward the left hip. If these strategies are ineffective, he will be referred for intra-articular hip injection. If he is refractory to all noninvasive and minimally invasive treatments, he will be referred for consideration of a total hip replacement.

*Discussion*: The synthesis of this case is shown in Fig. 88.6. The treatment target was his hip osteoarthritis. His pain signature consisted of severe self-reported pain and difficulty walking. His significant comorbidities included hypertension and difficulty walking/falls. Because his symptoms were initially attributed to the lumbar spine, he underwent an unnecessary lumbar MRI and was prescribed medications that resulted in significant adverse events, physical therapy that was ineffective, and a referral for spinal surgery that would likely not have relieved the "pain generator."

This case is presented to highlight the important contribution of hip osteoarthritis to low back pain. The hip-spine syndrome was first described in 1983 and refers to symptoms that exist in the setting of concurrent degenerative pathology in the hip and spine [95]. Three types of hip-spine syndrome were postulated: (1) *simple,* when history and physical examination clearly indicate whether the hip or the spine is the primary source of pain; (2) *complex,* when both the hip and the spine are responsible for pain; these cases are said to require ancillary investigations such as nerve root infiltration and intra-articular blocks of the hip joint to disentangle the primary source of pain; and (3) *secondary,* when altered hip function (e.g., flexion deformity with advanced OA) directly changes spinal biomechanics that cause low back pain. The contribution of hip OA to CLBP also is supported by more recent data. Specifically, total hip replacement surgery for patients with severe hip pain and advanced OA on x-ray

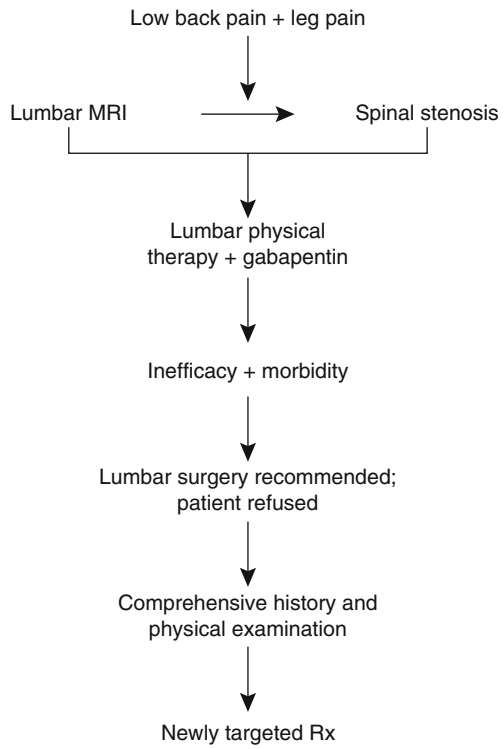


**Fig. 88.6** Synthesis of Case 4. For details, see text. *MRI* magnetic resonance imaging

reduces low back pain and improves overall spine function [96]. In patients with low back pain, diminished hip range of motion predicts poor outcomes following spinal manipulation [97] and after lumbar percutaneous electrical nerve stimulation (unpublished data). Preliminary data suggest that patients with self-reported hip OA respond less favorably to decompressive laminectomy for the treatment of lumbar spinal stenosis (LSS) than those without hip OA [98].

It is likely that many older adults have hip-spine syndrome that is both complex and secondary in which both the hip and spine are pain generators, but altered hip function causes abnormal spinal biomechanics and low back pain, that is, altered hip function adds insult to injury. Although severe hip flexion deformity may be absent, we hypothesize that underlying lumbar spondylosis makes the lower back vulnerable and, therefore, more modest alterations in hip function may be needed to cause low back pain. So, the lumbar spine is the weak link and the hip is the treatment target. Well-controlled studies are needed to test this hypothesis. Until definitive answers are available, practitioners must approach the older adult with low back and/or leg pain using a broad perspective to avoid unnecessary "diagnostics" and misguided/potentially harmful treatments. Table 88.3 highlights key history and physical examination differences between pain generated by lumbosacral degeneration and that associated with hip OA. It should be noted that hip x-rays alone cannot be used to make a diagnosis of clinically meaningful hip OA. Fewer than 50 % of patients with radiographic evidence of hip OA report pain [11]. A definitive diagnosis of hip OA should be based on a combination of clinical examination and x-ray findings [99, 100]. Thus, careful examination of the hip should be a routine part of evaluating all older adults who present with low back and/or leg pain.

## Treatment Guidelines

The overarching goal of treatment for the older adult with chronic pain is to optimize function and quality of life while minimizing the potential for adverse effects associated with treatment. To accomplish this goal, an integrative stepped-care approach that combines non-pharmacological and pharmacological modalities is recommended. Specific recommendations for treating older adults with nociceptive pain, neuropathic pain, and widespread pain are provided below.

### Nociceptive Pain

Figure 88.7 depicts an integrative stepped-care approach for the treatment of nociceptive pain. Topical preparations, cognitive behavioral therapy, interdisciplinary pain treatment, and complementary and alternative modalities (CAM) may be used at any step, either alone or in combination. The individual steps shown in Fig. 88.7 are arranged from treatments associated with relatively low risk (step 1) to those associated with high risk (step 7).

At the foundation of treatment are education, weight loss, exercise, and other physical therapy approaches (including assistive devices). Sometimes, these approaches are alone sufficient to accomplish desired outcomes. For the older adult with fibromyalgia who is capable of participating in aerobic exercise, no further treatment may be needed. For the patient with kyphosis related to vertebral compression fractures and associated lumbar strain, a four-wheeled walker often is very effective for reducing pain and improving mobility. Education should be targeted at ensuring realistic treatment expectations (i.e., pain reduction but not elimination and improved function despite the persistence of pain) and quelling any pain-associated fears (e.g., becoming crippled and/or losing independence because of pain, having cancer associated with pain).

To avoid risks associated with systemic medication, injections should be considered for the older adult with pain in one or two joints, for example, knee osteoarthritis (OA). Trigger point injections can be an effective adjunct for treating myofascial pain syndromes [101]. There is no strong evidence to guide the prescription of spinal injections for older adults with chronic low back pain (CLBP). In general, injection

**Table 88.3** Differentiation of lumbosacral from hip-generated low back/leg pain

| Feature | Lumbosacral | Hip | Comments |
|---|---|---|---|
| Pain location | Above pelvis; if comorbid spinal stenosis, pain may involve buttocks and/or legs | Most common referral patterns are buttocks, groin, and thigh | If sacroiliac joint syndrome (SIJS) complicates hip disease, SI pain can coexist with buttocks/groin/thigh pain |
| Leg pain | Present if comorbid spinal stenosis or knee/hip disease | Often | Radiculopathy pain typically extends the entire leg. Although hip pain can be referred to the lower leg and/or foot, most commonly it involves the buttocks, groin, and thigh |
| Groin pain | Absent | Often | SI pain can be referred to the groin, so if SIJS complicates lumbosacral pathology, groin pain can occur |
| Movements that aggravate pain | Spinal extension | Leg extension<br>Hip internal rotation | If comorbid SIJS, side lying and/or flexion may worsen pain |
| Movements that alleviate pain | Spinal flexion | Hip flexion<br>Hip external rotation | If spine and hip disease co-occur, response to movement patterns may be atypical |
| Posture | Spinal flexion | Leans forward, with flexion at the hip | When spine or hip disease is mild, there may be no obvious postural abnormalities |
| Associated symptoms | Paresthesias, radiculopathic pain, lower extremity weakness | Lower extremity weakness | If spine and hip disease co-occur, symptoms can overlap |
| X-ray findings | Poor predictive validity for pain | Poor predictive validity for pain | Degenerative disease of the lumbar spine exists in >90 % of older adults without low back pain [8], and spinal stenosis is not uncommon in older adults [9] |
| | | | 53 % of women with radiographic hip OA report no pain [11]. A definitive diagnosis of hip osteoarthritis should be based on ACR criteria [99] |

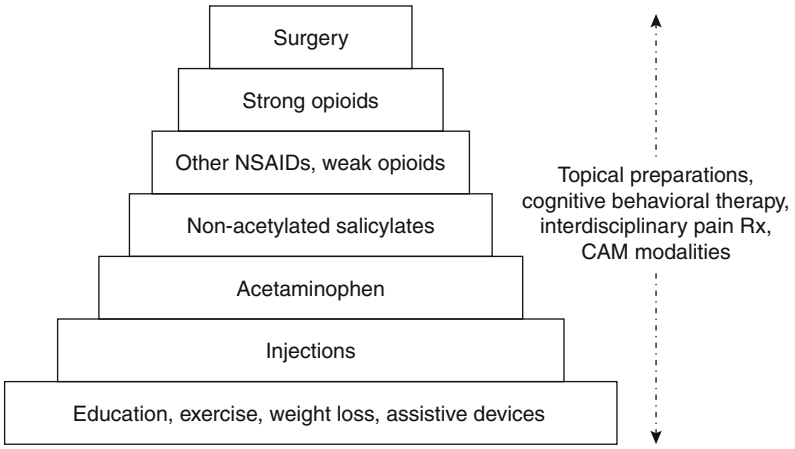


**Fig. 88.7** Stepped-care approach for the treatment of nociceptive pain. *CAM* complementary and alternative medicine, *NSAIDs* nonsteroidal anti-inflammatory drugs (Reprinted from: Weiner and Cayea [178], with permission from Debra Weiner and IASP Press)

therapies should be viewed as a tool to enhance compliance with rehabilitation efforts, which represent the mainstay of nociceptive pain treatment. For older adults with diabetes mellitus, patients should be instructed to monitor their blood sugar carefully following corticosteroid injections.

Systemic pharmacologic treatment of mild to moderate nociceptive pain should start with regularly scheduled acetaminophen because of its relatively safe side effect profile and few drug-drug or drug-disease interactions. Acetaminophen exerts its analgesic effect by weak, reversible, nonspecific cyclooxygenase inhibition, and, therefore, prostaglandin synthesis. It has no anti-inflammatory or antiplatelet effect and uncommonly causes gastrointestinal (GI) bleeding or nephrotoxicity [65]. An overdose of 10 g can cause liver failure and death. Hepatic injury can occur with lower doses when the patient drinks alcohol heavily or is taking hepatic enzyme

inducing medications (e.g., rifampin, carbamazepine, phenytoin, phenobarbital). Preexisting liver disease, malnourishment, fasting, or dehydration can also increase the risk of liver injury. Table 88.4 provides dosing guidelines, pharmacokinetics, key drug-drug and drug-disease interactions, and important adverse effects associated with acetaminophen and other medications used for nociceptive pain. To avoid breakthrough pain, it is important to dose analgesics around the clock [102].

When acetaminophen does not provide adequate analgesia or when an anti-inflammatory effect is needed, a nonacetylated salicylate such as salicylsalicylic acid should be considered [103]. As with all nonsteroidal anti-inflammatory drugs (NSAIDs), these drugs primarily promote analgesia via reversible inhibition of cyclooxygenase-2 that in turn blocks prostaglandin-associated sensitization of peripheral nociceptors [104]. Nonacetylated salicylates have a superior safety profile compared with other NSAIDs. They rarely cause GI bleeding. This is of particular clinical importance as adults over the age of 60 have a 3–4 % risk of bleeding while taking NSAIDs as compared to 1 % of the general population [65]. The nonacetylated salicylates also do not interfere with platelet function. Since many older adults are taking a daily aspirin for underlying diabetes or coronary artery disease, this latter benefit is also clinically relevant. These drugs can be combined with opioids if needed.

If a nonacetylated salicylate fails to relieve pain adequately, traditional NSAIDs or weak opioids can be considered. Because of the serious adverse events associated with NSAIDs, we advise that they be used only for brief periods in the setting of inflammatory disorders (e.g., a 7-day course of ibuprofen for an acute flare of gout or pseudogout). NSAIDs cause gastrointestinal bleeding, ulceration, and perforation. Additionally, because of renal prostaglandin inhibition with associated renal artery vasodilatation, NSAIDs promote fluid retention and may worsen or precipitate congestive heart failure, hypertension, and renal injury; cognitive dysfunction also may occur [65]. Although an NSAID can be combined with misoprostol or a proton-pump inhibitor to reduce the risk of gastrointestinal bleeding, we avoid the chronic use of NSAIDs in older adults whenever possible.

Recommended weak opioids include codeine and hydrocodone. The latter is more potent than codeine (10 mg of hydrocodone is equivalent to 60–80 mg of codeine). Like all opioids, they work by binding to mu receptors in the central nervous system. Their half-life is prolonged in patients with chronic kidney disease. Generally, older adults have an increased pharmacodynamic sensitivity to opioids [66, 105] and are more likely to experience the adverse effects of constipation, sedation, nausea, urinary retention, and cognitive impairment [106]. They are also more at risk for falling, especially if they have preexisting mobility impairment [107]. Because constipation with chronic opioid use is very common, practitioners should anticipate this and advise use of a stimulant laxative such as senna to patients at the first sign of constipation (e.g., 2–3 days without a bowel movement).

While opioids may be required at night, sleep quality may be affected by their nighttime use. Opioids both suppress rapid eye movement (REM) sleep and reduce total time spent in stage 4 (e.g., slow wave or "deep") sleep [108–110]. If sedation or daytime fatigue develops, initiation of methylphenidate (usually at starting doses of 2.5 mg daily or twice daily) can be considered, although large, well-controlled trials are lacking. The novel wake-promoting agents – modafinil and armodafinil – are treatment options for fatigue associated with chronic pain and the sedation commonly encountered with opioid pharmacotherapy. These agents may have safer side effect profiles than central nervous system stimulants, but care must be taken when prescribing for older adults because of potential cardiovascular and elimination concerns. Our recommendations, based on clinical experience, are to initiate treatment with half the recommended starting dose (50 mg/day for modafinil; 25–50 mg/day for armodafinil). Close attention should be paid to increases in blood pressure and heart rate with all of these agents.

Opioids also can cause hypogonadism because they bind to hypothalamic receptors and limit the production of gonadotropin-releasing hormones [111–117]. Estrogen and testosterone production is secondarily reduced resulting in hypogonadism [118–124]. While opioid-induced hypogonadism occurs in both sexes, it is more commonly recognized in men. The symptoms of hypogonadism in older adults include impotence in men and diminished libido in both men and women. Symptomatic improvement is seen after hormone supplementation [125]. Rat studies indicate that low testosterone is associated with increased pain sensitivity [126]. Preliminary evidence also suggests that hypogonadism may limit the antinociceptive properties of opioids [127]. At this time, there are no human studies demonstrating the effect of hypogonadism on pain sensitivity.

Tramadol is a weak mu opioid receptor agonist and blocks the reuptake of norepinephrine and serotonin. It has a similar side effect profile as the typical opioids. Tramadol should be used cautiously or not at all in patients taking other serotonergic medications because of its potential to contribute to serotonin syndrome. Typically used in neuropathic pain, this drug is described in more detail below.

If weak opioids are ineffective, a strong opioid should be considered. Among older adults, oxycodone, the combination of oxycodone/acetaminophen, and morphine are all used commonly. Long-acting preparations of oxycodone and morphine are appropriate in equianalgesic doses for long-term use. Alternative agents such as hydromorphone and

**Table 88.4** Oral analgesics for nociceptive pain

| Medication class | Medication | Recommended dosing | Pharmacokinetics | Key drug-drug interactions | Key drug-disease interactions | Important adverse effects |
|---|---|---|---|---|---|---|
| Other analgesic | Acetaminophen | 325–1,000 mg q 4–6 h<br>Maximum daily dose 4,000 mg | Metabolized via glucuronidation<br>Clearance may be reduced in frail older adults | Hepatic injury can occur with modest doses when concomitant use of hepatic enzyme inducing medications (e.g., rifampin, carbamazepine, phenytoin, phenobarbital). | None | Hepatic necrosis with acute 10 g ingestion or chronic use of >4 g/day.<br>Increased toxicity from chronic use occurs with heavy alcohol use, malnourishment, pre-exiting liver disease – decrease maximum daily dose to 2 g<br>Nephrotoxicity (dose dependent) |
| Non-acetylated salicylates | Salsalate | 500–750 mg bid<br>Maximum dose 3,000 mg/day | Metabolized by hydrolysis to salicylate; also metabolized via glucuronidation | No significant drug-drug interactions | See other NSAIDs below | Does not interfere with platelet function; GI bleeding rare |
| Other NSAIDs | Ibuprofen<br>Naproxen | 400 mg tid-qid<br>250–500 mg bid | CYP2C9/19<br>CYP2C9 and CYP1A2; clearance significantly reduced with advanced age | Concomitant use of NSAIDs with diuretics and antihypertensives may decrease their effectiveness. Use with corticosteroids and/or warfarin increases the risk of peptic ulcer disease. Increases concentration of lithium and methotrexate | Use NSAIDs with caution in patients with chronic renal failure, heart failure, hypertension, and peptic ulcer disease history | Risk of GI bleeding increased in persons ≥ 60 years. Cognitive impairment possible with higher doses.<br>These NSAIDs should be reserved for short-term use in the older adult. |
| Cyclooxygenase (COX-2) inhibitor | Celecoxib | 100 mg bid | CYP2C9/19 | Same as NSAIDs above | Same as NSAIDs above | Because of relatively long half-life, naproxen should not be first choice for older adults.<br>COX-2 inhibitor has less GI toxicity, but similar renal toxicity to other NSAIDS.<br>Given long half-life and perhaps greater cardiac toxicity make it not a preferred agent for older adults. |
| Weak opioids | Codeine<br>Hydrocodone | 15–30 mg q 4–6 h<br>5–10 mg q 4–6 h (alone or in combination with acetaminophen) | Prodrug metabolized by CYP2D6<br>CYP2D6;not studied in older adults | Few clinically significant drug-drug interactions. Quinidine can inhibit the analgesic effect of codeine. | For all opioids, increased risk of falls in patients with dysmobility. May worsen or precipitate urinary retention when BPH present. Increased risk of delirium in those with dementia.<br>Codeine has active renal metabolites that can accumulate with advancing age and renal insufficiency. | Because of increased sensitivity to opioids older adults at greater risk for sedation, nausea, vomiting, constipation, urinary retention, respiratory depression, and cognitive impairment. |

(continued)

**Table 88.4** (continued)

| | | | | | | |
|---|---|---|---|---|---|---|
| Opiate receptor agonist/SNRI | Tramadol | Initiate at 25 mg qd. Increase by 25–50 mg daily in divided doses every 3–7 days as tolerated to max dose of 100 mg Four times a day (QID). Renal dosing 100 mg Twice a day (BID). | Prodrug metabolized by CYP2D4, 2B6 and 2D6. | Sedative medications and other opioids. Risk of serotonin syndrome in combination with SSRI and triptans. Seizure risk with MAOI. Rare reports of interaction with warfarin and digoxin. Quinidine can inhibit the analgesic effect. | Seizure disorder (avoid if history of seizures).<br>Adjust dose with renal insufficiency; maximum dose 100 mg bid. | Seizures and orthostatic hypotension. Other side effects similar to traditional opioids including sedation, confusion, respiratory depression. |
| Strong opioids | Oxycodone (short and long-acting) | Start with 5 mg (short acting) q 4–6 h; after 7 days, determine dose requirements, then convert to long acting. | CYP2D6 | As above | As above<br>Oxycodone and morphine have active renal metabolites that can accumulate with advancing age and renal insufficiency. | As above<br>Stong opioids to avoid in older adults: pentazocine, meperidine. |
| | Morphine (short and long-acting) | Start with 2.5 mg q 4 h and titrate by 2.5 mg increments q 7 days. Convert to long acting after dosing requirements determined. | Large first pass effect and high hepatic extraction ratio results in higher serum levels and decreased clearance; glucuronidation to active renally cleared metabolites. | | | |
| | Hydromorphone | Start with 2 mg q 4 h. Increase after 7 days if needed. | Glucuronidation; not studied in older adults | | | Dose of opioid required (for weak, strong, and tramadol) can be reduced by combining with a non-opioid agent such as acetaminophen. |
| | Fentanyl transdermal | Start with 12 mcg patch q 72 h. If ineffective after 1 week, increase to 25 mcg. 48 h dosing interval may be required. | CYP3A4 | | | Constipation is very common with all opioids, but not universal; a stimulant laxative (e.g., senna) should be prescribed if needed. |
| | Methadone | Start with 1 mg q 12–24 h (po, buccal, sc). Titrate ≥ q 7d. | CYP3A4; not studied in older adults | Phentyoin can increase clearance | | Fentanyl patch, methadone and other sustained-release opioids should be avoided in those who are opioid-naïve.<br>EKG should be obtained and monitored in those on methadone, as may be associated with prolonged QT interval. |

fentanyl also can be considered. Although methadone has a long and variable half-life, it can be a very effective analgesic [128]. Meperidine and pentazocine should not be prescribed in the older adult because of enhanced toxicity. For the patient who benefits from opioids but has limiting side effects, an intrathecal opioid pump might be considered.

While many complementary and alternative modalities are not covered by third-party payers, the evidence base for their efficacy in older adults is growing. Data indicate that lumbar percutaneous electrical nerve stimulation is effective for the treatment of chronic low back pain (CLBP), although the minimally effective dose of electrical stimulation has not been determined [56, 57]. Preliminary data indicate that mindfulness meditation reduces pain interference with daily activities in older adults with CLBP [61]. Periosteal stimulation has short-term benefits in reducing pain and improving function in older adults with chronic knee pain and advanced osteoarthritis [129]. Tai Chi and hypnosis may help improve osteoarthritis-associated pain and functional limitations [130, 131]. Given the toxicities associated with pharmacological management of chronic pain in older adults, additional research is needed to expand the scope of proven complementary and alternative modalities that have a favorable risk profile [132].

Practitioners should be aware that vitamin D deficiency is not uncommon in older adults and can contribute to pain, wasting, weakness, and gait instability/falls [133, 134]. Over the years, the recommended serum vitamin D level has varied. Recent studies suggest that 25-OH vitamin D levels between 30–32 ng/mL are optimal to prevent fractures and secondary hyperparathyroidism [135–140]. For patients with vitamin D levels below 20 ng/mL, we recommend supplementation with 50,000 IU once weekly for 3 months and then serum levels should be rechecked. If the level is normal, the patient should be placed on 1,000 IU daily for maintenance. If the vitamin D level is between 20 and 30 ng/mL, patients may be supplemented with 1,000 IU daily. Other studies recommend more aggressive vitamin D supplementation with 50,000 IU biweekly for 3 months in patients with levels below 10 ng/mL. Supplementation with 50,000 IU once weekly for 3 months is recommended for those with levels between 10 and 32 ng/mL [135].

Vitamin D supplementation is well tolerated in older adults and may have considerable benefits. Vitamin D and calcium supplementation may reduce hip and non-vertebral fractures and fall risk [141–143]. A recent study of statin-associated myalgia demonstrated symptomatic improvement after vitamin D supplementation in deficient patients [144]. Other studies have demonstrated improvement of nonspecific muscle pain after vitamin D supplementation in deficient patients [145]. In one case series, chronic back pain and failed back surgery syndrome improved after vitamin D supplementation [146], although studies have had conflicting results. Despite contradictory data on the relationship between vitamin D levels and fibromyalgia pain [147], we routinely measure vitamin D levels in these patients and supplement if insufficient levels are found.

## Neuropathic Pain

An algorithmic approach to the treatment of neuropathic pain is depicted in Fig. 88.8. Monotherapy with an antidepressant or anticonvulsant is the standard-of-care first-line approach for generalized neuropathic pain. For severe pain, an opioid alone or combined with another drug may be necessary. Topical preparations and peripheral nerve blockade are effective for localized symptoms and may be combined with systemic treatments. Those with intractable symptoms may benefit from interventional treatments such as spinal cord and peripheral nerve stimulation.

Table 88.5 contains guidelines for dosing, pharmacokinetics, key drug-drug and drug-disease interactions, and important adverse effects associated with medications used to treat neuropathic pain. Gabapentin and pregabalin have no significant end-organ toxicities and are safe for long-term use in older adults. When initiating and/or titrating these medications, practitioners must be vigilant for the development of sedation, confusion, and/or gait unsteadiness. Starting with a low dose and titrating, these medications slowly can help to avoid these side effects. Weight gain and peripheral edema also occur not uncommonly.

Secondary tricyclic antidepressants (TCAs) are well studied for the treatment of neuropathic pain and provide effective analgesia at approximately 30–50 % of the antidepressant dose [148]. In general, caution should be exercised when prescribing this class of medications for older adults. Amitriptyline has the greatest anticholinergic potential and is contraindicated in older adults. If the practitioner wishes to prescribe a TCA, desipramine and nortriptyline are preferred agents. TCAs in general are contraindicated in patients with a history of myocardial infarction, QT prolongation, and/or bundle branch block, and a screening EKG should always be obtained prior to initiating them in older adults [149]. They also are contraindicated in patients with untreated narrow-angle glaucoma because of their potential to exacerbate this condition. Other commonly encountered anticholinergic side effects include sedation, confusion, dizziness, xerostomia, constipation, gait unsteadiness/falls, and urinary retention [107, 150]. For older adults with medical comorbidities that themselves contribute to these symptoms (e.g., urinary hesitancy in the older male with prostatism, poor cognitive function in the patient with dementia, gait unsteadiness typically related to multiple factors), an alternative to TCAs should be considered.

The newer serotonin and norepinephrine reuptake inhibitor (SNRI) antidepressants, duloxetine and venlafaxine, and

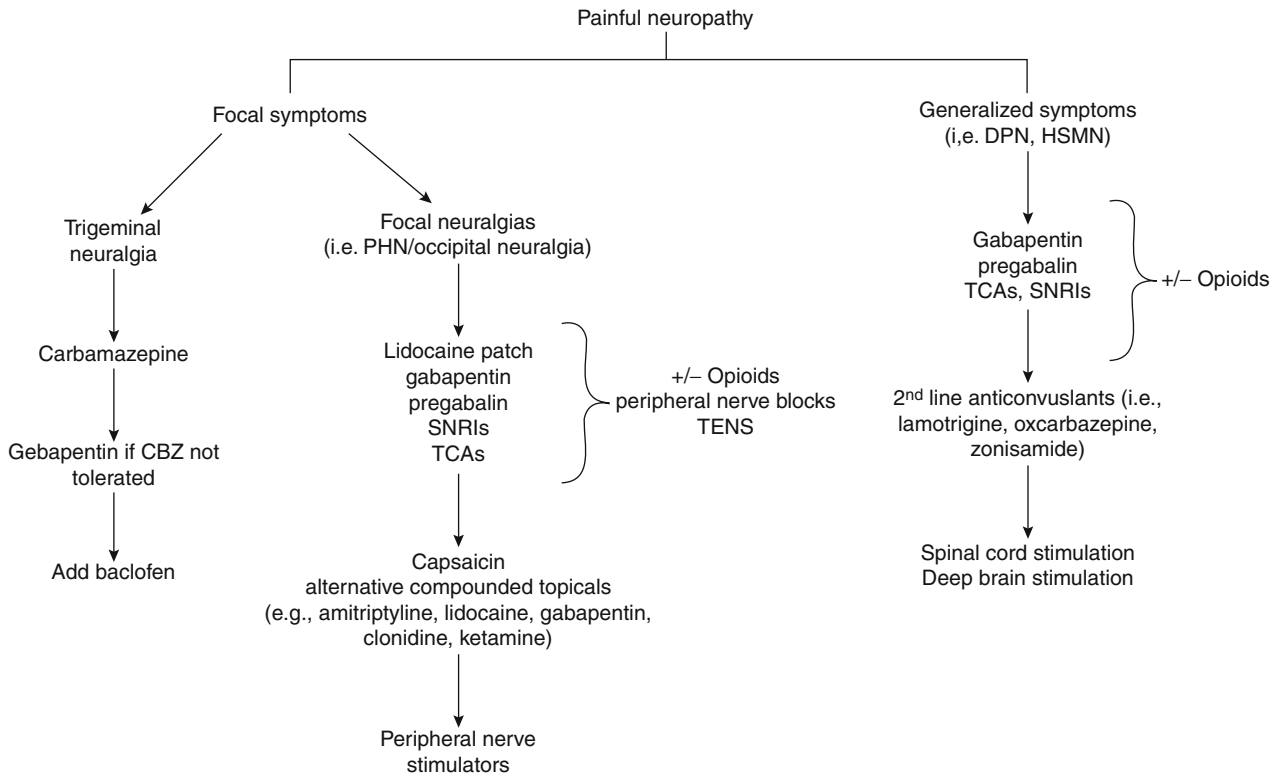


**Fig. 88.8** Algorithmic approach for the treatment of painful neuropathy. *CBZ* carbamazepine, *DPN* diabetic peripheral neuropathy, *HSMN* hereditary sensory/motor neuropathy, *PHN* postherpetic neuralgia, *SNRI* serotonin/norepinephrine reuptake inhibitor, *TENS* transcutaneous electrical nerve stimulation, *TCA* tricyclic antidepressant

o-desmethylvenlafaxine are effective for neuropathic pain and have fewer side effects than the TCAs [151, 152]. Duloxetine is Food and Drug Administration (FDA) approved for painful diabetic neuropathy. It is contraindicated for patients with uncontrolled narrow-angle glaucoma and should ordinarily not be prescribed to patients with substantial alcohol use or evidence of chronic liver disease. Nausea (especially during induction or dose escalations) and orthostatic hypotension are not uncommon adverse drug reactions. Venlafaxine, o-desmethylvenlafaxine, and duloxetine, in addition to causing orthostatic hypotension [153], can cause sustained elevations in blood pressure, may lower seizure threshold in patients with a history of seizure, and increase the risk of abnormal bleeding, especially when co-prescribed with NSAIDs, aspirin, or other drugs that affect coagulation.

If monotherapy with a first-line anticonvulsant or antidepressant provides suboptimal analgesia, these medications may be combined. Combining modest doses of gabapentin and morphine is more effective than larger doses of either drug alone [154]. Second-line anticonvulsants, lamotrigine, oxcarbazepine, and zonisamide, are effective for some types of neuropathic pain including painful diabetic polyneuropathy [155, 156].

Opioid analgesics are first-line options for moderate to severe nerve pain [157]. Traditional opioids as well as methadone, a long-acting opioid and N-Methyl-D-aspartic acid (NMDA) antagonist, are effective. Tramadol, a weak mu-receptor agonist with serotonin and norepinephrine reuptake blockade, also is effective for neuropathic pain [158, 159]. Serotonin syndrome may occur when tramadol is combined with other serotonergic medications (e.g., triptans, various antidepressants). Tramadol also lowers the seizure threshold and increases the risk of seizure in patients taking serotonin reuptake inhibitors, tricyclic antidepressants and other tricyclic compounds (e.g., cyclobenzaprine), and other opioids. At therapeutic doses, tramadol has no effect on heart rate, left-ventricular function, or cardiac index, although orthostatic hypotension has been observed.

While tramadol is currently approved for the treatment of moderate to moderately severe chronic pain in adults, a newer compound, tapentadol, is approved for the treatment of moderate to severe acute pain in adults [160]. The analgesic efficacy of tapentadol is thought to occur via mu-receptor agonism and norepinephrine reuptake blockade. The side effect profile of tapentadol is similar to tramadol, but given its recent release, it has not been as extensively used with

**Table 88.5** Oral analgesics for neuropathic pain

| Medication class | Medication | Recommended dosing | Pharmacokinetics | Key drug-drug interactions | Key drug-disease interactions | Important adverse effects |
|---|---|---|---|---|---|---|
| Anticonvulsants | Gabapentin | Initiate at 100 mg nightly. Increase by 100 mg weekly. | Renal elimination. Nonlinear pharmacokinetics. Plasma concentration increases disproportionately to dose | Other CNS/sedative medications | Dementia, ataxia | Confusion, dizziness, somnolence, peripheral edema, weight gain. Withdrawal syndrome with abrupt discontinuation |
| | | Renal dosing: CLcr 30–59 mg/min, titrate to 600 mg bid | | | | |
| | | CLcr 15–29 mg/min, titrate to 300 mg bid | | | | |
| | | CLcr <15 mg/min, titrate to 300 mg qd | | | | |
| | | Supplemental dosing after dialysis | | | | |
| | Pregabalin | Initiate at 25–50 mg nightly. Increase by 25–50 mg weekly up to 100 mg BID. Max dose 300 mg Once a day (QD) | Renal elimination. Linear pharmacokinetics (plasma concentration is dose proportionate) | Other CNS/sedative medications | Dementia, ataxia | Confusion, dizziness, somnolence, peripheral edema, weight gain |
| | | Renal dosing: CLcr 30–60 mg/min adjust dose to 150–300 mg QD. CLcr 15–30 mg/min adjust dose to 75–150 mg QD. CLcr <15 mg/min adjust dose to 25–50 QD | | | | |
| | | Supplement dose after dialysis | | | | |
| | Carbamazepine | Initiate at 50 mg nightly. Increase by 50 mg every week up to 100 mg BID. Target dose 200–600 mg QD. Max dose 1,200 mg QD. Adjust dose for serum levels (4–12 mg/L). Patients on multiple CNS medications may have toxicity at lower serum levels. | Metabolized by CYP450 3A4; induces CYP450 | CYP3A4 inhibitors increase serum CBZ levels. CYP3A4 inducers decrease serum CBZ levels<br>CBZ induces hepatic activity and can lower concentration of numerous drugs | | Slurred speech, gait instability, poor coordination; Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH); rare severe reactions: Stevens-Johnson syndrome, aplastic anemia, hepatotoxicity, drug-induced lupus |
| | | | | | | Monitor LFTs, CBC, and serum sodium after initiation and during treatment. |
| TCAs | Nortriptyline<br>Desipramine | 10 mg at night. Increase by 10 mg weekly. Max dose 50 mg at night | Metabolized by CYP2D6 | CYP2D6 inhibitors increase serum levels. CYP2D6 inducers decrease serum levels<br>Other CNS/sedative medications | Myocardial infarction and bundle branch block; seizures, narrow-angle glaucoma, prostatic hypertrophy; dementia, falls | Arrhythmia, prolongation of QT interval and conduction block. Severe cases may lead to torsades de pointes. EKG prior to use in older adults. Orthostatic hypotension, sedation, confusion, constipation, urinary retention, SIADH |

(continued)

**Table 88.5** (continued)

| Medication class | Medication | Recommended dosing | Pharmacokinetics | Key drug-drug interactions | Key drug-disease interactions | Important adverse effects |
|---|---|---|---|---|---|---|
| Dual reuptake inhibitors (SNRIs) | Venlafaxine | Initiate at 37.5 mg daily. Increase by 37.5 mg weekly up to 150 mg daily. Max dose 225 mg daily | Metabolized by CYP2D6 | Contraindicated within 14 days of MAOI use. May precipitate serotonin syndrome when combined with triptans, tramadol, and other antidepressants. | HTN and uncontrolled narrow-angle glaucoma. Precipitation of mania in bipolar disorder | Sedation/falls, insomnia, nausea, xerostomia, and constipation. Abrupt discontinuation may result in withdrawal syndrome. |
| | Duloxetine | Initiate at 20–30 mg daily. Increase to 60 mg after 1 week. Max dose 60 mg. Not recommended in ESRD or with CLcr < 30 mL/min. | Metabolized by CYP1A2 and CYP2D6 | CYP2D6 inhibitors; contraindicated within 14 days of MAOI use<br>May precipitate serotonin syndrome when combined with triptans, tramadol, and other antidepressants. | HTN, uncontrolled narrow-angle glaucoma, and seizure disorder. Precipitation of mania in patients with bipolar disorder | Nausea, dry mouth, sedation/falls, urinary retention, and constipation. Abrupt discontinuation may result in withdrawal syndrome and low risk of hepatotoxicity, but contraindicated with hepatic disease and heavy alcohol |
| Topical agents | Lidocaine patch 5 % | 1–3 patches topically 12 h on 12 h off. Only 3 ± 2 % of lidocaine patch absorbed. 95 % lidocaine remains in patch form | Hepatically metabolized<br>Unknown if lidocaine is metabolized in the skin. Negligible serum metabolite levels after topical application, that is, minimal systemic absorption | Use with caution in patients taking class I antiarrhythmics (tocainamide and mexiletine). | Severe hepatic disease and non-intact skin | Site reactions.<br>Symptoms of lidocaine toxicity are rare and include nausea, nervousness, tinnitus, metallic taste, confusion, and tremor. Toxicity seen with serum lidocaine levels of >5 mcg/mL. Serum levels with lidocaine patch typically 0.13 mcg/mL |
| | Capsaicin | Apply thin layer to affected area QID | Cutaneous action. Maximum effect noted after 4–6 weeks of use | None known | Irritation on non-intact skin | Skin reactions and burning. Avoid contact with eyes and sensitive skin areas. Respiratory irritation/cough if inhaled |
| Muscle relaxants | Baclofen | Initiate at 5 mg nightly. Increase by 5 mg every week as tolerated. Max dose 10 mg TID. Dose adjustment with renal insufficiency | Minimal metabolism; 85 % excreted unchanged in liver and feces | CNS depressants | Ataxia, renal disease, dementia, and seizures | Not recommended for older adults. Confusion, nausea, and sedation<br>Abrupt withdrawal syndrome with hallucinations, seizures, muscle rigidity, and high fever. If severe, may lead to rhabdomyolysis, multi-organ system failure, and death |
| Opioid analgesics | See Table 88.4 | | | | | |

*CBC* complete blood count, *CBZ* Carbamazepine, *CLcr* creatinine clearance, *CNS* central nervous system, *CYP* Cytochrome P450, *EKG* electrocardiogram, *ESRD* end-stage renal disease, *HTN* hypertension, *LFTs* liver function tests, *MAOI* monoamine oxidase inhibitor, *NSAID* nonsteroidal anti-inflammatory drug

older adults. The potential side effects associated with opioids are numerous and described earlier in this chapter.

Focal nerve pain is often amenable to treatment with peripheral nerve blockade, topical treatments, and transcutaneous electrical stimulation. Depending on the older adult's risk profile, these treatments may be chosen as first line for those with localized pain. Peripheral nerve blocks with local anesthetics and steroid are used to treat ilioinguinal, occipital, and postherpetic neuralgia. Complications from these interventions are rare and include bleeding and infection. The lidocaine patch and other compounded topical medications (e.g., gabapentin, clonidine, amitriptyline, ketamine either alone or combined) may be beneficial. Capsaicin relieves painful symptoms but may itself be painful and requires 4–6 weeks of use before taking effect.

As noted earlier in this chapter, treatment of comorbid myofascial pain (MP) in older adults with focal nerve pain may result in dramatic pain reduction, as evidenced by our clinical experience with a number of older adults who presented with refractory postherpetic neuralgia [51]. In these patients, pain reduction related to successful non-pharmacological treatment of MP afforded significant dose reduction or complete discontinuation of opioids.

Neuropathic pain secondary to trigeminal neuralgia (TN) is unique and may respond to treatment with carbamazepine (CBZ). Compared to other anticonvulsants, however, CBZ may be less well tolerated [161]. The risk of serious dermatologic reactions (e.g., Stevens-Johnson syndrome), aplastic anemia and agranulocytosis, and hyponatremia must be weighed prior to initiating treatment with CBZ. Gabapentin, while less effective for TN, is a reasonable alternative for those older adults who do not tolerate CBZ. Baclofen, a muscle relaxant, is effective for TN and may be combined with CBZ or gabapentin [162], but muscle relaxants generally should be avoided in older adults as highlighted in the 2012 [163].

Multidisciplinary pain treatment combining physical therapy, occupational therapy, and psychology may be beneficial for those with refractory symptoms. As for patients with nociceptive pain, those with neuropathic pain who benefit from opioids but have limiting side effects, an intrathecal opioid pump might be considered. Spinal cord or peripheral nerve stimulation is a final resort for those who fail systemic, topical, and other non-pharmacological treatments [164, 165]. Motor cortex stimulation may treat severe neuropathic pain involving the face or as a result of intracerebral pathology (i.e., stroke) [166]. A psychological evaluation is required prior to these invasive procedures.

## Widespread Pain

Fibromyalgia (FMS) syndrome, the classical condition defined by widespread musculoskeletal pain from which older adults suffer, affects 7 % of community-dwelling women aged 60–79 [167]. Diagnosis requires a history of pain in at least three of four body quadrants lasting at least 3 months and pain with palpation (using 4 kgf) at 11 or more of 18 specific points on the body [168]. Morning stiffness, fatigue, nonrestorative sleep, neuropsychiatric disturbances (e.g., impaired memory, depression), paresthesias, and irritable bowel and bladder symptoms commonly accompany FMS. Depression and/or anxiety should be screened routinely, given their common co-occurrence in FMS and their potential for interfering with analgesic efficacy and treatment adherence.

The first step in treating the older adult with FMS is education. This is especially relevant for older adults and their caregivers who may be puzzled and frightened by the presence of widespread pain; it may be interpreted as a life-threatening condition. A patient-centered care model should be adopted so that the patient, physician, and caregiver collaborate in developing a personalized treatment plan. After providing education, evidence-based non-pharmacological and pharmacological treatments should be implemented.

To date, there have been no non-pharmacological or pharmacological treatment studies of FMS restricted to older adults. Although pharmacological approaches are an important mode of treatment, there is no evidence to support long-term benefit and they should never be used without proven non-pharmacological approaches such as cognitive behavioral therapy and aerobic exercise [169]. When depression or anxiety is comorbid, an antidepressant should be utilized. Low-dose tricyclic antidepressants such as nortriptyline or desipramine improve both sleep quality and symptoms on the global assessment scale and lead to improvement in tender point score, pain, and fatigue [170]. Because of the anticholinergic and cardiac side effects noted above, it may be difficult to increase the dose of tricyclics to a level with antidepressant efficacy. Fluoxetine alone or in combination with amitriptyline also has beneficial effects [171]. We do not, however, recommend the use of either fluoxetine or amitriptyline in older adults. Fluoxetine has a long half-life, and as noted earlier in this chapter, amitriptyline is more sedating than other tricyclics and has the highest anticholinergic burden. Symptom improvement was not observed in a randomized, double-blind, placebo-controlled study of citalopram [172].

If depression or anxiety is comorbid with FMS and nortriptyline or desipramine is contraindicated, a serotonin norepinephrine reuptake inhibitor such as duloxetine or milnacipran would be suitable. In a 3-month study, compared to placebo, treatment with duloxetine (60 mg twice daily) resulted in more improvement on the Fibromyalgia Impact Questionnaire (FIQ) and a number of other outcomes, independent of its effect on mood [173]. Milnacipran, twice daily, improved pain and other outcome measures in 125 patients

with FMS over 12 weeks [174]. Duloxetine is not recommended for patients with end-stage renal disease (ESRD) or severe renal impairment (estimated creatinine clearance <30 mL/min). Milnacipran should not be used in patients with ESRD, and in those with creatinine clearance of 5–29 mL/min, the dose should be reduced by 50 %.

Pregabalin, discussed in the section on neuropathic pain, is one of three FDA-approved medications (duloxetine and milnacipran are the other two) for the treatment of FMS. Because of its anxiolytic properties, if symptoms or anxiety are prominent (and depression is not present), pregabalin may be a good first-line medication. Its molecular precursor, gabapentin, has proven efficacious in the treatment of FMS in mixed age adults [175].

Tramadol, discussed in the section on nociceptive pain, has efficacy in FMS for reducing pain and improving physical function [176]. Tramadol also has been found to be effective in treating the pain of osteoarthritis [177], a disorder that frequently coexists with FMS in older adults.

Cyclobenzaprine has strong efficacy evidence for reducing pain in FMS [169], but because of its strong anticholinergic potential, decreased clearance in older adults, and potential to disrupt cardiac conduction, it should be used cautiously [178].

## Future Directions

The field of pain and aging is in its infancy, having originated because of an obvious societal need rather than a distinct body of knowledge. To optimize the treatment of the burgeoning population of older adults, numerous questions must be answered: (1) What drives functional decline in older adults with chronic pain? How should future treatment be targeted to most effectively ameliorate this decline? (2) What is the efficacy and safety of pharmacological and non-pharmacological treatments for older adults? Studies must be designed that include adequate numbers of older adults to provide a meaningful answer to this question. (3) How should our health care resources be funneled to optimize benefits and decrease risks? Until health care policy changes, how can we improve the training of students and health care providers to evaluate and manage pain in older adults in a clinically effective and cost-effective way?

### Summary/Conclusions

Older adults with chronic pain should be thought of and cared for as older adults first and as pain patients second. Their management often requires the cooperation of an interdisciplinary team rather than a pain physician in isolation. Until an adequate evidence base exists to direct the treatment of these patients, care should proceed carefully and comprehensively.

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