Reviews in Medicine

Medicine in the elderly

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Introduction

The population is ageing, and old people are found in all wards. This review, written from the viewpoint of senior registrars in training, focuses on issues which we deem important.

The organisation of hospital services reflects past political decisions. Our specialty (geriatric medicine) exists because, at the start of the National Health Service, government placed responsibility for the chronic sick on the hospitals. From that decision our specialty, without a system or diagnostic base, emerged. In making choices we recognized that other disciplines may see our world differently. Nevertheless, the pot-pourri of topics that we have selected reflects our interests.

Geriatric medicine is a service discipline. Accepting that, it was tempting to begin this collection of reports with service organization. Yet we recognize the interest of our readers and begin with clinical matters. Those who reach the end will find we conclude with service matters, but between there and now, are contributions on skin ageing, pressure sores and fractures and metabolic bone disease; dementia and thrombolytic therapy and angiotensin converting enzyme (ACE) inhibitors.

These medical topics are then complemented by reviews of current practise relating to screening, stroke rehabilitation and the problem of carers.

Finally, the paper concludes with contributions on the organization of services and medical audit and we hope that our readers get as much enjoyment out of reading this review as we gained in writing it.

Skin ageing. Pathological and clinical changes

Skin changes associated with ageing (intrinsic ageing) are frequently confused with those due to environmental assault (extrinsic ageing) but recent research has helped to differentiate histological and clinical changes of intrinsic and extrinsic ageing.^{1,2} It is common to attribute the changes of extrinsic ageing to those of intrinsic ageing, as the latter occurs on readily visible skin and becomes more pronounced with advancing age.³

Intrinsic ageing

There is a flattening of the interdigitations between the dermal–epidermal interface and thus reduced adherence between the two surfaces, predisposing the ageing skin to blistering.³ Age-related skin changes include reduced proliferative activity in the epidermis and a slowed healing rate. Attenuated microvasculature predisposes to hypothermia, hyperthermia and prolonged or persistent contact dermatitis due to delayed clearance of allergens and irritants.³ Reduced sensation occurs and diminished elasticity due to changes in collagen predispose to tear-type injuries.

Langerhans' cells, which act as skin macrophages, are reduced in aged skin and further in chronically sun-exposed.³ The resultant impaired cell-mediated response increases the susceptibility to skin infections, particularly viral and fungal, though it reduces the potential for allergic contact dermatitis. The reduced cell-mediated immunity reduces the inflammatory response and patch tests reactions. Patch test sites for suspected allergic contact dermatitis should be assessed 3 weeks after application, in the elderly, as well as the routine follow-up at one week.³ The reaction of the tuberculin test is also reduced.⁴

Reduced immunosurveillance may account for a recently recognized increased susceptibility to cutaneous neoplasia. This is probably a result of

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abnormal antigen processing by the Langerhans' cells, affecting development of a suppressor T cell population, retarding tumour rejection.⁵ In addition, reduced numbers of melanocytes produce less melanin, thus compromising the ultraviolet barrier. B cell dysfunction in elderly skin is reflected by increased autoantibody formation and an increase in serum levels of IgA and IgG, predisposing to the development of pemphigus and pemphigoid.⁶

Ultraviolet B radiation converts 7-dehydrocholesterol in the skin to provitamin D3 which then undergoes spontaneous conversion to vitamin D3. Epidermal 7-dehydrocholesterol content and the release of active vitamin D into the blood are reduced by as much as 75% in the elderly.⁷

Extrinsic ageing

Skin changes from chronic sun exposure (photo-ageing) include coarseness, wrinkling, laxity and telangiectasia due to alterations in the connective tissue, mottled pigmentation due to melanocytic hyperplasia, benign hyperplasia and premalignant and malignant neoplasms. The majority of features associated with skin ageing are the result of sun-exposure and may account for more than 90% of the age-associated cosmetic problems of the skin.⁸ Eighty percent of UV-induced photoageing may occur within the first 20 years of life, raising the possibility that these changes could be reduced with effective sunscreens.

Actinic (solar) keratoses occur in photodamaged skin. These rough erythematous scaling lesions are the result of alternating zones of hyper- and parakeratosis overlying thickened or atrophic epidermis. The normal maturation pattern of the epidermis may be lost and occasional pleomorphic keratinocytes may be seen. The rate of transformation of actinic keratoses to squamous cell carcinomas is low. A survey of a randomized stratified sample of the population in South Glamorgan found a prevalence rate of 26%⁹ and, in a longitudinal survey, very few lesions (0.24%) underwent malignant change.¹⁰ Many of the lesions appear to disappear and this may provide information, in the future, of potential treatment.

There has been recent interest in the use of topical tretinoin (all-trans-retinoic acid) for partly reversing the structural damage of photoageing.^{11,12} It causes hyperplasia of the epidermis, eliminates dysplasia and atypia, including microscopic keratoses¹³ and leads to the formation of new collagen in the papillary dermis.

Pressure sores

Deep pressure sores develop as a result of pressure between a bony prominence and an outside resis-

tant surface. Recent measurements of tissue viability have shown the resultant high interstitial pressures and capillary deformity.^{14,15} This is distinct from superficial tissue distortion associated with humidity and friction that causes superficial dermal sores. Seventy percent of pressure sores occur in hospital within the first 2 weeks,¹⁶ indicating that they occur as a result of acute illness, though age-related skin changes predispose to pressure sores.¹⁷

Pressure ulcers usually result from a combination of extrinsic and intrinsic factors. Extrinsic causes, which are particularly important in patients admitted to hospital, include pressure, which is the most important, shearing forces, friction and moisture. Many current hospital regimes exacerbate pressure on the skin and increase the likelihood of patients developing sores. Intrinsic factors occur as a result of illness and include reduced sensation and mobility, sedation, pain, low blood pressure, peripheral vasoconstriction due to shock or heart failure, dehydration causing increased tissue deformity, septicaemia and nutritional deficiency.¹⁸

Elderly patients with femoral fractures are especially vulnerable to pressure sores and should be treated on low pressure support systems from the point of entry to hospital.¹⁹ Long periods left lying on unprotected trolleys in casualty and X-ray departments, or on operating theatres, increase their incidence.¹⁹

The Norton Score¹⁸ is one of several scales drawn up to combine risk factors to enable targeting of interventional measures. Physical condition, mental state, activity, mobility and incontinence are taken into account. A promising, more accurate score, the Braden Scale,²⁰ is being developed to be more specific and reduce the overlap between the physical and mental condition that occurs when predicting mobility and activity, and a nutritional assessment is also included.

The basic principles of preventative care are bed-rest with regular repositioning, relief of local pressure and the use of anti-pressure mattresses. In a patient's home, an emergency anti-pressure mattress can be constructed with pillows and cushions. Some types of alternating pressure air mattresses (APAM), such as Large-Celled Ripple or Alphabet Bubble pads, are suitable for emergency use in the community. In casualty departments, receiving trolleys must be protected by pillows or low-pressure mattresses. In the ward, patients should be nursed mainly in bed. Soft pressure-relieving mattresses that rely on low interface pressures²¹ may help to prevent superficial sores but are usually unable to prevent deep sores in sick patients unless they are repositioned regularly.²² Since 2 hourly turning may be impractical, anti-pressure mattresses, which reduce or eliminate the need for frequent repositioning can be helpful.

The latter include alternating-pressure air mat-

tresses (APAMs) which are relatively small and inexpensive and can be used in emergency situations, including the patient's home. They aim to prevent pressure damage over bony prominences by constantly changing the areas of support. The Pegasus Airwave Mattress,²³ which has a double bank of cells, can provide relief of pressure under even very heavy or contracted patients and those who have to be nursed in one position, such as orthopaedic patients on traction.²³ The latter bed is suitable in intensive care units as it can be immediately deflated in a cardiac arrest, can be used in the community, and is the most reliable APAM available in Britain today. Only those machines which can be guaranteed for one year's continuous use and have adequate warning systems in the event of failure should be used. Rigorous systems for supply and servicing are also essential.²⁴

Leg ulcers

A community study of a population of over one million found an incidence of active ulceration of 1.5/1,000.²⁵ The prevalence is, however, higher than this as ulceration has phases of healing and breakdown.²⁷ About 54% of ulcers are due to venous insufficiency and 10% are due to arterial ischaemia. Ultrasonography and photoplethysmography have shown that 27% of ulcers are due to a combination of arterial disease and venous insufficiency.²⁶ The ulcer is often heavily colonized by bacteria which are often not contributing to further damage and are not necessarily an indication for antibacterial treatment.²⁷ Conditions which may exacerbate or delay healing of the ulcer include anaemia, cardiac failure, diabetes mellitus and obesity. Severe limitation of ankle joint movement, deformity from fractures and neurological disease are frequent but less treatable contributory factors.²⁸ There is no case for supplementation with zinc and vitamins in the absence of a demonstrable deficiency.²⁹

Direct measurements of skin oxygen tension, using a surface oxygen electrode, have failed to demonstrate any block to diffusion as oxygen tensions are normal or high in recumbent ulcerated legs.³⁰ However, as a result of venous hypertension, the fall in oxygen seen on standing in normal limbs persists in affected limbs on exercise.³⁰ In normal legs, skin oxygenation is increased during exercise.³⁰

The use of an effective compression bandage is probably the most important single measure in the management of leg ulcers and attention to proper dressing and bandaging is likely to be more important than the choice of dressing.

The healing of a leg ulcer can be accelerated by

skin grafting. A recent new development, which may supersede grafting in many cases, is the use of sheets of keratinocytes grown in culture; good results have already been obtained.³¹ Once a venous ulcer is healed, measures should be taken to relieve venous hypertension and, if surgical correction is possible, this has been shown to reduce recurrences.³²

Systemic complications and skin disease

Systemic complications arise from widespread skin disease and can be fatal in the elderly.³³ The loss of barrier function in severe skin disease can cause loss of fluid and electrolytes, and the development of cutaneous and systemic infections. The latter is also worsened by the alterations of the normal host defence mechanisms. Impaired thermoregulation and increased cutaneous blood flow can occur with an increased cardiac output contributing to heart failure.

The main diseases responsible for systemic complications and necessitating intensive care are: toxic epidermal necrolysis, auto-immune blistering diseases, extensive viral infections, skin necrosis and erythroderma or exfoliative dermatitis.

Toxic epidermal necrolysis is a severe acute blistering disease with widespread sheet-like loss of epidermis. The incidence increases with age, probably because of increased drug use.³⁴ Sulphonamides, anticonvulsants and non-steroidal anti-inflammatory drugs are frequent culprits.³⁴ Age is an important adverse prognostic factor as is the area of necrolysis and a rise in blood urea.³⁵ Infection is the most important cause of death.³⁵ The death rate is around 30% and the basis of management remains symptomatic.³³

Auto-immune blistering diseases remain severe. The mortality rate in pemphigus has dropped to 5 to 10%.³⁶ Bullous pemphigoid should be considered more severe with a mortality rate around 20% at one year, related to the older age of these patients.³⁷ Sepsis is the main cause of death in both pemphigus and pemphigoid. Systemic steroids is the main treatment in both pemphigus and pemphigoid which can be used with adjuvant immunosuppressive therapy such as azathioprine.

The main complications of exfoliative dermatitis are infection and high output heart failure resulting from increased cutaneous blood flow, potentially dangerous in the elderly and those with previous cardiac disease.

Management of the above skin diseases includes careful attention to haemodynamic and electrolyte equilibrium, enteral high calorie protein-rich diet usually by tube feeding, the use of antibiotics only when there are direct or indirect signs of sepsis and

the alteration of the thermal environment to lower the energy expenditure. Prognosis can be assessed using an acute physiological score.³³ Age, extent of skin lesions, neutropenia, administration of high doses of corticosteroids and elevated blood urea have been found to be poor prognostic indicators in burns, toxic epidermal necrolysis and autoimmune blistering diseases.³⁷

Fractures

Bone is remodelled throughout life under the influence of local and systemic hormones. Osteoclasts resorb bone, to be replaced after 2 or 3 weeks by osteoblasts which, over 2 to 4 months, fill in the cavity forming a new structural unit.³⁹ Normally the resulting bone is as strong or stronger than before. In metabolic bone disease, it may be osteopaenic and thin or osteosclerotic and thick. In old age such disease commonly weakens bone causing pain and deformity.

The elderly have a stoical attitude to discomfort and disability and presentation may be with immobility. Fractures of weakened bone are frequent.^{40,41} By 80 years of age 15% of women will have suffered a Colles' fracture;⁴² and more than a quarter will have persistent pain and reduced grip strength 3 years afterwards.⁴³ The incidence of vertebral fracture is difficult to quantify, but if wedging is included, up to three quarters of elderly women are affected.^{41,44,45} The result may be deformity and pain requiring analgesia and nursing care, often necessitating hospital admission.

Proximal hip fractures are becoming increasingly common in the elderly.^{46,47} Thirty percent of sufferers will die within a year and 25% will have significantly more disability afterwards.⁴⁸ Consumption of orthopaedic resources contributes to the waiting list for routine operations.⁴⁹ Elderly patients, who sustain a proximal femur fracture, are likely to have multiple medical and rehabilitation problems.^{50,51} It is now clear that liaison or shared management by geriatricians and orthopaedic surgeons shortens the length of hospital stay and reduces the number of patients who subsequently require permanent nursing care.⁵²⁻⁵⁵ Some have opted for a liaison service with management on orthopaedic wards.⁵⁵ Others have developed joint 'orthogeriatric' wards to which patients may be admitted directly, transferred immediately post-operatively or else at a convenient subsequent occasion.^{52,54,56} Some advocate primary admission to a geriatric ward with orthopaedic surgeons operating from there. The key is rapid assessment with minimal delay before operation. Although acute management is best undertaken by surgeons, many of the patients will have an underlying cause

for the fall and early medical assessment is essential.

Involutional osteoporosis

Osteopaenic disease may be due to exogenous steroids or anticonvulsants, endocrine disorders such as Cushing's, hyperparathyroidism and hyperthyroidism, renal failure, malignant disease or calcium and vitamin D deficiency. Poor diet, minimal sun exposure, low skin levels of precursors of vitamin D together with impaired renal conversion of 25 hydroxy' to 1,25-dihydroxy-vitamin D contribute to the low calcium absorption seen in old age.⁵⁷⁻⁶¹ Patients with poor diet and low sun exposure should take supplements.^{62,63}

Involutional osteoporosis is the most important osteopaenic disease and the most common bone disorder of old age. There is loss of bone mass without detectable change in ratio of mineralized to non-mineralized matrix. The risk of fracture depends in part upon the frequency of falls and the effectiveness of protective reflexes,^{64,65} but critically upon bone mass. The greater the mass the less the chance of fracture.⁶⁶

Peak bone mass depends upon genetic factors, adequate nutrition and life style. It is achieved by early adult life with subsequent progressive loss with age. Therapy can be aimed at preventing loss and/or stimulating new bone formation.

In women there is an acceleration of bone loss at the menopause which is prevented by oestrogen therapy.⁶⁷ There is an initial increase in bone density as the remodelling space is filled in, thereafter bone loss is retarded so long as therapy continues.⁶⁸ It is still effective in retarding further bone loss in elderly osteoporotic patients.⁶⁹ Few women accept indefinitely the cyclical withdrawal bleeding of hormone replacement therapy and its life-long effects are unknown. There may be a role for indefinite administration to patients without a uterus but hormone replacement therapy is unlikely to continue into old age. Perimenopausal oestrogen replacement therefore delays rather than eliminates female involutional osteoporosis.

Thrice weekly calcitonin intra-muscularly is as effective as oestrogen in preventing bone loss⁷⁰ and its administration by nasal inhalation together with oral calcium supplements also prevents osteoporosis.⁷¹ The latter may represent an acceptable form of prolonged therapy but ideal dosage, long term side effects and the subsequent fracture rate are unknown.

Bone needs mechanical stress to maintain its integrity, cessation of regular exercise causing reduction in bone mass, conversely participation in an exercise programme, even after the menopause, can increase bone density.^{72,73} Exercise may stim-

ulate remodelling but extra calcium is likely to be required for mineralization. Recommended daily intake of calcium is 800 mg⁷⁴ for a 75 year old. The elderly often fail to ingest this amount as well as absorbing calcium poorly.^{59,60} To remain in calcium balance they need an intake averaging 1,400 mg calcium a day.^{75,76} Therefore it is wise to give dietary supplements of about 500 mg/day to elderly osteoporotic patients, probably with the vitamin D 400 IU to help absorption.⁷⁷

Osteoporotic patients need increased bone mass. Fluoride stimulates osteoblasts and people in areas with fluoride in the water were thought to have fewer fractures than those without.⁷⁸ It was hoped that fluoride therapy would increase skeletal mass. Its administration does increase density of cancellous bone, such as vertebrae, though at the expense of cortical. The result is increased numbers of long bone fractures in those treated. The evidence for a reduction in vertebral fractures is equivocal suggesting that the quality of the cancellous bone is poor.^{79,80} In addition therapeutic doses produce joint pains and gastrointestinal side effects which limit its use.⁷⁹

Anabolic steroids may be able to stimulate bone formation when combined with calcium supplements^{81,82} but evidence is not conclusive. Abnormalities in liver function, hirsutism, fluid retention and risk of hepatoma preclude their use.

Biphosphonates inhibit bone resorption.⁸³ Results of cyclical treatment using etidronate and calcium supplements show an increased vertebral bone mass coupled with decreased fracture rate without a loss in long bone density.⁸⁴ The effectiveness in those aged 75 or more, with established osteoporosis is unknown but biphosphonates are a promising future option for restoring bone mass.

Paget's disease of bone

Paget's disease of bone can be found in 6–8% of those over 55 years⁸⁵ affecting one or more bones. The primary disorder is of osteoclasts which are highly active and resorb bone far in excess of any need for remodelling. New bone is deposited, only for it to be removed. Successive waves of resorption and accretion result in enlarged bone with disorganized internal structure. There is pain, deformity and an increased fracture rate. Occasionally it may cause nerve entrapment or high output cardiac failure.

The majority of patients require no treatment, or at most analgesia. In severe disease the cytotoxic agent mithramycin is effective in reducing pain but usage is limited by concern about toxic effects on liver, marrow and kidneys. Though such problems are rare⁸⁶ it is reserved for intractable disease.

The ability of calcitonin to inhibit osteoclasts

makes it an ideal drug to treat the disease. Parenteral administration is effective in reducing the number of osteoclasts, improving pain and biochemical parameters and slowing disease progression.^{87,88}

Biphosphonates are powerful inhibitors of bone resorption and turnover.⁸³ They can be administered orally and etidronate is effective in relieving pain though there is little evidence of healing of osteolytic lesions.^{89,90}

Mild disease is managed with simple analgesia as required, bone pain may respond to biphosphonates, calcitonin is used when deformity, nerve entrapment or fracture seem likely. Mithramycin is reserved for resistant cases.

Dementia

Introduction

Over the last 20 years dementia has emerged to become a prime research topic. Cognitive impairment is no longer considered a part of normal ageing but as a pathological process. There has been a change of attitude away from the division into pre-senile dementia, with the assumption of an underlying pathological process and therefore with the hope of prevention or cure, and senile dementia with the assumption of an irreversible cause as part of the normal ageing process. Dementia affecting older subjects is now equally targeted for the research efforts of many diverse groups including geriatricians, neurologists and psychiatrists, as well as pure scientists. This review aims to bring together recent research from all these groups.

Alzheimer's disease

Despite a massive research effort, the aetiology of Alzheimer's disease (AD) remains obscure. However a great deal has been learned about the composition of the pathological hallmarks of the disease. It is now recognized that senile plaques and neurofibrillary tangles contain amyloid which is also found in relation to blood vessels in patients with AD. Particular research attention is being paid to the nature and origin of amyloid in AD. The A4 amyloids from plaques and from blood vessels are probably identical⁹¹ and are the breakdown products of a larger precursor molecule.⁹² The paired helical filaments of neurofibrillary tangles may also be A4 amyloid, although this remains controversial.⁹³ The association of AD and Down's syndrome, in which a significant proportion of patients develop dementia with the pathological changes of AD, and the localization of the gene coding for the familial form of AD to chromosome 21⁹⁴ directed much research interest towards a chromosomal abnormality. This in-

creased on localizing the gene coding for A4 amyloid to the same chromosome.^{92,95,96} However the finding that these two loci are separate^{97,98} and that the gene coding the amyloid precursor is identical in AD and normal controls has caused a waning of enthusiasm for a purely chromosomal aetiology.

The importance of vascular abnormalities in AD has recently been re-evaluated.⁹⁹ Cerebrovascular deposition of amyloid (cerebral amyloid angiopathy – CAA) is common in AD but is of uncertain importance as the distribution does not parallel that of plaques and tangles. It is also found in association with other disease processes such as Down's syndrome, Binswanger's disease and spongiform encephalopathy,¹⁰⁰ and in isolation as a cause of cerebral haemorrhage especially in non-hypertensive elderly subjects. The role of CAA in dementia therefore remains unclear. It may cause dementia directly by producing brain ischaemia or may be indirectly involved as a cause of the other pathological hallmarks of AD. The aetiological role of CAA in AD remains speculative, but the presence of these vascular changes may have prognostic significance. Periventricular lucencies on CT scanning (leuko-araiosis) are common in AD.¹⁰¹ These correlate with the post-mortem finding of CAA and these patients appear to have a worse prognosis with early death.¹⁰²

Aluminium has been implicated in the aetiology of dialysis dementia and is also found in the core plaques of AD.^{103,104} Although a causative link with AD has been postulated,¹⁰⁵ this remains unproven. The reason for aluminium deposition in plaques is uncertain, but it may be allowed to enter the brain by a gene affecting permeability of the blood–brain barrier.

Damage to the cholinergic pathways was the first to be demonstrated in AD, but it is now recognized that many neuronal pathways are involved. However, an attempt to manipulate the cholinergic system is still the most promising therapeutic approach in AD, probably because of the importance of cholinergic pathways to memory. Spectacular results with the anticholinesterase inhibitor tacrine hydrochloride (THA) were first described in 1986¹⁰⁶ but this study was then heavily criticized. A recent small pilot study with THA and the cholinergic precursor lecithin reported mainly cholinergic side effects in 6 out of 8 subjects, necessitating withdrawal in three.¹⁰⁷ In a recent double-blind cross-over study, THA and lecithin failed to improve mental or behavioural state, but did cause a small improvement in overall state as assessed by relatives and physicians, but 6 out of 67 dropped out because of hepatitis, 3 others developing hepatitis soon after stopping treatment, and 20 subjects had gastrointestinal side effects.¹⁰⁸ However the study methods were again heavily

criticized, and the place, if any, for THA and lecithin in the treatment of patients with AD must await the conclusion of better designed trials at present in progress.

Degenerative dementias

Degenerative dementias other than AD are less common and have therefore received less research attention to date. They appear to be a heterogeneous group clinically and pathologically, the most common being the dementia associated with Parkinson's disease (PD) and dementia of frontal-lobe type, which have recently been reviewed in more detail.¹⁰⁹

The relationship of dementia to Parkinson's disease (PD) is complex and much remains poorly understood. However, it is certain that dementia is more common in patients with PD than was originally thought, occurring in 15–20% of cases,^{110,111} and that this is not entirely due to the coincidental occurrence of two common disease processes. AD is twice as common in PD patients compared to the general population and is rather different histologically, with relatively fewer tangles.¹¹² Dementia may be a prominent feature of PD, even preceding the extra-pyramidal symptoms in 18%.¹¹³ Demented PD sufferers tend to be elderly with early memory loss, they may have psychotic features with delusions, hallucinations, depression and aggression, and 80% have a fluctuating confusion with mainly cortical features such as dysphasia, dyspraxia or dyscalculia.^{113,114}

The pathological hallmark of Parkinson's disease is the Lewy body. These round, acidophilic structures can be demonstrated in medium to large monoaminergic and cholinergic neurones in the substantia nigra of non-demented patients with PD and signify neuronal injury and fragmentation. Lewy bodies are also found in the substantia nigra or locus coeruleus of asymptomatic individuals,¹¹⁵ being present in 3.8% of non-PD subjects in their sixth decade and 12.5% of those in their ninth¹¹⁵ and their exact significance is therefore uncertain at present. In some patients with PD and the predominantly cortical dementia described above, Lewy bodies can be demonstrated in the cortex. The severity of this cortical dementia found in association with PD correlates to the number of Lewy bodies found in the cortex¹¹⁶ but they are also always found in the substantia nigra and brain stem and the term diffuse Lewy body disease has therefore been applied to this pathological pattern. It has been suggested that PD and diffuse Lewy body disease form a spectrum of clinical syndromes with a similar underlying pathological process, the clinical picture being dictated by the relative distribution of Lewy bodies in the cortex and substantia nigra.

Dementia of frontal-lobe type, also known as frontal lobe dementia of non-Alzheimer type, appears to be a distinct clinical entity.^{117,118} Patients tend to be younger than those with AD and half have a family history of dementia. They present with a marked change in personality and social behaviour with later impairment of speech, but with relative preservation of visuo-spatial skills and lack of neurological signs apart from primitive reflexes. They do not have the histological changes of AD and only a minority have had changes compatible with Pick's disease. There is atrophy of the frontal and temporal lobes with loss of neurones and spongiform change in the cortex associated with reduced cerebral blood flow to the frontal lobes compared to the reduction in flow to the parietal and temporal lobes seen in AD.¹¹⁹

Vascular dementias

Multi-infarct dementia (MID) is the commonest cause of dementia after AD with a significantly vascular component being demonstrated in around 25% of demented patients,^{119,120} although one study found 16 cases of MID out of 27 demented patients coming to post-mortem.¹²¹ It is of particular importance because controlling vascular risk factors such as smoking and hypertension may halt progression of the dementia.¹²²

Vascular dementias can be divided into predominantly cortical (such as MID), sub-cortical (such as Binswanger's disease) and diffuse (as in CAA). A particular clinical pattern of sub-cortical dementia is being increasingly recognized by neuro-psychiatric testing¹²³ with prominent impairment of mood, drive, attention and arousal. This may have a vascular aetiology, such as Binswanger's disease, or a degenerative aetiology, such as Parkinson's and Huntingdon's diseases, progressive supra-nuclear palsy and olivoponto-cerebellar degeneration.⁹⁹

Binswanger's disease has been recently reviewed.¹²⁴ Impaired memory and concentration, paranoia, dependence and mood changes such as euphoria, aggression or depression are common features with motor signs, a pseudo-bulbar palsy and bladder and gait disturbances. It is usually progressive but may plateau or even temporarily improve. It is often associated with hypertension and there may be a rapid decline with surgery, hypotensive events or arrhythmias. It is the result of patchy or diffuse ischaemic injury to deep white matter of the cerebral hemispheres. The clinical and radiological patterns are very similar to those of normal pressure hydrocephalus and it has been postulated that those who do not respond to shunting may in fact have Binswanger's disease.⁹⁹ At present it is not possible to say whether these are cause and effect, or are separate entities causing diagnostic confusion.

Computed tomography (CT) and magnetic resonance imaging (MRI) scanning in Binswanger's disease show symmetrical periventricular lucencies called leuko-araiosis.¹²⁵ This finding was initially thought to be specific to Binswanger's disease but is now known to be more widespread. One study found these changes in 35% of a demented group, including all patients classified as having MID, 33% with AD, 38% with a mixed aetiology and in 11% of normal controls, and demonstrated an association with vascular risk factors.¹⁰¹ There may be an association with disordered blood pressure control (either high or low) even in the non-demented,^{126,127} and post-mortem studies suggest this is due to sub-clinical vascular insufficiency.^{128,129} Leuko-araiosis in the non-demented may prove to be a sub-clinical stage of Binswanger's disease, although there is probably no neuro-psychiatric difference between non-demented subjects with and without these changes on brain imaging.¹³⁰

Transmissible dementias

Creutzfeldt-Jakob disease (CJD) is rare with an incidence of 0.5 cases/million/year,¹³¹⁻¹³³ but is of significance because of the invariably fatal outcome, the potential risk to medical and laboratory staff, and the recent emergence of a similar disease in cattle, bovine spongiform encephalopathy (BSE).¹³⁴ The latter has caused great public concern recently, although the equivalent disease known as scrapie has been recognized in sheep for many years. However, there is no support as yet for the transmission of CJD to man from animals, but the long incubation (15-20 years in man after injection of contaminated growth hormone)¹³⁵ and its resistance to heating will continue to cause concern.

CJD causes dementia but this is rarely an isolated presenting feature.¹³⁶ It is usual for the other features of visual disturbance, dysphasia, visuo-spatial disorientation and cerebellar ataxia to appear early in the course of the illness, so that differentiation from other dementias is rarely a problem beyond the initial stages of the disease.

The causative agent of CJD remains uncertain and the term prion (derived from protein infectious agent) has been coined for a postulated new kind of infectious agent containing little or no nucleic acid.¹³⁷

The human immunodeficiency virus (HIV) is neurotropic as well as lymphotropic and can therefore cause neurological manifestations in the absence of secondary infection of the central nervous system. A sub-acute encephalitis with malaise, loss of libido and psycho-motor retardation (i.e. predominantly sub-cortical features), with deterioration into a bed-ridden, cognitively impaired and incontinent state has been described in the

acquired immune deficiency syndrome (AIDS),^{138,139} and has been termed the AIDS dementia complex. It is common late in the disease, cognitive impairment being found in 46 out of 70 patients dying of AIDS who did not have focal or metabolic central nervous system disease, one third of the total being significantly impaired.¹⁴⁰ A more recent study showed an even greater incidence, with one third being cognitively impaired at diagnosis and two thirds at death.¹⁴¹ Again, this is unlikely to cause diagnostic confusion, but with increasingly elderly people contracting HIV infections, the diagnosis will have to be more widely entertained.

Diagnosis of dementia

The clinical differentiation of AD from vascular dementias is of more significance than mere professional curiosity. The possible halting of vascular dementias by attention to risk factors has already been mentioned, and this differentiation is likely to become of greater significance as further treatment regimes become available for both vascular dementias and AD. It also has great significance in the design and interpretation of research, as a relatively small error in initial diagnosis may invalidate the findings of otherwise perfectly planned studies.

Various studies have shown that CT and MRI scanning differentiate well between AD and MID, but only if compared to a clinical diagnosis.^{142,143} Similarly, ischaemic scores, notably that of Hachinski,¹⁴⁴ correlate well with clinically diagnosed MID.¹⁴⁵ There have been few studies comparing these tools with post-mortem studies however. Wade *et al.* found that 90% of demented patients with a post-mortem diagnosis of AD had a low Hachinski score (< 5) and 65% of those with high score had a significant vascular component to their dementia,¹²⁰ but even these results leave too wide a margin for error, and Homer *et al.* found the pre-mortem diagnosis was incorrect in 11 out of 27 patients coming to autopsy, concluding that the Hachinski score and CT were unhelpful in differentiating AD and MID.¹²¹ Ettlin *et al.* had their best results in diagnosing pure AD with a specificity of 80%, but if the relatively easily diagnosed dementias were excluded leaving only AD and MID, this fell to 65% with a sensitivity of only 47%.¹⁴⁶ Only the Hachinski score was useful in diagnosing MID, CT having a high sensitivity but low specificity, that is, it missed small infarcts, and neither could it diagnose mixed AD and MID.

Another approach uses purely clinical criteria to make a pre-mortem diagnosis of probable or possible AD, as described by the National Institute of Neurological and Communicative Disorders and Stroke and the Alzheimer's Disease and Related Disorders Association – NINCDS/ADRDA.¹⁴⁷ These criteria were recently used to make a pre-

mortem diagnosis in 50 demented subjects who then came to post-mortem.¹⁴⁸ Of the 32 patients diagnosed as probable AD, 24 had AD alone, 4 had AD as well as another pathological process and 4 did not have AD. Of the 18 patients diagnosed as possible AD, 10 had AD alone, 4 had mixed pathology and 4 did not have AD. From this study it would therefore seem that those patients satisfying the NINCDS/ADRDA criteria for probable AD do have AD in about 90% of cases. This might therefore be a useful technique to assess inclusion into a study of AD, although in 10% of cases the diagnosis will be incorrect. Like the other techniques it cannot identify those patients with mixed pathology. It cannot be recommended for general diagnostic use until it has been validated by post-mortem examination of those patients who are negative as well as positive for the criteria, that is when the sensitivity and specificity of the technique has been established. It should be noted that the NINCDS/ADRDA criteria are only an attempt to standardize present clinical criteria for the diagnosis of AD, and are not a new diagnostic method. They should therefore achieve the same success rate as a competent clinician albeit with improved consistency and reproducibility. There is also nothing new in achieving successful diagnosis of AD without the use of a CT scan. Indeed, in Ettlin's study, an attempt to combine a CT and clinical diagnosis was found to be less accurate than using either method alone.¹⁴⁶

Accuracy of diagnosis of dementia is improving, but the present inaccuracies continue to blight research into dementia, so that at the present time results of studies which use pre-mortem diagnosis alone must be interpreted with caution.

Thrombolytic therapy in the elderly

Thrombolytic therapy has become the standard primary form of treatment for acute myocardial infarction in young patients. A few studies have been specifically done in the elderly and have produced interesting findings, indicating both the usefulness and hazards of this therapy. Numerous studies, reviewing the natural history of myocardial infarction in elderly patients, have suggested that age is an important factor in the in-hospital and long term prognosis of acute myocardial infarction.^{149–151} The case fatality rate increases with advancing age and increasing age is also directly related to an unfavourable prognosis among the discharged, as well as being related to a high incidence of congestive heart failure and shock. Therefore the elderly would seem to have much to gain from the successful use of thrombolysis, but do they?

Although the 6-hour time window initially pro-

posed for using thrombolysis has now been extended up to 24 hours,^{152,153} the diagnosis of myocardial infarction may be difficult or impossible within this time limit. Kannel and Abbott have shown that 30% of all individuals diagnosed with acute myocardial infarction in the Framingham study had an unrecognized event that was detected only by serial electrocardiograms.¹⁵⁴ With increasing age the spectrum of symptoms occurring at the time of acute myocardial infarction also changes.¹⁵⁵ In spite of the above difficulties in recognizing an acute myocardial infarction within a given time, Brower and colleagues have demonstrated that the rate of perfusion is similar in all age groups.¹⁵⁶ Unfortunately most studies of thrombolytic therapy in acute myocardial infarction have excluded elderly patients. Nevertheless, some studies including or specifically addressing the elderly are of interest.

Ganz and Lew have reviewed their experience with streptokinase in patients over the age of 75. In their population in-hospital mortality was higher (33%) and appeared to be related to an increased incidence of anterior myocardial infarction, prior myocardial infarction and multivessel coronary artery disease.^{157,158} There was a twofold increase in the incidence of haemorrhage complication compared with younger individuals and this was associated with an increased mortality of 17% compared with 1% under age of 65.

In the ASSET study (Anglo-Scandinavian Study of Early Thrombolysis) recombinant tissue type plasminogen activator was compared with placebo in clinically diagnosed acute myocardial infarction (catheterization was not routinely performed at the time of, or after recombinant tissue plasminogen activator infusion), a significant reduction in mortality was seen in those over the age of 65 (10.8% vs 16.4%) although individuals over the age of 76 were excluded.¹⁵⁹

In the first TIMIS trial (thrombolysis in myocardial infarction) Chaitman found that mortality between age 65 and 76 was increased at 11.8% compared with 3.5% in individuals under 65.¹⁶⁰ While this represents a fourfold increase in mortality, the in-hospital survival rate for the elderly is comparable to that seen in the ASSET study. This supports the hypothesis that recombinant tissue plasminogen activator does benefit the aged population, despite major haemorrhage complications occurring more frequently with increasing age (26.1% of patients over age 70 compared with 11.5% for those under 65). The incidence of haemorrhagic complication in the elderly population without hypertension was similar to the younger population in Lew's series.¹⁵⁸ However, haemorrhagic complications were seen with a higher frequency in older women and in patients with diabetes mellitus or hypertension. In the first

GISSI trial there was no statistically significant benefit in survival demonstrated in patients over the age of 65 although the trend in 21-day mortality (28.9%) appeared to be reduced when compared with placebo (33%).¹⁶¹ No analysis by age subset was performed to see if the overall incidence of haemorrhagic complication was directly related to age.

In the second ISIS study (International Study of Infarct Size) there was a 20% reduction of in-hospital mortality for individuals over the age of 65.¹⁶¹ AIMS (The Aspic Intervention Mortality Study) reported similar results with an estimated reduction in mortality at 57% in one year.¹⁶²

It should be noted that for those over age 80 no data exist to establish whether thrombolysis will indeed result in decreased myocardial infarct mortality or increased mortality due to higher frequency of intracranial haemorrhage. The few studies done which included patients over age 75 have reported no reduction in mortality with acute myocardial infarction treated with intravenous streptokinase. It may be reasonable not to treat patients aged 75 or older with intravenous streptokinase especially women and those who have a history of diabetes mellitus or systemic hypertension.¹⁶³

Under the age of 75, while the use of thrombolysis cannot be recommended in all elderly with acute myocardial infarction, careful screening should help to minimize the complications of therapy and identify those individuals most likely to benefit from it. However, despite the lack of conclusive thrombolytic data in patients over age 75, transmural anterior wall myocardial infarction, recurrent or subsequent myocardial infarctions may be considered for thrombolysis as this age group has a poor prognosis with increased mortality.¹⁶⁴

In the light of the current lack of sufficient data to identify precisely those individuals for whom thrombolysis is indicated, up to what stage after their infarcts and with which agent, a pragmatic approach has to be adopted within each department since so many well-designed and extensive trials are still in progress, policies must be reviewed frequently within departments, to take account of the rapidly expanding knowledge. The debate about these issues should not be allowed to distract attention from important practicalities of implementing thrombolysis in clinical practice. Once the decision is made to institute active treatment of myocardial infarction in a department, an operational policy must be drawn up identifying inclusion and exclusion criteria as to who should make the decision and what thrombolysis will be administered; until the door to needle time is minimized other issues probably represent the icing on the cake.

Use of angiotensin converting enzyme (ACE) inhibitors in the elderly

In young patients ACE inhibitors are now well established in the treatment of hypertension and heart failure, but how relevant are ACE inhibitors in this group, particularly with regard to their efficacy and safety? They act on the renin/angiotensin/aldosterone axis, and the plasma levels of all three are reduced with advancing age.¹⁶⁵ Therefore, it has been argued that efficacy of ACE inhibitors would be comparatively blunted in elderly patients.

There are wide differences in individual response to ACE inhibitors and plasma renin has not always proved an accurate predictor of this response,¹⁶⁶ particularly with regard to pre-treatment plasma renin levels and reduction of blood pressure.

The fall in blood pressure after administration of ACE inhibitors in salt replete normals and hypertensives with low levels of plasma renin has undermined the view that ACE inhibitors act solely by preventing the formation of circulating angiotensin. As ACE inhibitors also lower blood pressure after bilateral nephrectomy there must be an alternative mechanism of action.¹⁶⁷ Several recent investigations, largely conducted in animals, have shown that, in addition to circulating renin and angiotensin, there is endogenous renin and angiotensin in many tissues, such as brain, heart, vascular tissue and kidney.¹⁶⁸ There is also additional evidence that renin and ACE are not the only tissue enzymes that convert angiotensinogen to angiotensin, tissue plasminogen activators calhepsin and elastase also have converting enzyme and angiotensinogenase activity.¹⁶⁸ Despite uncertainty about the mechanism, it is now apparent that ACE inhibitors do lower blood pressure and are well tolerated in the elderly.^{170–171} Possibly the long-term effects of an ACE inhibitor on arterial pressure are to some extent independent of the renin–angiotensin system. Whether the antihypertensive effects parallel the activity of the intracellular renin–angiotensin system in vascular smooth muscle needs further investigation.

With regard to chronic heart failure ACE inhibitors produce symptomatic and haemodynamic improvement and may improve the life of severely affected patients. They are amongst the drugs which can reverse cardiac hypertrophy¹⁷³ and have been shown to decrease the incidence of cardiac arrhythmias¹⁷⁴ which are more frequent in elderly patients. They have also been claimed to increase blood flow to target organs such as kidney, brain and heart, which already have an age-related decreased blood flow. This may produce a greater improvement in the patient's quality of life than with drugs such as propranolol and methyl dopa.¹⁷⁵

Caution must, however, be taken in the use of these drugs in the aged. Pharmacokinetic studies

have identified changes in drug handling that appear to be directly or indirectly related to age. These changes include a reduction in the rate of biotransformation and clearance of ACE inhibitors in older subjects and a higher plasma concentration,¹⁷⁶ although there is no difference in sensitivity between young and old.¹⁷⁷ In addition, adverse reactions during treatment in the elderly may be slightly more frequent than in younger patients.

Serious complications do occur; severe hypotension one or two hours after the first dose of captopril has been widely recognized.¹⁷⁸ It is postural and usually responds to lying down although sometimes intravenous fluid is necessary. After the first dose of enalapril hypotension may be delayed for 6–8 hours and may last 24–48 hours.¹⁷⁹ Cerebral circulation in the elderly may be more susceptible to systemic hypotension and ageing is accompanied by the loss of homeostatic mechanisms. Therefore hypotension may be accompanied by serious cerebral ischaemia. In practice it is usually possible to predict those patients most susceptible to hypotension, patients on high doses of diuretics and hyponatraemic patients. These patients will be most safely started on ACE inhibitors in hospital.

In addition to this well-known problem numerous reports have shown that ACE inhibitors can cause a reversible deterioration in renal function, the cause of which is more complex. The majority of susceptible hypertensive patients have been shown to have bilateral renal artery stenosis, renal disease or a solitary kidney but patients with impaired renal function in whom stenosis of the main renal artery has been excluded by angiography may also be at risk, presumably due to intra-renal artery disease. This observation lends indirect support to Bender's proposal that renal artery impairment could be a consequence of disruption of glomerular autoregulation.^{179a} Whether the adverse reactions are related to the drugs themselves or to the general characteristics of elderly hypertensives has not been established. Likewise, whether or not the hypotensive action of converting enzyme inhibitors is age-related is as yet unknown.

On the basis of available data it may be predicted that adverse effects such as hypotension and exacerbation of pre-existing renal failure could pose problems in the elderly. Empirically, however, this class of drugs seems to have a useful role but it would seem good clinical practice to start with a very low dose and carry out a renal function test within a week of starting treatment or changing doses.

Stroke rehabilitation

The WHO recently reviewed stroke prevention, diagnosis and therapy.¹⁸⁰ The high cost of rehabilitation necessitates selection of patients who will recover satisfactorily only through intensive rehabilitation, recover without rehabilitation or recover poor function irrespective of input. The relative contributions of spontaneous and assisted recovery have 'yet to be satisfactorily determined'. Much work has been done in defining factors which determine prognosis and outcome.

Measurement of the manifestations of stroke at the levels of impairment, disability and handicap is crucial to progress in rehabilitation.¹⁸¹ Functional disability is most relevant to the patient in terms of independence, and numerous assessment scales exist and continue to be developed. The Barthel Index¹⁸² has been validated, and is amenable to statistical manipulation. It has been widely used and has been proposed as the standard for both clinical and research use.¹⁸³ Modifications have been suggested but not generally adopted. Criticisms of lack of sensitivity¹⁸¹ are balanced by ease of use.

In a review of 33 selected studies,¹⁸⁴ adverse predictors of functional outcome were concluded to be previous stroke, older age, urinary and bowel incontinence. Two general approaches were identified – single factor correlation, and multiple regression analysis. Sixty three percent of studies measured function with methods of no reported reliability or validity.

Urinary incontinence is an independent prognostic indicator,¹⁸⁵ only 3% of 362 patients continent on admission dying in the first month compared to 22% of those who were occasionally incontinent and 46% of catheterized patients. Eighty five percent of patients with severe hemiparesis continent on admission were home at 6 months compared to 33% of those incontinent. Prognosis may be enhanced through successful treatment of incontinence, perhaps by improving motivation and self-respect. Despite the many causes of incontinence, lack of awareness due to apathy and urgency of micturition due to bladder instability are the predominant problems,¹⁸⁶ which may be managed using incontinence charts, bladder training and where appropriate drugs.

Arm function alone is as good a predictor of discharge within the first month as a multivariate model,¹⁸⁷ multivariate models improving prognostication of death by 6 months to only 64% accuracy, irrelevant to clinical decisions. Initial Barthel Index score is a good predictor of functional outcome but arm and leg paresis are almost as accurate.¹⁸⁸ Sequential sitting balance is also highly correlated with Barthel Index.¹⁸⁹ Improvement in motor deficit occurs mainly in the first 48

hours after stroke, with recovery over the following month predominantly in those with milder deficit.¹⁹⁰

What after rehabilitation? One hundred and twenty-nine of 234 patients admitted from home in Sweden¹⁹¹ were discharged to independent living (including 53% judged as severe strokes), 88% remaining in their own homes at 12 months. In Chicago,¹⁹² 79% of 432 selected patients were discharged home and 85% remained at home after 3 months. Walking and transfers improved although overall functional ratings decreased slightly between discharge and follow-up. Crude outcome measures are also revealing. Discharge to long-term care in Montreal¹⁹³ was up to eight times more likely from teaching hospitals as from teaching affiliated hospitals. Time to outcome was shorter in the former perhaps indicating pressure for early discharge. Curiously the reduction in death rate approximates on superficial inspection to the increased rate of discharge to long-term care.

In an ongoing community based study of first strokes¹⁹⁴ which has provided a model for other studies,¹⁹⁵ 221 of 487 patients¹⁹⁶ were managed in the community, of whom 23% had severe strokes and 43% were living alone. In the same region, in the month following stroke less than 40% saw a physiotherapist and less than 30% saw an occupational therapist.¹⁹⁷ The incidence of 'non-stroke' lesions (e.g. subdural haematoma) was 1.5%,¹⁹⁸ and only 1 of 77 progressive or fluctuating strokes. Correct diagnosis is clearly essential in targeting rehabilitation.

The 'Bobath concept' of rehabilitation is based on restoration of normal tone and automatic postural control using a variety of inhibitory and facilitating reflexes, and improving voluntary control of movement by reinforcing sensory input and feedback. It has gained widespread adherence in the UK. The techniques have been refined empirically¹⁹⁹ and recent emphasis has shifted from static postures to reflex inhibiting movement patterns, with the use of 'key points' of control, in particular the trunk, to reduce spasticity more generally. A motor learning model,²⁰⁰ emphasizes learning through practice and repetition. Although justified on neurophysiological principles, these and other similar approaches are essentially empirical. Sensorimotor Integrative Treatment in occupational therapy has been compared with functional treatment, which uses adaptive and compensatory approaches.²⁰¹ Using sensorimotor tests sensitive to the outcome aims, for example, imitation and sequencing of postures, no differences were identified. Pragmatic and eclectic approaches are used in practice, particularly in the elderly, where rapid mobilization is paramount.

Biomechanical methods remain unproven. Spinal cord stimulation using implanted electrodes has been reassessed in stroke,²⁰² with apparent im-

provement in motor performance following 5–7 days of stimulation, disappearing 24 h after cessation and reappearing on resumption. Only sensory impaired patients benefitted. Functional electrical stimulation has aroused renewed interest.²⁰³ In an uncontrolled trial of initiation of ambulation using 6 channel stimulation in combination with conventional rehabilitation, gait indices improved in those patients who were otherwise able to walk only with support some months after stroke. The clinical significance remains to be demonstrated.

The debate over the value of specialized stroke rehabilitation continues.^{204,205} Specialized stroke units are not yet recommended.²⁰⁶ Optimal duration and intensity of rehabilitation are not yet established, but at present as little as 5% of patients' waking hours are spent in therapy.²⁰⁷ Additional weekend physiotherapy may hasten favourable outcome.²⁰⁷

Geriatric day hospital

Since the first purpose built geriatric day hospital opened in Oxford in 1958, over 350 have been opened, and Department of Health policy recommends a norm of 2 places per 1000 population over age 65.

A norm of near 0.5 places was suggested due to consistently low ($\leq 63\%$) occupancy in the early years of one unit.²⁰⁹ Discharges and new patient attendances increased fivefold with reduced attendances per patient keeping the occupancy rate almost stable, probably due to introduction of formal medical involvement and multidisciplinary management. Sixty percent occupancy was also observed²¹⁰ in another region where 48% of patients were referred primarily for social care.

General practitioner controlled day hospitals in community hospitals are a feature of the Oxfordshire region. Thirty three percent of referrals are for social reasons alone compared to 2% for consultant-led day hospitals.²¹¹ Indices of patient turnover and therapeutic activity were significantly higher in the District General Hospital units.

The objectives of day hospitals²¹² are medical and nursing care, rehabilitation, relief of carers, and social. One study found these objectives achieved in only one third of patients.²¹³ A joint finance scheme between Health and Local authorities²¹⁴ allowed cost comparison of a specialist day care setting for the 'frail elderly' (£25.25 to £28.45 daily) with a social centre (£7 to £14.40) and a day hospital (£38.90 to £41.40). The effectiveness of all three in meeting objectives such as relieving carer stress and providing basic essential services (e.g. bathing) was considered equivalent for a large overlap group of patients. Further evaluation of the cost effectiveness of day hospital

care is desirable.²¹⁵ Available evidence suggests attendance for treatment up to twice weekly provides value for money.²¹⁶

Screening the elderly

Interest in screening the elderly has been revived with the introduction of the new general practitioner contract which requires that patients over 75 years of age must be visited annually by a member of the practice. This requirement poses a number of questions: who should be screened?; what should they be screened for?; how can this best be achieved in practice? and what benefits does it achieve?

Health screening the elderly is not a new idea – the concept was introduced 35 years ago with the Rutherglen project.²¹⁷ In the 1960s a study of elderly patients in general practice identified a large number of unknown medical and social needs and suggested that regular screening, possibly by a health visitor, may be beneficial.²¹⁸ Several subsequent papers have described similar surveys.^{219–221}

These studies have been conducted by identifying the total over-75 population from age–sex registers. However, it is well recognized that up to 90% of elderly people will consult their general practitioner at least once a year. In fact, on average, they consult 6.5 times a year.²²² Is the group who do not see their doctor a high risk group worth targeting? Evidence would not support this view. A postal questionnaire of elderly consulters and non-consulters found the latter group to have less sociomedical problems.²²³

If screening is to be carried out on patients when they consult their doctor, it requires asking about problems other than that for which the patient is seeking advice. This may not be welcomed by the patient. Several authors make the point that screening the elderly should include functional assessment.^{224–226} However, a short consultation in a surgery may not be the optimum setting for detecting functional problems. Certainly within a hospital setting it has been shown that doctors rate poorly in functional assessment of the elderly.²²⁷

What, if any, specific disorders should be screened for? Opinion on this varies. A recent article from the USA²²⁸ suggested that screening the elderly should include a full history, examination including sigmoidoscopy and cervical smear, and a battery of tests including biochemical profile, full blood count, thyroid function, stool occult blood testing and mammography. Urinalysis, electrocardiography and CT head scanning could possibly be included. By contrast, a recently reported survey from Winchester²²¹ used only a disability rating scale administered by volunteers.

One of the criteria which makes any screening test justifiable is whether it has a high sensitivity and specificity.²²⁹ Urine testing for infection using a

leucocyte esterase and nitrite stick has been shown to be sensitive and specific.²³⁰ However, it must be shown that detecting asymptomatic urine infection in this age group is of benefit, and as yet this is unproven.²³¹

In psychogeriatric practice, screening for syphilis serology has become routine. Is this justified? A large study of psychogeriatric patients²³² showed that, out of 800 patients, only 25 had positive serology. Furthermore, when referred to venereologists, no treatment was advised in any of the cases. The authors point out that there have been no long-term follow-up studies of elderly mentally ill patients after receipt of anti-syphilitic treatment to guide clinicians on whether screening is useful in this age group.

The abolition of free eye testing in 1989 has raised the question of whether the potential for picking up undiagnosed glaucoma and other potentially treatable ophthalmic disorders in the elderly has diminished.²³³ Certainly there has been a substantial fall in the number of high-street eye tests carried out. It is known that 75% of new cases of open-angle glaucoma referred to hospital are detected by their optician, so it seems likely that new cases may now be missed. However, another author points out that the accuracy of tonometry and ophthalmoscopy in the diagnosis of glaucoma is not yet well established.²³⁴ Thus, widespread screening for the disease may not be cost-effective or beneficial.

In planning any screening programme the logistics of who should carry out the assessment need to be considered. Health visitors have been used in some studies^{219,220} but if this were to be a widespread practice, their numbers would have to be increased substantially. Untrained volunteers have been used to administer questionnaires,²²¹ but it could be argued that nursing or medical personnel may detect useful clues from patients, such as appearance and gait, which a lay person would not.

It may be argued that, as yet, we have no good evidence to support the value of screening programmes in the elderly.²²⁶ Most studies have shown that screening results in an increased use of hospital and social services,^{219–221} though total days spent in hospital are reduced, perhaps implying that detecting and treating problems early saves time in the long run. One study²²¹ suggested that less time was spent in institutional care in the screened group. However, the resultant increased requirement for community social services will require improved resources.

In conclusion, screening the elderly for a battery of specific medical problems is as yet of unproven benefit. However, assessing their functional performance when they consult for other reasons may be useful if solutions to the identified problems can be provided.

Elder abuse

In 1988 the British Geriatrics Society held a conference to discuss the problem of elder abuse in the UK. It was well attended and widely reported.²³⁵ 'Granny battering' was first described in this country in 1975,²³⁶ the same year that a large survey was reported of carers in the community which concluded that most 'carers support elderly dependents at great cost to themselves and with inadequate support from community services'.²³⁷ Nearly 10 years later, a social worker provided a descriptive account of elder abuse from a social case-work view,²³⁸ but there has been no systematic prevalence study of abuse in this country until recently.²³⁹

There is greater awareness of the problems of elder abuse in North America. The National Committee for the prevention of Elder Abuse was formed in the USA in 1986 and the *Journal of Elder Abuse and Neglect* is its official publication.²⁴⁰ In 1988 the Council on Scientific Affairs recommended that the American Medical Association should establish a multidisciplinary task force to develop approaches to intervention and prevention of elder abuse, to coordinate 'mutually supportive constituencies' and develop diagnostic and treatment guidelines.²⁴¹ All of these are much needed in the UK.²⁴²

Research on abuse has been hampered by the lack of precise and accepted definitions.²⁴³ Concepts of abuse vary. It is anomalous that with their heightened interest in elder abuse American doctors are increasingly tying their elderly down. If you are over 75 and admitted to hospital in America you have a 1 in 5 chance of being tied down by your wrist, waist and chest.²⁴⁴ Americans are critical of our readiness to use psychotropic medication to control unacceptable behaviour. Using definitions of physical violence, verbal aggression and neglect, large scale studies in America have shown a prevalence rate for overall maltreatment of 32 elderly persons per thousand in the community.²⁴⁵

State legislation in America enforcing notification of suspected elder abuse has allowed for easier identification of cases, and a case-controlled study suggests that the abuser is powerless and dependent on the elder for emotional or financial support, the victims are not necessarily more disabled, and the abuser is psychiatrically disturbed and misuses drugs and alcohol.²⁴⁶ Carers generally use more psychotropic drugs than the general population or other elderly people but this depends on their own characteristics rather than those of the dependent.²⁴⁷ The quality of the premorbid relationship is important in caring for the elderly mentally infirm at home²⁴⁸ and has been shown to be an important risk factor in domestic abuse of patients receiving geriatric inpatient respite care in this country.²³⁹

There are many assessment instruments for the

screening and detection of elder abuse.²⁴⁹ Some of the screening techniques used in the United States may be usefully applied over here.²⁵⁰ Once abuse is identified, the research suggests shifting the emphasis from the abused to the abuser with psychological counselling and support groups. There are no systematic procedures or uniform national guidelines for social workers to follow in cases of suspected elder abuse in this country, although the British Association of Social Workers, along with Age Concern, the British Geriatrics Society and the police have recently published a pamphlet on abuse intended for health and social services staff.²⁵¹ They have no right of access to the home, no duty to intervene and no powers to remove a person to a place of safety or summon parties to a case conference. Recommendations to rectify this and proposed guidelines on how to manage suspected cases have been made.²⁵²

Abuse should be prevented whenever possible, and now that risk factors for abuse are known, proposed caregivers should be screened before decisions to take up the burden of caring for a dependent are made. Guidelines are available for this purpose.²⁵² No-one, asked the question 'can she be left alone now?' should reply without very careful consideration and referral to a social worker for assessment of the situation.

Carers needs are by now well known.²⁵⁴ Respite care is frequently mentioned but may not be the panacea it is thought to be. A controlled study of respite care for the carers of those suffering from Alzheimer's disease have shown no reduction in caregiver burden or improvement in caregiver mental health although satisfaction with the service was high.²⁵⁵ Respite, combined with counselling and guidance on coping techniques has been shown to reduce distress in carers²⁵⁶ as has emotional support alone.²⁵⁷ Carers have set out what needs to be done in the 'Carers Charter'²⁵⁸ and a book has been produced for carers and professionals working with them by the King's Fund Informal Caring Support Unit. It details a range of projects to gain recognition for carers and improve community services, and contains information on most self-help and voluntary organizations.²⁵⁹

Audit in geriatric medicine

One of the widely accepted, yet still controversial, aspects of the recent Government White Paper 'Working for Patients'²⁶⁰ was the emphasis on the importance of medical audit. Medical audit is a mechanism for assessing and improving the quality of patient care, enhancing medical education and identifying ways of improving the efficiency of clinical care. A cycle of audit has been described whereby practice is observed, standard of practice is

defined, and the observed practice is compared. Changes are then implemented and the practice is reobserved.²⁶¹ To be effective, audit must be adequately organized and resourced both at regional and local level.²⁶²

But what of audit in geriatric medicine? The commonest method is case note review and guidelines for this have been issued by The Royal College of Physicians.²⁶³ However, in good geriatric medical practice there are aspects of patient care which are not covered by the suggested format. Attempts have been made to improve audit by producing a pro-forma geared specifically to elderly patients.²⁶⁴ This included analysing the documentation by medical staff of a detailed functional history, level of continence, memory impairment and social history, and so on.

The International Classification of Diseases has been used in audit, but in the elderly it has limitations because it has no classification for health problems such as immobility, falling or incontinence. These are multifactorial, and thus a system incorporating the concepts of impairment, disability and handicap may be more useful.²⁶⁵ This should in turn enable us to measure more relevant outcome parameters, particularly in the field of rehabilitation. However, any audit must allow for the fact that the data is a static representation of a dynamic process.²⁶⁶

Outpatient clinics are a standard practice in other specialties. They may not be as appropriate in the elderly, but there has been little objective evaluation. A study from Nottingham²⁶⁷ of geriatric outpatient clinics showed that management was altered in only 37% of patients, and a new problem or diagnosis made in only 16%. They suggest that review at home by a geriatric liaison nurse might be more appropriate in some cases, particularly for recent ward discharges.

In recent years it has been emphasized that a large proportion of geriatric beds should be on a district general hospital site, but the impact of this has been little studied. In Canterbury an assessment ward on a district general hospital site was newly established without changing total bed or staff numbers.²⁶⁸ Audit showed that there was a 33% increase in throughput with more active investigation and treatment and a tendency to shorter length of stay, and an increase in transfers from other disciplines with a shorter time interval to transfer.

Questions remain as to whether audit necessarily changes behaviour for the better. Feedback provided for clinicians may be passive or active. Passive feedback such as written statistical information without suggestions for improvement produces minimal change. Active feedback, involving discussions amongst clinicians, may be more effective.^{261,269} An example of this is a general practice study on digoxin prescribing.²⁷⁰ Audit of all patients on

digoxin in a number of practices was made and then a protocol on optimum use and monitoring of the drug was sent to all the prescribers. A year later the audit was repeated. Improvements occurred only in the practices in which the doctors were involved in conducting the study and the authors concluded that 'audit may change only the auditors'.

Audit in geriatric medicine is well-known. Since the early sixties services for the elderly and mentally ill have been reviewed by the Health Advisory Service and the common practise is for a multi-disciplinary team to conduct detailed visits to health districts. Teams identify problems such as lack of privacy, homely surroundings and personal possessions in long stay wards, but often the advice is ignored, despite the fact that many of the recommendations require no extra funds.²⁷¹

Thus it remains unclear whether long term benefit will come from clinical audit even if the optimum format is established.

Organization of geriatric services

No two departments in the UK operate in the same way. Some operate a traditional secondary referral system; others run an acute age-related service.²⁷² Some are fully integrated with general medicine sharing junior staff,²⁷³ others are needs related.²⁷⁴ All have catchment areas, and the local provisions of hospital and community services can change dramatically across a single boundary.

Consultants in geriatric medicine agree that the elderly benefit from access to modern technology.²⁷⁵ But individual proponents argue fiercely for and against the merits of different systems.²⁷²⁻²⁷⁴ Despite the bewildering variety, most systems work well, and flexibility to local needs is an advantage. Within units, division of wards by function into acute, rehabilitation and long stay improves efficiency.²⁷⁶

Research comparing geriatric services is therefore more easily conducted by examining component parts of a service.

Intrahospital referrals and geriatric medicine

The traditional referral pattern focuses on the individual patient. One consultant team refers to another according to the patient's ward, home address, admitting specialty or personal preferences. There are no studies to evaluate how well this works for the elderly patient with multisystem problems, the expectations of the two teams may not match. The consultant in geriatric medicine may be asked to 'take away' patients perceived as inappropriately placed. This may reflect ageist attitudes but 'bed blocking' is not unique to the elderly and may not be as large a problem as previously thought.²⁷⁷ Prolonged stays in hospital²⁷⁸ and readmissions^{279,280}

frequently reflect a patient's underlying diagnosis. Discharge planning with appropriate social support is valuable in reducing unnecessary time spent in hospital.^{281,282}

Intraspecialty liaison is effective. Following the original combined geriatric orthopaedic unit²⁸³ many others have been set up^{284,285} and variations have developed.²⁸⁶ Prospective studies show that patients benefit from the department of geriatric medicine's rehabilitative and medical skills and skilled interventions in shortened length of stay and increased likelihood of return to the patient's own home.²⁸⁴

Formal links between geriatric medicine and other specialties are less common. Isolated examples exist and there is a geriatric surgical unit whose most tangible benefit is low mortality from gastrointestinal haemorrhage.²⁸⁷ Ambivalent relationships may exist between departments of general and geriatric medicine, but they can work well together without integration.²⁸⁸

There are few UK studies of co-operation between disciplines. Studies in North America have focused on multidisciplinary geriatric consultation teams which operate within hospitals and are open to referrals from all specialties.²⁸⁹⁻²⁹⁴ The most consistent benefits are prolonged survival, reduced annual medical care costs, and reduced use of acute hospital facilities.²⁹⁵ Inconclusive results may be due to different methods of evaluation, different service team composition or selection of patients.²⁹⁶ Multi-institutional randomized controlled trials could resolve this issue;²⁹⁷ however, it seems that the efforts of geriatric consultation teams are best targeted towards the frail elderly (those with pre-existing disability prior to intercurrent illness). Furthermore, the teams must be empowered to implement and follow up the rehabilitation programme devised for an individual patient.²⁹⁵

Domiciliary visits

Domiciliary visits, whereby a general practitioner (GP) invites a consultant into a patient's home to give a specialist opinion, predate the NHS. Since 1948 the annual number has trebled and in 1988 the cost was estimated at £20 million per year.^{298,299} Analysis of domiciliary visits reveals a high rate in geriatric medicine – although the contribution from other specialties is significant and sometimes unexpected.³⁰⁰ More surprising, however, is the tenfold variation between consultants in the same specialty and even within the same region.

Consultants receive a special fee from the NHS per visit. The 'gentlemen's agreement' between consultant and GP is less well defined; 20% of GPs never use the service: of those that do, only 5% think all visits should be combined and 20% believe

there is no need to accompany the consultant at all.²⁹⁸

Both parties may request inappropriate visits. Alleged abuses include requesting visits to facilitate admission; using visits to screen all referrals to the geriatric department; and financial gain. Conversely, a domiciliary visit can provide a consultant opinion; give valuable information about a patient in the context of their home and family; promote education and understanding between hospital and community doctors,²⁹⁹ and prevent unnecessary and uneconomic admissions to hospital.³⁰²

Domiciliary visits still await proper evaluation. Without this, managers and budget-holding general practitioners needing to make financial savings may decide to dispense with them altogether. Perhaps standardization would maximize use of a geriatricians' time. Alternatively, more innovative methods of community assessment may supersede them such as outpatient clinics in GPs surgeries³⁰³ or residential settings³⁰⁴ or even mobile clinics.³⁰⁵

Continuing care

Long stay care of the chronic aged sick once formed the major workload of the geriatrician.³⁰⁶ Now its role is smaller, but still important. From April 1991, District Health Authorities will be free to commission long stay care from private nursing homes, and there is concern that if there is no consultant responsibility the standards of care will fall.³⁰⁷

Patients must be properly assessed prior to admission to a long stay unit;³⁰⁸ the expensive facilities, are, after all, for life. Consultants in geriatric medicine, because of their special expertise and skills, should fulfil this commitment. Yet whose responsibility should it be for maintaining the standards of institutional care?

Studies have tried to compare nursing homes

with long stay wards.³⁰⁹ Elsewhere, model longstay units have been created within the NHS.³¹⁰ One major study, set up by the Department of Health, involves three experimental nursing homes.^{311–315} In the experimental nursing homes, more residents were engaged in meaningful activities and verbal interactions during the day than in comparable long stay wards. But the price to be paid was a lowered standard of rehabilitation: 25% of the control group were discharged.

Privatized long term care may not be better or cheaper than the NHS. Better premises do not necessarily mean better quality of care³¹⁶ and most old people are not 'value for money'.³¹⁷ The duration of terminal dependency has been increasing for the last 30 years,³¹⁸ and unless the rehabilitation is continued the UK may not maintain one of the lowest rates of institutionalization of elderly people.

The future

At the time of writing the Hospital Service is being reorganized. In every health district administrative sections representing the providers and the purchasers are being established. Chinese walls are being set up between the two groups wherein one side is not expected to know what the other side is thinking. These methods may work in business but time alone will tell whether they will bring to the next generation of elderly a better service than is now being provided.

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