

Selected abstracts from: Geriatric medicine symposium 2008

HOW TO MANAGE THE 'DIZZY' OLDER PATIENT

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Balance symptoms occur commonly in all age groups, but are particularly common in the elderly. They occur more typically in females than males.¹

The most common reason for impaired balance in the elderly is global degeneration of the vestibular system, but benign paroxysmal positional vertigo (BPPV) is actually more common in the elderly.²

The history is the most important part of the assessment of balance patients, but it can be unreliable and inconsistent.³ Imaging, particularly magnetic resonance imaging (MRI) scans, is valuable in excluding serious central pathology.

Vestibular rehabilitation is helpful in patients with reduced balance function. Drug treatment helps disordered labyrinthine function. Very few balance patients, particularly those who are elderly, benefit from surgery.

References

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Keywords Assessment, balance symptoms, elderly patients, treatment, vestibular rehabilitation

Declaration of interests None declared.

ASSESSING AND TREATING OSTEOARTHRITIS IN THE ELDERLY

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Osteoarthritis (OA) is ubiquitous in the elderly population and causes very significant morbidity, both medical and social, often causing chronic pain and contributing to reduced mobility and social isolation.

In this presentation I briefly review the epidemiology and pathological basis of OA, and explore some of the more unusual patterns of presentation as well as the potential diagnostic 'traps' into which physicians can fall.

The use of novel imaging methods is also addressed, and more complex related conditions such as Milwaukee shoulder are discussed.

The second part of the talk explores the utility of surgical versus conservative approaches to the management of OA in the elderly, and discusses the National Institute for Health and Clinical Excellence (NICE) guidelines in this domain.

Finally, we review the latest data on analgesics and COX-2 inhibitors, with particular reference to their safety, particularly the evidence (for example, the 'MEDAL' studies) in relation to cardiovascular risks associated with their use.

Further reading

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Keywords Cyclo-oxygenase inhibitor, imaging, osteoarthritis

Declaration of interests None declared.

NEW THERAPIES FOR AGE-RELATED MACULAR DEGENERATION

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Neovascularisation (Nv) in the macular retina is a common feature of the condition termed age-related macular degeneration (AMD). The onset of NvAMD is accompanied by severe central visual loss, which can become permanent from structural damage and fibrosis within the retina and supporting tissues.

The past two years have seen the introduction of therapies using monoclonal antibodies that inhibit mediators of neovascularisation. Inhibitors of the potent growth stimulant vascular endothelial growth factor (VEGF) have been found to be highly effective therapeutic agents that have revolutionised visual outcomes.

Major phase three clinical trials have revealed that the inexorable decline of central vision in eyes with NvAMD can be halted in more than 90% of cases treated. Even

more importantly, some 40% of eyes treated experience significant visual gains. This was accomplished in the pivotal trials through monthly intravitreal injections of ranibizumab (a monoclonal antibody that inhibits all isoforms of VEGF). The treatment was given monthly for up to two years. Such a treatment regime imposes huge burdens on the NHS and for the patient, who has to undertake monthly visits to the local provider unit. Ranibizumab has an excellent safety profile. However, there are indications that the treatment will result in a small excess of adverse systemic vascular events, such as stroke and myocardial infarction. Nonetheless, patients appear willing to accept the small excess in risk of a systemic vascular event in the future in order to maintain or improve their current visual status.

For a population of one million with the ageing profile of the UK, the annual incidence of NvAMD is around 300–350 new cases. If all of these patients are commenced on a course of ranibizumab with monthly assessment and treatment for two years, the total number of episodes (assuming that assessment and treatment are undertaken at the same visit) for the local eye unit will be in excess of 7,000 once a steady state is reached. Thus investment for additional clinic capacity and retinal expertise along with appropriate retinal imaging technology is urgently needed.

The major challenges at present are the optimisation of the treatment regime, particularly with a view to maximising the retreatment intervals; case selection for safe cessation of therapy; case selection for safe cessation of continuous monitoring; and the setting up of a VEGF treatment registry to monitor adverse events on a national scale.

The National Institute for Health and Clinical Excellence has undertaken a comprehensive assessment of the clinical and cost-effectiveness of ranibizumab in treating NvAMD. The final appraisal determination document is yet to be published, but the draft guidance contains the following salient points:

1. Ranibizumab is clinically effective in NvAMD.
2. It is cost-effective, particularly in second eyes.
3. Its cost-effectiveness when used to treat the first eye is still being debated.
4. Treatment is indicated for NvAMD in eyes without permanent structural damage to the fovea.
5. Treatment should be stopped if there is no prospect of visual recovery.

The introduction of anti-VEGF monoclonal antibodies and other molecules has revolutionised the management of NvAMD. These developments have been rapid and threaten to overwhelm current service provision.

Further reading

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Keywords Neovascular age-related macular degeneration, ranibizumab, VEGF

Declaration of interests Professor Chakravarthy has been a consultant to and received travel support from the following pharmaceutical companies who manufacture products involved in treating NvAMD: Novartis, Pfizer, JERINI, Allergan.

IMPROVING THE MANAGEMENT OF FRAIL ELDERLY PEOPLE

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In most Western countries, most people in hospital are elderly, so what makes a given patient ‘geriatric’? Lacking an age-defined rule, most geriatric medicine services aim to care for elderly people who are frail, i.e. at an increased risk of adverse health outcomes.¹

Frailty is comprehensible and measureable. Against other methods, my colleagues and I count the number of things that individuals have wrong with them.² To standardise the count, we divide the number of deficits present in an individual by the number of items that were counted; this gives a frailty index. In routine care, a frailty index can be derived from counting the deficits revealed by comprehensive geriatric assessment.³

The more things someone has wrong with them, the more likely they are to be frail. As their frailty index approaches 0.67, they have characteristics of a complex system at the edge of failure.⁴

The frailty index is one ‘clinical state variable’ – a single number that summarises an individual’s clinical state. Other state variables reflect high-order functions – divided attention, opposable thumbs, upright bipedal ambulation and social interaction. Their failures are the

'geriatric giants' of delirium, functional decline, falls and social withdrawal.

Geriatricians are specialists in managing complexity. We reject the 'one-thing-wrong-at-once' approach to embrace the complexity of our patients. Complex systems analysis can help us understand how to improve care.

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Keywords Complex systems analysis, frailty, frailty index

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HOW TO ASSESS AND MANAGE DELIRIUM

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Delirium is a severe neuropsychiatric syndrome characterised by acute deterioration in cognition and other mental functions. It is frequently precipitated by acute illness or trauma and mainly occurs in older people with baseline comorbidities. Delirium affects 15% of general hospital inpatients and at least 25% of inpatients in geriatric units. It is independently associated with multiple adverse outcomes including increased length of stay, loss of independence and increased mortality.

The diagnosis depends on (a) assessment of the patient's mental state and (b) informant history. Mental status is assessed by a combination of interview and formal cognitive testing. Informant history may reveal the onset of acute decline as well as pre-existing cognitive impairment.

Acutely, delirium may indicate life-threatening illness such as pneumonia, and rapid screening for such conditions is essential. Further assessment involves documentation of relevant predisposing and precipitating factors. All factors that might adversely affect brain function should be addressed. Thus, as well as treating the presumed proximal cause, potentially toxic drugs should be discontinued and, where possible, predisposing factors ameliorated. Additionally, drug treatment for agitation and/or psychotic symptoms may be required. Delirium that does not resolve in a few days may require second-line investigations. Persistent delirium (longer than one month) occurs in around 25% of patients; here other diagnoses such as depression or dementia should be considered.

Delirium may indicate dementia and is also a risk factor for future dementia. Patients with delirium should be screened for dementia, and those not meeting criteria for current dementia followed up with cognitive testing, although this is not yet standard practice.

Further reading

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Keywords Acute confusional state, delirium, dementia

Declaration of interests None declared.

UPDATE ON ASSESSMENT AND MANAGEMENT OF COGNITIVE IMPAIRMENT

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As public awareness of the importance of memory impairment grows, people are presenting earlier to the medical profession. Memory clinics, in whatever form, have become more established over the past decade. This has also contributed to an earlier presentation of people with memory complaints, which can create difficulty with diagnosis. This problem causes concern for people who have memory loss associated with ageing and those who have more significant memory loss which, although mild, is in fact likely to be early dementia. This is recognised in clinical guidelines that advocate early referral.

The typical approach to investigation and diagnosis relies upon a symptomatic approach and then an assessment of what type of dementia may be present. The diagnosis itself is often made when there is a definite decline in function from a previous level. As there is no universally recognised test for any of the dementia syndromes, diagnosis is based mainly on clinical grounds, with a comprehensive initial interview with patient and carer, cognitive, behavioural and functional assessment and blood testing. Access to neuroimaging is variable and while MRI is the recommended modality, unless analysed in a major centre it will not be diagnostic. The future assessment and diagnosis is likely to involve clinical assessment together with enhanced neuroimaging and biomarker assays. For example, cerebrospinal fluid analysis is routine in many European centres.

It should be possible to indicate, with reasonable confidence, the type of dementia that may be involved. Generally, Alzheimer's disease (AD) is the most common, followed by AD with cerebrovascular disease, vascular dementia and dementia associated with Parkinson's disease or diffuse Lewy body disease. In older people there is increasing recognition that a mixture of pathology may be involved. It is clear that a significant percentage of older people who are 'cognitively normal' have

plaques and tangles and infarcts in the brain. The more abnormal the pathology in the brain the more likely it is that dementia will manifest. In particular, the prevalence of cerebrovascular disease in older people with dementia is noticeable. Despite this, most studies indicate that Alzheimer's pathology, better defined as plaques and tangles, is present in 80–90% of older people who present to memory clinics.

Despite the recommendations about early referral and diagnosis in the UK, it is estimated that 33–50% of cases are not diagnosed. Similarly, when it comes to treatment, the National Audit Report showed that in 2004 only 18% of those eligible for treatment were in fact treated.

Treatment guidelines suggest that treatment be based on the condition that is the predominant cause of the dementia, which seems in the vast majority of cases in later onset disease to be of a plaque and tangle aetiology (i.e. AD).

There is also a global consensus that early intervention with licensed drug treatments is best in order to at least maintain existing function. The options include the cholinesterase inhibitors donepezil, rivastigmine and galantamine, while memantine is licensed for moderate to severe dementia. Rivastigmine capsules are licensed for dementia associated with Parkinson's disease, while galantamine receives mention in Canadian and Scottish guidelines for AD with cerebrovascular disease. None of these medications is licensed for treatment of vascular dementia. In terms of cholinesterase inhibitors, all appear equivalent (Cochrane review) and it is acknowledged that they produce small improvements in cognitive function, activities of daily living and behaviour. Once-daily preparations are available for donepezil (one titration step), galantamine (two titration steps) and rivastigmine as a transdermal preparation (one titration step). Rivastigmine remains available as a twice-daily capsule (three titration steps).

These medications seem fairly safe. The main contraindications are significant ECG abnormality, significant obstructive lung disease and peptic ulcer. The principal adverse events seen in practice are nausea and vomiting, while major adverse events seen less frequently include syncope and leg cramps. Memantine produces small effects and seems to impact on the development of agitation. Memantine is more effective than placebo when added to donepezil and may be useful when cholinesterase inhibitors are contraindicated or not tolerated. Memantine is generally well tolerated.

The issue of treatment withdrawal is very difficult. It is not clear that guidance based on cognition alone is helpful in this situation. Once again, a global assessment is necessary. Treatment should not be withdrawn if the optimum therapeutic dose has not been achieved or where there are unrealistic expectations of treatment benefits. It is clear

that patients at more severe stages of disease may be unlikely to benefit from the continuation of therapy, and this should be discussed with carers (and patients if possible). It is not possible to predict who is deriving continued benefit. Audit suggests that 30–40% of patients whose medication is discontinued need to have that medication reintroduced because of significant decline either in cognition or behaviour. It remains unclear if patients who decline despite treatment or those in more severe stages of disease should have their treatment changed to another cholinesterase inhibitor or memantine or if memantine should be taken additionally or treatment withdrawn. This is the subject of an MRC-sponsored study.

Keywords Alzheimer's disease, cerebrovascular disease, cholinesterase inhibitors, dementia associated with Parkinson's disease, diffuse Lewy body disease, donepezil, memantine, memory impairment dementia syndromes, plaques and tangles, rivastigmine and galantamine, treatment withdrawal, vascular dementia

Declaration of interests Peter Passmore has been a member of speaker bureaux in international meetings and conferences for Eisai, Janssen-Cilag, Lundbeck, Novartis and Pfizer, and national meetings in the UK for Bayer, BMS, Astra Zeneca, Sanofi-Synthelabo, MSD, Eisai, Shire, Novartis and Pfizer. He has been an advisor internationally for Bayer, BMS, Eisai, Janssen-Cilag, Novartis, Pfizer and Sanofi-Synthelabo and in the UK for Bayer, BMS, Astra Zeneca, Sanofi-Synthelabo, MSD, Eisai, Shire, Lundbeck, Novartis and Pfizer.

HYPERTENSION IN OLDER PEOPLE – ARE WE OVERTREATING?

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The benefits from treating elevated levels of blood pressure (BP) in the elderly in terms of reducing strokes and cardiovascular events have been well established. Recent data from the HYVET trial have extended the upper age at which benefit can be achieved to above 80 years of age.¹

Although hypertension is defined as a sustained systolic BP of 140 mmHg or more, no intervention trials in elderly hypertensives have recruited such subjects. Patients recruited to such trials have had systolic BPs of 160 mmHg or more. It still remains to be established what are the real benefits of treating subjects with systolic BPs of 140–160 mmHg.

Treatment rates for elderly hypertensives, defined as those with BPs of 140 mmHg or above, varies by country, with rates as low as 18% to as high as 73%. The optimal target level for systolic BP has also not been established for elderly hypertensives. Reported control rates (using a definition of 140 mmHg or lower) also varies greatly from 1% to 30%.

There are concerns about reducing BP levels too much, particularly in the elderly. Although often considered as a risk factor for falls, in a systematic review this was not found to be the case.² The INVEST trial, which included patients with established coronary artery disease, showed an increase in mortality with achieved systolic blood pressures below 140 mmHg.³

At present, the evidence does not support the proposal that we are overtreating hypertension in the elderly. However, more research is required to determine the optimal target for such individuals, and in the meantime it would be sensible not to lower BP too much in those with established coronary artery disease.

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Keywords Control, elderly, hypertension

Declaration of interests Dr Beckett is employed by Imperial College London from a grant provided by Servier.

ANTICOAGULATION IN OLDER PEOPLE: FOCUS ON ATRIAL FIBRILLATION

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Atrial fibrillation (AF) is a major contributor to stroke and thromboembolism. Antithrombotic therapy is beneficial, and substantial trial data supports the use of anticoagulation therapy for moderate–high risk subjects. The most recent meta-analysis¹ concluded that when compared with control, adjusted-dose warfarin and antiplatelet agents reduced stroke by 64% (95% CI, 49–74%) and 22% (CI, 6–35%), respectively. Adjusted-dose warfarin was also more efficacious than antiplatelet therapy (relative risk reduction, 39% [CI, 22–52%]) (12 trials, 12,963 participants).

The most recent comparison of warfarin versus aspirin comes from the Birmingham Atrial Fibrillation Treatment of the Aged Study (BAFTA).² This was a UK-based randomised trial of warfarin (INR 2–3) versus aspirin 75 mg daily in elderly (age >75 years) patients with AF, which was conducted by general practitioners in the primary care setting. In this trial, 973 elderly patients (mean age 81) were followed up for an average of 2.7 years, and the results showed a primary event rate of 1.8% per annum in the warfarin arm, versus 3.8% per annum in the aspirin arm (relative risk, RR 0.48 [95% CI 0.28, 0.80, $p=0.003$], NNT for one year to prevent one primary event, 50). For the

endpoint of strokes per se, the annual risk was also significantly reduced with warfarin vs aspirin (1.6 vs 3.4%, RR 0.46 [0.26–0.79]; $p=0.003$), with the most benefit seen for severe or disabling stroke rather than fatal stroke. Importantly, the risk of major haemorrhage was similar in both treatment groups in BAFTA. Thus, warfarin was more effective than aspirin for stroke prevention among elderly patients with AF, with no significant difference in major bleeding between warfarin and aspirin.

The concern with anticoagulation in the elderly is bleeding. Hylek et al.³ reported that the cumulative incidence of major haemorrhage for elderly patients (≥ 80 years) was 13.1 per 100 person-years and 4.7 for those aged <80 years ($p=0.009$). Within the first year, 26% of elderly patients stopped taking warfarin; stoppage due to perceived safety issues accounted for 81% of this group. The risk of stroke substantially increases with age, but so does the bleeding risk.

In summary, the use of anticoagulation in the elderly requires attention to the risk–benefit ratio, balancing the risks of bleeding versus the reduction of mortality and morbidity of thromboembolism.

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Further reading

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Keywords Anticoagulation, antiplatelet therapy, aspirin, atrial fibrillation, bleeding, stroke prevention, warfarin

Declaration of interests None declared.

HEART FAILURE IN OLDER PEOPLE

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People are living longer, largely due to declining cardiovascular mortality, but with an attendant rise in cardiac morbidity – in particular congestive heart failure, which is well recognised as a disease of older patients.

Heart failure in older patients is complex. Comorbidity increases with age and is associated with a greater likelihood of hospitalisation, for which congestive heart failure is the most common cause above the age of 65. In addition, older patients admitted to hospital with

heart failure have higher in-hospital and one-year mortality. Unfortunately, medication uptake in heart failure diminishes with increasing age.

The impressive evidence base for treatment in 65-year-old men contrasts with an average age of 76 in community studies of heart failure, in which 50% are women. The treatment of older patients must rely upon post-hoc meta-analysis, which suggests benefit with ACE inhibitors and beta blockers. Only one formal randomised control trial, the SENIORS study (2005), has specifically looked at heart failure in this age group. The advantage of more recent technology, including cardiac resynchronisation therapy, is less certain, with doubts over the benefit of implantable defibrillators above the age of 75.

Beyond medical and device management there is considerable need for 'social' treatment. Isolation is common among older people, with depression prevalent and a lack of social input associated with poor prognosis. There is a small evidence base that exercise classes, such as t'ai chi programmes, may particularly benefit an older population. Disease management programmes may also be of particular benefit in the older patient.

Heart failure with preserved ejection fraction remains a central conundrum. Community studies find that ejection fraction is distributed in a unimodal fashion. Echocardiography, the gold standard diagnostic intervention, is well suited to the detection of cardiac dilatation and provides a numerical assessment of reduced ejection fraction. However, heart failure may also be present when ejection fraction appears 'normal' (or preserved), although the underlying pathology is disputed. Such heart failure with preserved ejection fraction may now be the most common form of inpatient heart failure. Survival appears equally poor whether ejection fraction is preserved or reduced, suggesting that the clinical diagnosis may drive prognosis. Research in this area is scant but increasing.

Heart failure can be prevented, with meta-analysis of blood pressure lowering trials finding significant reduction in heart failure. The recent HYVET study has provided important confirmation of these observations in a truly elderly population, and deserves thorough attention.

In conclusion, heart failure remains a clinical diagnosis reflecting an archetype of chronic disease that requires a co-ordinated approach to treatment, supervised by specialists and unhindered by healthcare boundaries. The prevalence and complexity of heart failure will increase, as will demand on healthcare services. Fully integrated, co-ordinated services are increasingly important, with adjunctive remote monitoring likely to have a major role.

Keywords Evidence, heart failure, older patients, preserved ejection fraction

Declaration of interest Dr McIntyre has received reimbursement for conference attendance, lectures and for advising members of the pharmaceutical industry on the treatment of heart failure.

WHY DO OLD BONES BREAK?

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Low trauma fractures are an important cause of excess mortality, substantial morbidity and health and social service expenditure for older people. The incidence of these fractures rises with advancing age, reflecting an increase in skeletal fragility and in the incidence of falls. The risk of fracture is determined not only by skeletal factors, such as bone mineral density (BMD), bone turnover, trabecular architecture, skeletal size and geometry, but also by non-skeletal factors associated with a propensity for falls.

The diagnosis of osteoporosis is usually made with BMD measurements, but there is growing interest in assessing the absolute risk of fractures, using BMD and clinical risk factors. The World Health Organization has developed a Fracture Risk Assessment Tool (FRAX) for determining the ten-year risk of hip and major osteoporotic fractures in men and women (www.shef.ac.uk/FRAX), which is likely to be used to identify people at high risk of fractures and so target treatment.

A number of treatments have been shown in large clinical trials to increase BMD and decrease the risk of fractures, including bisphosphonates, raloxifene, strontium ranelate, teriparatide, calcium and vitamin D.

Although skeletal fragility and low trauma fractures are common in older people, effective treatments are now available that decrease fracture risk. The challenge is to identify those at highest risk, in whom to target treatment.

Further reading

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Keywords Bisphosphonates, bone mineral density, calcium, fractures, Fracture Risk Assessment Tool (FRAX), osteoporosis, raloxifene, strontium ranelate, teriparatide, vitamin D

Declaration of interests Professor Francis has received research funding in the past year from Novartis. He has also served as an adviser to Procter & Gamble, Roche/GSK and Servier & Shire.