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Pharmacological Management of Pain in Older People

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ABSTRACT

The prevalence of pain increases with advancing age. Pain is often undiagnosed or under-treated in older patients, particularly those with cognitive impairment, communication problems or residents of aged-care facilities. The most definitive approach to the management of pain is to treat the underlying cause. As this is often not feasible in the elderly, the emphasis then shifts to symptom control. Pharmacological approaches form the mainstay of therapy. They are convenient and often cost-effective, and may be used alone, or in combination with physical and psychological therapies.

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INTRODUCTION

Despite its increased prevalence, pain is not an inherent part of the ageing process. The greater prevalence of pain in the elderly is largely related to the increased burden of pathology. Untreated pain has many adverse consequences. It may lead to decreased mobility, functional impairment, sleep disturbance, weight loss and depression. It may compromise the effectiveness of rehabilitation programs and the person's ability to remain in their own home. In advanced dementia, behaviours such as resistance, aggression and irritability may be expressions of pain, yet attributed to the dementia.

Pain is an inherently subjective experience. Pain is not solely an unpleasant sensory phenomenon, it is also an emotional experience.¹ The patient's self-report is the most reliable method of evaluating the impact of pain. Pain is often unrecognised and under-treated especially in those with cognitive impairment, communication problems or residents of aged-care facilities. The inability to communicate verbally does not negate the possibility that an individual is experiencing pain and is in need of suitable pain relieving treatment.

The most definitive approach to the management of pain is to treat the underlying condition. This is often not feasible in older patients, at which stage the goal shifts to symptom control. Although pain cannot be eradicated in all circumstances, good outcomes can be achieved with increased vigilance for the presence of pain, using available options, and making adjustments according to the response to therapy.

PREVALENCE

Prevalence studies of persistent pain often report levels exceeding 50% in community-based samples and up to 80% in aged-care facilities.^{2,3} An Australian community-based study reported 17% of males and 20% of females experienced pain every day for 3 months in the 6 months prior to the interview.⁴ About 65% of those with pain reported some degree of interference with daily life. The prevalence of persistent pain

peaked in the 65 to 69 year age group in males at 27% and in the 80 to 84 age group in females at 31%. A study undertaken in nursing homes in New South Wales reported 28% of residents had pain at the time of interview, yet in many instances no analgesic was given; 41% of residents were not included in the study as they were unable to communicate.⁵ No conclusions were drawn as to what proportion of this group had pain.

Joint, foot and leg pain become more prevalent with age. Conversely headaches peak between 40 and 50 years, and facial, dental, abdominal and chest pain becomes less prevalent in old age. Age-related prevalence of back pain varies among studies and no definite conclusions can be drawn.³

Women have a significantly higher prevalence of pain when compared to men of similar age. As females represent a larger proportion of the older population, this has a disproportional impact on the age-related prevalence rates of pain. The magnitude of gender differences varies according to the disease, for instance, rheumatoid arthritis, osteoarthritis, headache, and fibromyalgia are more common in women, whereas gout, ankylosing spondylitis, and coronary heart disease are more common in men. Biological factors have less impact on the gender difference in the elderly as they do during reproductive years. Social and lifestyle factors need to be considered too.

PAIN PERCEPTION

Evidence from many psychophysical studies suggests a small, but potentially important age-related increase in pain threshold. This may be associated with under-reporting of pain and an increase in the risk of undiagnosed disease or ongoing injury. Reduced efficacy of the endogenous analgesic system and greater sensitisation of pain processing pathways may contribute to greater persistence and anatomical spread of pain.⁶ In contrast to the reduced sensitivity, once pain is experienced there is an age-related decline in the ability to tolerate severe pain.⁷

Silent myocardial infarction is a well-recognised entity in older people occurring in 30 to 40% of cases, and pain may not be a major feature of peritonitis or intestinal obstruction in older individuals.⁸ The absence of pain in the elderly should not be taken to indicate the absence of a serious condition usually associated with pain in a younger person.

UNDER-TREATMENT OF PAIN

Despite advances in pain control and proliferation of pain management guidelines, pain in older people is often under-treated. The presence of cognitive impairment, communication problems and residence in aged-care facilities increase this risk.

The elderly often under-report their own pain, putting it down to getting old and stoically suffer in silence not wanting to bother carers. They may also be concerned that the pain may be a symptom of a serious disease that they would prefer not to know about. Medical and nursing staff often fail to inquire adequately about the presence of pain. Some dismiss pain that does not have an identifiable medical reason.⁹ Documentation of the presence of pain is often poor. Older people receive less analgesia than younger individuals with similar conditions; and

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those with cognitive impairment receive significantly less than cognitively intact individuals.¹⁰

The adage of safe prescribing for older people, 'start low and go slow' may lead to under-treatment of pain. The frequency of clinical review and dose escalation should be determined by the level of acuity and level of distress. Patients with severe pain may need review and dose adjustment a number of times per day. In contrast, if pain has been present for some years a number of weeks can be allowed between dose adjustments.

Doctors may under prescribe analgesics because of fear of aggravating other medical problems, such as constipation and cognitive impairment, and concerns about polypharmacy. Analgesics have the potential for affecting cognition, however neuropsychological function may also be affected by unrelieved pain.¹¹ Patients with under-treated pain following a hip fracture were 9 times more likely to develop delirium than patients whose pain was adequately treated. Inadequate doses of opioids increased the risk of delirium in this population.¹² The co-administration of analgesics with different modes of action may decrease the dose requirement of each drug, and hence dose-related adverse effects.¹³ Paracetamol is often co-administered with codeine, and in postoperative pain management the co-administration of paracetamol has been shown to reduce morphine requirements.¹⁴ The combination of morphine and gabapentin is more effective for post-herpetic neuralgia than either alone.¹⁵

EVALUATION

The gold standard for evaluation of pain is the individual's self report. The pain history should focus on the onset and temporal pattern of the symptoms, site and quality of the pain, the severity, aggravating and relieving factors, and the impact that pain is having on lifestyle. If the problem is complex the assessment may need to take place over several consultations. A collaborative history from a family member is often most helpful. The reliability of the history may be affected by the chronicity of the pain, comorbid conditions, past interventions and alterations in cognition. The assessment should include functional, social and psychological aspects and where possible the individual should be assessed within their own environment.

The most commonly used pain measurement tools focus on pain severity. These tools include the visual analogue, numerical and word descriptor scales. With a visual analogue scale, patients indicate the severity of pain by placing a mark on the appropriate point along a 10 cm line, marked at one end 'no pain' and at the other 'most severe pain'. With a numerical descriptor scale, patients indicate pain severity by selecting a number from 0 to 10, with 10 being the most severe pain. With a word descriptor scale, patients are asked to select the most appropriate adjective to describe their pain from a list such as, no pain, mild pain, moderate pain, severe pain, very severe pain and worst possible pain. These tools can be repeated a number of times daily to evaluate response to treatment.

Multidimensional tools provide further information about the impact of the pain experience on the individual. The McGill Pain Questionnaire has been validated for an older population, and includes measures of the sensory, cognitive and evaluative aspects of the pain experience.¹⁶ It is largely used for research purposes. The Brief Pain Inventory is a widely used tool that is available in many languages.¹⁷ It measures the severity and location of pain, as well as its impact on sleep, mood, activity, mobility, socialisation, depression and anxiety. It has recently been modified specifically for use in residential care facilities.¹⁸

To ensure that pain is being appropriately identified and documented, and the adequacy of treatment assessed, the US Department of Veterans Affairs has mandated that pain is reported as the fifth vital sign every time pulse, blood pressure, temperature, and respiration are recorded in all of its facilities.¹⁹

PAIN IN DEMENTIA

During the early to middle stages of dementia, communication abilities tend to remain sufficiently intact for the verbal self-report of pain. Individuals with cognitive impairment may be able to inform about the presence or absence of pain, but may have difficulty with more complex questions, such as identifying the location and quality of the pain, diurnal variation, and aggravating and relieving factors.

The ability to complete simple pain measurement tools is often retained by patients with moderate to severe dementia. Eighty-three per cent of nursing home residents with an average mini-mental status examination score of 12/30 were able to complete at least one self-report pain measure with the word descriptor scale having the highest completion rate.²⁰⁻²²

A multidimensional instrument, The Resident's Verbal Brief Pain Inventory is a modified version of the Brief Pain Inventory developed by the Australian Pain Society (APS) specifically for use in aged-care facilities including subjects with moderate degrees of dementia.¹⁸

Patients with severe dementia may be capable of verbal self-report, although as communication ability deteriorate there is increasing role for observational pain scales. There are a number of validated observational scales for this population.²³ The presence of pain or discomfort is inferred from the observation of certain behaviours. The APS recommended Abbey Pain Scale is a 4-point scale that is used to rate six domains of observation: vocalisations, facial expressions, body language, behavioural changes, physiological changes and physical changes.²⁴ This and other pain measures appropriate for this population are published in *Pain in Residential Aged Care Facilities: Management Strategies*.¹⁸

EXPECTATIONS OF THERAPY

Despite advances in pharmacotherapy and interventional approaches, pain cannot be eliminated in all situations. The average response to opioids for persistent non-malignant pain is 30%, and most trials of neuropathic pain are based on a 50% reduction in pain severity, not total eradication. Good communication is necessary to ensure that patients have realistic expectations. A mismatch between the patient's expectations and the outcome may lead to loss of confidence in the practitioner, disenchantment, mood disturbances, medication abuse, doctor shopping, and vulnerability to exploitation.

When treating cancer pain, alleviating symptoms is the main goal, whereas in persistent non-cancer pain the goal is to keep the patient functional (physically and mentally) with improved quality of life. Patients with ongoing pain following a comprehensive evaluation and adequate trial of therapy may need to come to terms with the fact that the pain may be persistent. The treatment goal shifts from aiming for a cure, to one of restoration of function and psychological state. Medications are adjusted to ease the pain to tolerable levels without causing intolerable adverse effects.

MANAGEMENT

The type of therapy is often determined by the personal preferences of the patient and the type of practitioner they choose to consult. The most effective approach to the management of persistent pain often involves a combination of pharmacological and non-pharmacological therapies. If a treatment fails to adequately control the pain then other therapeutic options should be considered. Where appropriate, interventional techniques such as intra-articular steroid may provide pain relief without systemic adverse effects. There is an increasing evidence base for the efficacy of some traditional and non-Western approaches.

Pharmacological Approaches

Pharmacological approaches are the mainstay of most interventions for pain as they are convenient and often cost-effective. Many patients will have initiated treatment prior to seeking professional consultation. Selection of therapy requires an understanding of age-related pharmacokinetic and pharmacodynamic changes, and needs to take into account any coexisting diseases and other medications, including those obtained without prescription. The choice of drug is based on the balance of the potential efficacy with the potential for harm.

The timing of drug administration is important. Predictable episodes of pain brought on by certain activities (e.g. wound dressings, weight-bearing) is known as incident pain. It may occur on a background of continuous pain or the patient may otherwise be pain free. This is best managed with pre-emptive short-acting analgesia. In contrast, pain that has been present for more than 3 to 6 months is referred to as chronic or persistent pain. It may be intermittent or continuous. It is best managed with regular long-acting analgesics. Transient exacerbations of pain occurring on a background otherwise satisfactorily controlled pain, known as breakthrough pain, is managed with short-acting analgesics administered on an 'as required' basis.

The WHO analgesic ladder was developed to provide a rational three-step approach for the management of cancer-related pain. It is widely used for non-cancer pain. Step 1 uses non-opioid analgesia for mild pain, Step 2 uses weak opioids with or without non-opioid analgesics for pain of moderate severity, and Step 3 uses strong opioids for severe pain. Adjuvants may be used at any stage if required. This has provided a useful guide for clinicians, although recently the appropriateness of this approach has been questioned.²⁵ The stepwise approach does not address how to deal with pain that is severe at the onset, or how to integrate procedural techniques into a management strategy. Increasingly Step 2 is being bypassed going directly to a low dose of a strong opioid such as oxycodone.²⁵

Non-Opioid Analgesia

Paracetamol is the preferred first-line analgesic for mild to moderate pain.²⁶ The recommended adult dose is 0.5 to 1 g 4-hourly to a maximum of 4 g/day. The duration of action of paracetamol is short and the analgesic effect reaches a plateau following a single dose of 0.6 g. When pain relief is not sustained it may be better to give a lower dose more frequently up to the maximum daily dose. Alternatively, extended-release paracetamol may be considered (maximum dose two 665 mg tablets 6- to 8-hourly up to a maximum of 6 tablets in 24 hours, or 3990 mg). The preference for paracetamol as the first-line analgesic is not based on its efficacy or convenience of dosing, but its better safety profile. It can be safely used on a long-term basis. Hepatotoxicity is very rare in standard therapeutic doses. The risk is increased in malnourished individuals and with chronic alcohol consumption. Most cases of paracetamol-induced hepatotoxicity are due to deliberate or accidental overdose. The availability of a plethora of paracetamol containing analgesics and cold and flu preparations without prescription increases the risk of inadvertent paracetamol overdose.

If paracetamol is insufficient to control pain the options include adding codeine or a non-steroidal anti-inflammatory drug (NSAID) particularly if there is an inflammatory component to the pain. A number of meta-analyses have shown superiority of NSAIDs over paracetamol for pain relief in osteoarthritis. The benefits are relatively modest, and the risk of adverse events associated with NSAID use is of concern. NSAIDs are associated with toxic gastrointestinal effects, decreased renal

perfusion, decreased glomerular filtration rate, oedema, hypertension, interstitial nephritis and vascular events. The risks of gastrointestinal bleeding with an NSAID are increased in those 65 years or older, with a history of peptic ulcer disease or upper gastrointestinal bleeding, concomitant use of oral glucocorticoids or anticoagulants, and the presence of comorbid medical conditions. Where the risk of gastrointestinal bleeding is high a COX-2 selective NSAID should be considered. Alternatively, a proton pump inhibitor may be co-administered with an NSAID for gastro-protection.²⁷

Following mounting evidence of increased risk of vascular events, rofecoxib a COX-2 selective NSAID was withdrawn in 2004. Subsequent meta-analysis has revealed a 42% relative increase in the incidence of serious vascular events with COX-2 selective NSAIDs compared with placebo, largely due to a twofold increased risk of myocardial infarction.²⁸ The cardiovascular risk appears to be dose related. There is an increased risk with high-dose celecoxib, but not with commonly used doses.²⁹ Several other NSAIDs are associated with increased cardiovascular risk, including diclofenac, meloxicam, indomethacin, and ibuprofen. Naproxen does not appear to increase cardiovascular risk.³⁰ It is unclear whether aspirin abolishes NSAID-related cardiovascular risk but concomitant use of aspirin appears to eliminate any gastrointestinal benefit of COX-2 selective NSAIDs questioning the logic of prescribing them in high-risk patients.

Opioid Analgesia

An opioid may be considered if an NSAID is not appropriate, not tolerated or fails to control the pain. Codeine is a weak opioid commonly used in combination with paracetamol or aspirin, either self-administered or prescribed. Codeine is devoid of analgesic activity until metabolised by CYP2D6 to morphine. Approximately, 8% of Caucasians and 2% of Asians are genetically deficient in CYP2D6 and obtain little pain relief from codeine. In addition, a number of commonly prescribed drugs, such as haloperidol, amitriptyline, fluvoxamine, fluoxetine and paroxetine, inhibit CYP2D6. Despite its widespread use there is little evidence to support the efficacy of codeine used on a long-term basis. There are no reported trials of the most common combination of paracetamol with 8 mg of codeine. A single dose of 60 mg codeine when added to paracetamol produces an increased analgesic effect of 5%. A daily oral dose of codeine 240 mg is equivalent to 45 mg of oral morphine per day. This dose is associated with severe constipation.³¹ Codeine has a ceiling dose effect, above which further dose increases will not increase analgesia.

The efficacy of tramadol 100 mg and paracetamol 1 g/codeine 60 mg is comparable. Tramadol is a centrally acting synthetic analgesic that has a dual mode of action. It has weak affinity for the mu opioid receptor. It also inhibits reuptake of serotonin and noradrenaline. Structurally, it is not an opioid and it is partially antagonised by naloxone. Tolerance and dependence are uncommon. It is of benefit in nociceptor (musculoskeletal) and neuropathic pain.^{32,33} Its usefulness is lessened because of a high potential for adverse effects and drug interactions. For older people the recommended maximum dose is reduced to 300 mg/day. The most common adverse effects are nausea, vomiting, dizziness, constipation, sweating, tiredness, and headache. Over 20% of trial patients were unable to tolerate tramadol although the adverse effects tend not to be life threatening and are reversible on cessation. Tramadol may precipitate serotonin syndrome by itself, but particularly when used with other serotonergic drugs, such as selective serotonin reuptake inhibitors and monoamine oxidase inhibitors. Signs of serotonin syndrome include confusion, agitation, fever, sweating, ataxia, hyper-reflexia, myoclonus and diarrhoea.³⁴

Despite its popularity among some patients the use of dextropropoxyphene is discouraged. Combinations of paracetamol with dextropropoxyphene are no more effective than paracetamol alone.³⁵ Dextropropoxyphene is structurally related to methadone, and is associated with physical and psychological dependence. Its major metabolite is cardiotoxic. Chronic high dose use has been associated with psychosis and convulsions.

When other analgesics do not provide sufficient relief a strong opioid may be considered. Opioids have a well-established role in the management of acute pain and in palliative care. The role of opioids in the management of persistent non-cancer pain raises special concerns. Opioids are effective for the management of persistent neuropathic (post-herpetic neuralgia, diabetic neuropathy) and musculoskeletal pain (osteoarthritis) with similar efficacy. The mean decrease in pain intensity is in the order of 30%. About 80% of patients experienced at least one adverse event, with constipation (41%), nausea (32%) and somnolence (29%) being most common.³⁶ These benefits have largely been demonstrated in short-term studies. There are no long-term randomised controlled trials of opioid use. A large cross-sectional population study comparing long-term opioid users with non-users found that opioid therapy did not achieve the desired outcome in any one of the key goals of pain relief, quality of life and functional status.³⁷ Due to the nature of the study no causative relationship could be established between the worse outcomes of individuals on opioid therapy but it does raise some concern.

Older adults have increased sensitivity to opioids achieving equivalent analgesia at lower doses. Adverse effects are more common. Sedation, nausea and vomiting are usually transient, but constipation persists and should be managed effectively. Opioids may cause confusion particularly in those with a pre-existing dementia and increase the risk of falls in patients with impaired balance or mobility.

Most opioids are equally efficacious for persistent pain when given in equi-analgesic doses. Any opioid that produced fewer adverse effects than morphine, at a dose that provided the same degree of analgesia, would be an improvement. For most clinically important adverse effects, there are no comparative data at equi-analgesic doses to allow recommendation of any of the alternatives.³⁸ There is a trend to initiate controlled release opioids for management of persistent pain rather than initial dose titration using an immediate release preparation. Parenteral opioid administration should be avoided for persistent non-cancer pain.³⁹

The choice between controlled-release morphine and controlled-release oxycodone appears largely one of personal preference. If one is not well tolerated then it is worth trying the other. Oral oxycodone has a higher oral bioavailability than oral morphine, which may result in less interindividual variation in the bioavailability.⁴⁰

Transdermal opioid patches offer steady-state medication delivery with the convenience of less frequent dosing. Two transdermal opioid patches are available in Australia, fentanyl and buprenorphine. They have not been proven to be more effective than oral opioid preparations but may be suitable for patients who cannot take oral opioids because of adverse effects. Adverse effects are similar to those of oral opioids. The most common side effect is constipation, but it appears less frequently than with oral morphine. The delayed onset of action makes them unsuitable for acute pain. The onset of side effects including delirium may be delayed by a number of days following the initiation of therapy resulting in the association with the opioid patch not being immediately recognised. Patients

should be advised against the application of heat directly to a transdermal opioid patch (e.g. hot water bottles, heat pads, electric blankets) as this may result in a toxic increase in blood concentration of the opioid. Care must be taken to ensure the old patch is removed prior to applying a new patch.

Fentanyl transdermal patches need to be changed every 72 hours. The equivalent morphine dose to the fentanyl 25 µg/hour patch is approximately 120 mg of oral morphine over 24 hours. Fentanyl transdermal patches should not be initiated in opioid naive patients with non-cancer pain. Steady state blood levels may not be achieved for 24 to 72 hours after initial application. Drug effects continue after removal of the patch; fentanyl concentrations fall by 50% over 17 hours.⁴¹ Fentanyl is metabolised primarily in the liver and therefore may be more suitable for patients with renal failure than other opioids.

Buprenorphine transdermal patches need to be changed every seven days. Steady state concentration is not achieved until day 3 after initial application; other analgesics may be needed in the interim. There is a ceiling dose to its analgesic effect above which there is no further analgesic effect. The equivalent dose of transdermal buprenorphine and oral morphine has not been established. The manufacturer suggests that the dose range covered by the three patch strengths may be equivalent to oral morphine up to 90 mg/day. Buprenorphine transdermal patches are indicated for persistent non-cancer pain. They have not been evaluated in cancer pain. Due to its antagonist properties, buprenorphine may induce withdrawal symptoms and antagonise analgesia in patients using other opioid agonists. The effects of buprenorphine are only partially reversed by naloxone. After removal of the buprenorphine transdermal patch, concentrations decline approximately 50% in 12 hours.

Pethidine should also be avoided for prolonged use. The accumulation of its toxic metabolite norpethidine that may result in seizures, agitation, irritability, tremors, twitching and myoclonus. Methadone has a long and variable half-life making it difficult to use in older adults.

Co-analgesia

A number of drugs, known as adjuvants or co-analgesics, are often used in pain management, although their usual role is other than pain relief. The most widely used co-analgesics are the tricyclic antidepressants (TCAs) and antiepileptics. Other adjuvants such as ketamine and mexilitene are best reserved for pain specialists. Adjuvants may be used alone or in combination with conventional analgesics.

TCAs tend to be used for constant, burning neuropathic pains, whereas antiepileptics are better for sharp, shooting pains (although the evidence to support these statements is not strong). There is robust evidence supporting the effectiveness of TCAs in treating neuropathic pain, particularly amitriptyline. They produce analgesia more rapidly and at a lower dose than their antidepressant effect, in patients with or without depression. TCAs are not well tolerated by older people. The most common adverse effects are sedation, postural hypotension, falls and anticholinergic effects such as dry mouth, blurred vision, constipation and urinary retention. The starting dose should be low, for instance amitriptyline 10 mg *nocte*, increasing every 3 to 5 days to 50 or 75 mg. A trial of at least 2 weeks is required before deciding on effectiveness.⁴² Selective serotonin reuptake inhibitors are better tolerated than TCAs but are not effective for neuropathic pain. Some recent studies with the serotonin and noradrenaline reuptake inhibitors venlafaxine and duloxetine (unavailable in Australia) have demonstrated significant pain relieving effect in painful diabetic neuropathy, but not equivalent to amitriptyline.^{43,44} It is worth trying another class of antidepressant if a TCA is not tolerated.

There are surprisingly few trials to support the widespread use of antiepileptics for neuropathic pain. Gabapentin and pregabalin are of proven efficacy for painful peripheral neuropathies and post-herpetic neuralgia, and lamotrigine has been demonstrated to be of some benefit in post-stroke pain. Carbamazepine is the drug of choice for trigeminal neuralgia. Common adverse effects of antiepileptics are dizziness and somnolence that may limit clinical use, particularly in the elderly.⁴⁵

The management of neuropathic pain is difficult. Pain is rarely eradicated. It is often unresponsive to first-line analgesics such as paracetamol and NSAIDs, and may not be responsive to opioids. A 50% reduction in pain is used as the end point in most studies, although a smaller outcome may still be considered beneficial. There is no single treatment that works for all conditions. If the algorithm for the selection of medication for peripheral neuropathic pains such as post-herpetic neuralgia and painful peripheral neuropathies were based solely on pain relief, then the order would be TCAs first, followed by opioids, tramadol then gabapentin/pregabalin. If the criteria was based on a broader range of criteria including side effects, quality of life, dependency potential and cost, then the order changes. Finnerup et al. recommend initiating therapy with lignocaine 5% patches as they reduce pain without systemic side effects. These are not readily available in Australia. The next option is to try gabapentin/pregabalin or a TCA provided there are no contraindications. If the pain is not adequately controlled, then swap over to the other agent. The next step is tramadol or oxycodone.⁴³ A recent study reported the combination of gabapentin and morphine was more effective for neuropathic pain than either drug alone.¹⁵ Cost of medication is often a critical factor resulting in the selection of carbamazepine, and if not tolerated valproate, despite the lack of data supporting these drugs over the others.

Non-Pharmacological Approaches

There is a wide range of non-pharmacological approaches for the management of pain that may be used individually or as an adjunct to pharmacotherapy. Simple measures such as massage and hot packs are very popular. Physical techniques including exercise training, strengthening and balance exercises have general health benefits in addition to any improvement in pain. Psychological strategies including cognitive behavioural therapy have demonstrated efficacy in older populations. Advances in surgical techniques now enable older and frailer individuals to undergo procedures such as joint arthroplasty, often resulting in excellent pain control and functional restoration.

Where standard therapy has failed, practitioners may wish to involve other therapists or consider referral to a multidisciplinary pain clinic. There are few pain clinics that have the expertise to manage frail older patients with multiple medical conditions.⁴⁶ Treating older people the same way as younger adults without addressing their specific needs is bound to fail.

Complementary and alternate therapies are widely used, encompassing a wide range of biological and traditional treatments. Some treatments have been subject to critical appraisal, at times with conflicting results. The Rotta preparation of glucosamine was found to be superior to placebo for pain and function in osteoarthritis, whereas non-Rotta preparations failed to show benefit.⁴⁷ Some herbal remedies have demonstrated similar efficacy to NSAIDs.⁴⁸ Most clinical trials support the safety of complementary and alternate therapies, although adverse interactions of dietary supplements and herbal therapies with prescription medications are of concern. More detailed data about various complementary and alternate modalities are available on the National Centre for Complementary and Alternate Medicines web site <www.nccam.nih.gov>.

CONCLUSION

Pain is often undiagnosed or under-treated in older patients. The most definitive approach to the management of pain is to treat the underlying cause. This is often not feasible, leaving the individual at risk of ongoing pain, with consequences on function, mood and quality of life. All health professionals involved in the care of older individuals need to be even more vigilant about this common problem. General frailty and medical comorbidities make the selection of therapy more difficult. Adverse effects of therapy are more common. These challenges should not create insurmountable barriers to effective pain management.

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