GM conference report

The Ageing Patient: today and tomorrow

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The next conference is on Thursday 10th October 2013, again with Professor Passmore as the chair. Further details can be found on page 23 and at www.gmjournal.co.uk

Management of stroke

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In the UK, stroke consumes 7% of the entire NHS budget. Aspirin is the mainstay of treatment in stroke and it works across the board of atherothrombosis with a risk reduction of 25%.[1]

Patients with atrial fibrillation have a five-fold increased risk of stroke and a two-fold increased risk of death. What can we do about atrial fibrillation? We can give patients antiplatelets yet most healthcare physicians would prescribe warfarin as that has a risk reduction of 70%.[2] Another study noted that net clinical benefit significantly favoured warfarin in patients aged ≥75 years, suggesting that age should not be perceived as a barrier to anticoagulant prescription.[3]

Although warfarin is undoubtedly effective for stroke prevention in patients with atrial fibrillation, the elderly, in particular, face the risk of major haemorrhage during therapy.

Warfarin though is significantly more effective than dual antiplatelet therapy. In the ACTIVE-W trial, patients were allocated randomly to receive warfarin therapy (target INR 2.0–3.0) or clopidogrel (75mg/day) plus aspirin (75–100mg/day) and were intended to be followed for approximately two years. The trial was stopped early because of clear evidence of the superiority of warfarin therapy. Major bleeding rates were similar in both treatment groups.[4]

There have been some novel anticoagulants that have been released into the market recently but currently only two have been approved. These are rivaroxaban, which is a direct factor Xa inhibitor and dabigatran, which is a direct factor IIa inhibitor.

The RE-LY trial was a randomised, phase III, single-blind, non-inferiority study of dabigatran versus warfarin. The primary efficacy was the composite of all-cause stroke or systemic embolism.[5] Stroke or systemic embolism occurred in 183 patients receiving 110mg of dabigatran (1.53% per year), 134 patients receiving 150mg of dabigatran (1.11% per year) and 202 patients receiving warfarin (1.69% per year). Both doses of dabigatran were non-inferior to warfarin ($p<0.001$). The 150mg dose of dabigatran was also superior to warfarin, but the 110mg dose was not.

Rates of haemorrhagic stroke were 0.38% per year in the warfarin group, compared with 0.12% per year in the group that received 110mg of dabigatran and 0.10% per year in the group that received 150mg of dabigatran.

Another study called ROCKET AF compared rivaroxaban with warfarin. This was a phase III, prospective, randomised, double-blind, double-dummy, active-controlled, multicentre, event-driven study.[6] It compared rivaroxaban with warfarin (titrated to INR 2–3) for the prevention of stroke and systemic embolism in patients with non-valvular atrial fibrillation plus either a history of stroke, TIA or systemic embolism, or at least two moderate risk factors for stroke (CHADS2 score ≥2).

The rivaroxaban dose was 20mg once daily—except for patients with moderate renal impairment (creatinine clearance 30–49 ml/min) at baseline who received a reduced dose (15mg once-daily).

In patients with atrial fibrillation, rivaroxaban was noninferior to warfarin for the prevention of stroke or systemic embolism. There was no significant between-group difference in the risk of major bleeding, although intracranial and fatal bleeding occurred less frequently in the rivaroxaban group.

Thrombolysis is another treatment option and aims to reverse the ischaemic penumbra.

1. Antithrombotic Trialists' Collaboration. *BMJ* 2002; **324**: 71–86
2. Hart RG, et al. *Ann Intern Med* 2007;**146**: 857–67
3. Singer DE, et al. *Ann Intern Med* 2009; **151**: 297–305
4. ACTIVE Investigators. *Lancet* 2006; **376**:1903–12
5. Connolly SJ et al. *N Engl J Med* 2009; **361**: 1139–51
6. Patel MR et al. *N Engl J Med* 2011; **365**: 883–91

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Dignity and care in care homes

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The issue of dignity in care homes is more difficult than it should be as undignified care is what is recognised whilst good care goes largely unnoticed. Indeed, it is fair to say that care homes are only in the news when things go wrong, and then the blame game starts. Then it is usually the care workers and managers that are held accountable, but too often there are other factors that are not as easily understood or held to account.

Many years ago I conducted a small pilot (and unpublished) study seeking to understand individuals' attitudes following admission into care homes. I found that residents tended to become less depressed over a two to three month period, but during this time they would become more anxious. When I interviewed a sample of residents further I found that they were less depressed because they were more comfortable than they had been in hospital or at home. However, they were more anxious because they didn't know what was going to happen next.

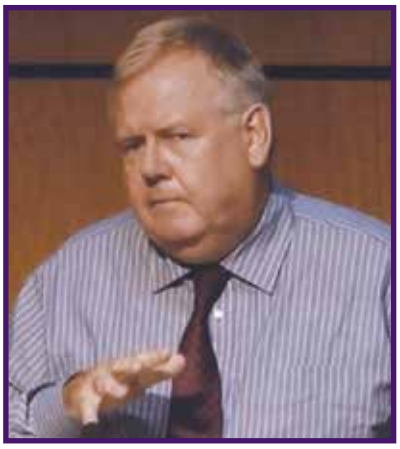
There are more than three care home beds for every NHS bed in the UK and the NHS has a dependence on the care sector that it doesn't really understand or manage. Essentially, if the care home sector "catches a cold", the NHS is likely to catch pneumonia. Yet, despite being a major sector in health and social care, care homes don't have a policy-centre lead.

Over 80% of the adult social care budget is committed to care homes and the understandable priority is to prevent admission to care homes yet for healthcare commissioners care beds are a hugely more affordable option than acute care.

Typically, 70–75% of care beds are reliant on funding by the local authority. A further tranche are self-funded largely as a result of means testing and around 15% are under contract to the NHS—either for intermediate or fully funded long-term care. For operators, the rate of admission and discharge or death through these NHS-funded beds is quite distinct from the traditions of long-term social care, and the risks both in care quality and costs are greater. Over the next 5–10 years, there will be a series of predictable and potentially avoidable crises in care, and dignity (or lack of) will be a common theme.

A good starting point is the matter of "purpose". While we accept that prevention of frailty is a prime medical goal, it is also important to understand life trajectories. For people with long-term neurological conditions, that form the core population of care homes, primary prevention is largely irrelevant as is "end of life care". These patients will have a typical length of stay of approximately 20 months. This is a new medical "twilight zone" where patients may be unresponsive to much evidenced-based care, but their quality of life and dignity will greatly depend on their medical as well as care management. The medical purpose needs to be clear and "the best quality of life possible in such a reframed life" seems appropriate.

Prescribing is one area that needs serious thought. Two extremes of prescribing in care homes are recognisable—an institutional and a personalised



approach. Institutional approaches may be hallmarked by high rates of antipsychotic prescribing and supplemental feeds accompanied by low levels of analgesic and antidepressant use. Personalised approaches may be recognised by low levels of antipsychotic use, higher levels of analgesics (as required), higher rates of antidepressant prescribing and much lower levels of supplemental feeds.

Supplemental feeds are a big issue in care homes and a huge amount of the NHS budget is being squandered on them. Often, a tick box approach to malnutrition risk is the cause. In reality, more care time to support feeding may be the solution.

Structural approaches to issues in care are important, but other issues that need attention include the training and licensing of care staff, the role of professional nursing, and the responsibilities of commissioners and providers. While many of these issues have strong advocates, they are still largely unco-ordinated. This leads me to return to the need for the care home sector to have a greater recognition, design and support as a means to generate dignity.

Parkinson's disease: across the ages

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Pharmacological treatment of Parkinson's disease (PD) has historically focused on the motor symptoms of PD.

The beginning of treatment of PD began in 4500 BC when the disease was recorded as Kampavata—a shaking disease. James Parkinson wrote his famous essay on the "Shaking Palsy" in 1817, which established Parkinson's as a recognised medical condition.

It was another half a century before there was any treatment and in 1867 the age of the anticholinergics began. For a century this was all there was to manage the "shaking palsy". In 1912 the Lewy body was identified. This gave us our first pathological basis for PD.

Then in 1960, the theory of a dopaminergic deficit in PD was actually nailed down. A year later levodopa was tried in PD, but it was poorly tolerated and it led to huge neuropsychiatric problems. It wasn't until 1967, when a different approach was used by George Cotzias of using small amounts and titrating up. This led to the first theory of how to treat PD—"start low and go slow". This approach is often followed today.

In 1969, dopa-decarboxylase inhibitors were launched, which was another giant leap in treatment as this allowed doctors to use much smaller doses of levodopa, which reduced the side effects. Among these side effects were motor fluctuations. Patients can tolerate treatment of levodopa at first but then as time goes on the therapeutic stage gets narrower and narrower especially for patients in the complex stage of the disease.

Too much levodopa and the patient experiences dyskinesia and too little then the patient can switch off. There are a number of approaches to managing these motor fluctuations. This includes taking smaller doses more frequently. However, this becomes increasingly inconvenient and many patients can't stick to it. Concerns about these fluctuations and a feeling that the drug might be toxic led to a shift away from levodopa.

In 1974, bromocryptine, the first oral dopamine agonist, was tested, and a new approach to treating PD followed. The non-ergot dopamine agonists and COMT inhibitors then came along with deep brain stimulation treatment for patients with severe PD.

In early 2000, things shifted again with the discovery that dopamine agonists were associated with impulse control disorders such as hypersexuality, shopping and "punding."

Another issue was recognition that the ergot-derived dopamine agonists (although very effective) could cause cardiac-valve regurgitation.[1] This risk was particularly high among patients who had taken daily doses of pergolide or cabergoline that exceeded 3mg; the risk was increased only among those who had taken either drug for six or more months. The risk was not increased among patients treated with other ergot-derived dopamine agonists or with dopamine agonists that are not derived from ergot. This led to a wholesale shift to non-ergot drugs.

Continuous dopaminergic stimulation then became the watchword with the launch of continuous intestinal infusions and the rotigotine patch followed by rasagiline, a monoamine oxidase inhibitor.

The approach now is rational polypharmacy and the feeling is that we don't put all our eggs in one basket—a lot of patients are on three PD drugs at smaller doses. It is rare to see people reaching top doses of levodopa.

1. Schade R, et al. *N Engl J Med*. 2007; **356**(1): 29-38.

Advances in understanding the ageing process

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There is no genetic programme for ageing. We age because in our evolutionary past it would have been too expensive to build a body that might last for ever.

Ageing is caused primarily by damage. Longevity is regulated by resistance/repair. There are multiple mechanisms in this that are inherently stochastic (influenced by chance). The ageing process, therefore, is functional impairments in organs and tissues leading to age-related frailty, disability, and disease, which are caused by accumulation of cellular defects and random molecular damage.

One question this raises is why is the aged cell (or organ) more vulnerable to pathology? This is because damage drives the ageing process and this damage accumulates from day one. Each cell division is accompanied by inevitable somatic mutation.

Since the very beginning of the study of the biology of ageing it was clear that one of the main determinants of ageing was located in the nucleus and that is the telomeres. Telomeres protect chromosome ends—they shorten with cell division (end-replication problem); and this is accelerated by biochemical stress. Critically short telomeres cause growth arrest. Prematurely short telomeres are linked with increased risk of age-related disease and diminished survival.

In 2007, a major discovery was published in *PLoS Biology*[1] showing for the first time that interactions between mitochondrial dysfunction and telomere erosion were of primary importance in driving cells to age. This has transformed the field and led to a series of recent exciting discoveries about the molecular networks that are involved.

Other factors influencing longevity and health span include genes and nutrition, lifestyle, environment, socioeconomic status and attitude.

In the Nutrition and Survival: EPIC-Ageing Study, (which included 76,707 men and women aged 60-plus with no coronary heart disease, stroke or cancer at enrolment) a Mediterranean diet was associated with increased survival among older people.[2] This diet includes high intakes of vegetables, fruits and cereals; moderate to high intake of fish; low intake of meat; low intake of saturated fatty acids; high intake of monounsaturated fatty acids (olive oil); low to moderate intake of dairy products, principally cheese and yoghurt; and modest intake of alcohol (mostly wine).

Exercise also significantly improves health across the life course and delays diseases linked with ageing.

The Newcastle 85+ Study[3] is a prospective study in 1041 individuals all born in 1921 that looks at the biological, clinical and psychosocial factors associated with healthy ageing. Started in 2006, it has been funded for seven years and the study aims to: assess, in great detail, the spectrum of health in the oldest old; examine the associations of health trajectories and outcomes with biological, clinical and social factors as the cohort ages; identify factors which contribute to the maintenance of health and independence and advance understanding of the biological nature of human ageing.

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2. Trichopoulou A. *BMJ* 2005; **330**: 991
3. Collerton J, et al. *BMJ* 2009; **339**: 4904

End of life care in diabetes

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Patients with diabetes who are at the end of life have a unique set of care needs related to both health and social care. However, end of life diabetes care has been recognised as an area lacking quality standards and guidance on best clinical practice and commissioning.[1]

Palliative care concentrates on reducing symptoms of a disease or slows its progress rather than curing. It aims to improve quality of life by reducing or eliminating pain and other physical symptoms as well as enabling the patient to ease or resolve psychological and spiritual problems with support from their family.

Its importance is gaining recognition, but it has always been part of diabetes treatment objectives; diabetes is associated with life-threatening diseases such as vascular disease, dementia, chronic kidney disease, and malignancy.

Palliative care in diabetes management can be straightforward—one makes a comprehensive assessment and asks if the proposed intervention will do more good than harm. Sometimes management is difficult as there are no randomised trials in this area and healthcare professionals, patients and families are no longer striving for good glycaemic control.

Markers for death within the next six to 12 months include multiple comorbidities, non intentional weight loss of >10% over six months, and general physical decline. Other markers include a serum albumin <25g/l; dependence in most activities of daily living; repeated hospital admissions with little improvement, and patient getting worse despite increasing treatment.

Management includes removing unnecessary drugs such as statins, and relaxing glycaemic targets to allow for weight loss and poor appetite. Communication with patients, family and healthcare professionals is crucial.

Diet can be relaxed with the use of "build up" drinks but healthcare professionals must be aware that these can raise glucose levels and there is some evidence that they do not help.[2] Dietetic advice is valuable but asking the patient what they want to eat and obtaining that food (however possible) seems to work best.

If a patient is at end of life and has type 1 diabetes, the treatment regime can be simplified (eg. twice daily fixed mixture). The insulin dose can be reduced as weight and appetite decrease. If a mentally competent patient requests withdrawal of insulin, this should be respected.

If a patient is at end of life and has type 2 diabetes, tablet medication can be reduced and often withdrawn. If they are insulin treated, this can often be reduced and stopped.

Other problems in end of care in diabetes include intra-abdominal malignancy, which may delay gastric emptying causing poor absorption of oral agents. Pain is also a factor as is thirst, thrush and confusion. Patients are also at higher risk of gastrointestinal bleeds increased by aspirin, steroids and SSRIs.

The advice on steroid use in end of life diabetes care is to keep them to a minimum. Healthcare professionals need to keep a good record of steroid dose, diabetes treatment and the effect on the patient. Steroids raise the glucose levels over the afternoon and evening, and treatment must be targeted at these times.

1. www.diabetes.org.uk/upload/Position%20statements/End%20of%20Life%20Diabetes%20Care%20Stategy.pdf
2. www.bmj.com/content/315/7117/1219.full

Obesity and cardiometabolic disease

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Abdominal obesity predicts adverse outcomes such as sudden death. The Foresight report (2007) referred to a "complex web of societal and biological factors that have, in recent decades, exposed our inherent human vulnerability to weight gain".[1]

In this document more than 52 so-called "system-maps" defining the causes of obesity were reported. It also included a number of calls to action for healthcare professionals who deal with obese patients. One such call to action estimated that the cost of diabetes management (which will run to billions by 2050) could be significantly reduced if healthcare professionals were highly engaged in the management of obesity. Sadly, an accompanying paper found that there is no evidence that obesity can be successfully treated.

What does then play a role in obesity? In the Paris Prospective study, 7079 asymptomatic, middle-aged men were stratified for sagittal abdominal diameter (SAD) and BMI, and followed for clinical outcomes for an average of 23 years. The measured outcome was sudden death.[2]

The risk of sudden death increased in parallel with increases in abdominal obesity. These data suggest that SAD, the measure of abdominal obesity, was superior to BMI in predicting the risk of an adverse clinical outcome for most of the study participants.

Epigenetics also plays a role in obesity. This is "the branch of biology which studies the causal interactions between genes and their products, which bring the phenotype into being". A foetus will get a massive amount of information from its mother about the world it's going to live in once it's born and the mother's body composition, diet, lifestyle, all tell her baby about the world in which she lives. Bad diet during pregnancy can suppress healthy cells in the foetus so what your mother does during pregnancy can predispose you to premature death and diabetes due to obesity. It is irrelevant what genes a baby is born with; what matters is which, and how much those genes are expressed, in deciding whether a child has a long and healthy life, or a short obese one.

Obesity is a risk factor for many diseases, such as diabetes mellitus, hypertension, stroke, and heart and renal disease. Despite this relationship, obese people with these diseases in some circumstances may live longer than their normal-weight counterparts. This conundrum has been called the "obesity paradox". In one study, it was fitness that altered the obesity paradox. Overweight and obese men had increased longevity only if they registered high fitness.[3] The obesity paradox means that healthcare professionals need to assess elderly people individually to ascertain whether losing weight is a good thing.

Obesity in primary care involves identification, screening, management and treatment. Treatment includes orlistat. Studies have shown that orlistat produces greater weight loss than diet alone.[4] During four years of treatment, orlistat plus lifestyle intervention significantly decreased the progression to type 2 diabetes compared with placebo plus lifestyle intervention. Cumulative incidence of diabetes was 6.2% with orlistat and 9.0% with placebo, corresponding to a 37.3% decrease in the relative risk of developing diabetes with orlistat.

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2. Empana JP, et al. *Circulation* 2004; **110**: 2781-5
3. McAuley PA, et al. *Mayo Clin Proc* 2010; **85**(2): 115–21
4. Sjöström L, et al. *Lancet* 1998; **352**: 167–72

Chronic pain and the ageing patient

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The population is ageing and we are set for a "time bomb" of the oldest and most vulnerable compared to their younger counterparts. There has been a shift in the age distribution of the world's population and the proportion of this population over 65 years old will rise from 7.4% to 16.4% by 2050.

Pain is a common problem in this age group but it shouldn't be assumed to be "part of getting older". There are a number of barriers to effective pain management in older patients and these include attitudes held by healthcare professionals and the older population themselves.

Fundamental to the principals of pain management, the first step is pain assessment and being older does not preclude the use of pain assessment tools, even in the presence of cognitive impairment.

Pain is the most frequently reported symptom in over 50% of community-dwelling patients and over 80% of nursing home residents. In addition, 19% of older persons admitted to hospital have moderate or extreme pain and cancer is the second leading cause of death in patients over 65 years old.

According to a care homes study,[1] there is a reluctance to report pain in the older population and an acceptance that being in pain is normal. There is also a low expectation from medical interventions. In addition, there is a fear of chemical and pharmacological interventions and a lack of awareness of potential strategies.

Extracts taken from "listening events" and interviews held with older people who suffer pain by Help the Aged include[2]: "*Pain is exhausting... you have to walk slowly. You have to stop and make an excuse or pretend to look in a shop window so that you can put your hand on the window and rest a moment. It's humiliating*" and "*pain is frustrating because you can't do things for yourself … everything's a challenge.*"

Pain can be assessed using a pain assessment scale such as the Visual Analogue Scale (Scott & Husskisson 1976); Verbal Descriptors (Gracely et al 1981); and the Faces Scale (Whaley & Wong 1987).

With older patients, healthcare professionals need to listen carefully to what words are used. A patient may deny pain but admit to discomfort, aching and soreness. Questions to use include: Do you hurt anywhere? Are you uncomfortable? How does it affect you? Most importantly we need to believe the patient.

Intuitive signs of pain include facial expression (grimace), verbal expression (groaning, moaning), protected position—rigid, limited movement, restlessness, agitation and physiological signs such as clammyness, sweating, paleness, and raised blood pressure.

Management of pain in older adults includes pharmacological, interventional, psychological, self-management, exercise/activity/assistive devices and complementary therapies.

There are many areas that require further research, including pharmacological management where approaches are often tested in younger populations and then translated across. Prevalence studies need consistency in terms of age, diagnosis and terminology, and further work needs to be done on evaluating non-pharmacological approaches as the impact of pain can be fairly substantial with this group.

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2. Kumar A & Allcock N. Help the Aged—Pain in Older People: Reflections and Experiences from an older person's perspective (2008)

How to organise a good memory service

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There are four priority areas in dementia, which are: good quality early diagnosis and intervention for all; improved quality of care in general hospitals; living well with dementia in care homes; and reduced use of antipsychotic medication.

A memory service is a good way of achieving these four priority areas. The Memory Services National Accreditation Programme is useful for helping set up a memory service. This is a set of standards for a range of healthcare professionals to assure and improve the quality of the service.

The overarching principles that have guided the development of the standards are that: people with memory problems/dementia have fair access to assessment, care and treatment on the basis of need, irrespective of age, gender, social or cultural background, and are not excluded from services because of their diagnosis, age or co-existing disabilities/medical problems. Also that people with memory problems/dementia and their carers should receive a service that is person-centred and takes into account their unique and changing personal, psychosocial and physical needs.

The memory service works closely with other professionals, agencies and providers to support the processes of assessment and diagnosis.

It should also provide timely access to assessment and diagnosis to ensure that a diagnosis of dementia is made only after a comprehensive and holistic assessment of the person's needs by appropriate professionals, either within the service or elsewhere.

The memory service should also be designed and managed so that the respect and dignity of people with memory problems/dementia and their carers is preserved. Additional tests and investigations should be carried out in accordance with individual and clinical need.

It should also be able to offer appropriate support, advice and information to people with memory problems/dementia and their carers at the time of assessment and diagnosis, as needed.

Many areas now have memory services so a bigger issue for a lot of patients is about local modifications of the service, which ensure the most cost-effective delivery of services. Another question is how much of this service could be done in primary care? The expectation is that GPs will refer patients on to a memory service once the patient has been diagnosed.

GPs should also offer treatment and advice on general health, driving and finances. Advance care planning and cognitive rehabilitation should also be taken into account. With the evolution of disease, patients should have a point of contact for advice and support and access to day/respite care especially if there is challenging behaviour.

In an acute hospital setting there should be cognitive testing on admission and recognition of delirium. A care pathway for patients with dementia should be implemented with appropriate staff training. Whereas in the nursing home setting, factors to be considered include diagnosis, neuropsychiatric symptoms, psychotropic drugs and palliative care.

The essentials in my view are to have an experienced specialist, experienced nurse, occupational therapist and social worker, and that will go a long way to covering the basics. I think that the four priority areas will make a big impact and the changes will be enormous in England.