

MANAGEMENT OF PAIN IN PERSONS WITH DEMENTIA

Pain is a common medical condition in older persons; especially residents in long term care (LTC) settings. Pain is defined as a sensory and emotional experience associated with actual or potential tissue damage. Chronic persistent pain occurs in 24% of LTC residents while only 29% are free of all pain. Most, i.e., 74%, demented nursing home residents have some pain and the majority, i.e., 70%, is untreated or under-treated. Pain can have multiple origins; however, discomfort produced by musculoskeletal disease is the most common problem in the older person, e.g., arthritis 42%, bone fracture 12%. Untreated or under-treated pain can produce significant suffering as well as agitation and behavioral problems in persons with dementia. Regular administration of acetaminophen can reduce agitation in more than one-half of agitated, demented patients with pain. Assessment and management of pain is an important responsibility of any clinical management team. Dementia patients are less likely to receive analgesics despite the fact that they experience suffering equal to cognitively intact individuals.

Neuroanatomy of Chronic Pain

The therapy for pain should target each level within the nervous system that produces the noxious sensory stimulus. The brain perceives pain via a complex series of emotional and intellectual responses to pathways that begin at the level of the pain-sensing organ and end with our interpretation of the sensory stimulus (**See Table 3**). Pain sensors are located throughout the skin, joints, bones, and organs within the body. The pain associated with a burn, bruise, or broken bone is perceived through specialized nerve endings in each tissue. The sensory nerve impulse is transmitted to the spinal cord via peripheral nerves. The spinal cord receives the raw information and transmits the sensation to a relay station in the base of the brain termed the thalamus where the information is organized. The thalamic neurons then relay the organized information to the brain region that integrates sensory information called the parietal lobe. The parietal lobe recruits limbic systems to judge the level of distress and develop an emotional response to this discomfort, e.g., temporal and frontal lobes. Malfunction at any level in the pain circuit can produce misinterpretation of painful or noxious stimuli. Visceral sensations from bladder, bowel, stomach, gallbladder, etc. are transmitted to a distinct brain region buried beneath the temporal lobe called the insular cortex. Alzheimer's disease does not damage sensory pathways from the body and internal

organs; however, brain regions that interpret these messages are altered by the disease. Stimulation or damage at each sensory processing level can produce specific pain syndromes that require distinct therapeutic interventions. The interpretation of pain in dementia patients is complicated because they misidentify, under-recognize, over report, or ignore painful stimuli due to damaged cortical centers that integrate sensory information, i.e., parietal lobe, insular cortex. The normal intellectual expressions of pain, such as verbal complaints or help-seeking, are replaced by agitation, hostility, and aggression.

Pain Assessment in Dementia

The assessment of pain in the demented patient requires a review of medical records to determine the timing and sequence of the painful experience as well as a careful physical examination. Demented patients require a mental status examination to determine their ability to interpret pain, or ask for medication, as well as exclude depression or anxiety that might intensify the painful sensation. Chronic pain can produce depression and many patients experience less discomfort when treated with antidepressants. Tricyclic antidepressants like Elavil are helpful in younger patients but this medication causes severe confusion in demented patients. Other TCAs with fewer side effects, e.g., desipramine and nortriptyline, are as effective as Elavil at equivalent doses.

Recognition of pain in demented patients usually requires direct observation as these individuals frequently suffer from receptive and expressive aphasia. Tense body language, sad facial expressions, fidgetiness, loud perseverative verbal outbursts, and immobilization of specific body parts may indicate pain. Facial expressions that include clenched teeth, widely opened eyes, or tightly shut eyes may suggest the patient is experiencing significant pain. Distress or agitation during attempts at repositioning, transfer, or ambulation may indicate unrecognized arthritic or orthopedic pain. Episodic pain with secondary diaphoresis may indicate angina. Some verbal outbursts may result from chronic, untreated or under-treated pain. The frequency and intensity of these pain symptoms must be documented in the record. Pain charts are often helpful.

Management of Acute and Chronic Pain in Dementia

The first step in pain management is assessment of the discomfort. Acute pain syndromes commonly follow injuries, surgical procedures, etc. and require standard analgesic or narcotic management. Acute pain syndromes are expected to last for brief periods of time, i.e., less than six months. Pain that persists for over six months is termed chronic pain. Chronic non-malignant pain requires a more

complex strategy to minimize the use of narcotics and maximize non-pharmacological interventions. Acute pain rarely produces other long-term psychological problems, such as depression, although acute discomfort will produce distress manifested by acute anxiety or agitation in the demented patient. Mildly demented patients can become agitated or anxious with pain because they rapidly forget explanations or reassurances provided by staff. Amnesic individuals may forget to ask for PRN non-narcotic analgesics such as acetaminophen and these patients need regularly scheduled medications. Disoriented patients do not realize they are in a health care facility and aphasic patients may not comprehend the staff's inquiry about pain symptoms.

The symptoms of pain expressed by patients with moderate to severe dementia include anxiety, agitation, screaming, hostility, wandering, aggression, failure to eat, and failure to get out of bed. A small number of demented individuals with serious injury may not complain of pain, e.g., hip fractures, ruptured appendix, etc. Assessment of pain in the demented patient requires verbal questioning and direct observation to assess for behaviors that suggest pain. Standardized pain assessment scales should be used for all patients; however, these clinical instruments may not be valid in persons with dementia or psychosis. The past medical history may be valuable in assessing the demented resident. Individuals with chronic pain prior to the onset of dementia usually experience similar pain when demented, e.g., compression fractures, angina, neuropathy, etc. These individuals can be monitored carefully and non-narcotic pain medication can be prescribed as indicated, e.g., acetaminophen on a regular basis, anticonvulsants for neuropathy.

Management of Chronic Pain

The management of pain in any person requires careful consideration about the contribution of each component of the pain circuit to the painful stimulus (**See Table 3**). Neuropathic pain is produced by dysfunction of the nerve or sensory organ that perceives and transmits noxious stimulus to the level of the spinal cord. Radicular pain, i.e., pain occurring in a specific nerve pattern, is more consistent with dysfunction of a specific nerve, e.g., sciatica that radiates down the back of the leg. Persons with serious back disease may have herniated discs that compress specific nerve roots. This pain is often positional and produces spasms of the musculature in the back.

Damage to the spinal cord can produce chronic, neuropathic or non-localizing pain. This discomfort is commonly seen in persons with traumatic back injuries. Strokes

in the thalamus can produce a chronic pain syndrome called thalamic pain by misinforming the brain that a painful stimulus has been received.

The brain interprets pain in a highly organized systematic pattern. Discrete brain regions interpret and translate painful stimuli from specific body regions, e.g., arm, leg, etc., misfire in that discrete brain region will misinform the person that pain or discomfort is being experienced in that limb or part of the trunk. A person who loses a limb from trauma or amputation may continue to experience painful sensations in the distributions for that limb termed *phantom limb pain*.

Management of chronic pain involves three elements (1) physical interventions, (2) psychological interventions, (3) pharmacological interventions. Physical interventions include basic physiotherapy that incorporates warm or cool compresses, massage, repositioning, electrical stimulation and many other treatments. Dementia patients need constant reminders to comply with physical treatments e.g., using compresses, sustaining proper positioning, etc., and many do not cooperate with some interventions, like nerve stimulators or acupuncture. Physical interventions are particularly helpful in older persons with musculoskeletal pain regardless of cognitive status. Psychological interventions usually require intact cognitive function e.g., relaxation therapy, self-hypnosis, etc. Demented patients generally lack the capacity to utilize psychological interventions; however, management teams should provide emotional support to validate the patient's suffering associated with pain. Demented patients may experience more suffering from pain than intellectually intact individuals because they lack the capacity to understand the cause of their discomfort. Fear, anxiety, and depression frequently intensify pain.

Pharmacological management begins with the least toxic medications and follows a slow progressive titration until pain symptoms are controlled. Clinicians must distinguish between analgesia and euphoria. Some medications that appear to have an analgesic or pain relieving effect actually have an euphoric effect, which diminishes the patients' concern about perceived pain. The goal of pain management is to remove the suffering associated with the painful stimulus rather than making the patient euphoric or high to the point where they no longer care whether they experience pain. Euphoria-producing medications can cause confusion, irritability, and behavioral lability in patients with dementia. Narcotic addiction is not a common concern in dementia patients as these individuals have a limited life expectancy and rarely demonstrate drug-seeking behaviors.

Pharmacological interventions always begin with the least toxic, i.e., least confusing, medications. A regular dose of acetaminophen up to 4 grams per day will substantially diminish most pain and improve quality of life. Clinical studies show that regular Tylenol reduced agitation in over half the treated patients. Chronic arthritic pain with inflammation of the joints may also respond to non-steroidal anti-inflammatories (NSAIDS) or Cox-2 inhibitors. The gastrointestinal toxicity associated with NSAIDS is greater than that of Cox 2 inhibitor medications. Patients who fail to respond to non-narcotic analgesics should receive narcotic-like medications, i.e., tramadol. Patients who fail to respond to maximum doses of tramadol, i.e., 300 mgs per day, may require narcotic medications.

Calcitonin is effective in some chronic pain associated with osteoporosis and fractures. Osteoporotic fractures are common painful complications of aging and produce significant distress in demented and non-demented individuals. Studies demonstrated that 50 to 100 units of nasal calcitonin would substantially reduce discomfort associated with fractures. Calcitonin suppositories were also effective for treatment of long-term bone related pain.

Opiate Management Of The Demented Resident

The prescription of opiates for dementia patients requires attention to the need to relieve distress caused by pain and the potential for medication toxicity. The World Health Organization has proposed a three-step analgesic ladder for use in pain caused by cancer, which may be a useful guide in patients with dementia, as well. Pain treatment starts with non-narcotic medications, and moves up the ladder as indicated by patient response and tolerability of therapies. At any step in the ladder, co-prescription of non-opiate analgesics (acetaminophen, NSAIDS, COX-2 inhibitors), as tolerated, can provide synergistic relief and reduce the doses of opiate necessary for good pain control.

The choice of specific opiate compounds depends on the situation. For intermittent pain, short-acting immediate release opiates may be sufficient (Step 2 on the WHO ladder). Many of these are available in preparations combined with acetaminophen or an NSAID. Codeine preparations provide a relatively mild analgesic effect, but can be as “constipating” as stronger narcotics. Preparations containing meperidine, pentazocine, and propoxyphene can worsen confusion in dementia patients and should generally be avoided in this population. Mixed agonist-antagonist medications (e.g., butorphanol, nalbuphine, pentazocine) are problematic for use in this setting, particularly if the patient has been exposed to other opiates.

Hydrocodone, morphine, and oxycodone preparations are preferred for pain requiring intermittent or short-term opiate treatment.

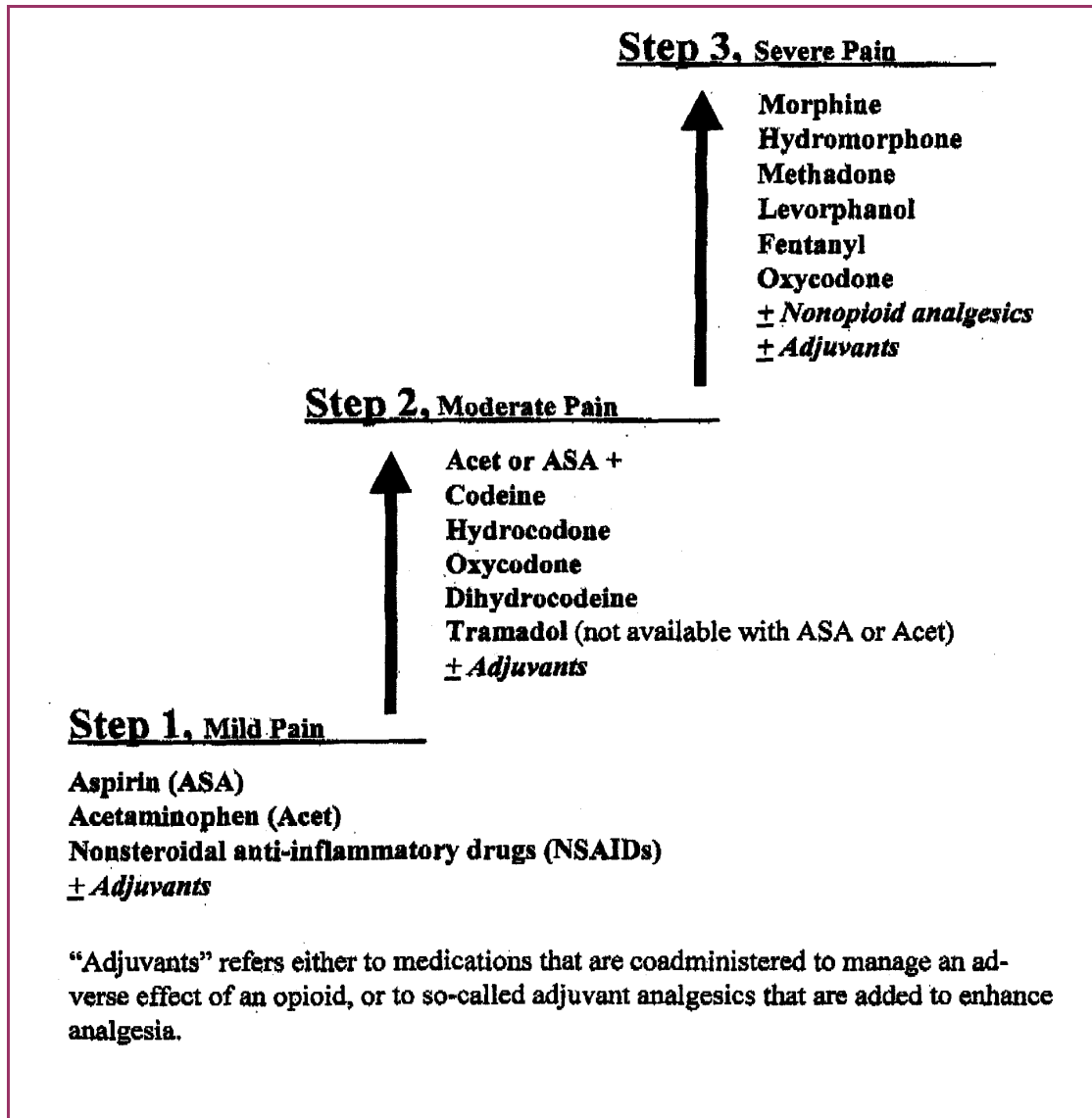
Severe pain that is refractory to less aggressive measures can be treated with WHO Step 3 agents such as fentanyl, hydromorphone, morphine, or oxycodone. Patients with persistent severe pain require continuous dosing around-the-clock to avoid breakthrough pain. To avoid every 2 to 4 hour dosing around the clock, sustained release preparations are used once the patient's 24-hour dose need is determined using immediate-release preparations. Breakthrough (or rescue) doses should also be provided as a PRN; a dose of an immediate-release preparation available every 2-4 hours PRN is ideal for breakthrough dosing. A breakthrough dose of 10% of the total 24-hour sustained-release dose is a good rule of thumb. If more than one or two breakthrough doses are needed on a regular basis, the sustained-release dose should be adjusted. The goal of opiate therapy for severe pain is to control as much of the pain as possible with the sustained-release medication. Common side effects of opiate therapy include constipation, dry mouth, nausea, and sedation. Constipation is sufficiently common in elderly patients with dementia and prophylactic treatment (e.g., stool softeners) is indicated when opiate therapy is initiated. Bowel function should be closely monitored while on opiate therapy to avoid impaction that may produce agitation or diminished oral intake.

Chronic analgesic therapy should be continued in severely demented patients unless re-evaluation shows resolution of pain. Severely aphasic patients must be carefully observed for non-verbal expression of pain after discontinuation of medication. A slow taper off opiates is indicated to avoid withdrawal symptoms in tolerant patients and to allow monitoring for re-emergence of pain.

Dose conversion tables (examples attached) can be used to convert from one opiate to another (if the first agent is poorly tolerated) or to calculate an appropriate breakthrough dose of an immediate-release preparation for sustained-release agents without an immediate-release form (e.g., fentanyl).

Table 1

WHO 3-Step Ladder



In 1986, the World Health Organization (WHO) developed a 3-step conceptual model to guide the management of cancer pain. It provides a simple, well-tested approach for the rational selection, administration, and titration of a myriad of analgesics. Today, there is worldwide consensus favoring its use for the medical management of all pain associated with serious illness.

Table 2

NARCOTIC DOSING RANGES AND EQUIVALENT DOSES

STANDARD GERIATRIC DOSE		EQUINANALGESIC DOSES OF OPIOID ANALGESICS		
ANALGESIC	DOSE RANGE (MG)	ORAL/RECTAL DOSE (MG)	ANALGESIC	PARENTERAL DOSE (MG)
Codeine	15-60mgm PO q4-6hrs	100	Codeine	60
Hydrocodone	2.5-5mgm PO q4-6hrs	15	Hydrocodone	-----
Hydromorphone	1-2mgm PO q4-6hrs	4	Hydromorphone	1.5
Oxycodone	2.5-5mgm PO q6hrs	10	Oxycodone	-----
Levorphanol	2-3mgm PO q12hrs	2	Levorphanol	1
Methadone	2.5mgm PO q8-12hrs	10	Methadone	5
Morphine	10-30mgm PO q4hrs	15	Morphine	5
Fentanyl	SEE PDR	-----	Fentanyl	0.1

This table demonstrates routine dosing ranges and equivalent doses among the commonly prescribed narcotic.

Data from Geriatric Dosage Handbook.

Table 3

CORRELATING THE ANATOMY OF PAIN TO THERAPY

The Pain System in Dementia				
Neurological Level Generating Pain Signal	Function of Level	Classic Symptoms of Dysfunction	Change by Dementia AD or Other	First Line Therapy (When level is damaged)
Pain receptors Sensory nerves	Receive and Relay Pain	Diminished sensation or burning, painful sensation *Neuropathic pain	AD – No change ETOH- ↑ or ↓	Anticonvulsants
Spinal Cord	Relay individual pain signals	Neuropathic or visceral	None	Anticonvulsants
Thalamus	Collect and relay pain signals	↑ or ↓ pain *Thalamic pain syndrome *Phantom limb pain	Unclear	Anticonvulsants for thalamic or phantom limb pain
Parietal and Insular Cortex	Localize and interpret pain message	↑ or ↓ pain *Phantom limb pain	↓ Ability to recognize by all dementia	*Analgesics *Other meds appropriate to pain source
Limbic System	Reaction to Pain	Distress, agitation, hostility, apathy, depression	↑ or ↓	*Analgesics *Mood stabilizers *Behavior mgt.