J R Coll Physicians Edinb 2005; **35:**156–163

Abstracts: St Andrew's Day Geriatric Medicine Symposium 2004

ABBREVIATIONS Age-related macular degeneration (AMD), dopamine agonists (DAs), non steroidal anti-inflammatory drugs (NSAIDs), Parkinson's disease (PD), randomised controlled trials (RCTs), selective serotonin reuptake inhibitors (SSRIs), tricyclic amines (TCAs), World Health Organisation (WHO)

DAY 1

SESSION 1
PARKINSON'S DISEASE

Chairman: Dr S Pound, Consultant Geriatrician, Queen Margaret Hospital, Dunfermline, Scotland

Has this patient with tremor got Parkinson's disease?

Dr G Macphee, Consultant Geriatrician, Southern General Hospital, Glasgow, Scotland

Email graeme.macphee@sgh.scot.nhs.uk

Abstract Diagnosis of PD is often straightforward when a classical resting pill rolling tremor occurs in association with asymmetrical rigidity and bradykinesia. Nevertheless, other types of tremor may occur in PD and this can be confusing, particularly when they emerge in the absence of other clamant signs of PD. Other atypical Parkinsonian syndromes such as multiple system atrophy and progressive supranuclear palsy as well as drug-induced and vascular causes may all mimic PD. Awareness of atypical features, i.e. 'red flags' to other disorders, is crucial for accurate diagnosis.

The most useful approach to assessment of tremor is clinical, utilising information from history and examination. Initial description is based on the circumstances during which tremor occurs, i.e. static or resting tremor, or during muscle action, including postural and kinetic tremor. Further classification is determined by duration of symptoms, frequency, exacerbating and relieving factors and particular attention to current drug, family and medical history. Some tremors are associated with dystonia or emerge only during specific tasks, e.g. writing. The most common tremor in clinical practice is essential tremor and differentiation from PD may be very difficult in some patients if mild extra pyramidal signs are present. Utilisation of new imaging techniques such as FP-CIT SPECT scanning may be helpful in such indeterminate tremor syndromes.

References

1 Macphee GJA, Stewart DA. Parkinson's disease. *Rev Clin Gerontol* 2001; **11**:33–49.
2 Macphee GJA. Diagnosis and differential diagnosis of Parkinson's disease In: Playfer JR, Hindle JV (editors). *Parkinson's disease in the Older Patient.* London: Arnold; 2001.
3 Barker R, Burn DJ. Tremor. *ACNR* 2004; **4(1):**13–14.

Sponsors None.

Declaration Dr Macphee has received honoraria from GE healthcare (formerly Amersham Biosciences) for consultancy and lectures.

Drug therapy in a 75-year-old

Dr D Stewart, Consultant in Elderly Medicine, Mansion House Unit, Glasgow, Scotland

Email david.stewart@gvic.scot.nhs.uk

Abstract The treatment of PD remains symptomatic; no drug has been proven to alter disease progression. When starting drug therapy, there has been a change in emphasis away from improving morbidity as the only goal of treatment towards strategies designed to prevent or reduce long-term complications.

When deciding on a treatment strategy for a 75-year-old patient, considerations including the degree of disability, co-morbidity and life expectancy are important. Anticholinergic agents are commonly associated with cognitive side-effects and are best avoided. Selegiline has a modest anti-Parkinsonian effect and may be appropriate for some patients with mild disease. When significant disability emerges the choice of initial treatment is between levodopa and DAs. Levodopa remains the most effective drug in relieving morbidity but is associated with motor complications in many patients with long-term use. Initial monotherapy with DAs has been shown to reduce or delay the occurrence of these complications. Dopamine agonists are not suitable for all as they are more likely to cause adverse effects in vulnerable patients, particularly those with pre-existing cognitive problems. Careful patient selection is essential. In time all patients will require levodopa, but the dose should be kept to the minimum required to achieve satisfactory relief of symptoms.

Motor complications of longer-term levodopa therapy include fluctuating motor response ('wearing off' and 'on-off') and dyskinesia. Wearing off is manifested by end-of-dose deterioration and can be managed by adjunctive

CME

therapy with a DA, selegiline or a Catechol-O-methyltransferase inhibitor such as entacapone. Dyskinesia can be difficult to manage; strategies include attempts to reduce levodopa dosage, adjuvant DA therapy and the use of subcutaneous apomorphine. Amantadine can have an antidyskinetic effect but is often poorly tolerated in the elderly.

No two 75-year-old patients are the same; decisions regarding treatment need to be made on an individual basis by a clinician experienced in the management of PD.

References

1 Bhatia K, Brooks DJ, Burn DJ *et al.* Updated guidelines for the management of Parkinson's disease. *Hosp Med* 2001; **62(8):**456–70.

2 Macphee GJA, Stewart DA. Parkinson's disease. *Rev Clin Gerontol* 2001; **11:**33–49.

Key words Anticholinergic agents, antidyskinetic effect, amantadine, entacapone, levodopa, PD, selegiline.

Sponsors None.

Declaration Dr Stewart has received honoraria from GlaxoSmithKline, Pharmacia and Orion Pharma for consultancy and lectures.

Mental health problems in Parkinson's disease

Dr G McLean, Consultant Psychiatrist for the Elderly, Falkirk & District Royal Infirmary, Falkirk, Scotland

Email gmclean@fvpc.scot.nhs.uk

Abstract Parkinson's disease is a progressive neurological disorder arising primarily from the degeneration of dopaminergic neurones in the substantia nigra. However, there is an associated degree of degeneration in the brainstem serotonergic and noradrenergic systems as well as the basal forebrain cholinergic systems which play an important permissive and compounding role in the aetiology of cognitive disturbances and mood changes. Quality of life is determined not just by these disease-related symptoms, but also by response to various therapeutic strategies. A better understanding of the pathophysiology of various signs and symptoms in PD will help determine the most effective therapeutic interventions in individual patients.

Psychiatric symptoms frequently co-exist with PD and are often underdiagnosed and poorly treated. Accurate assessment and treatment of these conditions will lead to improved function, better quality of life, reduced length and frequency of hospital stays and lower levels of institutionalisation.

Depression and anxiety are the most common psychiatric accompaniments to PD. They occur in 40–60% of patients and are unrelated to the severity of motor symptoms. The picture is often complicated by the overlap of clinical symptoms. They are primarily the result of a reduction in brain catecholamines and dopamine in the ventral tegmentum. Most current treatment strategies are aimed at replacing these amines by reuptake inhibition. However, the TCAs and SSRIs both carry additional risks in terms of worsened cognition and movement disorder. A novel antidepressant, mirtazapine, may offer a better alternative.

Psychotic symptoms occur in about 30% of patients and may be early or late. They vary in intensity and duration. They are primarily drug-induced and result from overstimulation of the dopaminergic neurones in the mesolimbic and mesocortical systems of the brain. The main intervention is aimed at reduction of dopamine-inducing drugs, but occasionally antipsychotics are required. A delicate balancing act ensues between maintenance of function and reduction of psychologically distressing symptoms. A new generation antipsychotic, aripiprazole, a D2 stabiliser, may offer a useful alternative to the current range of atypicals.

The prevalence of dementia in PD is about 40%. The risk of developing dementia in patients with PD is five times that of non PD controls. The symptoms can be sub cortical or cortical and may be further complicated by the increased risk of psychosis in this group. New research suggests that these patients benefit both cognitively and behaviourally from the implementation of a cholinesterase inhibitor.

Parkinson's disease is therefore a complex condition with a high level of psychiatric co-morbidity. The presentation will examine how best to identify these and introduce some new treatment strategies aimed at reducing psychological distress and maintaining physical wellbeing.

Key words Aripiprazole, colinesterase inhibitor, depression and anxiety, mirtazapine, reuptake inhibition, SSRIs, TCAs.

Sponsors None.

Declaration No conflict of interest declared.

SESSION 2
BALANCE AND VISION

Chairman: Dr S Pound, Consultant Geriatrician, Queen Margaret Hospital, Dunfermline, Scotland

New hope for old eyes?

Professor B Dhillon, Consultant Ophthalmologist, Princess Alexandra Eye Pavilion, Edinburgh, Scotland

Email bal.dhillon@luht.scot.nhs.uk

Abstract Age-related macular degeneration is the leading cause of partial sight and blind registration in the UK and

CME

many other developed countries. It has a negative impact on quality of life and independent functioning. Sight loss caused by AMD increases exponentially from the age of 70–85 years.[1] Early detection of 'wet AMD' and understanding the impact of AMD on visual functioning are key to effective management and rehabilitation. Quality of life studies in Edinburgh demonstrate the impact of AMD, the benefits of photodynamic therapy in a well-defined subgroup of patients and the effect of surgical intervention for cataract co-morbidity.[2,3] New initiatives in AMD treatment and rehabilitation offer the prospect of new hope for old eyes.

References

1 Owen CG, Fletcher AE, Donoghue M *et al.* How big is the burden of visual loss caused by age related macular degeneration in the United Kingdom? *Br J Ophthalmol* 2003; **87:**312–17.
2 Armbrecht AM, Aspinall PA, Dhillon B. A prospective study of visual function and quality of life following PDT in patients with wet age-related macular degeneration. *Br J Ophthalmol* 2004; **88:**1270–3.
3 Armbrecht AM, Findlay C, Aspinall PA. Cataract surgery patients with age-related macular degeneration. *J Cataract Refract Surg* 2003; **29:**686–93.

Key words AMD, photodynamic therapy.

Sponsors None.

Declaration No conflict of interest declared.

CME

MARJORIE ROBERTSON LECTURE

Chairman: Professor N Douglas, President, Royal College of Physicians of Edinburgh, Edinburgh, Scotland

Intermediate or indeterminate care: Evidence based community rehabilitation

Professor G Mulley, Professor of Elderly Medicine, St James's University Hospital, Leeds, England

Email Graham.Mulley@leedsth.nhs.uk

Abstract In this review I will define rehabilitation and intermediate care, trace the origins and evolution of intermediate care and describe the polarity of views on this major whole-system change in the medical and social care of older people in England. I will describe the main drivers to change, with particular emphasis on the National Beds Enquiry.

The main part of the presentation is an analysis of the systematic reviews and other critiques of this scheme. The evidence for and against ways of reducing admissions to hospitals and care homes and organising early discharge will be assessed. Though described as an 'evidence-free zone' intermediate care does have an evidence base, though most of the studies are methodologically unsound and many aspects have not been evaluated. However, the results of RCTs may not be music to the ears of politicians, policy makers or managers.

The evidence will be summarised and a way forward for geriatricians and their teams will be proposed.

References

1 Alternatives to hospital care. *Age Ageing* 2001; **30(Suppl 3)**.
2 British Geriatrics Society. Guidance for Commissioners and Providers of Health and Social Care (BGS Compendium Document D4). In: *Compendium of guidelines, policy statements and statements of good practice*. Revised 2001. www.bgs.org.uk/compendium/comd4.html
3 *The national beds enquiry.* London: Deprtament of Health; 2000. http://www.dh.gov.uk/PolicyAndGuidance/OrganisationPolicy/SecondaryCare/NationalBedsEnquiry/fs/en
4 Parker G. *What works and what doesn't in intermediate care.* Presented at the BMA conference intermediate care – options and opportunities, 18 March 2002. http://www.prw.le.ac.uk/nccsu/BMA101.pdf
5 www.NICE.org.uk
6 Cochrane Database Systematic Reviews www.clinicalevidence.com

Key words Care homes, intermediate care, National Beds Enquiry, rehabilitation and systematic reviews.

Sponsors None.

Declaration No conflict of interest declared.

SESSION 3

Chairman: Dr A Elder, Consultant Geriatrician, Western General Hospital, Edinburgh, Scotland

Debate: This house believes that use of statin treatment in the over 80s is inappropriate (For the motion)

Professor C Gray, Professor of Clinical Geriatrics and Associate Clinical Sub Dean, University of Newcastle upon Tyne, England

Abstract Not available at the time of going to press.

Debate: This house believes that use of statin treatment in the over 80s is inappropriate (Against the motion)

Dr Chris Isles, Consultant Physician, Dumfries & Galloway Royal Infirmary, Dumfries, Scotland

Abstract Not available at the time of going to press.

DAY 2

SESSION 1
THE PAIN PROBLEM

Chairman: Dr N Colledge, Consultant Geriatrician, Liberton Hospital and Royal Infirmary, Edinburgh, Scotland

Understanding pain

Dr W Macrae, Consultant Anaesthetist and Pain Specialist, Ninewells Hospital, Dundee, Scotland

Email w.a.macrae@dundee.ac.uk

Abstract Pain is defined by the International Association for the Study of Pain as 'an unpleasant sensory and emotional experience, associated with actual tissue damage or described in terms of such damage'.

The importance of this definition is that it recognises the emotional component of pain and acknowledges that for pain to be experienced by a person it is not necessary to have appreciable tissue damage.

Physiology Our modern concept of how the body appreciates pain goes back to Descartes. He suggested that physical injury produced a 'perturbation' that was transmitted through the nerves to the spinal cord and then up to the brain. He likened it to a bell tower, you pull the rope at the bottom and the bell rings at the top. This model of the nervous system persists today in the mind of many people, even some doctors.

The publication of the Gate Control Theory in 1965 by Melzack and Wall showed that the spinal cord is not a passive junction box for passing on messages to the brain. Rather it acts as a modulator, letting information through in some circumstances, blocking it in others. In 1976, Melzack and Wall showed that there is descending control from the brain stem and higher centres which also modulates the spinal gating mechanism.

Since then there has been an explosion of interest in the physiology of pain with important developments at the peripheral receptor, the neuron itself, the spinal cord and brain. What has emerged from all this work is that not only is the nervous system more complex than we imagine, it is probably more complex than we *can* imagine. Each primary afferent neuron communicates with over a thousand other neurones, in some cases over a hundred thousand other neurones. The nervous system is not hard wired, but is constantly changing. It is these changes, neural plasticity, that are most significant for us as pain doctors.

The idea that the adult nervous system can change has only recently been appreciated. The mechanism is complex and probably involves several pathways and many receptors. The details of this are still being elucidated, but what is important from our point of view is that it shows how patients may have symptoms that the old theories could not explain. These include allodynia (pain from a stimulus that is not normally painful), hyperalgesia (more pain than you would expect from a painful stimulus) and dysaesthesia (unpleasant sensory symptoms either spontaneous or evoked).

In the past few years advances in imaging have allowed researchers to look at how the brain is working in real time. Work with amputees has shown that the primary sensory cortex (and the motor cortex) remap after loss of a body part. More recent research has shown that remapping is more widespread than previously thought and appears to occur even after acute pain. In patients with back pain cortical reorganisation has been found. There was a shift and potentially an extension of the representation of the back in the primary somatosensory cortex into adjacent cortical territory. The amount of reorganisation was highly positively correlated with the chronicity of the pain problem.

We should not therefore doubt patients when they tell us about their pain, even when their descriptions appear to contradict our understanding of neurophysiology.

Clinical implications There are many types of pain. This has significance for management. The main division is:

- Pain from damaged tissue, relayed through a normal nervous system – nociceptive pain.
- Pain where the damage is in the nervous system itself and the pain is arising in the damaged nerves, not the tissue where the pain is felt – neurogenic pain.

There may be other distinct types of pain in addition, such as inflammatory pain.

The importance of this is that these different pains are mediated through different pathways in the nervous system and they use different chemical transmitters. Drugs that help one type of pain may not therefore help another. The classic example of this is morphine, which is excellent for nociceptive pain but less useful in neurogenic pain. Fortunately, there are drugs that help neurogenic pain, the tricyclic anti-depressants such as amitriptyline and the anti-convulsants such as gabapentin, sodium valproate and carbamazepine.

This brings us to the topic of the psychology of pain. If the nervous system can change in response to injury, how about learning? Animal work has shown that classical and operant conditioning can produce changes in the way the nervous system works. Work on humans has shown that this also applies to us! Patients who have illnesses are subjected to all sorts of subtle pressures that will influence their behaviour. We can grasp that psychological mechanisms are at work, but recent studies suggest that the changes may not just be behavioural, and they may involve changes to the central nervous system. Whether this means new connections being made, or merely the strengthening or weakening of existing connections, we do not know. This opens the way for new approaches to the management of pain, and perhaps many other symptoms. Some of this work goes against our intuition and may be hard to come to terms with. For example, Herta Flor has

CME

shown that patients with solicitous spouses do worse than those with spouses who appear less caring!

Conclusions If we are to improve our treatment of patients with pain we must first stop making value judgements about patients and start believing them. We must do our best to understand the underlying physiology and pathology and put into practice the recent advances in treatment.

References

1 Knost B, Flor H, Birbaumer N. Pain behaviors, spouse responses and somatosensory evoked potentials of chronic pain patients during acute pain tests. *Zeitschrift fur Klinische Psychologie-Forschung und Praxis* 1999; **28:**242–7.

2 Flor H, Breitenstein C, Birbaumer N, Furst M. A psycho-physiological analysis of spouse solicitousness towards pain behaviors, spouse interaction, and pain perception. *Behavior Therapy* 1995; **26:**255–72.

Key words Allodynia, amitriptyline, amputees, back pain, carbamazepine, chronicity of the pain, dysaesthesia, gabapentin, Gate Control Theory, hyperalgesia, morphine, neural plasticity, neurogenic pain, nociceptive pain, psychology of pain, remap, sodium valproate, TCAs.

Sponsors None.

Declaration No conflict of interest declared.

CME

Assessment of pain in dementia

Dr D Forsyth, Consultant Geriatrician, Addenbrooke's Hospital, Cambridge, England

Abstract Not available at the time of going to press.

Control of cancer pain

Dr B Dymock, Associate Specialist in Palliative Medicine, Roxburghe House, Royal Victoria Hospital, Dundee, Scotland

Email barbara.dymock@tpct.scot.nhs.uk

Abstract

Background One in three people will be diagnosed with cancer during their lifetime. Cancer is a disease that affects mainly older people, with 64% of cases occurring in those aged 65 and over. The typical patient with cancer is approximately 60 years old, with multiple medical problems, taking several medications simultaneously, and his or her carer is often older than 60. Many older cancer patients have concerns and convictions that can prevent them from receiving and accepting the pain relief they need. Some healthcare professionals harbour misconceptions about treating pain in older people, resulting in inadequate care and suffering.

Methods: Expert consensus opinion supports a three-step approach to treating cancer pain by oral medication (the WHO Ladder): non-opioid agents, followed by low-potency opioids and, if necessary, high-potency opioids. Adjuvant drugs should be used in combination with opioids, which may result in synergistic effects producing better pain relief at a lower dose of opioids, hence the patient may experience fewer opioid side-effects. Morphine is the 'gold standard' strong opioid in cancer pain. Experience from Specialist Palliative Care indicates that in approximately 20% of patients with pain this three-step WHO approach may be ineffective. In these resistant cases four further steps are proposed: Step four: Alternative opioids and/or alternative routes of administration; Step five: NMDA antagonists such as Ketamine; Step six: Interventional procedures such as neural blockade or neuraxial drug administration. The effect of global suffering on pain management, and non-pharmacological methods of pain control also need to be considered in any cancer management plan.

Conclusions Follow the WHO Analgesic Stepladder:

- Begin with low doses and titrate slowly.
- Remember adjuvants, especially in neuropathic pain.
- Be aware of additive effects.
- Remember cancer pain is multifactorial and needs multimodal management.
- Seek specialist palliative care advice if pain remains uncontrolled.

References

1 Portenoy RK. Pain management in the older cancer patient. *Oncology* 1992; **6(2):**86–98.

2 Ferrell BA. Pain evaluation and management in the nursing home. *Ann Intern Med* 1995; **123(9):**681–7.

3 Ferrell BR, Ferrell BA (eds). *Pain in the elderly.* Seattle: IASP Press; 1996.

4 Ferrell BR, Rhiner M, Ferrell BA. Development and implementation of a pain education program. *Cancer* 1993; **72:**3426–32.

5 *Control of Pain in Patients with Cancer.* SIGN publication No 44. Edinburgh: SIGN; 2000.

6 Bernabei R, Gambassi G, Lapane K *et al.* for the SAGE Study Group. Management of pain in elderly patients with cancer. *JAMA* 1998; **279:**1877–82.

7 Cleeland CS, Gonin R, Hatfield AK *et al.* Pain and its treatment in outpatients with metastatic cancer. *N Engl J Med* 1994; **330(9):**592–6.

8 Paice JA, Penn RD, Shott S. Intraspinal morphine for chronic pain: a retrospective multicenter study. *J Pain & Symptom Manage* 1996; **11(12):**71–80.

9 Miguel R. Interventional treatment of cancer pain: the 4th step of the WHO analgesic ladder? *Cancer Control* 2000; **7(2):**149–56.

Key words Cancer, older patients, pain and WHO analgesic Stepladder.

Sponsors None.

Declaration No conflict of interest declared.

SESSION 2
CHRONIC PAIN IN THE ELDERLY

Chairman: Dr N Colledge, Consultant Geriatrician, Liberton Hospital and Royal Infirmary, Edinburgh, Scotland

Acupuncture and pain

Dr J Spiller, Consultant in Palliative Medicine, St. John's Hospital, Livingston and Marie Curie Hospice, Edinburgh, Scotland

Email juliet.spiller@mariecurie.org.uk

Abstract

Background Pain is common in the elderly and is associated with functional limitations, fatigue, sleeping problems, depression and reduced quality of life. Conventional analgesics are often poorly tolerated in this age group and may cause life-threatening interactions and side-effects. Patients are often keen to explore non-drug measures such as acupuncture.

Methods Human and animal studies indicate that acupuncture afferent pathways engage the descending inhibitory system. Release of various neurochemicals including endogenous opioids may also contribute to its analgesic effect. Only two studies have specifically looked at acupuncture use in the older population and both examined its use in chronic back pain.

Results In 2003, Meng *et al.* showed significant reductions in disability score, analgesic use and drug side-effects which were sustained for four weeks beyond the five-week treatment period.[1] In 1999, Grant *et al.* showed significant reductions in pain scores and analgesic use for both acupuncture and Transcutaneous Electrical Nerve Stimulation with a slight further improvement for acupuncture at four weeks' follow-up.[2]

Conclusions Acupuncture can be a useful adjuvant analgesic in the elderly and can help to minimise drug use and side-effects.

References

1 Meng CF, Wang D, Ngeow J *et al.* Acupuncture for chronic low back pain in older patients: a randomized, controlled trial. *Rheumatology* 2003; **42(12):**1508–17.

2 Grant DJ, Bishop-Miller J, Winchester DM *et al.* A randomized comparative trial of acupuncture versus transcutaneous electrical nerve stimulation for chronic back pain in the elderly. *Pain* 1999; **82:**9–13.

Key words Acupuncture, older patients, pain.

Sponsors None.

Declaration No conflict of interest declared.

SYDNEY WATSON SMITH LECTURE

Chairman: Professor N Douglas, President, Royal College of Physicians of Edinburgh, Edinburgh, Scotland

Management of non malignant pain

Professor BA Ferrell, Associate Professor, David Geffen School of Medicine at UCLA, Los Angeles, USA

Email bferrell@mednet.ucla.edu

Abstract Pain is a common problem in older people. For treatment purposes, it may be helpful to classify pain based on the physiological mechanism for pain perception. The two most common pathophysiological mechanisms of pain perception are nociceptive and neuropathic. Nociceptive pain is caused by stimulation of pain receptors in the peripheral nervous system. Common sources include arthritis, inflammation, pressure, tissue deformity, strain and trauma. Neuropathic pain is caused by a pathophysiological process in the nervous system that is perpetuated long after any disease insult or injury. Examples of neuropathic pain include peripheral neuropathies, complex regional pain and thalamic pain syndromes. Although most pain syndromes do respond to traditional analgesic drugs, including acetaminophen and NSAIDs for mild to moderate pain, and opioids for severe pain, neuropathic pain may respond uniquely to some non-traditional drugs such as anticonvulsants, antidepressants or antiarhythmic drugs. It is important to remember that nociceptive pain does not respond to these non-traditional analgesic medications.

Choosing appropriate analgesic drugs for older people requires knowledge of physiological and pharmacological changes that occur with ageing. These age-related changes have important implications for distribution, metabolism and excretion of drugs that often have longer half-lives and narrower therapeutic ratios in older people. Older people have an increased fat-to-lean body ratio that often results in a larger lipid storage compartment and a longer half-life for many lipid soluble drugs. It is well known that older people lose about 1% of glomerular filtration each year after age 30. Thus an 80-year-old person may have lost 50% of their renal function that is not reflected in the serum creatinine because of a concomitant loss of muscle mass. Oxidation of drugs via the cytochrome system in the liver is variable in older people, but conjugation and first pass kinetics are usually well preserved. Common disease states also affect medications in the elderly. Hypoalbuminaemia may affect those drugs that are highly protein bound. Dehydration, atrophic gastritis, *Helicobacter pilori*, constipation and a variety of other common diseases can complicate analgesic drug therapy. In these circumstances it is important to know the advantages and limitations of commonly used analgesic drugs.

Although it is beyond the scope of this presentation to review all known analgesic drugs, it is important to review a few of them. Acetaminophen is probably the safest drug for mild to moderate pain. Non steroidal anti-inflammatory drugs should be avoided in high doses for long periods of

CME

time because of multiple potential problems that are increased in older people including gastric toxicity, nephrotoxicity and several drug–drug and drug–disease interactions. Opioids are probably under-utilised in many older people and may have fewer long-term problems for some older people compared to NSAIDs. Newer anticonvulsant drugs such as NSAIDs and others may be the first drugs to try for neuropathic pain because of their low side-effects. Traditional tricyclic anti-depressants are not first-line choices today because of their higher incidence of anticholinergic effects. It is important to remember that most non-traditional analgesic drugs for neuropathic pain are only partially effective and they are rarely effective as a single line therapy. There are a few drugs that should be avoided in older people. There is no justification for the use of placebos. Meperidine has an active metabolite that can cause seizures. Mixed opioid agonists-antagonists often have narrow therapeutic windows and may be associated with more delirium. Caution should be taken with those drugs that have extreme half-lives. New medications and new evidence are appearing often and it is important to keep abreast of the latest new findings.

References

1 AGS Panel on Persistent Pain in Older Persons. The management of persistent pain in older persons. *J Am Geriatr Soc* 2002; **50:**S205–S224. http://www.americangeriatrics.org/products/positionpapers/JGS5071.pdf

Key words Acetaminophen, neuropathic, nociceptive, NSAIDs, pain perception.

Sponsors None.

Declaration No conflict of interest declared.

CME

SESSION 3
RISK MANAGEMENT

Chairman: Dr D Farquhar, Consultant Physician, St. John's Hospital, Livingston, Scotland

But will it do me any good, Doctor?

Dr S Maxwell, Consultant Physician, University of Edinburgh, Edinburgh, Scotland

Abstract Not available at the time of going to press.

Preventing falls in institutions

Dr D Oliver, Senior Lecturer, Elderly Care Medicine, University of Reading, Reading, England

Email d.oliver@reading.ac.uk

Abstract

Background Falls in hospitals and care homes are common and problematic for individuals, institutions, governance and risk management. They bring about physical, psychological and functional morbidity for patients, anxiety, guilt, complaint and litigation for staff and relatives, and excess cost and bed occupancy for institutions. Hence the imperative that 'something must be done' to prevent them, yet interventions should be based on the available evidence. And, in turn, this is just one element in a minefield of practice which also involves ethics, law, cultural, political and professional values and priorities, and problems translating research into practice. A review of the evidence, gaps and limitations is a good starting point, however.

Methods The speaker's previous work in this area will be described, including a Department of Health-funded systematic review in progress and previous reviews and original papers.

Results: Seventy-one studies (of various designs) have been reported in which prevention of falls or injuries is an endpoint. This includes six RCTs of fall/injury-prevention in hospitals, 14 RCTs of hip-protectors in care homes and eight RCTs of fall/injury-prevention in care homes, together with two RCTs of calcium and vitamin D in care homes. There is also a number of 'before and after' studies in hospital and large observational cohort studies, which yield useful information. There are a further 52 studies of risk factors and fall risk assessment tools in institutions that form part of a separate systematic review. A range of interventions is described, including:

- structured risk/risk-factor assessment with tailored medical or nursing interventions to follow;
- staff and patient education;
- use or removal of bedrails or other physical restraints;
- environmental safety/built environment;
- physiotherapy;
- gait and balance training;
- alarms and assistive technology; and
- bone protection and bone strengthening.

Many trials use several of these in the intervention group, so that determining attributable benefit from each element is difficult.

Subsidiary evidence is also useful in designing effective interventions (e.g. on phenomenology of falls, reduction of inappropriate prescribing of 'culprit' drugs and safe or smart building design).

Interpretation of the evidence is further confounded by the great heterogeneity of settings, populations and outcome measures – all of which suggest caution in interpreting apparently positive findings and in extrapolating evidence to one's clinical setting.

Performing trials in this area is also problematic. Few cluster-randomised trials have allowed adequately for clustering. 'Before and after' studies do not adequately allow for confounders. Individual randomisation often leads

to exclusion of the very groups at highest risk (i.e. those with dementia, delirium, frailty or acute co-morbidity) leading to low external validity of findings for translation to typical populations. No trials of fall prevention have been sufficiently powered to detect reductions in fracture rate. Hip protector studies are confounded by poor adherence and by lack of effect in trials using individual as opposed to cluster randomisation. Finally, few trials have looked at the negative effects (e.g. on rehabilitation or autonomy) of falls prevention or at economic evaluation.

Conclusions

a There is good evidence on the patterns (where, when, how and why?) of people falling in institutions.
b There is reasonable and growing evidence for multifaceted interventions to prevent falls in care homes and some emerging evidence for this in hospitals. Meta-analysis may help us to pick out the most effective components.
c There is no consistently validated falls risk assessment tool which is operationally useful. Is it better to use structured assessment for reversible risk factors – to be reapplied following each fall.
d There is a weight of evidence that physical restraint or cotside use does not prevent injurious falls and may lead to injury or death, and that 'chemical restraint' in the form of sedatives or hypnotics increases fall rates. There are also ethical concerns about infringement of autonomy.
e There is some evidence for routine supplementation of care home residents with high dose calcium and vitamin D preventing falls and fractures.
f There is no clear evidence for the untargeted use of hip protectors. Though they might be beneficial in high-risk clients, these are the very group least likely to adhere to their use.
g There is no clear evidence for the use of exercise as a single intervention in long-term care – though it meets with good adherence and has many other benefits and is a component of a number of successful interventions.
h There is no clear evidence for the use of validated risk assessment tools in correctly classifying fallers and non-fallers, though a handful of common reversible risk factors may be targeted.
i There is no clear evidence on the effectiveness or acceptability of alarms or other assistive technology approaches to falls prevention.
j There is no clear evidence for medication review and adjustment as a single intervention.

The above information should be taken into account in designing intervention strategies in clinical practice.

Adequately powered, well-designed clinical trials are required to address some of the gaps in the evidence base outlined. However, pursuing falls prevention should not be driven by a risk averse, excessively custodial approach and should never be at the expense of promoting patients' rehabilitation or autonomy. Proactive public relations are required by institutions in this regard. It may be that some falls are preventable, but many are an inevitable consequence of morbidity or of promoting functional independence.

References

1 Oliver D. Preventing falls and injuries in care homes. *Age Ageing* 2004; **33:**532–5.
2 Oliver D. Prevention of falls in hospital. Agendas for research and practice. *Age Ageing* 2004; **33:**328–30.
3 National Institute for Clinical Excellence. *Clinical Practice Guidelines for the Assessment and Prevention of Falls in Older People*. 2nd draft. London: NICE; 2004. Available at www.nice.org.uk
4 Gillespie LD, Gillespie WJ, Robertson MC *et al. Interventions for preventing falls in older people*. Cochrane Database Syst Rev. 2003.

Key words Accidental injuries, care homes, ethics, evidence, falls, fractures, heterogeneity, hospitals, interventions, law, limitations, outcomes.

Sponsors None, but ongoing systematic review funded by Department of Health Accidental Injury Prevention Programme. Dr Oliver also chaired the NHS Information Authority Falls Dataset Group.

Declaration No conflict of interest declared.

SESSION 4
OSTEOPOROSIS

Chairman: Professor M McMurdo, Professor of Ageing and Health, University of Dundee, Dundee, Scotland

Recent studies of calcium and vitamin D in older people – results and implications

Dr R Francis, Reader in Medicine (Geriatrics), University of Newcastle upon Tyne and Honorary Consultant Physician, Freeman Hospital, Newcastle upon Tyne, England

Abstract Not available at the time of going to press.

Case studies in osteoporosis

Dr D Farquhar, Consultant Physician, St. John's Hospital, Livingston, Scotland

Abstract Not available at the time of going to press.

Dr S Ralston, Director of the Institute of Medical Sciences and Professor of Medicine and Bone Metabolism, University of Aberdeen, Aberdeen, Scotland

Abstract Not available at the time of going to press.

Dr R Francis, Reader in Medicine, University of Newcastle upon Tyne, Newcastle, England

Abstract Not available at the time of going to press.

CME