GENERAL CARDIOLOGY

Heart disease in the elderly

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We are an aging population. It is estimated that 20% of people in Europe will be over 65 years of age in the year 2000. The proportion of the population over 80 years, the so-called "old old", is increasing most rapidly. Life expectancy at all ages is also increasing. At 65 years life expectancy ranges from 14.9 to 18.9 years and at 80 years from 6.9 to 9.1 years for men and women, respectively. Cardiovascular disease is the most frequent single cause of death in persons over 65 years of age[1], and most importantly it is responsible for considerable morbidity and a large burden of disability, particularly in the community.

Cardiovascular pathologies such as hypertension and cerebrovascular disease, and heart diseases such as coronary artery disease, arrhythmias, and heart failure, increase in incidence with increasing age.[w1] The aging process itself also effects the cardiovascular system. It is difficult to differentiate "normal" aging, which is inevitable, from age related pathology, which is potentially preventable or treatable.[w2] Age related changes are most likely to be seen in the "old old" who have escaped cardiovascular pathology earlier in life. This group demonstrates the dual processes, often interacting, of biological aging of the cardiovascular system and age related pathology. This combination modifies the pathophysiology of disease such that knowledge of that condition and treatment thereof, derived from studies in "young old" (65–75 years) are not readily applicable to the "old old". Older patients differ from "trial" patients of any age by virtue of their other comorbidities and multiple drug usage, which are invariably exclusion criteria for entering treatment studies.

Age related structural changes

Cardiac

- Increased left ventricular wall thickness, independent of any increase in blood pressure.[2] This is attributed to hypertrophy of individual myocytes with a progressive loss of myocyte numbers. There is also an accumulation of interstitial connective tissue and, in the "old old", accumulation of amyloid deposits.[w3]
- Increased fibrosis and calcification of the valves, particularly the mitral annulus and the aortic valve.[w3] [w4] Recently it has been shown that aortic sclerosis without outflow obstruction is not the benign condition once thought and is associated with significantly increased cardiovascular and total mortality.[w5]
- Loss of cells in the sinoatrial node. By 75 years of age, only 10% of the cells that were present at 20 years remain. There is loss of muscle cells and mild increases in fibrous tissue in the internodal tracts. The remainder of the conducting system is also affected but to a lesser extent.[w4] These changes occur in the absence of coronary artery disease.

Vascular

- Increased stiffness of peripheral and central arteries, caused by proliferation of collagen cross links, smooth muscle hypertrophy, calcification, and loss of elastic fibres.[w6]
- Increase in number of sites for lipid deposition cause endothelial changes that reduce laminar blood flow. These changes are independent of atherosclerosis.[w6]
- More diffuse coronary artery changes. The earliest changes usually appear in the left coronary artery during youth or adulthood, whereas the right and posterior coronary arteries do not usually become involved until after the age of 60 years.

Age related functional changes

Left ventricular systolic function

- In patients carefully screened to exclude coronary artery disease and hypertension, there is little change in left ventricular systolic function with increasing age, although cardiac output may decrease in parallel with a reduction in lean body mass.[1] [w7] The determinants of cardiac output which may be influenced by age include heart rate, preload and afterload, muscle performance, and neurohormonal regulation.
- Increases in heart rate in response to exercise or stress caused by non-cardiovascular illnesses, particularly infections, are attenuated with increasing age.[3] [w8] Stroke volume increases only by "moving up" the Frank Starling curve.[w9] [w10] Thus end diastolic volume increases. These age related changes in cardiac response to exercise are mimicked by β adrenergic blockade,[w11] but β adrenergic agonists do not reverse this aging process.[w12] The decline in exercise performance with age may additionally relate to peripheral factors, blood flow, and muscle mass rather than being solely the consequence of cardiac performance changes.

Diastolic function

- The rate and volume of early diastolic filling decrease with age.[1] [w13] The structural changes described above account for some

Features of a normal aging process[w2]

- Universal within the species
- Intrinsic to the individual
- Deleterious to survival
- Progressive and irreversible

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of this decline, but recent findings of partial reversibility with calcium channel blockers[4] or exogenous angiotensin II receptor antagonists[w14] illustrate the dynamic and therefore potentially reversible nature of the process.

- The aged heart requires atrial contraction to maintain adequate diastolic filling, so atrial fibrillation, so common in older people, has a disproportionate effect on cardiac function.[w7]
- Reduced ventricular compliance results in higher left ventricular diastolic pressures at rest and during exercise.[w8 w13] As a result, pulmonary and systemic venous congestion may occur in the presence of normal systolic function.[w15] With increased afterload on the left ventricle, left ventricular hypertrophy occurs, even in the absence of hypertension or aortic stenosis.[w16 w17] Diastolic dysfunction, at least in the early stages, may be a feature of normal aging. Later, however, it is a pathological process leading to significant left ventricular hypertrophy. At this stage coronary heart disease, hypertension or other pathology is probably involved.
- Age related decreases in capillary density and coronary reserve may cause myocardial ischaemia and thus further diastolic abnormalities in the absence of coronary atherosclerotic disease.[2 w18] Age associated decreases in the rate of maximal capacity of calcium sequestration by the sarcoplasmic reticulum and/or an age associated increase in net trans-sarcolemmal calcium influx may also contribute to diastolic ventricular abnormalities.[w19 w20]

Heart failure

Chronic heart failure is a disease of "old old" people, and unlike coronary artery disease, the incidence continues to rise with increasing age. Only 17% of people with heart failure are less than 65 years of age,[5] yet most of the interventional studies of the treatment of chronic heart failure have focused on this minority group and extrapolated the results to the older majority.[w21 w22] "Diastolic" heart failure is probably the primary haemodynamic dysfunction in the elderly. Among patients over 80 years of age with clinically defined heart failure, up to 70% have preserved systolic function,[w21] whereas probably less than 10% of patients below 60 years of age have preserved systolic function.[6] It is important to be aware of this high prevalence of diastolic dysfunction as it has implications for treatment. Over 75% of elderly patients with heart failure have hypertension and/or coronary artery disease, and patients with diastolic dysfunction may present with decompensated heart failure caused by uncontrolled blood pressure or progression of ischaemic heart disease.[w23]

Table 1 Evidence of benefit for symptoms and mortality for drugs used in heart failure in patients more than 75 years of age

	Benefit		
Drugs	*Yes*	*No*	*Unknown*
Diuretics	Symptoms	–	Mortality
Digoxin	Symptoms	Mortality	–
ACE inhibitors	Symptoms/Mortality	–	–
AIIAs	–	Mortality	Symptoms
β Blockers	–	–	Symptoms/mortality
Spironolactone	–	–	Symptoms/mortality

ACE, angiotensin converting enzyme; AIIAs, angiotensin II receptor antagonists.

Clinical presentation

The symptoms and signs of heart failure are similar in young and elderly people, but non-specific presentation is more common in the elderly.[1 w24] Community based studies have indicated that perhaps up to half of "old old" patients with activity limiting heart failure are undiagnosed and therefore untreated.[7] There is an expectation by old people themselves, by their relatives, and unfortunately by some of their doctors that many of the features of heart failure in older people are a result of normal aging—"what do you expect at his age?" Patients may present with confusion, depression, fatigue, weight loss, immobility or "social crisis".[w25] Patients with chronic heart failure caused by systolic dysfunction tend to present with gradual worsening of daytime symptoms and paroxysmal nocturnal dyspnoea, whereas those with diastolic dysfunction may present with a more abrupt onset of symptoms.

It is difficult to distinguish between systolic and diastolic heart failure clinically. Some patients may have a combination of both, especially in later stages of the disease. As in younger patients, a normal 12 lead ECG virtually excludes significant heart failure. All older patients should have access to echocardiography to aid diagnosis. This will allow diagnosis of causal factors such as valvar lesions, and provide an assessment of haemodynamic (systolic versus diastolic) function. Normal or preserved systolic function in heart failure has been assumed to imply diastolic dysfunction, but this is no longer adequate as we now have guidelines from the European Society of Cardiology giving clear echocardiographic criteria for diastolic heart failure—at least applicable to the "young old" patient.[8]

Management

Diuretics

The management of systolic heart failure in elderly patients, as in younger patients, involves the use of diuretics, vasodilators, and oxygen supplementation (table 1). Most elderly patients will require a loop diuretic because of the age related and heart failure mediated reduction in glomerular filtration rate. Thiazide diuretics are ineffective when the glomerular filtration rate is less than 30–40 ml/min. There is an age related decrease in total body potassium content, as predicted by a proportional reduction in lean body mass, but not in plasma potassium concentrations.[w26] Potassium retention is more of a problem than hypokalaemia in older patients, particularly with the combined use of angiotensin converting enzyme (ACE) inhibitors and potassium sparing diuretics or supplements. Additionally potassium sparing diuretics should be avoided in the elderly because of the increased risks of

Adverse effects of diuretics in older people

- Incontinence
- Urinary retention
- Hyponatraemia
- Hyperkalaemia

hyponatraemia and uraemia as well as hyperkalaemia. While metabolic adverse effects of diuretics should be appreciated the physical effects are invariably more important to the older patient. Thus for the patient whose mobility is impaired by arthritis, Parkinson's or even heart failure itself, being rendered incontinent by high dose loop diuretics does not aid compliance. Similarly acute retention in both men and women does not create a good doctor–patient relationship. It is best therefore to start with a low dose and titrate upwards gradually on the basis of effect on body weight, for example.

ACE inhibitors

ACE inhibitors have demonstrated haemodynamic, functional, and mortality benefits in heart failure patients.[w27 w28] Unfortunately they are often under prescribed to older patients or, if given, are administered at suboptimal doses.[5] First dose hypotension is not a particular problem in older patients since low dose initiation of ACE inhibitors, preferably with short acting captopril, has become standard practice. Renal function should be monitored closely following their introduction.[w29] Cough, which may be poorly described and confused with early paroxysmal nocturnal dyspnoea, is a frequently seen problem in elderly patients. In old age generalised atherosclerosis may increase the risk of renal artery stenosis, thus increasing the risk of renal failure precipitated by ACE inhibitors. The place of angiotensin II receptor antagonists in routine management of heart failure patients of any age remains problematic. The beneficial effect of losartan seen in ELITE I was borne out in ELITE II, but losartan was shown to be no better than captopril in this larger study. Both trials, however, demonstrated that older patients can successfully be recruited to intervention studies of the treatment of heart failure.[9 w30]

Inotropes

Data on the use of digoxin in systolic heart failure with normal sinus rhythm are equivocal,[w31] but there is some evidence of benefit for patients with severe heart failure; fewer clinical deteriorations, hospital admissions, and emergency visits have been reported with digoxin.[w32] Digoxin must be used with caution in the elderly because of its narrow therapeutic window,[w33] and it is advisable to monitor blood concentrations especially if circumstances change and the patient develops pre-renal uraemia (influenza, chest infection, dehydration, haematemesis). Other possible inotropes cannot be recommended for routine use in elderly patients at the present time.

Other pharmacological agents

The absence of older patients in the recent studies of β blockers and spironolactone in heart failure make it difficult to assess their benefits in older patients. It is likely, however, that the older heart failure patient will benefit but this requires confirmation. For patients intolerant of ACE inhibitors the usual practice has been to use a combination of hydralazine and isosorbide mononitrate.[w34 w35] While there are no direct comparisons of hydralazine, isosorbide mononitrate and angiotensin II receptor antagonists, it is likely that the latter will be better tolerated in older patients.

General measures

Bed rest is discouraged in elderly patients because the risk of thromboembolism and physical deconditioning far outweigh any advantages.[10] Compression stockings should be used and anticoagulation should be considered in the immobile patient with severe failure and/or atrial fibrillation to guard against the development of deep venous thrombosis and embolisation.[w36] Hopefully the period of anticoagulation should be limited until mobility is restored. The European Society of Cardiology guidelines stress the importance of general lifestyle measures which are probably of equal benefit in the older patient.[11] Fluid restriction is not usually necessary and may be dangerous, as many elderly patients have poor oral intake when ill. Salt restriction is often difficult as many elderly patients survive on convenience foods in which the salt content is high. The elderly also suffer from "cardiac cachexia"—loss of fat free mass.[w37] The mechanism is unclear, although fat absorption is impaired,[w38] but there is no evidence of gastrointestinal protein loss.[w39] Apoptosis in skeletal muscle is common in patients with heart failure.[w40] It is often not noticed until the oedema has subsided in response to diuretic treatment.

The social burden of heart failure on elderly patients is largely ignored. Many older patients live alone and the onset of heart failure drastically reduces independence. The provision of a home help, a shopper, aids or appliances may substantially improve quality of life and maintain patients in their own homes. These measures are as important as drug treatment of many older patients.

Support clinics

Studies have shown the effectiveness of nurse led, patient focused support clinics for heart failure patients by reducing hospital readmissions, improving compliance with medications, enhancing quality of life, and improving patient education.[w41–43] Our own simple studies of older unselected patients discharged from hospital demonstrate significant improvements in exercise capacity, quality of life, drug adherence, and a 60% reduction in readmissions in the three months after hospital discharge. A more intensive nurse led multidisciplinary intervention in the USA improved quality of life and reduced rehospitalisations of older selected patients with heart failure.[12] Attention to the delivery of support to older heart failure

patients may be more beneficial and cost effective than adherence to the latest drug usage guidelines.

"Diastolic" heart failure

The management of heart failure with preserved systolic function is not as clear as that of heart failure in which systolic function is impaired. Treatment objectives should be to improve ventricular relaxation and filling. Attention to aggravating factors (such as hypertension, atrial fibrillation, anaemia, and left ventricular outflow obstruction) is the first step in management. There are no trials of treatment of diastolic heart failure so drug recommendations can only be made on an empirical basis. Certainly patients benefit symptomatically from diuretics. However, hypovolaemia and reduced preload secondary to over vigorous diuresis should be avoided. Thus loop diuretics should be started low and monitored carefully. Digoxin is not indicated and may be harmful.[w33] Agents that may improve ventricular filling in diastole include β blockers, calcium channel blockers, and perhaps ACE inhibitors.[9] Calcium channel blockers have been shown to improve left ventricular diastolic filling, whether impairment is age associated, or caused by ischaemic heart disease or hypertension.[w13 w20 w44] Studies on efficacy in diastolic heart failure of β blockers and ACE inhibitors are awaited.[w45]

Prognosis

Heart failure caused by diastolic dysfunction has a better prognosis in terms of mortality than systolic heart failure.[13] The five year mortality rate with systolic heart failure is about 50%.[w46] In elderly patients and those with severe heart failure the mortality at one year is at least 30%. Older males have higher mortality than females and white men have a 10% greater risk of death than black men.[w47] These figures underscore the importance of secondary prevention strategies and the early detection and treatment of heart failure in older patients. Prognosis also depends on the presence of other cardiovascular comorbidities such as ischaemic heart disease, hypertension, and vascular complications of diabetes.[w48] Additionally older patients suffer multiple pathologies which have to be taken into account when planning management or assessing prognosis.

Congestive heart failure is the most common cause of hospital admission in elderly patients in the USA, with patients subject to frequent readmissions.[w49] Poor compliance with medication, particularly diuretics, is an important factor and has been reported in up to 50% of elderly patients with congestive heart failure.[w50]

Coronary artery disease

Coronary artery disease increases in incidence with aging. Age itself is an independent risk factor for coronary artery disease.[w51 w52] Sixty per cent of all deaths attributed to acute myocardial infarction are in patients over 75 years of age.[w53] A necropsy study of patients 90 years of age and over revealed that 70% of subjects had one or more coronary vessels occluded.[w54]

In recent years, coronary artery disease mortality has declined in elderly patients, but to a lesser extent than in younger patients. Risk factors are similar to those in younger patients and equally modifiable.

Myocardial infarction

Older patients may describe the typical central chest pain of myocardial infarction, but are as likely to present with dyspnoea without pain.[w55] As in congestive heart failure, they may also present non-specifically with confusion, syncope, vertigo, or epigastric pains.[w56 w57] In the Framingham study, 42% of myocardial infarctions were noted to be clinically silent (asymptomatic) or unrecognised in men aged 75–84 years compared with only 18% in men aged 45–54 years.[w58] The proportion of unrecognised myocardial infarctions was higher in women. Some studies have reported that up to 60% of myocardial infarctions may be unrecognised in the very old.[w59] Unrecognised myocardial infarction patients are more likely to be hypertensive, have diabetes, and smoke, and have a lower prevalence of preceding angina.[w60 w61]

Risk factors are similar in both sexes, but women have a less favourable psychosocial risk profile, including living alone, which adversely affects prognosis.[w62] Older people and women of all ages present later to hospital.[w63 w64] One study reported that patients over 80 years of age delayed more than 6.5 hours in calling paramedics, compared with a 3.9 hour delay in younger patients.[w65] Older patients are twice as likely as younger patients to have non-Q wave myocardial infarctions.[14] Up to 40% of elderly patients do not have typical ST elevation or Q waves on their ECG at presentation.[w65] Right ventricular infarction is also more frequent in older patients,[15] and mortality rates as high as 75% have been reported in this group.[w66]

Management

Unfortunately, elderly patients have been largely excluded from the randomised controlled trials of treatments for myocardial infarction. Pooled data from several large placebo controlled trials show that the absolute reduction in mortality is at least as great in older as in younger patients.[w67] It is no surprise that elderly patients are less likely to receive thrombolysis than younger patients given the differences in presentation. Contraindications are similar to those for younger patients, but elderly patients are more likely to experience adverse effects from treatment. There is an excess of eight haemorrhagic strokes per thousand in patients over 75 years given thrombolytics, and it may occur more frequently with recombinant tissue plasminogen activator (rt-PA) than streptokinase.[w68] The decision to administer thrombolysis and the choice of agent used must therefore depend on the overall assessment of risk versus the potential benefit.

Aspirin should be administered to older patients as for younger patients. In ISIS-2, the absolute benefit from aspirin was greatest in patients over 70 years of age.[w69] In the absence of contraindications, early use of β blockers reduces mortality post-myocardial infarction at all ages.[w70 w71] ACE inhibitors should be given to haemodynamically stable patients, particularly older patients, within 24 hours if there are any signs of heart failure, and possibly routinely in the presence of a large anterior myocardial infarction.[w72 w73] Other treatments including nitrates and oxygen should be administered as for younger patients. Smaller doses of morphine are recommended for elderly patients.

Evidence to support routine use of unfractionated heparin in acute myocardial infarction is lacking, but its benefit is clear in unstable angina. The low molecular weight heparin, enoxaparin, was superior to unfractionated heparin in reducing recurrent angina, myocardial infarction, and death in patients with unstable angina or non-Q wave infarction.[w70 w74]

In acute infarction the role of primary percutaneous transluminal coronary angioplasty (PTCA) in this age group has not been evaluated, apart from one small study in which mortality compared favourably between PTCA and streptokinase.[16] There are very little data available on the use of intracoronary stents in older people.

Prognosis

Hospital mortality for myocardial infarction patients over 70 years of age is at least three times that of younger patients.[w75 w76] Older patients are at high risk for major cardiovascular complications. Women have a higher crude mortality associated with myocardial infarction.[w77] Poor prognosis in this age group is multifactorial. Numerous studies have shown that the therapeutic approach in older patients is unjustifiably less aggressive than in younger patients and potentially beneficial drugs are underused.[w78 w79]

Secondary prevention

Risk factors should be identified in older patients, and modified if possible. Lifestyle measures such as diet, smoking, and exercise should be addressed whatever the age of the patient. It is surprisingly easy to persuade older patients who have smoked cigarettes for half a century to give up if they are given all the information. Hypertension, especially isolated systolic hypertension, should be treated rigorously while monitoring for adverse drug effects, which are more common in the older patient. Patients should be screened for impaired glucose tolerance or non-insulin dependent diabetes mellitus, and both treated to maintain normal glycaemia. Patients with a raised low density lipoprotein (LDL) cholesterol should receive dietary advice and a statin as for younger patients. Pravastatin reduced coronary events and mortality in patients up to 75 years of age with average cholesterol over a five year follow up period.[17] Cholesterol may be a poorer predictor of coronary events in older people as plasma concentrations are influenced by comorbidity. Patients over 75 years have not been recruited to the major lipid lowering trials. However, because of the accumulation of other perhaps non-modifiable risk factors, intervention is probably still warranted.[w80]

Rehabilitation

All older patients who are not seriously cognitively impaired should have access to cardiac rehabilitation, with the aim of maintaining peak physical functioning and personal independence. Staff in cardiac rehabilitation programmes have to be aware of the patients' comorbidities and modify programmes and support as appropriate. Exercise training programmes have been found to improve endurance and functional capacity in older people after myocardial infarction.[18] In spite of advanced age older patients, rather than healthy subjects, can and will change well-entrenched lifestyles. Even after 20 years relative inactivity, patients can and will increase exercise if given good reasons and "permission" to do so and can be made aware of personal benefits.

Angina

Lifestyle limiting angina has a prevalence of around 16% in people over the age of 65 years.[w81] The diagnosis in older patients can be difficult. It is overdiagnosed because too little attention is paid to obtaining a precise history or the diagnosis is a legacy from many years ago. Many "old old" patients had angina in their 60s which they seem to have outgrown in their 80s. This may be due to almost subconscious avoidance of precipitating factors. It is more acceptable for older subjects to adopt a sedentary lifestyle and thus avoid provoking symptoms on exercise. This may also lead to underdiagnosis of angina. Standard treatment should be followed in this age group as for younger patients but adverse drug effects (postural hypotension, negative inotropism, and oedema) may limit drug options. In this situation, or where patients are resistant to maximal medical treatment, referral for PTCA or surgery is indicated.[w82] While PTCA or surgery confirm no survival benefit, recent evidence suggests that elderly patients benefit from symptom relief more than younger patients with aggressive surgical intervention.[w83] They should therefore, at the very least, be referred for assessment and not denied the potential benefits because of age alone.

Arrhythmias

Atrial fibrillation

Five per cent of people over the age of 65 years have chronic atrial fibrillation (AF)[w84] approximately half of whom do not have associated myocardial disease—so called lone AF. Loss of diastolic filling caused by AF may compromise left ventricular filling to such an extent that it precipitates heart failure. Except in recent onset AF chemical or electrical cardioversion to sinus rhythm is not usually an option because of high relapse rates, though careful

selection may improve maintenance of sinus rhythm.[w85] Failing this rate control with digoxin is a less effective alternative,[w86] although the addition of a calcium channel antagonist or β blocker may improve rate control during exercise.[w87]

AF is associated with cerebral embolisation and stroke.[w88] The risk of embolisation increases with the addition of other factors such that cardioversion or anticoagulation becomes warranted. These risk factors include previous transient ischaemic attack/stroke, heart failure, hypertension, diabetes,[w89] and most importantly age over 75 years.[w90] AF associated with any one of these risk factors requires anticoagulation.[w91] The latter risk factor alone causes logistic problems for primary care in the management of long term warfarin treatment in the community.[w92]

Several placebo controlled trials have shown the efficacy of anticoagulation in non-rheumatic AF. More than 1000 patients (mean age 67 years) were entered into a placebo comparison with aspirin or warfarin. The trial had to be stopped prematurely at 29 months because of significant benefits of anticoagulation.[w93] A comparison of warfarin and aspirin by the same group showed similar thromboembolic benefits in patients older than 75 years compared with younger patients.[w94] However, bleeding complications with warfarin were higher in the older patients. Novel ways of delivering anticoagulation treatment need to be explored. While adverse effects and risks from anticoagulation do increase with increasing age, benefits still invariably outweigh adverse effects for carefully selected patients.[w95 w96] It should be appreciated that the majority of old people with AF are not so frail and decrepit that they should be denied the undoubted benefits of anticoagulation. To fail to anticoagulate on the basis of age alone is "ageism" and poor medicine.[w22] Anticoagulation with warfarin is probably not indicated in patients with cognitive impairment sufficient to compromise compliance, in patients subject to recurrent falling, and in patients with a history of recent gastrointestinal bleeding (within three months) or potentially serious drug interactions. In patients where full anticoagulation with warfarin is likely to cause potentially severe adverse effects aspirin will deliver less benefit at less risk.

Bradyarrhythmias

Older patients benefit from appropriate physiological pacing which can be provided at a relatively modest cost.[w97] Unfortunately older people in the UK receive pacemakers at approximately half the rate of similar aged patients in the rest of Europe,[w98] and when they do receive a pacemaker it tends to be a bottom of the range model.[w99] Quality of life benefits, especially improvement in cognitive function of older paced patients,[w100] require primary care physicians and generalists to be alert to possible bradyarrhythmias and refer early for appropriate assessment and treatment.

Table 2 Prevalence of different forms of hypertension in a community sample of people aged more than 65 years[w102]

	Screening	
Type of hypertension	*Visit 1*	*Visit 2*
Isolated systolic	19.1	4.2
Isolated diastolic	5.7	1.0
Combined	9.8	3.9
Total	52.2	10.3

Hypertension

Epidemiology

Blood pressure rises with increasing age. The NHANES III survey showed almost linear increases in systolic and diastolic blood pressures from early adult life through to about 65 years of age.[w101] Thereafter systolic levels continue to rise but less so, especially in women, while diastolic levels tends to fall progressively. Thus isolated systolic hypertension becomes the predominant type of hypertension in the "old old"[w102] (table 2). As the latter workers showed, the very high prevalences of raised blood pressure in older people decrease with repeated measurements, presumably because of the "white coat" phenomenon. This confirms that the diagnosis of hypertension in older people should not be based upon a single measurement. More than half the population over 85 years of age will be "hypertensive".[w101]

Raised blood pressure in old people is not a benign condition. Hypertension is the major risk factor for strokes, heart failure, coronary heart disease, and peripheral vascular disease.[w103] Isolated systolic hypertension is particularly related to strokes and less so to coronary events. Diastolic pressures may be inversely related to subsequent mortality, implying that pulse pressure may be the best predictor of the adverse effects of raised blood pressure in old people.[w104] Paradoxically low blood pressure is also associated with high mortality in older people but the relation is only short term—that is, within three years of diagnosis. Thereafter, low blood pressure is a predictor of survival.[w105] Presumably the short term observation is a reflection of comorbidity and increased frailty near death.

Treatment

Meta-analyses have quite clearly demonstrated the benefits of treating old people with any type of hypertension, but especially those with isolated systolic hypertension.[w104 w106] Indeed the number needed to treat to prevent one major cardiovascular event decreases significantly with decreasing age, at least under the age of 80 years. Treatment prevents stroke much more than coronary events.[w104 w107] The target blood pressure should be the same as in younger patients—that is, 140/90 mm Hg—and probably lower in that significant proportion of older patients (25%+) with non-insulin dependent diabetes mellitus.[w108]

There remains the problem of the "old old". No adequately controlled treatment study of hypertensive patients aged more than 80 years

Trial acronyms

ELITE: Evaluation of Losartan in the Elderly
HYVET: Hypertension in the Very Elderly
ISIS: International Study of Infarct Survival
NHANES: National Health and Nutrition Examination Survey

has been reported.[w22] Very few "old old" patients have been included in wider age range studies, and where they have the numbers are so small that treatment recommendations cannot be made. There is no reason to believe that "old old" hypertensive patients would not benefit from blood pressure lowering, but it is likely that the increased incidence of adverse drug effects may act as a counterbalance. The results of the HYVET study of hypertensive patients over 80 years of age are eagerly awaited.[w109]

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