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ORIGINAL ARTICLE

Seven-year follow-up of blood pressure in the Healthy Old People in Edinburgh (HOPE) cohort

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The relationship between blood pressure and health in old age is complex and influenced by socio-economic factors. The Healthy Old People in Edinburgh cohort were initially disease-free and untreated, providing a sample in which directionality in this relationship could be examined. Subjects' health status, medication use and blood pressure was ascertained at baseline, after 4 years, and again after 7 years. Socio-demographic and socio-economic data were also collected. A total of 603 subjects were seen at baseline, 429 at 4 years and 301 at 7 years; complete blood pressure data were available for 294. Mean blood pressures were 157/85 mm Hg, 159/87 mm Hg and 162/86 mm Hg at baseline, 4 years and 7 years respectively. When subjects with diagnosed hypertension were excluded, the presence of disease ($P = 0.009$) and medication use ($P = 0.047$) at 7 years were associated with a relative reduction in blood pressure over time. For these subjects disease was predicted by deprivation index of residential area (OR 1.24, 95% CI 1.10–1.40 per Carstairs unit) and occupational group (OR 0.85, 95% CI 0.74–0.97 per major group). In this cohort disease, excluding hypertension itself, significantly attenuated the age-related rise in systolic blood pressure; the longer disease has been present the less the increase. In addition, socio-economic variables are important predictors of blood pressure change in those with disease. Deprivation index of residential area was a better predictor of disease than previous occupation in these subjects who had retired over a decade previously.
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Introduction

Throughout most of human adult life both systolic and diastolic blood pressure (SBP/DBP) increase with age. Towards the end of life, however, DBP remains much the same whilst SBP continues to rise.[1] One leading hypothesis that explains this observation is that the differential elevation of SBP relates to reduced arterial compliance.[1] Elevated SBP is a risk factor for stroke and other vascular diseases in old age. Such vascular diseases exhibit a clear gradient of risk across social class and other indices of socio-economic status.[2] In our follow-up of the Healthy Old People in Edinburgh (HOPE) cohort at 4 years we observed that, paradoxically, SBP rose significantly less in retired unskilled workers compared with other occupational groups.[3] We considered that one explanation of this was that subjects who had been manual workers were more likely to have covert disease attenuating the rise in SBP. For the whole group that had their blood pressure remeasured after 4 years, those subjects who had developed disease in the follow-up period failed to show the expected increase in SBP. We now report outcome at 7 years that allows this hypothesis to be tested.

A further question that arises from the disease-related attenuation of SBP rise in old age is the duration of this effect. Does disease permanently depress BP or does it rise again after the acute or semi-acute phase? Disease in elderly people is heterogeneous and multiple pathology is common, so that the answer to this question may be diagnosis-specific, especially for those diagnoses that lead to the use of drugs with antihypertensive actions. Investigation of the complex interaction between disease and BP requires longitudinal data. The HOPE study provides unique data because the sample were healthy and on no medication at baseline thus avoiding any confounding influence of lagged effects of disease or drugs pre-dating the study period. We therefore sought to model the effects of disease and medication on BP over time in this initially healthy cohort.

Subjects and methods

The HOPE cohort comprises 603 community-resident disease-free, untreated subjects tested in 1990–1991 aged 70 years and over.[4] Written informed consent was obtained from all subjects. By Wave 2 in 1994–1995, 69 subjects had died, 15 were too unwell to test, 46 refused to be seen, 19 failed

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to reply, 12 had moved out of the area and 13 were untraceable. A total of 429 (71%) subjects were revisited.[5] For the 1997–1998 follow-up (Wave 3), subjects were contacted via their general practitioners (GPs), and all available general practice notes screened to ascertain GP-reported illness and medication use. Subjects who had moved were traced via Lothian Primary Care Division, and cause of death for those subjects who had died ascertained from death certificates held at Register House, Edinburgh. Subjects were seen by the trained research nurses (SI and SC) who had visited them previously. The research nurses were blind to Wave 1 and Wave 2 BP measurements.

Wave 3 of the HOPE study was planned to be identical to Wave 2 with collection of socio-demographic, health, BP and cognitive data.[5] Blood pressure was measured at home with the subject sitting after at least 30 min rest. Presence or absence of specific diseases and medication use was enquired about directly from subjects and from primary care casenotes. Diagnoses were categorised as before: no disease; cardiovascular; cerebrovascular; neoplasia; hypertension; diabetes; thyroid disorder; dementia; other vascular; other single diagnosis; multiple diagnoses. For the purposes of analysis, subjects with a diagnosis of hypertension, either alone or with other disease, were specifically identified. Thus, as in previous waves of the study, classification was pragmatic, based on the recorded and reported clinical data. Statistical analysis was performed with the SPSS 8.0 statistical package.

Results

Description of sample available at 7 years follow-up

Of the 429 subjects available for follow-up at Wave 3, 57 had died, 16 were too unwell for cognitive testing, 43 refused, one failed to reply, nine had moved out of the area and two were untraceable. Disease outcome was available for 404 of the 429 subjects seen at Wave 2 either directly, from primary care notes or death certificate entries. A total of 301 (71% of those available, 50% of original sample) subjects were seen again, but reliable BPs could only be recorded for 294 (103 male, 191 female) subjects of mean age 81.4 (s.d. 3.9, range 76–93) years. A total of 56.1% of subjects were retired white-collar workers (SOC groups 1–4), 29.1% retired skilled or semi-skilled blue-collar workers (SOC groups 5–7), 9.6% retired unskilled workers (SOC groups 8–9) and 5.6% unclassifiable (ex-armed forces, etc). Of the 294 subjects with BP data, 157 (53.4%) remained disease free at Wave 2 and 103 (35.0%) at Wave 3. A total of 176 (59.9%) subjects were on no regular medication at Wave 2 and 117 (39.8%) at Wave 3. Hypertension had been diagnosed in 37 (12.6%) of the 294 subjects by Wave 2 and in 53 (18.0%) by Wave 3.

Description of blood pressure change in all available subjects

Mean SBP and DBP for the 294 subjects with BP data at each Wave are shown in Table 1. SBP rose throughout the period, whilst DBP rose initially, but fell later. Change in BP over time was significant for both SBP (F = 4.10,[2,292 df], $P = 0.018$) and DBP (F = 3.72,[2,292 df], $P = 0.025$), but there was a significant difference of time on SBP compared with DBP (test [SBP *VS* DBP] by time interaction F = 6.28,[2,292 df], $P = 0.002$).

Table 1 Mean, standard deviation (s.d.), minimum and maximum of range systolic (SBP) and diastolic (DBP) blood pressures for baseline (1), 4-year follow-up (2) and 7-year follow-up (3) of the 294 surviving and evaluated HOPE subjects

	Min	*Max*	*Mean*	*s.d.*
Age at baseline (yrs)	70	85	74.8	3.8
SBP1 (mm Hg)	100	220	157	24
SBP2 (mm Hg)	100	220	159	20
SBP3 (mm Hg)	105	230	162	24
DBP1 (mm Hg)	50	120	85	10
DBP2 (mm Hg)	60	120	87	10
DBP3 (mm Hg)	60	110	86	10
Education (yrs)	7	19	11	2.7
Period 1–2 (yrs)	3.23	5.23	4.21	0.18
Period 2–3 (yrs)	2.09	4.33	3.01	0.45

Socio-economic status and blood pressure change

We next tested hypotheses relating disease and socio-economic status to blood pressure change. Table 2 shows BPs for five groups: those who remained disease-free at Wave 3 ($n = 81$); those who had incident disease between Waves 1 and 2 excluding hypertension ($n = 100$); those who were disease-free at Wave 2 but had recognised disease at Wave 3 excluding hypertension ($n = 57$); those with hypertension diagnosed between Waves 1 and 2 ($n = 37$); those who were disease-free at Wave 2 and had hypertension diagnosed between Waves 2 and 3 ($n = 13$). For those subjects with hypertension, there was no significant effect on BP levels over time (F = 2.32,[2,11 df], $P = 0.14$) for those diagnosed between Waves 2 and 3, but a significant change over time was detected for those diagnosed between Waves 1 and 2 (F = 5.00, [2,35 df], $P = 0.012$) with a significant difference between the change in SBP compared with DBP (F = 4.10,[2,35 df], $P = 0.025$). Presence of disease at Wave 2, excluding hypertension, had a significantly attenuated the rise in BP over time, (F = 4.09,[2,254 df], $P = 0.018$), but there was no differential effect over time on SBP compared with DBP (F = 1.40,[2,254 df], $P = 0.25$). There was no significant effect on BP levels over time was detected for those subjects who were disease-free at Wave 2 but with incident disease between Waves 2 and 3, excluding hypertension, compared with those subjects who remained disease-free throughout follow-up (F = 1.16,[2,141 df], $P = 0.32$). Neither age nor sex were significant covariates of the effects of disease status on BP levels.

For the 294 subjects with BPs measured at each Wave, there was no significant effect of SOC, categorised by major occupational groups, on BP level over the 7-year follow-up (F = 1.04,[18,564 df], $P = 0.41$) nor differentially on SBP compared with DBP over time (F = 1.03,[18,564 df], $P = 0.77$). Other

Table 2 Systolic (SBP) and diastolic (DBP) blood pressure (mm Hg) at Waves 1,2 and 3 for HOPE subjects by time of onset of disease (excluding hypertension) and hypertension (HYP)

	Disease-free (*n* = 81)	*Disease Waves 1–2*[a] (*n* = 100)	*Disease Waves 2–3* (*n* = 57)	*HYP Waves 1–2*[a] (*n* = 37)	*HYP Waves 2–3* (*n* = 13)
SBP1	150 (23)	157 (21)	151 (23)	179 (20)	172 (19)
DBP1	84 (8)	84 (9)	82 (12)	95 (12)	91 (8)
SBP2	155 (18)	159 (21)	156 (19)	167 (18)	180 (19)
DBP2	87 (9)	86 (11)	86 (9)	91 (9)	95 (11)
SBP3	164 (25)	159 (23)	158 (19)	168 (26)	172 (28)
DBP3	87 (10)	84 (10)	86 (8)	89 (9)	94 (9)

[a]Denotes groups with significant changes in SBP and DBP over time.

indices of socio-economic status, years of full-time education (F = 0.12,[2,291 df], $P = 0.88$) and Carstairs deprivation index of residence (F = 1.29,[2,289 df], $P = 0.28$), were non-significant when entered as covariates either individually or in combination with each other and SOC. Similarly, no significant effect on BP level over time was detected for SOC (F = 0.99,[18,152 df], $P = 0.48$), education (F = 0.45,[2,84 df], $P = 0.64$) or deprivation (F = 0.25,[2,82 df], $P = 0.78$) for those subjects who remained disease-free throughout follow-up.

Age was a significant contributor (F = 5.07,[2,97 df], $P = 0.008$) to BP level over time in those subjects with disease at Wave 2, excluding hypertension. Correcting for age, a trend for SOC on BP level over Waves 1–3 was detected in this group (F = 1.49,[18,178 df], $P = 0.097$). Neither education nor deprivation had significant effects. For the period of established disease between Waves 2 and 3, SOC had a significant effect (F = 2.45,[9,90 df], $P = 0.015$) on BP (Table 3), but age, education and deprivation did not.

For the smaller number of subjects who were disease-free at Wave 2, but had incident disease between Waves 2 and 3 excluding hypertension, a similar pattern emerged. Neither SOC (F = 1.35,[18,94 df], $P = 0.17$) nor any other socio-demographic variable had a direct effect on BP level over the whole follow-up period, but SOC had a significant differential effect on SBP compared with DBP (test [SBP *vs* DBP] by time interaction, F = 2.15, [18,94 df], $P = 0.009$). No other socio-demographic variable had a significant differential effect on BP over time. The effect remained significant for the period between Waves 2 and 3 (F = 2.32,[9,47 df], $P = 0.03$). The differential effect of SOC on BP reflected the relative stability of DBP in all SOC groups, whilst SBP rose substantially in most subjects (SOC groups 1–7, mean 148 mm Hg to 158 mm Hg) but failed to rise much in unskilled workers (SOC groups 8 and 9, mean 157 mm Hg to 159 mm Hg). Blood pressure rise was more marked in the earlier disease-free period compared with the period with the incident disease. Examining socio-demographic variables together with disease status and medication use in the whole group excluding those with diagnosed hypertension, two variables, presence of disease at Wave 3 (F = 4.87,[2,228 df], $P = 0.009$) and medication use (F = 3.09,[2,228 df], $P = 0.047$) were the only significant contributors.

Table 3 Means (and standard deviations) of systolic and diastolic blood pressures at Waves 2 and 3 for subjects with incident disease between Waves 1 and 2, excluding hypertension

Occupation	*Systolic BP Wave 2* (*mm Hg*)	*Diastolic BP Wave 2* (*mm Hg*)	*Systolic BP Wave 3* (*mm Hg*)	*Diastolic BP Wave 3* (*mm Hg*)
White collar SOC 1–4 (*n* = 48)	158 (23)	84 (9)	161 (22)	85 (9)
Skilled and semi-skilled SOC 5–7 (*n* = 34)	153 (18)	87 (12)	155 (22)	83 (10)
Unskilled SOC 8–9 (*n* = 12)	171 (19)	88 (13)	166 (22)	85 (9)

Disease and blood pressure change

We next tested secondary hypotheses relating specific disease categories to change in BP. For those developing disease between Waves 1 and 2, diagnostic category, excluding hypertension but examining all others discretely in the model, had no significant effect on BP levels at Waves 2 and 3 (F = 0.38,[13,86 df], $P = 0.97$). Within the group who were disease-free at Wave 2 but developed disease between Waves 2 and 3, specific diagnostic category or combination of categories, excluding hypertension, had no significant effect on BP levels over time between Waves 2 and 3 (F = 1.14,[9,48 df], $P = 0.36$). Parsimoniously categorising disease into heart disease, neoplasia, other single diagnosis or multiple diagnoses also showed no significant effect, although there was a trend (F = 2.53,[3,54 df], $P = 0.067$) towards BP falling in those with heart disease (163/88 mm Hg to 157/84 mm Hg) and multiple diagnoses (168/88 mm Hg to 156/82 mm Hg) compared to the other categories.

Outcome of SOC groups 8 and 9 who were disease-free at 4 years follow-up

We noted the differential effects of SOC on SBP and DBP in those who remained disease-free at Wave 2 and were re-evaluated at Wave 3 over the period between Waves 2 and 3. We then examined outcome

of all disease-free subjects in SOC groups 8 and 9 compared with the other SOC groups. Overall, SOC group had no effect on mortality, illness too severe to be seen and other non-evaluated outcomes (χ^2 = 69.5, P = 0.27), disease category (χ^2 = 177.9, P = 0.34) or specific diagnosis of hypertension (χ^2 = 7.16, P = 0.62) at Wave 3. However, compared with other SOC groups, retired unskilled workers were less available for evaluation at Wave 3 (χ^2 = 24.7, P = 0.001), but were no more likely to have died (Fisher's exact test P = 0.28), or have developed disease (Fisher's exact test P = 0.37). Only one retired unskilled worker was diagnosed with hypertension between Waves 2 and 3. For those subjects diagnosed with hypertension by Wave 2, SOC had no significant effect on BP levels between Waves 2 and 3 (F = 0.57, [7,29 df], P = 0.77). No other socio-demographic variable had a significant effect either.

Blood pressure and socio-economic status as risk factors for disease between 4- and 7-years follow-up

Finally, having examined the effects of disease on BP, we examined the effects of BP and socio-demographic variables on the risk of subsequent disease between Waves 2 and 3. We performed logistic regression for all subjects, dead or alive, in which outcome was known, comparing those subjects developing disease between Waves 2 and 3 with those remaining disease-free. The optimum model (χ^2 = 17.81, P = 0.0005) comprised deprivation (OR 1.20, 95% CI 1.07–1.35 per Carstairs deprivation unit), SOC (OR 0.86, 95% CI 0.76–0.98 per SOC group) and Wave 1 SBP (OR 1.01, 95% CI 1.00–1.03 per mm Hg). No other variable added significantly to this model. Excluding those subjects with hypertension at Wave 3, the optimum model comprised deprivation (OR 1.24, 95% CI 1.10–1.40 per Carstairs deprivation unit) and SOC (OR 0.85, 95% CI 0.74–0.97 per SOC group) only. For those subjects diagnosed with hypertension between Waves 2 and 3, the optimum model comprised a single variable, Wave 2 SBP (OR 1.06, 95% CI 1.03–1.10 per mm Hg).

Discussion

The results from the 7-year follow-up of the HOPE cohort build on those available at 4 years.[3,6] SBP continues to rise in those subjects who remain free from disease as they enter their ninth decade. However, DBP in these healthy subjects fell back to baseline levels after the initial rise over the first 4 years. Disease, excluding hypertension itself, significantly attenuates the rise in SBP; the longer disease is present the smaller the increase (Table 2). Furthermore, BP was depressed more if the disease required regular drug treatment. There is heterogeneity within disease with regard to its effect on BP with a trend for subjects with heart disease or multiple pathology to experience falls in both SBP and DBP. Age and gender had little effect on BP change in the HOPE cohort, but we confirmed the findings at 4 years[3] that socio-economic variables are important predictors for those subjects with disease. For those with prolonged exposure to disease at 7 years, SOC influenced both SBP and DBP, with BP falling in subjects with unskilled occupations (Table 3). A differential depression of SBP compared with DBP was noted for subjects with a relatively short exposure to disease. The attenuated SBP rise in subjects with recent disease and from an unskilled background suggests that the similar attenuation seen in disease-free subjects at 4 years may have been due to covert disease at that time. However, we did not detect any increase in subsequent mortality or morbidity in the disease-free subjects of SOC groups 8 and 9 compared with the rest of the cohort. This may be because these subjects were significantly less likely to be evaluated at 7 years, and any excess morbidity to have occurred in those for whom we have no data.

The combined effects of socio-economic status and disease on BP in the HOPE cohort can therefore be considered in two stages. There are the additional effects of occupation in those elderly subjects summarised above. But there are the effects of socio-economic status on risk of disease itself. At 7 years, the significant predictors of disease in this initially healthy cohort are previous occupation, deprivation index of area of residence and initial systolic BP. Excluding hypertension as a disease, we are left with greater residential deprivation increasing risk and, paradoxically, lower social class reducing risk of disease. Both deprivation score and SOC are indices of socio-economic status and usually correlate in the same direction as each other with health status in younger adults. For these subjects, about 20 years post-retirement, occupation is likely to reflect historic socio-economic status, whilst deprivation index of residence may represent a more current measure of resources. Explanations of this paradox are: that people of higher occupational status living in relatively deprived areas (1) have chosen to invest less in housing over a prolonged period or (2) represent post-retirement socio-economic 'drift'. In our previous reports we found that significant predictors of disease at 4-years follow-up were age, gender and initial SBP.[5,6] At that stage we examined SOC but not deprivation index. Reviewing disease outcome at 4 years including deprivation, but excluding those subjects with hypertension, the optimum logistic regression models comprises age (OR 1.13, 95% CI 1.08–1.18 per year), gender (OR 0.60, 95% CI 0.41–0.87 women compared with men), baseline SBP (OR 1.01, 95% CI 1.00–1.02 per mm Hg) and deprivation (OR 1.07, 95% CI 1.00–1.14 per Carstairs index unit). SOC did not add significantly to this model. This model favours the post-retirement 'drift' hypothesis, with deprivation index of residence being a better socio-economic predictor of disease in this initially healthy cohort. The independent effect of SBP on disease, excluding hypertension itself, introduces another pathway and further paradox. On the one hand rising SBP is associated with health, but on the other hand high SBP increases the risk of subsequent disease.

In summary, the following pathways link BP and disease in the HOPE cohort. Initially, when the subjects were in their mid-seventies, disease was more

likely if they were older, male, lived in a deprived area or had a high SBP. Disease led to a reduction of both SBP and DBP, and this reduction was greater the longer the exposure to disease and if the disease was severe enough to require drug therapy. Blood pressure fall was greatest if they had an unskilled occupation. For those remaining free from disease as they entered their ninth decade, disease was most likely if they lived in a more deprived area than expected for their occupational background. Those who developed disease had a fall in SBP relative to DBP. SBP continued to rise for those remaining in good health throughout follow-up, but DBP remained about the same. Blood pressure levels in these people, now more likely to be living in relatively non-deprived areas, was not significantly affected by any socio-demographic or socio-economic factor.

Many studies in the elderly investigate the effect of BP on risk of subsequent disease. Pulse pressure is reported as a significant predictor of poor outcome[7] and associated with left ventricular hypertrophy.[8] Some investigators note that low BP is also a poor prognostic feature,[9,10] and others that a U-shaped relationship exists between BP and disease.[11–13] Such a U-shaped relationship would be predicted by the HOPE data for samples comprising a case-mix of subjects with and without disease: high SBP in disease-free subjects carries a poor prognosis, but the presence of disease itself predicts later mortality and is associated with a reduction in SBP. Indeed, in a sample of elderly subjects in Leiden, the paradoxical increased risk of low BP on mortality was fully explained by health status.[9] The authors comment that adjustment for confounding variables is rarely made in examining the relationship between BP and mortality in the elderly. In discussing their findings they state, 'It is unclear how much of this relation may be a result of poor health causing lower blood pressure (for example, by impairing cardiac function), or if it may have occurred because those who live to age 85 despite having high blood pressure are a highly selected group of healthy individuals'. Our data suggest that both explanations contribute to their observations.

Although our data help to disentangle the complex relationships between BP, socio-economic status and disease, attrition even in this initially healthy cohort results in some of the detected relationships being based on relatively small numbers of subjects. Hence, at present, conclusions must be tentative. Moreover, the HOPE cohort is deliberately highly selective. These data may not be relevant to the majority of old people who have some disease before 70 years of age. Nevertheless, more and more people are entering their eighth decade in good health. Studies in other settings would also help examine the influence of socio-economic variables. The Carstairs index of deprivation[14] is specifically designed for Scotland and calculated for small areas. The HOPE data fail to provide a specific causal pathway between deprivation index and health outcome. Other indices of socio-economic status would be required in other locations, and these might highlight specific factors contributing to risk. This would be particularly useful to examine the effects of any post-retirement 'drift'. Any post-retirement 'drift' might be of especial importance for the small number of subjects lost to follow-up because they moved out of the area. For this hypothesis to be tested, studies in well-defined, relatively stable populations may reduce attrition, but the study area needs to be large enough to allow adequate internal migration to look for 'drift'.

Identifying specific socio-economic factors related to poor health outcome in old people may provide opportunities for effective, focussed interventions. At first sight age and sex, the two principal variables related to risk of disease, appear less obvious candidates for intervention. However they, too, may be acting as proxies for age- or sex-related variables that could be influenced. Evidence for benefit of intervention for the other significant health predictor, SBP, are well established in the young old.[1] Benefit in the oldest old is yet to be clearly established. The HOPE data suggest that SBP may not be a significant predictor of disease in the oldest old. One hypothesis consistent with our observations that explains this is the exhaustion of subjects susceptible to the adverse effects of raised SBP. In other words, risk of hypertension-related disease is not homogeneous in the elderly, but distinct 'at-risk' groups exist. Indeed, there may be a group whose survival free from disease into the ninth decade is aided by elevated SBP.

The HOPE study confirms the importance of age, sex, socio-economic status and SBP as predictors of disease risk in the elderly. The data also confirm that disease attenuates the rise in SBP associated with aging. However, relationships between these variables are complex and multi-directional. An understanding of these relationships is essential to clinicians wanting to evaluate either the effects of BP on disease or *vice versa*. Future research should seek to identify those variables related to age, sex or socio-economic status that both predict health status and are amenable to intervention.

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References

1 Starr JM, Bulpitt CJ. Hypertension. Chapter 26. In: Ebrahim S (ed). *Epidemiology of Old Age*. BMJ publications, London, 1996.
2 Black D *et al*. Inequalities in health: the Black report. Penguin: London, 1988.
3 Starr JM *et al*. Blood pressure and ageing: longitudinal cohort study. *BMJ* 1998; **317**: 513–514.
4 Starr JM, Whalley LJ, Inch S, Shering PA. Blood pressure and cognitive function in healthy old people. *J Am Geriatrics Soc* 1993; **41**: 753–756.
5 Starr JM *et al*. Age-associated cognitive decline in healthy old people. *Age & Ageing* 1997; **26**: 295–300.
6 Starr JM *et al*. Blood pressure and mortality in healthy old people: the r-shaped curve. *BMJ* 1996; **313**: 1243–1244.

7 Mitchell GF *et al*. Sphygmomanometrically determined pulse pressure is a powerful independent predictor of recurrent events after myocardial infarction in patients with impaired left ventricular function. SAVE investigators. Survival and Ventricular Enlargement. *Circulation* 1997; **96**: 4254–4260.
8 Gardin JM *et al*. Left ventricular mass in the elderly. The Cardiovascular Health Study. *Hypertension* 1997; **29**: 1095–1103.
9 Boshuizen HC, Izaks GJ, van Buuren S, Ligthart GJ. Blood pressure and mortality in elderly people aged 85 and older: community based study. *BMJ* 1998; **316**: 1780–1784.
10 Hakala SM Tilvis RS. Determinants and significance of declining blood pressure in old age. A prospective birth cohort study. *Eur Heart J* 1998; **19**: 1872–1878.
11 Clausen J, Jensen G. Blood pressure and mortality: an epidemiological survey with 10 years follow-up. *J Hum Hypertens* 1992; **6**: 53–59.
12 Tuomilehto J *et al*. Low diastolic blood pressure and mortality in a population-based cohort of 16913 hypertensive patients in North Karelia, Finland. *J Hypertens* 1998; **16**: 1235–1242.
13 Kannel WB, D'Agostino RB, Silbershatz H. Blood pressure and cardiovascular morbidity and mortality rates in the elderly. *Am Heart J* 1997; **134**: 758–763.
14 Carstairs V, Morris R. Deprivation and Health in Scotland. Aberdeen University Press: Aberdeen, 1991.