CARE OF THE ELDERLY

— the ageing process in a nutshell

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Ageing affects most, if not all, of the bodies' systems and organs. The third part of this month's special feature gives an overview of the ageing process, looking at some of the main changes associated with growing old

The face of an elderly patient

Is ageing a natural state to be borne, a disease to be cured where possible or a natural event that can be considered as a disease where steps may be taken to lessen its adverse effects?[1] The debate remains unresolved, although the latter view seems to be the most relevant to practice today. Whatever the best description of ageing, it is a fact that ageing affects most, if not all, of the bodies organs and systems. Importantly, ageing does not necessarily lead to impairment, because the body often successfully compensates for the associated changes. Set out below is an overview of the main systems affected by ageing. It is, however, worth remembering that ageing processes interact with each other and should not be considered in isolation.

HEART

Most heart cells (myocytes) lack the capacity for regeneration (ie, they are terminally differentiated). Myocytes lost through ageing are therefore not replaced, leading to hypertrophy of the remaining cells. This causes characteristic changes in the cardiac architecture resulting in stiffer and less compliant ventricles. This is not a problem at rest, because compensatory reduced early diastolic filling allows cardiac output to be maintained. However at times of reduced ventricular loading (eg, dehydration, blood loss, diuretic or vasodilator use) the compensation may not be enough and the change in cardiac architecture can become symptomatic.

Loss of the pacemaker cells in the sinoatrial node and the calcification of the conduction system contribute to the common rhythm disturbances seen in old age (eg, atrial fibrillation, sick sinus syndrome and heart block). As arterial walls age, collagen and elastin crosslink. This reduces compliance and contributes to the development of isolated systolic hypertension and left ventricular hypertrophy.[2]

Blood levels of catecholamines also rise with age. This desensitises the heart to noradrenergic stimulation and thereby limits the maximum achievable heart rate. Sodium conservation is decreased and baroreceptor response changed, alterations which can predispose individuals to orthostatic and postprandial hypotension. There are age-related changes in the response of the endothelium to physical and chemical stimuli that limit its vasodilatory properties.

ENDOCRINE CHANGES

Changes in hormonal levels play a large part in the ageing process.[1,3] For women, the loss of oestrogen at the menopause is accompanied by long-term effects of increased cardiovascular disease, loss of bone mass and cognitive impairment. In men, testosterone decline is less universal but has been associated with muscle weakness, anaemia, lower bone mass and mood disturbances. The decrease in oestrogen and testosterone levels results from a reduction in their universal precursor in peripheral tissues — dehydroepiandrosterone [DHEA] and its sulphate [DHEAS] — and has been termed the "adrenopause". The use of DHEA supplements is controversial but has been linked with wide-ranging reversal of the signs of ageing.

The synthesis of other hormones is also affected. Levels of growth hormone (GH) and insulin-like growth factor I (IGF-I) reduce with age. This (termed the "somatopause") is associated with a decrease in muscle and bone mass and an increase in fat mass. Insulin secretion is

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impaired with age (reducing to 50 per cent of normal levels in those aged over 80 years), affecting both the early and late responses to glucose load. Peripheral insulin resistance is increased with age and combined with poor diet, physical inactivity (eg, as a result of decreased skeletal muscle mass and frailty) and decreased lean body mass, puts the elderly at high-risk of all the complications associated with diabetes.

Thyroid dysfunction is largely caused by autoimmunity and, despite being more common in the old, should not really be considered a part of ageing.

Skeletal muscle

Skeletal muscle is lost with increasing age.[4] There is muscle hypotrophy as a result of a decline in muscle cell size and number. There may also be changes in the quality and strength of the contractions achievable. Overall this adds up to an impairment of motor function, which weakens and slows movement. This puts the elderly at great risk of falls and injury and is often an early indication of increasing frailty. It is a major cause of morbidity and mortality in elderly people.

Bones and joints

Decreased skeletal mass (ie, bone mineral density) is seen with ageing and, in women, is accelerated for a while after menopause.[2] There are age related declines in the transport and absorption of calcium and a reduction in vitamin D activity secondary to decreasing renal function. Elderly people also tend to have less calcium in their diet, which can compound the situation. The balance of osteoclast and osteoblast activity is disturbed, with osteoclast activity predominating. Osteoarthritis is also seen with increasing age as loss of chondrocytes and proteoglycans leads to malformation of the ends of bones. These bony changes can cause the loss of normal joint function which can only be remedied by joint replacement.

Care of the elderly networking groups

UK Clinical Pharmacy Association (UKCPA) Care of the Elderly Group (national coverage) and the London Specialist Pharmacy Services' Older Peoples Pharmacy Network (London, South East and Eastern Region) exist to promote networking and to offer continuing education. Membership of either group is open to pharmacists from all areas of practice via Derek Taylor (UKCPA — dataylor@tinyworld.co.uk) or Christine Masterson (London OPNet — christine.masterson@nwlh.nhs.uk).

Prosthetic hip joints: age-related changes to joints can make hip replacement surgery necessary

Central nervous system

The brain, like the heart, has limited capacity for repair after an insult.[5] Insults include age-related changes such as an accumulation of neurofibrillary tangles, senile plaques and large amounts of amyloid protein (intrinsic insults), changes that have been associated with dementias and cognitive impairment. Extrinsic insults (ie, secondary to other age-related changes) include vascular changes leading to hypertension and stoke.

Vision is affected by age. The light-sensitive compounds in the retina are constantly being replaced. Lysosomes break them down but some insoluble fragments persist and are stored in secondary lysosomes in the underlying epithelium. It is these stored fragments that contribute to macular degeneration and blindness. Changes in the lens also occur. Only the cells at the extreme edge of the lens divide so that the centre consists of some of the earliest embryonic tissue.[6] In order to remain transparent, the lens depends on anaerobic glycolysis and is under high osmotic stress because it is the "driest" tissue in the body. The lens is therefore susceptible to mechanical and metabolic insults which result in the loss of optical clarity that with time produces a cataract.

Regarding hearing, there is an age-related cumulative loss of both the hair cells of the cochlea and nerve fibres of the cochleal nerve, which produce the hearing loss associated with old age.

Immune system

Age-related changes to the immune system include an increase in the levels of early cytokines such as interleukin-6 (IL-6), IL-1b and tumour necrosis factor. Auto-antibody production also increases and there is a decrease in phagocytosis and the generation of freeradicals. This tends to blunt the response to infections and allows latent infections (eg, tuberculosis) to re-emerge.

Conclusion

Ageing is associated with many changes to the body. Compensatory mechanisms often exist, and so impairment does not always result, although some loss of function in some systems is almost inevitable.

References

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The details of one of the authors of the article on intervention monitoring (p201–2) of the May issue of *Hospital Pharmacist* were incorrect. Dr Walters was practitioner pharmacist for North Tees and Hartlepool NHS Trust at the time of writing, but is now employed by South Tees Hospitals Trust and Middlesbrough PCT.