Blood pressure in the elderly

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SUMMARY. The distribution of blood pressure levels in elderly patients is described and the evidence that higher levels are associated with morbidity at this age is reviewed. In the absence of definitive evidence about the results of treating hypertension in the elderly, the physiological factors that have to be taken into account are surveyed with particular reference to aggravating cerebral ischaemia.

More potent hypotensive agents should be avoided in older patients and in practice the decision on whether to treat may depend more on the clinical context than on the level of the blood pressure itself.

Introduction

Cross-sectional studies in most communities show a steady rise of blood pressure in both sexes with increasing age (Hamilton *et al.*, 1954). Men usually have higher pressures until about 45 years of age, when higher pressures occur in women. Exceptional communities are found where this does not occur. These are usually primitive populations, living unsophisticated lives (Lowenstein, 1961) free from the stresses of civilised living, but also perhaps on diets low in sodium (Prior *et al.*, 1966) or with high levels of parasitic infestation (Maddocks and Vines, 1966).

In patients over 70 years of age the evidence is conflicting. Some studies suggest that the pressure continues to rise (Anderson and Cowan, 1959; Droller *et al.*, 1952). Others show that the systolic or the diastolic or both pressures level off or even fall slightly in older people (Russek and Zohman, 1946; Master *et al.*, 1958). In some of these patients a fall may be associated with the onset of congestive cardiac failure (Brown *et al.*, 1963). Differential mortality could account for some of the change in gradient (Edwards *et al.*, 1959). Loss of elasticity in the arterial tree causes a disproportionate rise in systolic pressure (O'Rourke, 1970) producing the characteristic rise in pulse pressure of old age.

Definitions

There has been much discussion on what the ' normal ' level of blood pressure in the elderly should be and argument has waged between those who consider that the upper limit of normality should be re-set with increasing years (sometimes by using formulae like ' 100 plus the age ' or ' 113 plus one third the age ') and investigators like Alvarez and Stanley (1930) who state that " a (systolic) pressure of 140 mm mercury is just as abnormal in an old man as in a young one."

This debate has been confused by ambiguity in the meaning given to the word ' normal.' Sometimes it is used to refer to the middle part of the frequency distribution and at others to levels of blood pressure associated with a relatively low risk of disease. Robinson and Brucer (1939) in an analysis of 11,383 insured persons first excluded all those with blood pressures over 140/90 as ' definitely hypertensive ' and went on to define ' normal ' blood pressure as levels below 120/80. They stated that " normal blood pressure does not rise with age." Their manipulations, however, prejudge the issue instead of proving it. Master *et al.* (1958) in trying to establish ' normal ' levels of blood pressure

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in old-age, analysed 5,757 reports from physicians in the United States, but since all patients with " evidence of cardiovascular disease " were excluded the significance of the norms produced seems limited. Perhaps it is more profitable to eschew the word ' normal ' in relation to hypertension and talk about defined risks in defined groups of the population.

Risks

How dangerous is hypertension in the elderly? Most prospective studies show a striking correlation of high levels of blood pressure with cardiovascular morbidity and mortality. Thus, the evidence from Framingham has been analysed by Kannel over the first 18 years and includes individuals up to 62 years of age at the start of the period of observation. He has reported the association of stroke (Kannel *et al.*, 1970), coronary artery disease (Kannel *et al.*, 1971), and congestive cardiac failure (Kannel *et al.*, 1972) with systolic, diastolic, and mean blood pressure in 5,192 men and women and shown that the correlation with systolic pressure increased with advancing age for all these conditions while that with diastolic pressure waned.

Colandrea *et al.* (1970) in a large population of retired people in California examined individuals with isolated systolic hypertension (systolic pressure of more than 160 mm Hg and diastolic pressure under 90 mm Hg) and found a six-fold mortality from cardiovascular disease compared with normotensive individuals from the same population. The old shibboleth that systolic hypertension in the elderly is an innocent finding appears no longer tenable.

Miall and Chinn (1974) have shown an increase from all cardiovascular deaths in the age range 65–74 with elevations of systolic or diastolic blood pressure, especially in males. Another prospective study (Chapman *et al.*, 1971), carried out over 15 years on 1,859 male civil servants in Los Angeles from 20–70 years of age when it started, showed, contrary to the findings of Framingham, little evidence of a relationship between blood pressure and coronary artery disease in any age group. The author showed, however, an increased liability to stroke in those with high blood pressure, but did not publish an age analysis.

The difficulties inherent in interpreting cross-sectional data are illustrated by a cardiovascular survey in a random sample of older people in Edinburgh, carried out by Kitchin *et al.* (1973). Evidence of cardiovascular disease (questionnaire and electrocardiogram) was associated with a lower prevalence of diastolic and diastolic-and-systolic hypertension than in a group without such evidence, but isolated systolic hypertension was the same in both groups. The low diastolic pressure in the affected group could have been due to associated myocardial disease, to an arrhythmia, or to some other common causal factor. Cutler (1967) in another cross-sectional survey of people over 60 years of age showed that a history of stroke was associated with elevated systolic pressure, but not with elevations of diastolic pressure or serum lipids.

The standardised mortality ratio for hypertension falls with increasing age and this fact has been adduced as evidence for the harmlessness of hypertension in older people (Fry, 1974). The standardised mortality ratio $= \frac{\text{observed mortality}}{\text{expected mortality}}$ and relates mortality due to hypertension to mortality from all causes and, as other threats to life increase, the relative risk of hypertension decreases. Nevertheless, this does not give any indication of the absolute impact, the total number of deaths involved, which may continue to rise while the standardised mortality ratio falls. Neutra (1974) has shown this trend in the Build and Blood Pressure Study, but the data here do not go beyond patients of 55 years of age. He calculates from the data of the Veterans' Administration Co-operative Study that treatment of 100 patients below 50 would prevent eight morbid events, while treatment of the same number of patients above this age would prevent 25 morbid events.

No trials have yet been reported on the treatment of older patients with hypertension. A multi-centre trial organised by the European Working Party on High Blood Pressure in the Elderly (E.W.P.H.E.) (Amery and De Schaepdrijver, 1973) was started in 1972 and has just completed its pilot stage. This trial is using hydrochlorothiazide and triampterene supported by alpha methyldopa. A trial in my practice is using β adrenergic blockade as a first treatment, followed by bendrofluazide. Both these trials will last for five years.

Principles of treating elderly patients

In the absence of definitive information, what are the theoretical principles for the treatment of hypertension in the elderly? Has the damage to end-organs or the degree of atheroma proceeded to the point at which no improvement is likely to occur? Carter (1970) in a trial of therapy in stroke survivors was not able to show an improvement in prognosis in patients over 65 years of age, but the numbers of patients in this group were too small to draw significant conclusions.

McKeown (1965) in a study of 1,500 necropsies in elderly people failed to find the relationship between antemortem blood-pressure levels and the degree of atheroma in these patients that he found in younger subjects. On the other hand, Evans (1965) in a careful study of the arteries of elderly patients dying in a mental hospital found a highly significant correlation of atherosclerosis, especially that of the cerebral arteries, with records of previous hypertension and noted that this was clearer if the levels of blood pressure during the last two years of life were excluded. He argued that these last years often included unrepresentative levels due to the terminal illness. The reliability of records of blood pressure examined retrospectively in this way, however, is open to question. Cardiac failure can certainly be improved by the treatment of hypertension in elderly patients.

Risks of treatment

How dangerous is hypotensive therapy in this age? It has been postulated that older people may need a high blood pressure to maintain cerebral perfusion in the presence of narrowing of the arterial tree. Indeed Dickenson has elaborated this hypothesis to account for essential hypertension at all ages, relating it to narrowing of the vertebrobasilar system and assembling the relevant evidence (Dickenson, 1965). Gubner (1962) mentions an unpublished case of De Bakey's in which re-establishing blood flow in a vertebral artery not only eliminated vertigo, but reduced blood pressure to normal levels. Conversely the rise of blood pressure that commonly occurs after strokes (Adams and Merrett, 1961) may be an attempt to maintain cerebral perfusion. Autoregulation of cerebral blood flow is abnormal in hypertension, the lowest pressure at which cerebral perfusion can be adequately maintained being higher than in normal people, so that they tolerate falls of blood-pressure less well (Fazekas *et al.*, 1953).

Whether this mechanism is affected by age itself or by the presence of stenosis of extra or intracranial vessels is not known, but seems likely. In normal individuals cerebral blood-flow declines with advancing age (Kety, 1956; Lovett Doust, 1972), so increasing vulnerability to critical ischaemia. It is considerations such as these that have encouraged suspicions that lowering blood pressure in susceptible elderly subjects might increase the rate of cerebral infarctions. No clinical evidence has been found to support this supposition, however. Meyer and his colleagues (Meyer *et al.*, 1968) measured cerebral blood flow before and after hypotensive therapy in 13 patients between 48 and 78 years old, all of whom had cerebrovascular symptoms and found an *increased* cerebral blood flow in nine and a fall of cerebrovascular resistance in all of them. Kendell and Marshall (1963) attempted to reproduce transient ischaemic attacks in patients subject to them by drastic lowering of blood pressure, but failed. Moreover, a number of trials of hypotensive therapy in stroke survivors have been reported (Adams and Merrett, 1961;

Marshall, 1964; Carter, 1970; Beevers *et al.*, 1973; Hypertension-Stroke Co-operative Study Group, 1974) and in no instance has therapy been shown to increase the likelihood of cerebrovascular attacks. The pilot stage of the E.W.P.H.E. trial has also not produced any evidence of more cerebrovascular attacks in the treated group (Amery and De Schaepdrijver, 1975).

Despite the paucity of evidence that permanent damage is likely to accrue from hypotensive therapy in the elderly, it would be wise to be cautious in the presence of widespread atheroma or with oxygen desaturation or hypercapnia (both of which impair autoregulation). Certainly diffuse hypertensive brain disease with dementia can deteriorate if the patient is put on hypotensive therapy (Hughes *et al.*, 1954). Sudden falls of blood pressure also may be prejudicial to adjustment (Loell and Schneider, 1944). Even without therapy Johnson *et al.* (1965) noted spontaneous falls in blood pressure with changes of position in old people, especially in those with evidence of cerebrovascular disease.

The type of agent may also effect the likelihood of side effects (Bauer, 1974). Thiazide diuretics have been most often used, combined with potassium supplements or potassium-sparing diuretics. Hypovolaemia is sometimes encountered, especially at the onset of therapy if there is a vigorous diuresis. Hyperuricaemia and hyperglycaemia may also present problems by aggravating underlying gout or diabetes.

B *adrenergic-blocking drugs*

B adrenergic-blocking agents, which have recently been used widely in the treatment of hypertension in younger patients, have been the subject of only one report in the treatment of elderly hypertensives (Eisalo *et al.*, 1974). This purported to show that one of these agents (alprenolol) produced a fall of both systolic and diastolic blood pressure during a three month period in a group of people aged 61 to 89 years living in a Finnish home for the elderly. Unfortunately there was no run-in period, nor were controls used, so that the significance of the modest falls of blood pressure achieved is difficult to evaluate. Three out of 38 patients developed congestive heart failure during the first week and therapy had to be withdrawn. This is a possible disadvantage in a sector of the population with a high incidence of left ventricular failure. Another danger would be the exacerbation of bronchial asthma or heart block. On the other hand, if β blockers were shown to offer protection from deaths due to coronary artery disease, their use in elderly patients might be worth these risks.

Treatment with methyldopa

Another study of treatment in elderly patients using methyldopa and hydrochlorothiazide (Corman and Skversky, 1973) also had no controls and there was only a short run-in period. Larger falls of blood pressure were, however, reported than in the Finnish patients. Only minimal side effects were experienced, but five (out of 22) patients developed a significant fall in haemoglobin over three months which did not rise to pre-treatment levels on stopping the drug. The Coombs' test remained negative in all the patients. Other workers have reported drowsiness and depression in patients on methyldopa therapy.

Stronger sympatholytic agents should be avoided in older patients as they are liable to cause severe postural hypotension (Whitfield, 1972). Finally, it should be emphasised that, in treating older patients, the context may be more significant than the disease. Some patients are old at 65 and concomitant illness may make the treatment of hypertension inappropriate. Nevertheless, if it can be shown that strokes and coronary disease can be made less likely, it may be possible to improve significantly the quality of life in the decades after retirement and prevent some of the disablement of old age.

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