Assessment and Understanding of Pain in Patients With Dementia

Jeff C. Huffman, MD, and Mark E. Kunik, MD

The literature on pain in dementia patients is reviewed. A summary of methods for assessment of pain in demented elderly persons and an examination of studies that used such methods are included. In addition, literature theorizing a decrease in affective pain in this population is discussed; management of pain in such patients is not discussed extensively. Research reveals 3 major findings: (a) a moderate decrease in pain occurs in cognitively impaired elderly persons, (b) communicative dementia patients’ reports of pain tend to be as valid as those of cognitively intact patients, and (c) assessment scales developed thus far for noncommunicative patients require improvement in accuracy and facility. Many questions about pain in dementia patients remain, and the continued development of valid pain assessment techniques is a necessity.

Key Words: Elderly persons, Alzheimer’s disease, Scales

Pain, a significant problem in elderly persons, has become the topic of multiple studies investigating its detection, causes, and treatment. Pain control is necessary not just for relief of suffering, but also because of the adverse health events related to pain. Poor pain control in the settings of trauma and surgery has been shown to significantly increase mortality and morbidity (Cousins, 1991). Pain has also been linked to sleep disturbance, impaired ambulation, and possibly malnutrition (Dworkin, Von Korff, & LeResche, 1990; Ferrell, Ferrell, & Osterweil, 1990; Magni, Schifano, & De Leo, 1985; Roy, 1986) and has been clearly linked to depression, especially in elderly persons (Moss, Lawton, & Glicksman, 1991; Parmelee, Katz, & Lawton, 1991). Further, it has been shown that pain and stress inhibit immune function and enhance tumor growth in laboratory animals (Liebeskind, 1991). A single exposure to stress/pain in laboratory animals has been shown to decrease NK cell cytotoxic activity and increase tumor metastasis (Ben-Eliyahu, Yirmiya, Liebeskind, Taylor, & Gale, 1991; Yirmiya et al., 1991).

Pain is an especially important topic in elderly persons because of its high prevalence and uneven treatment in this population. Numerous studies (Ferrell et al., 1990; Scherder & Bouma, 1997; Sengstaken & King, 1993) have shown that pain is underestimated and undertreated by healthcare teams, and pain is most undertreated in elderly persons, especially those who cannot communicate their pain. Several studies have shown that the number of elderly persons experiencing pain exceeds the number of younger persons with pain (Simons & Malabar, 1995). A study of several hundred noninstitutionalized rural elderly persons revealed that 86% had had pain at some time in the previous year and 59% had had multiple pain complaints. Further, in the majority of cases, the pain had impacted the patients’ ability to perform usual activities such as work or errands in the past 2 weeks (Mobily, Herr, Clark, & Wallace, 1994). Other studies of this population reported comparable (Lau-Ting & Phoon, 1988; Roy & Thomas, 1986) or somewhat lower prevalence of pain (Brody & Kleban, 1983; Moss et al., 1991; Roy & Thomas, 1987), but all identified pain as being present in a significant portion of elderly persons and having a substantial impact on their lives.

Treatments for pain in elderly persons are underutilized. A study of 97 institutionalized elderly patients revealed that approximately three quarters of such patients had pain and that only 15% of those with pain had received analgesics in the last 24 h. Pain management strategies other than medication were very limited in scope (Ferrell et al., 1990), despite evidence that simple measures like physical exercise have been shown to significantly improve pain management in older persons (Chow, Harrison, & Dorman, 1989; Ettinger et al., 1997; Ferrell, Josephson, Pollan, Loy, & Ferrell, 1997; Khalil, Abdel-Moty,
that severely demented patients (especially those with Alzheimer’s dementia; DAT) may have a decrease in the affective component of pain because of memory loss and/or the functional loss of certain brain areas associated with emotion and expectation. Most studies relating cognitive impairment and pain have shown decreased pain with decreasing cognitive function; however, such patients’ ability to report pain also decreases with severity of dementia, and it has thus far been difficult to determine whether the decreased pain report is due to less pain or lessened ability to report such pain.

In this article we review the literature on the study of pain in dementia patients. Pain mechanisms and the theoretical alteration of such mechanisms in certain dementia patients are examined. Studies describing the relationship between pain and cognitive impairment are outlined, and articles detailing pain assessment methods are reviewed. Finally, areas of further research are suggested and discussed.

### Pain Mechanisms

At one time, pain was thought to be a relatively simple mechanism, with peripheral nociceptive fibers communicating with the parietal somatosensory cortex for pain interpretation, with a direct return circuit for a withdrawal reflex. However, as pain has become better understood, and the phenomena of chronic pain, central pain, and phantom limb syndromes have been further studied, it has become clear that multiple cortical and subcortical inputs act to modulate the pain state and have the ability to intensify or attenuate reactions to noxious stimuli or to create a pain state in the absence of such stimuli.

Specific nerve fiber types (Adelta, associated with sharp, fast pain, and C, associated with slow, dull pain) located at the periphery are associated with pain transmission to the spinal cord and somatosensory cortex. However, as first outlined in Melzack’s gate theory of pain (Melzack & Casey, 1968) and since expanded, sensations created by noxious stimuli are modulated at spinal cord and midbrain levels. Further study has shown that the peripheral mechanism of pain activates subcentral mechanisms in the thalamus and then progresses to the reticular activating system, an area that processes input from the cortex, cranial nerves, limbic system, hypothalamus, and locus ceruleus as part of a diffuse modulatory system. This finding lends credence to the idea of affective components of pain, because the locus ceruleus and limbic system are associated with anxiety, fear, and primitive emotion. The hypothalamus also appears to be involved in such affective pain, because stress has also been shown to modulate pain via hypothalamic-adrenal systems. In addition, the receptive system of the dorsal horn in the spinal cord and every higher level is influenced in a direct manner by attention, expectancy, learning, anxiety, and fear (Calliet, 1993; Craig, 1994; Ferrell, 1991).

Nociceptive event-related cerebral potentials, a physiologic correlate to pain, are event-related electrical signals of brain activity that are recorded from the scalp of laboratory volunteers.
when precisely controlled stimuli are delivered. The event-related potentials have been shown to be affected by expectation and levels of arousal, further supporting the concept that such emotional states affect pain perception (Bromm, 1985).

Of the previously mentioned structures, the limbic system seems most tightly involved in the relaying of emotional input into pain perception, with linkages to the dorsal horn, thalamus, and cortex via ascending and descending tracts. Further pain-modulating processes occur in the midbrain, hypothalamus, and periaqueductal gray, with the periaqueductal gray receiving significant input from the frontal cortex; this frontal cortex involvement has been displayed on positron-emission tomography (PET) scans, which show frontal activation by noxious stimuli but no such activation by nonnoxious stimuli (Calliet, 1993; Fine & Ashburn, 1998). The presence and importance of these central, modulatory pain mechanisms is borne out by the presence of central pain that is unresponsive to the interruption of classical pain tracts and phantom limb syndromes in which pain persists in an amputated limb. Memory obviously plays a significant role in these central pain mechanisms, affecting anxiety, expectation, and fear. Time elapsed since a previous pain experience can impact the perception of a similar pain experience (Wachtler-Shikora & Perez, 1982). In sum, central nervous system mechanisms are believed to account for 20–50% of chronic pain syndromes (Bouckoms & Hackett, 1997).

Despite multiple studies of pain in elderly persons, it remains unclear whether they experience extensive physiologic changes in pain perception. The evidence obtained thus far, though, does not strongly indicate any large baseline decrease in pain threshold or intensity (Collins & Stone, 1965; Harkins & Chapman, 1977; Schumacher, Goodell, Hardy, & Wolff, 1940; Sherman & Robillard, 1964; Tucker, Andrew, Ogle, & Davison, 1989; Woodrow, Friedman, Siegelbaum, & Collen, 1972). Some researchers believe that the pain experience may be altered to varying degrees in dementia patients because the dementing illnesses can affect many of the brain areas crucial to the central modulation of pain. DAT is often implicated in such discussions. The somatosensory cortex is relatively unaffected in DAT, and hence the sensory/discriminative quality of pain is likely preserved in such patients. This preservation of sensation is supported by two early studies of pain thresholds in dementia patients that showed that dementia patients’ pain thresholds as measured by a pain withdrawal reflex were not significantly different than pain thresholds in cognitively intact elderly persons (Cornu, 1975; Jonsson, Malhammar, & Waldton, 1977); further, Cornu found that the dementia patients showed changes in behavior consistent with changes in perception and stimulus localization when presented with noxious stimuli, further supporting the idea that pain sensation was intact in such patients (Cornu, 1975). Another study of primitive nociceptive reflexes actually showed a significant increase in such reflexes in dementia patients when compared with nonde-mented elderly controls matched for age and sex (Vreeling, Houx, Jolles, & Verhey, 1995). Hence the sensory component of pain appears to be intact.

However, the pain areas involved in affective pain are often affected to a significant degree in DAT, which suggests that this component of pain may be altered in DAT patients. Neuropathologic studies of DAT patients have revealed varying degrees of atrophy in the amygdala (a key component of the limbic system) along with consistent, significant frontal cortex atrophy and neurofibrillar pathology (Morris, 1997). Other neuropathologic studies of DAT patients have revealed amyloid plaques in the amygdala, hypothalamic tracts/nuclei, and thalamic tracts/nuclei (Rudelli, Ambler, & Wisniewski, 1984). These changes are quite variable in nature, and thus changes in pain perception are likely quite variable (Farrell, Katz, & Helme, 1996).

Fisher-Morris and Gellatly (1997) described two case reports and a small-scale, anecdotal survey report of 49 DAT patients who failed to exhibit normal pain perceptions. The first case report involved a communicative DAT patient with a fungating, friable cancerous mass on the chest wall and zoster about the spine who felt touch without pain and had no changes in vital signs upon dressing changes of the friable lesion or manipulation of either region. The second report concerned a communicative DAT patient who was walking pain-free within hours of surgery for placement of dynamic hip screws for a femur fracture. The patient went home within a week, ambulatory without physical therapy or walking frame. The survey of DAT patients’ caregivers included unconfirmed reports of similar communicative DAT patients who dipped fingers into boiling water, had zoster recurrences, and experienced other events associated with significant pain but felt no pain. Although this article, largely made up of unconfirmed reports, clearly is not a rigorous study of the topic, the reports might represent the far end of a spectrum of decreased pain in DAT patients and may be worthy of further investigation.

The impact of Lewy body dementia on the pain experience is even less well understood. Lewy bodies, however, have been shown to occupy the locus ceruleus, hypothalamus, and frontal cortex (Lapallo & Sakla, 1998), suggesting some impairment of affective pain perception. Prominence of psychotic features early in this disorder may cause further changes in how pain is perceived in these patients. It is also difficult to generalize about pain perception in multiinfarct dementia, mostly because of the widespread variability in affected brain regions. Infarcts within the prefrontal cortex do appear to attenuate emotional and motivational responses to nociceptive stimuli (Price, 1988), and infarcts of the somatosensory cortex predictably impair pain sensation.

In addition to actual neuropathological involvement in specific brain regions, the affective component of pain may be affected by the overall memory loss of these patients. Memory loss would surely have an impact on the components of pain mediated by expectation and learning, as well as likely having ef-
fects on anxiety and fear. The effects that these alterations might have is unknown; however, they could reduce pain by reducing expectation and anxiety or could lead to the development of an indefinite acute pain state, as proposed by Farrell and colleagues (1996), because of lack of habituation to painful stimuli and thus an inability to adapt to the stimuli.

**Studies of Pain in Cognitively Impaired Elderly Persons**

A number of studies in elderly persons comparing pain and cognitive ability have been performed. Such studies vary widely in their patient population, methods of pain assessment, and control populations. Many of these studies excluded patients who were noncommunicative, and most examined cognitively impaired elderly persons in general as opposed to patients with specific diagnoses, for example, DAT. However, consistent trends with regard to the relationship between pain and cognitive function seem to persist across many of these studies despite the significant differences in methodology and patient population.

One such consistent trend is that measured pain seems to decrease with worsening cognitive impairment. Cohen-Mansfield and Marx (1993) measured pain using a simple numerical scale in 408 nursing home elderly persons and found a significant inverse correlation between pain and cognitive impairment as measured by the Brief Cognitive Rating Scale. Similarly, a study of 758 institutionalized responsive elderly persons showed a negative association between cognitive impairment and reported pain intensity (Parmelee, Smith, & Katz, 1993). A study of elderly patients who had undergone surgery for hip fracture showed a significant inverse correlation between cognitive impairment and pain as measured by a verbal and an observational scale (Feldt et al., 1998), and a fourth study of 132 community-dwelling elderly persons showed a decrease in reported pain over the last 24 h in patients with senile dementia compared with those with normal cognitive function (83% vs 63%), but the decrease was not statistically significant (Brody & Kleban, 1983).

Another study of demented patients receiving lumbar punctures revealed a post-lumbar-puncture headache rate of 2.0%, significantly less than reported incidences in all comers (24–39%) or elderly persons (4–9%), and the rate of reported headache decreased with the severity of dementia (Blennow, Wallin, & Hager, 1993). Researchers compared community-dwelling DAT patients with cognitively intact elderly persons with regard to the 25 most common symptoms in elderly persons and found that DAT patients had significantly fewer complaints of 5 of the 25 symptoms, including 1 (joint pain) of 5 purely pain-related symptoms (McCormick et al., 1994). Finally, cognitively impaired patients, specifically those with DAT, have been shown to have both less joint pain and less nonsteroidal anti-inflammatory drug (NSAID) use than cognitively intact elderly persons despite no significant difference in the number of osteoarticular disorders between the two groups (Breitner et al., 1994; Lucca, Tettamanti, Forloni, & Spagnoli, 1994; McGeer, McGeer, Rogers, & Sibley, 1990; Scherder & Bouma, 1997). In contrast to the previous studies, one study of 97 communicative nursing home patients revealed no association between Mini-Mental State Examination (MMSE) and pain report (Ferrell et al., 1990), although clearly the weight of the evidence suggests decreased pain with decreased cognitive function.

Potential explanations for the apparent decrease in pain with declining cognitive function are numerous. Most of these studies used verbal self-report as a measure of pain, which could be impacted by both memory loss and dysphasia, a common symptom of dementia. These factors could lead to a significant underreporting of symptoms, which would account for the findings. Two previous studies of elderly out-patients evaluated for dementia revealed previously unrecognized illnesses in nearly one half of the participants, a result quite possibly due to diminished pain report in these persons ( Larson, Reifler, Sumi, Canfield, & Chinn, 1986; Teri & Wagner, 1991). Another confounding factor in studies looking specifically at DAT patients can be the decreased medical burden (but not decreased mortality) sometimes found in patients diagnosed with DAT (Semla et al., 1993), probably due to exclusion criteria necessary in the diagnosis of DAT. This factor certainly might account for decreased pain report; however, many of these studies involved cognitively impaired participants as a group without regard to diagnosis. Possible reasons for decreased NSAID use include those mentioned previously as well as the possibility that disorders requiring NSAIDs may be more often seen as a marginal problem in patients with DAT and treated more conservatively. A final and important consideration for both decreased verbal pain report and NSAID use is that cognitively impaired patients may actually feel less pain, as discussed previously, because of a decrease in the affective component of pain perception.

Along with this relationship between pain and cognitive function, a second consistent finding in the previous studies is that cognitively impaired patients, although perhaps having statistically less pain than their cognitively intact counterparts, still had significant untreated pain. In each of the previous studies, most elderly persons in a variety of settings had some unrelieved pain (66–80% among all comers in the last three studies). An important finding in the study of responsive institutionalized elderly persons by Parmalee and colleagues (1993) was that when specific physical complaints in these patients were investigated for possible physical causes of pain, there was no difference between cognitively intact and impaired persons with respect to the percentage of patients found to have an identifiable cause for the complaint. The authors thus concluded that, although cognitively impaired persons have fewer pain complaints, their complaints are just as reliable and valid as those of cognitively intact elderly persons.
Methods of Pain Assessment in Cognitively Impaired Elderly Persons

Whereas a small collection of studies have surfaced regarding pain in cognitively impaired elderly patients able to communicate, patients whose demening illnesses are so severe that they preclude meaningful communication have typically been excluded from studies because of the difficulty of objective pain assessment in this population. Despite the inherent difficulties of such assessment in this population, a number of methods and scales have been developed that measure pain in noncommunicative patients. In addition, in studies of significantly demeaned patients who are able to communicate, researchers have attempted to find the most effective methods to accurately and reproducibly determine pain in these patients. In this section we review the literature on pain assessment in both communicative and noncommunicative elderly persons.

A study of communicative patients with moderate to severe dementia found that 62% of patients reported pain with the remainder denying pain or unable to respond. Of those patients reporting pain, 83% were able to use at least one of five available scales (Downie et al., 1978; Fishman et al., 1987; Melzac, 1975; Nelson et al., 1987; Ohnhaus & Adler, 1975) to quantify their pain, showing that when healthcare providers are patient, allowing time for demented patients to assimilate clues and using visual cueing techniques to aid their patients, quantification of pain is often possible, even in this difficult population (Ferrell, Ferrell, & Rivera, 1995).

Noncommunicative patients pose an even greater challenge in pain assessment. Studies of noncommunicative patients without dementia have revealed behaviors that can be matched with later verbal reports of pain. A report of 30 postanesthesia patients revealed that certain facial expressions, patient sounds, and muscle tension were significant indicators of pain (Mateo & Krenzischek, 1992). Similarly, a study of patients with chronic temporomandibular disorder having facial pain revealed specific facial movements (lowered brows, closed eyes, orbicularis oculi movement) that strongly correlated with verbal self-report of pain and specifically with measures of the affective components of pain. However, the actions tended to be generally idiosyncratic, and no single facial action was seen in all patients (LeResche & Dworkin, 1988). Examination of facial expression during nonfacial pain in female college students also revealed specific facial movements (lip parting, orbicularis actions, cheek raise) associated with the pain but that again were not universal (Craig & Patrick, 1985). Other facial studies (Boucher, 1969; Prkachin, Currie, & Craig, 1983) have shown specific facial expressions as indicators for different affects and for different intensities of pain. Although these studies appear somewhat useful in detecting pain in noncommunicative persons, the moderate lack of agreement among studies and the lack of universality of any one facial action make the use of such cues difficult.

Studies and narrative articles regarding pain assessment in noncommunicative elderly persons are becoming more prevalent. One such narrative article described the use of changes in facial expression, body movements, and daily activities for identification of behavioral changes possibly associated with pain in cognitively impaired patients (Parke, 1992). Parke noted that researchers must learn a given patient’s idiosyncratic baseline activities to be able to document change; similarly, another narrative article focused on the individuality of pain behaviors in demeaned elderly persons that, in some cases, may seem paradoxical (e.g., a patient who ceased moaning when in pain; Marzinski, 1991). A study of screaming in nursing home residents found that the best predictors of screaming were pain, cognitive impairment, and social network, showing that verbal agitation may be decreased by pain control and, likewise, pain may be identified with use of screaming as an assessment tool (Cohen-Mansfield, Werner, & Marx, 1990). A small study of 8 elderly patients in a skilled nursing facility on psychotropic medication for “difficult behavior” found that 5 of these patients had a decrease in problem behavior that allowed their psychotropic medications to be discontinued when they were placed on scheduled acetaminophen (Douzjian et al., 1998).

One study possibly useful in assessing pain in demeaned elderly persons is a study of communicative and noncommunicative elderly patients that used an amalgam of nonverbal pain behaviors adopted from an observation method for chronic low back pain patients (Keeffe & Block, 1982). The researchers found that, by locating these behaviors, they were able to use pain treatment modalities to eliminate the patients’ identified pain behaviors in 96.2% of cases (Simons & Malabar, 1995). In a similar study researchers constructed a scale, the DS-DAT (Discomfort Scale-DAT), to measure discomfort. This scale measured nine items, including patient sounds, facial expressions, and body postures, over a 5-min period to determine discomfort. The scale was then administered to patients with severe DAT before, during, and after fever episodes and was found to effectively detect differences in discomfort caused by the fever episodes (Hurley, Vollicer, Hanrahan, Houde, & Vollicer, 1992). However, J. Miller and colleagues (1996) attempted to use this scale on acutely confused elderly patients and found that, although the tool was generally accurate, it required significant training and was too complex for routine nursing care.

One thus far underused pain detection mechanism is the use of nociceptive-event-related cerebral potentials (NEPs). NEPs have been shown to be an effective physiologic correlate to pain (Carmon, Dotan, & Sarne, 1978; Carmon, Friedman, Coger, & Kenton, 1980; Chatrian et al., 1982; Coger et al., 1980), and have been tested specifically in elderly persons and appear to be a reliable objective measure of their pain (Gibson, Gorman, & Helme, 1991). Further, NEPs are affected by motivational pain components such as expectation and fear, and thus they would be
appropriate for the demented population, who theoretically have intact peripheral and somatosensory mechanisms but possibly decreased affective components of pain. Thus far, no studies using NEPs have been completed specifically on cognitively impaired elderly persons.

Future Directions

First and foremost, future research on pain in elderly dementia patients hinges on the development of better pain assessment tools, especially for patients who are noncommunicative. The DS-DAT and similar scales appear to have some promise as pain assessment tools in this population, but the development of nonverbal behavior scales that are even more objective and easier to use will greatly enhance research. In addition to these scales, the use of NEPs in this patient population is potentially very useful as an objective and accurate marker for pain. In general, more widespread use of established pain assessment tools in communicative patients, the development of better scales for noncommunicative patients, and further examination of NEPs in demented patients should be three major goals for researchers attempting to better quantify pain in elderly demented patients.

In addition to better tools for pain assessment, further advances in the understanding of brain function and the mechanism for pain will aid researchers’ ability to predict changes in pain perception in demented patients. Studies of brain response to noxious stimuli using functional imaging techniques are warranted, especially in those patients with dementing illnesses. Functional imaging modalities such as PET, single-photon emission computed tomography (SPECT), and functional magnetic resonance imaging (fMRI) are becoming more available to researchers, and they can use such modalities to gain a better understanding of pain perception and how such perception may differ in demented populations. Neuropathological studies of patients with such illnesses will also continue to provide insight into areas of the brain affected by dementing diseases and how these effects may distort feelings of pain.

Further studies of the relationship between pain and cognitive impairment will also aid in understanding of the clinical effects of dementia on pain. As pain assessment methods improve, the relationship between pain and functional cognitive deficits can be more accurately studied. In addition, the inclusion of noncommunicative patients—often the most severely affected and potentially the most useful with regard to research—would add much to the fund of knowledge about pain in such patients and would allow clinicians to better help this often overlooked and undertreated population. Finally, grouping of study patients into more specific dementia diagnostic categories, such as DAT, Lewy body dementia, and so on, will allow researchers to detect specific changes in pain perception for a given dementia diagnosis. Such studies, when correlated with neuropathologic results, may also give researchers a better understanding of pain mechanisms in general by allowing them to note clinical changes in pain perception and then to see the specific neuropathology that caused such changes. Hence, with more specific diagnosis and more accurate pain assessment, researchers can further the understanding of pain in each dementia population.

In conclusion, pain in elderly persons, and especially cognitively impaired elderly persons, is a significant problem with high prevalence and little research regarding its causes, detection, and treatment. The treatment of such pain, through accurate detection and appropriate measures, not only relieves suffering but also improves immune function, mood, sleep, and nutritional status. Research in this area is likely to be rewarding both in a scientific sense, allowing more insight into the mechanisms of the brain and of the multilayered pathways of pain perception, and in a clinical sense, allowing physicians to relieve pain in patients whose pain had previously gone undetected.

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