Geriatrics and Gerontology Elsewhere

Pain management in dementia: so far, not so good

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Pain is highly prevalent in the aging population. Individuals with neurological disorders such as dementia are susceptible patient groups in which pain is frequently under-recognised, underestimated, and under-treated. The inability to successfully communicate pain in moderate-severe dementia is a major barrier to effective treatment and several observational studies indicate that pain is under-treated among cognitively impaired elderly people.

Pain has been related to neuropsychiatric symptoms in dementia, such as agitation, aggression, mood syndrome and sleep problems. Adequate pain management has been demonstrated as possibly effective in mediating or alleviating those symptoms.

Recent guidelines (American Geriatric Society 2009, British Geriatric Society 2013) recommend a comprehensive, disease-specific assessment to determine appropriate treatment for each individual. Whereas in old patients data on pain management are becoming more consistent, we still lack clinical evidence in those affected by dementia.

In this narrative review, we summarize the best-available evidence regarding the aetiology, assessment and treatment of pain in people with dementia. Further large-scale trials of treatment approaches in people with dementia are needed to improve clinical guidance for the diagnosis and treatment of pain in these fragile individuals.

Key words: Pain, Dementia, Alzheimer disease, Elderly, Opioids

INTRODUCTION: CRITICAL ISSUES ABOUT PAIN IN DEMENTIA

Dementia is a "clinical syndrome due to disease of a progressive nature, which leads to disturbances multiple higher cortical functions, including memory, thinking, orientation, comprehension, calculation, learning capacity, language and judgment" [1]. Dementia affects approximately 44 million people worldwide, and this is expected to double every 20 years as the population ages (Fig. 1). One-third of people with dementia reside in nursing homes [2].

Ample evidence shows that aging is associated with a high rate of painful conditions, irrespective of cognitive status. Chronic pain is common in older people, affecting about 60% of people aged more than 65 years and functionally impairing 45%-80% of older people in nursing homes [3]. The most common types of pain are musculoskeletal, such as arthritis, or neuropathic pain as result of diabetes or stroke [4].

The number of patients with dementia who will

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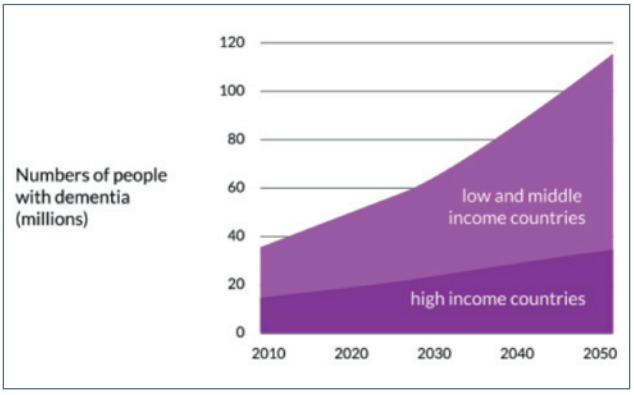

Figure 1. Expected growth of people affected by dementia in 2050.
Modified from Ferri et al. The Lancet 2005. Alzheimer's Disease International (2009), World Alzheimer's Report.

experience painful conditions is therefore likely to increase and the majority of them experience persistent pain lasting 6 months or longer [5]. Despite the high prevalence of pain in affected individuals, the assessment and management of their perceived pain is difficult due to the frequent loss of cognitive and communicative abilities. This begs the question of exactly how patients with dementia and other chronic cognitive neurodegenerative disorders perceive pain [6].
Little is known about the relationship between dementia and the neurophysiology of pain. Dementia is associated with central nervous system changes such as the destruction of cortical neuronal cells and depletion of cortical chemical neurotransmitters. Nociceptor response and transmission of pain sensation, however, are not thought to be affected by these physiological changes. In particular, the somatosensory cortex, crucial to the central modulation of pain, is largely unaffected by dementia [7] (Tab. I).
Patients with dementia may express their pain in ways that are quite different from those of elderly people without dementia. Particularly in the more severe stages of dementia, the complexity and consequent inadequacy of pain assessment leads to the under-treatment of pain itself [8].
Several observational studies indicate that pain is undertreated among cognitively impaired elderly people [9 10]. A major problem associated with the under-treatment of pain in the elderly who are cognitively impaired is the challenging nature of pain assessment [11]. The under-treatment of pain therefore has been associated with withdrawal, sleep disturbance, increased disability, and depression [12]. In addition, failure to adequately treat pain could compromise the effectiveness of rehabilitation therapies and may irreversibly alter the patient's ability to remain in their homes.
It has been reported that fewer analgesics are prescribed for the oldest category of cancer patients (over 75 years) than for younger patients, and low cognitive performance was one of the independent predictors of this finding [13]. In addition, it has been shown that among patients with history of hip fractures, those in advanced stages of dementia receive significantly less opioid analgesics than cognitively intact patients do [14].
All these observations stress the importance of increasing our knowledge of pain pathways, recognition and management in this specific, fragile population.

PAIN PERCEPTION IN ALZHEIMER DISEASE

Alzheimer's Disease (AD) is primarily a disorder of the neocortex, while the somatosensory cortex is relatively unaffected by the histological changes that are the hallmark of this disorder. Furthermore, although some senile plaques have been identified in the diencephalon it is unlikely that thalamic nuclei are significantly affected in AD. Consequently the sensory/discriminative quality of pain perception might be expected to be preserved in cases of AD, although distortions of sensation associated with parietal dysfunction may occur [15 16]. On the

Table I. Characteristics of dementia subtypes (modified from International AsD. World Alzheimer Report 2009. London: Alzheimer's Disease International, 2009).

Dementia subtypes	Early characteristics symptoms	Neuro-pathology	Proportion of dementia cases
Alzheimer's Disease (AD)*	Impaired memody, apaty and depressione, gradual onset	Cortical amyloid plaques and neurofibrillary tangles	50-75%
Vascular Dementia (VaD)*	Similar to AD, but memory less affected, and mood fluctuations more prominent Physical frailty Stepwise onset	Cerebrovascular disease Single infarcts in critical regions, or more diffuse multi-infarct disease	20-30%
Frontotemporal dementia	Personality changes Mood changes Disinhibition Language difficulties	No single pathology – damage limited to frontal and temporal lobes	5-10%

* Post mortem studies suggest that many people with dementia have mixed Alzheimer's disease and vascular dementia pathology, and that this 'mixed dementia' is underdiagnosed.

contrary, intralaminar thalamic nuclei, which represent a strategic pathway of the non-discriminative medial pain system, are early and progressively affected by the AD-related cellular changes [17]. As a consequence, emotional and affective function may be altered, and this is related to conspicuous neuronal and synaptic loss in prefrontal and limbic regions [18].

A detailed study on patterns of brain structural deficits across the cortex in AD suggested a spatially complex model of different atrophic patterns as AD progresses. Three main features were observed: 1) the overall deficit pattern spreads through the brain in a temporal-frontal-sensorimotor sequence, with a time lag in the right hemisphere; 2) the left hemisphere degenerates faster than the right; this asymmetric loss rate increases the existing asymmetry in cortical gray matter found in healthy elderly subjects; 3) some philogenetically older brain systems are spared late in the disease (e.g., sensory-motor cortices) [19]. With regard to pain perception, all brain regions related to medial system are affected during AD. Those pathways related to lateral pain pathways, such as the primary somatosensory cortex, are also preserved in late stages of AD [20].

Several studies observed a dissociation between sensory/discriminative pain perception, usually elaborated through the lateral pathway, and affective/emotional pathways, generally mediated by the medial system. A detailed and systematic study by Benedetti et al. showed a dissociation of pain perception in AD patients. By comparing AD patients with normal subjects of the same age, no differences in stimulus detection and pain thresholds were found, whereas a clearcut increase in pain tolerance was present in AD patients. They found pain thresholds to be unchanged, whereas pain tolerance increased according to the severity of the disease. A correlation between pain tolerance and the severity of AD, measured by means of neuropsychological (MMSE) and neurophysiological tests (spectral EEG), was also performed. There was a straightforward correlation between MMSE scores and pain tolerance demonstrating that the more severe the cognitive impairment the higher the tolerance to pain. What emerged from this analysis was that pain tolerance can be estimated by the degree of impairment of both MMSE and EEG. As a generalisation, MMSE scores ranging from 10-19 tend to follow the rule of thumb: 'the more severe the MMSE and EEG changes, the higher the tolerance to pain' as pain tolerance tends to co-vary in both MMSE's and EEG's. These findings show that, whereas the sensory-discriminative component of pain is maintained in AD patients, pain tolerance is altered and depends on cognitive and affective factors [21].

Pain perception and autonomic responses to pain are known to be altered in dementia. As a proof of that, neither stimulus detection nor pain threshold was correlated to cognitive status and a decline in brain activity [22]. In contrast, there is a correlation between autonomic responses and the deterioration of both cognitive functions and brain electrical activity. In particular, the heart rate increase after pain stimulation was correlated to the presence of slowed brain electrical activity (delta and theta frequencies). This correlation was also found for the anticipatory heart rate increase just before pain stimulation. These results indicate that pain anticipation and reactivity depend on both the cognitive status and the frequency bands of the electroencephalogram. On the contrary, both stimulus detection and pain threshold are not affected by the progression of AD. These findings clearly show that, whereas the sensory-discriminative components of pain are preserved even in advanced stages of AD, the cognitive and affective functions, which are related to both anticipation and autonomic reactivity, are severely affected. This sensory-affective dissociation is well correlated with the neuropathological findings in AD, mentioned above [23].

The degree of progression of AD could deeply influence the functioning of the medial and lateral pain systems. Cole and colleagues studied pain responses and compared them with cognitive function evaluations through f-MRI after mechanical stimulation in patients early stage dementia. They observed that medial and lateral systems were preserved in AD patients and controls had similar results [24].

It has been observed therefore that in early stages of AD both networks – medial and lateral – work correctly. The next step, clinically, is to understand the influence of severe cognitive loss on pain perception in this patient population.

PAIN PERCEPTION IN VASCULAR DEMENTIA

In comparison to AD, where brain lesions are topographically constant, vascular dementia (VaD) is a heterogenous disorder. In fact, vascular lesions could affect different brain regions and consequently determine different effects on pain perception. As a consequence it is not possible to predict how a vascular dementia patient feels pain.

However it's known that VaD is mainly determined by white substance lesions. These lesions becomes clinically relevant when they block afferent fibres, for example thalamus-cortex projections, and cause a deafferentation syndrome and consequently hyperalgesia [25].

Scherder and colleagues observed that patients with VaD showed an increase affectivity in pain perception due to several conditions such as osteoarthritis, fractures and diabetic neuropathy [26]. Another study showed

that periventricular hyper-density is correlated to an increase of pain perception's affectivity [27]. Therefore it is reasonable to observe that patients with VaD could feel pain differently, showing a more affective/emotional component.

PAIN PERCEPTION IN FRONTO-TEMPORAL DEMENTIA

In fronto-temporal dementia (FTD) a degeneration of frontal and temporal lobes occurs. As frontal lobes have a central role in pain elaboration, pain experience is altered. Some studies described loss of pain consciousness as one of remarkable behavioural symptoms of FTD, useful for differential diagnosis with other sub-type dementias.

Therefore, it can be hypothesized that stricter neuroradiological criteria may play a central role in demonstrating an increase in pain tolerance as well as improved detection of neuroanatomical changes. Such measures may improve the identification of patients with compromised medial pain system areas. In particular, areas such as the anterior cingulate cortex, the insula, and the prefrontal cortex show signs of atrophy in FTD.

The observed changes in pain processing in FTD patients depend on how the diagnosis of FTD is performed. The clinical diagnosis alone, mainly based on neuropsychological testing, may fail to isolate the specific frontal and temporal impairment. Conversely, neuroimaging techniques may reveal specific frontal and temporal anatomical changes that are more specifically related to some aspects of pain processing [28].

These findings differ in part from those described in AD. In fact, while a selective loss of the affective components of pain have been reported in AD patients with conserved aspects of the sensory-discriminative component, herein, studies also found elevated pain thresholds in FTD patients [29]. Pain threshold is a sensory-discriminative aspect of pain sensation, which is mediated by the lateral pain system, while pain tolerance represents an affective aspect that is mediated by the medial pain system (Tab. II).

CHALLENGING PAIN ASSESSMENT IN DEMENTIA

In clinical practice, pain in elderly verbal patients – with or without mild cognitive impairment – is usually assessed by means of self-reports. Self-reporting is considered as the 'gold standard' in pain assessment. A broad range of self-report scales is currently available to assess pain in the elderly. The great majority of these have been developed and tested in very different settings before being applied to elderly people with dementia. The most frequently assessed component of pain is pain intensity [30].

Commonly used measures of pain intensity include Visual Analogue Scales, Verbal Rating Scales, Numeric Rating Scales and Facial Pain Scales. It is generally worth noting that elderly people find it difficult to use self-report scales correctly and no single self-report scale seems appropriate for all elderly people, especially with dementia. For instance these scales typically assess only sensorial-discriminative characteristics of pain instead of emotional and affective ones. Approaching pain in non-verbal patients by using self-reports scales is inadequate, as dementia patients feel pain differently depending on the sub-type of dementia and individual communication deficits [4].

The progression of dementia severely compromises the ability to communicate, and verbal reports of pain tend to become less reliable. Other approaches, such as observational and surrogate reporting, are necessary in patients with moderate and advanced dementia. Over the past decade a number of observational tools for use with nonverbal older adults with dementia have been developed. These tools focus on the assessment of non verbal behaviours such as facial expressions, paralinguistic vocalizations, guarding, bracing, changes in

Table II. Relation between neuropathology and results of experimental and clinical studies in subtypes of dementia on motivational-affective aspects and presence or intensity of pain (modified from Scherder et al., 2005).

	Motivational-affective aspects of pain *(medial system)*			**Presence or intensity of pain** *(lateral system)*
Condition	Neuropathological involvement	Experimental and clinical results	Neuropatholical involvement	Experimental and clinical results
Alzheimer's disease	Degeneration of thalamic intralaminar nuclei	↓ Decreased	Unaffacted	Unaffacted
Vascular dementia	De-afferentation	↑ Increased	Not examined	Not examined
Fronto-temporal dementia	Degeneration of prefrontal cortex	↓ Decreased	Not examined	Not examined

social behaviour, changes in sleeping patterns, aggressive behaviour, changes in psychomotor activity, and others [31 32]. While the validity of several of these tools has been supported, many of the behaviours assessed by pain assessment tools also represent manifestations frequently present in psychogeriatric disorders, such as delirium, depression or Parkinson disease [33].

Another weakness of this approach is the considerable inter-individual variability between patients with dementia in their expression of pain via behavioural demonstration. This consideration is immensely important, given the presence of atypical behaviour in different types of dementia and the frequently associated concomitant neuropsychiatric disorders [34 35].

In non verbal patients the autonomic responses do not seem to be useful in pain assessment, as these reactions do not directly represent pain intensity. As previously decribed, AD patients show reduced autonomic responses, but they can discriminate between a tactile or pain experience [18].

Clinical guidelines for older adults have been published by the American Geriatric Society (AGS) panel from 1998, with regular updates in 2002 and 2009. The latest version also includes recommendations for accurate pain assessment in patients with dementia [36] (Tab. III).

Although a standardized assessment tool is not yet available for widespread use, a comprehensive approach to pain assessment is recommended in non-verbal older adults with dementia.

Table III. Common pain behavoirs in elderly patients with dementia according to AGS Panel on persistent pain in older persons.

1. Facial espressions	• Slight frown; sad, frightened face • Grimacing, wrinkled forehead • Closed or thightened eyes • Any distorted expression • Rapid blinking
2. Verbalizations, vocalizations	• Sighing, moaning, groaning • Grunting, chanting, calling out • Noisy breathing • Asking for help • Verbally abusive
3. Body movements	• Rigid, tense body posture, guarding • Fidgeting • Increased pacing, rocking • Restricted movement • Gait or mobility changes
4. Changes in interpersonal interactions	• Aggressive, combative, resisting care • Decreased social interactions • Socially inappropriate, disruptive • Withdrawn
5. Changes in activity patterns or routines	• Refusing food, appetite change • Increase in rest periods • Sleep, rest patern changes • Sudden cessattion of common routines • Increased wandering
6. Mental status changes	• Crying or tears • Increased confusion • Irritability or distress

PAIN MANAGEMENT IN DEMENTIA. PRACTICAL AND CLINICAL ISSUES

The issue of pain treatment in literature is lacking in some key areas but the evidence does provide consistent support for specific approaches to address pain. Randomized controlled trials (RCTs) and systematic reviews show a high level of agreement around the value of treatment with acetaminophen as a first-line approach, aligning with the recent and current guidelines from both the AGS and British Geriatric Society (BGS) [36 37]. The value of stepped treatment approaches to address pain is greatly enhanced in the literature, starting with a comprehensive medical and personalized non-drug 'comfort' approaches before escalating to pharmacological treatment [38 39].

Despite this, non steroidal anti-inflammatory drugs (NSAIDs) are widely prescribed for pain in the elderly, and those drugs must be used with caution in older people. This is due to the high risk of potentially serious and life-threatening side effects, as prostaglandins have a pivotal role in the normal human physiological functions of the GI tract, renal and cardiovascular systems, as well as others. Of note, data about hospital admission due to adverse drug reactions is shocking in its scope (23.5% in the elderly population) [37].

Opioid use in older people may be associated with less risk than that of NSAIDs, particularly in those older people who are at particular risk of NSAID-related events. As there is marked inter-patient variability in efficacy and tolerability of individual opioids, if there is no analgesic response or significant adverse events with one opioid, switching or rotation may be considered. Opioid analgesics should be considered an effective alternative therapy for older patients with moderate-to-severe pain or for pain that impairs functioning and quality of life, instead of NSAIDs. Additionally, they should be administered as part of a comprehensive pain management strategy. Although older people tend to require lower doses than younger individuals, opioid effects do not appear to vary with age and careful dose titration based on individual response is required [40].

Many side effects, such as sedation, nausea and vomiting, may be worse during opioid initiation or dose

escalation, and may resolve after 2 or 3 days [41]. On the other hand, constipation does not readily improve and may be managed with laxative therapy or a peripheral opioid antagonist (such as oral prolonged-release naloxone). In this regard, a recent open-label prospective study of our group has demonstrated that low-dose of oxycodone/naloxone is effective and well tolerated for treatment of moderate-to-severe chronic pain in geriatric population. In particular, this opioid agonist-antagonist combination reduced the impact of pain on daily activities with significant improvement in daily functioning; of note, no changes were observed in cognitive status and bowel function. Besides its effectiveness, these data indicate that low-dose opioids may be reasonably considered a safe analgesic option in fragile patients [42 43].

Of note, some adjuvant drugs treating associated conditions such as tricyclic antidepressants and some anti-epileptic medicines have been shown to have a significant beneficial effect on the attenuation of neuropathic pain [41].

Therefore a recent research by Dublin and colleagues has revealed that people with the heaviest opioid or NSAID use had slightly higher dementia risk than people with little or no use. These results may reflect an effect of chronic pain on cognition. Although opioids have other risks, little evidence of long-term cognitive harm specific to opioids was found [44].

Despite this data and the high prevalence of pain in dementia, the quality of pain management is currently lacking in clinical practice.

Recent interventions in pain management have been effective in reducing both pain and behavioural symptoms in dementia [45]. Manfredi and colleagues investigated effect of opioid analgesics on pain intensity and behavioural disturbances in these specific subpopulations. They evaluated 25 patients with agitation assessed by Cohen-Mansfield Agitation Inventory. Of the 25 subjects, 13 showed significant reduction of agitation after 4 weeks [46].

In a placebo-controlled crossover trial with 25 patients, Chibnall et al investigated the efficacy of acetaminophen on emotional well-being and behaviour and they reported significant improvement in activities but found no effect on agitation [47].

Another RCT in 114 patients with behavioural disturbances were assigned randomly to either a serial trial intervention (STI) of stepped assessment and treatment or usual care; results indicate that the STI approach improved behavioural symptoms significantly, although the effect of analgesics was not reported [48].

A clear limitation in the existing literature is the lack of large RCT studies with pain intensity as the main outcome. To date, no large-scale pain intervention studies have focused upon improvement of pain intensity as a key outcome.

Interestingly, a recent study on 352 patients with dementia showed that a stepwise protocol on pain intensity in nursing homes significantly reduced pain intensity as assessed through observational scales (Mobilization-Observation-Behaviour-Intensity-Dementia-2 Pain Scale). Remarkably it was also observed that administration of acetaminophen also improved ADL function [49].

Moreover in a prospective observational study on 53 elderly patients with cogntive impairment Petrò and colleagues proved oxycodone/naloxone to be effective in improving control of pain and behavioral symptoms associated to dementia, with a favorable safety and tolerabiltiy profile and no bowel interference [50].

Indeed, further large-scale trials of treatment approaches in people with dementia are needed to improve clinical guidance.

PAIN AND ITS RELATION WITH BEHAVIOURAL PSYCHOLOGICAL SYMPTOMS OF DEMENTIA

In addition to cognitive decline, dementia is commonly accompanied by neuropsychiatric symptoms, which largely overlap with the term “behavioural and psychological symptoms of dementia” (BPSD). The aetiology of the neuropsychiatric symptoms is poorly understood. It is suggested that the cause is multifactorial, based on chemical, anatomical and transmitter changes in the brain. Other hypotheses speculate that it’s related to physical diseases, or to unmet needs such as boredom, fear or pain. There is an association between pain and neuropsychiatric symptoms in patients with dementia. A considerable number of items in current pain tools for patients with dementia overlap with those in neuropsychiatric inventories.

Some of these BPSDs appear to be dictated by the severity of pain, with more severe pain resulting in reduced wandering and pacing but increased aggressive responses. Importantly, these symptoms may also respond to analgesic treatment in clinical trials, with the largest RCT reporting improvement in agitation and aggression following stepped treatment of pain. As a matter of fact, verbally agitated behaviours such as complaining, negativism, repetitious sentences and questions, constant requests for attention, cursing or verbal aggression have been found to respond to pain treatment [49].

However the literature on whether pain is associated with agitation in people with dementia is inconsistent. Volicer et al. [51]. found in a longitudinal study of 2032 Dutch nursing home residents that pain was not highly

related to agitation and pain scores did not change in proportion to agitation scores. Other studies have found an association between agitation and pain in hospital-dwelling people with dementia [52].

In a recent prospective study, pain was found to predict the development of aggression [53]. Further investigations have suggested that pain can manifest as agitation or aggression in people with dementia and that pain treatment may ameliorate these symptoms [54].

A recent study confirmed the efficacy of pain treatment in ameliorating mood symptoms Those patients received individual daily pain treatment with acetaminophen, extended release morphine, buprenorphine transdermal patch or pregabaline for 8 weeks. Mood symptoms, including depression, were found to significantly improve with pain treatment, emphasizing the importance of more rigorous treatment of pain in agitated people with dementia [55].

Current literature shows no improvement in psychotic symptoms such as deliria and hallucinations with the treatment of pain in these populations. This defines a clear and typical impact of pain on behaviour of Dementia patients.

The impact of pain on BPSD is of particular importance since these symptoms are commonly treated with antipsychotic medications. Antipsychotic drugs are associated with considerable and severe side effects, including worsening of cognitive decline, Parkinsonism, stroke and death. Despite recent reductions in the use of these drugs, prescription levels remain high, and are often outside their licensed use [56].

Another similar concern is raised on the treatment of sleep disorders. Several studies have suggested that the use of sedative drugs masks the underlying causative pain [57]. This issue highlights the concern about inappropriate drug prescription in the elderly and the consequent risk of polipharmacy, which might be avoided by an accurate assessment and treatment of the underlying pain.

However, in patients with dementia, if the presence of pain is uncertain, an analgesic intervention may be warranted to evaluate the presence of pain. If the interventions appear to provide pain relief, pain may be assumed as the likely cause and the length of intervention protracted.

DISCUSSION AND FUTURE PERSPECTIVES

When considering pain management in dementia, clinicians are frequently faced with situations that question the very nature of suffering. The inability of individuals to accurately convey their feelings complicates our efforts to identify and measure the nature of their discomfort. The diminished capacity of individuals with dementia to advocate for themselves, however, increases the duty of care of those who are charged with this responsibility. There is a high risk for under-treatment of pain in dementia, particularly in non-communicative patients with white matter lesions, but also in dementia patients who report less prevalent and intense pain. Older adults with dementia receive less pain medication than those who are able to communicate, even though they are just as likely to experience painful illnesses [9].

In fact, findings from clinical and experimental pain studies do not suggest that pain is less frequent and intense even if it's no longer reported. On the contrary, the impact of dementia on pain processing varies in patterns and quality, depending on the type of pain, neuropathology and stage of dementia.

The most important finding is that according to type of dementia, patient could experience different pain perception related to specific brain damage [16-18]. It's reasonable to conclude that consequently for each type and stage of dementia, there is a specific pattern of pain thresholds and detection. Critically, treating physicians should consider the unique pattern of cognitive degeneration in managing the pain in each and every patient with dementia.

Assessment of pain in non-verbal older adults with dementia remains a challenge for clinicians and researchers. Future pain assessment in this vulnerable population may reveal opportunities for recognizing pain through enhanced brain imaging techniques and monitoring of pain-related chemical substances currently under study. However, for the immediate future our focus must be on strategies to assist clinicians recognize pain with readily available methods and resources. A great priority is to raise awareness of pain presence and potential indicators to screen for potential pain.

Critically, very poor literature is present on pain treatment in older persons with dementia at this time. Adequate pain control in patients with dementia depends on good pain evaluation and may express itself in an improvement in behaviour and activities of daily life, since verbal communication about pain is not reliable.

It is essential that implementation and continuous education and training programs be developed, implemented, and evaluated to ensure the effective use of any new tool. These are prerogative steps for better management. There is a great need to provide support and clear guidance for clinicians and other health professionals.

"Start slow and go slow" is the key word in pain therapy in geriatrics, but the benefit–risk ratio of any analgesic treatment must also be kept in mind. Further studies in older persons with dementia with regard to the therapeutic approach and the link between pain and

behavioural symptoms would be of great importance in order to clarify these issues.

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