Stroke

ORIGINAL CONTRIBUTION

# Cardiovascular Health and Stroke in Older British Men

## Prospective Findings From the British Regional Heart Study

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**BACKGROUND AND PURPOSE:** Research exploring the utility of cardiovascular health (CVH) and its Life's Simple 7 (LS7) components (body mass index, blood pressure [BP], glucose, cholesterol, physical activity, smoking, and diet) for prevention of stroke in older adults is limited. In the British Regional Heart Study, we explored (1) prospective associations of LS7 metrics and composite CVH scores with, and their impact on, stroke in middle and older age; and (2) if change in CVH was associated with subsequent stroke.

**METHODS:** Men without cardiovascular disease were followed from baseline recruitment (1978–1980), and again from re-examination 20 years later, for stroke over a median period of 20 years and 16 years, respectively. LS7 were measured at each time point except baseline diet. Cox models estimated hazard ratios (95% CI) of stroke for (1) ideal and intermediate versus poor levels of LS7; (2) composite CVH scores; and (3) 4 CVH trajectory groups (low-low, low-high, high-low, high-high) derived by dichotomising CVH scores from each time point across the median value. Population attributable fractions measured impact of LS7.

**RESULTS:** At baseline (n=7274, mean age 50 years), healthier levels of BP, physical activity, and smoking were associated with reduced stroke risk. At 20-year follow-up (n=3798, mean age 69 years) only BP displayed an association. Hazard ratios for intermediate and ideal (versus poor) levels of BP 0.65 (0.52–0.81) and 0.40 (0.24–0.65) at baseline; and 0.84 (0.67–1.05) and 0.57 (0.36–0.90) at 20-year follow-up. With reference to low-low trajectory, the low-high trajectory was associated with 40% reduced risk, hazard ratio 0.60 (0.44–0.83). Associations of CVH scores weakened, and population attributable fractions of LS7 reduced, from middle to old age; population attributable fraction of nonideal BP from 53% to 39%.

**CONCLUSIONS:** Except for BP, CVH is weakly associated with stroke at older ages. Prevention strategies for older adults should prioritize BP control but also enhance focus beyond traditional risk factors.

**Key Words:** blood pressure ■ cardiovascular health ■ cholesterol ■ life's simple 7 ■ middle age ■ older age ■ prevention ■ stroke



Stroke remains a major global cause of morbidity and mortality.[1] Its incidence rises sharply with age.[2] In the United Kingdom, >25 billion pounds ($30 billion) are spent annually on stroke care and stroke related disability. With more adults surviving to older ages, this burden is expected to increase.[3]

Primary prevention is seen as the best approach to reducing the burden of stroke.[4] The European Stroke Organisation and Stroke Alliance For Europe emphasize both risk factor modification and improved stroke risk assessment as means to improve primary prevention in their latest European Stroke Action Plan.[5] A risk factor

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## Nonstandard Abbreviations and Acronyms

| | |
|---|---|
| **AF** | atrial fibrillation |
| **BMI** | body mass index |
| **BP** | blood pressure |
| **BRHS** | British Regional Heart Study |
| **CVD** | cardiovascular disease |
| **CVH** | cardiovascular health |
| **HR** | hazard ratio |
| **LS7** | Life's Simple 7 |
| **MI** | myocardial infarction |
| **PA** | physical activity |
| **PAF** | population attributable fraction |
| **Q20** | 20-year follow-up |

based model of cardiovascular health (CVH) was developed by the American Heart Association in 2010 for prevention of cardiovascular disease (CVD) and stroke.[6] CVH is measured using 7 traditional health metrics: smoking, body mass index (BMI), physical activity (PA), diet patterns, total cholesterol, blood pressure (BP), and fasting glucose, referred to as Life's Simple 7 (LS7). Population prevalences of ideal, intermediate, or poor levels of each metric, and of summary CVH scores based on all 7 metrics, have been explored in association with a range of CVD outcomes[7,8] to identify metrics that can be targeted as part of health promotion programs.

Most studies, however, have either evaluated stroke as a combined end point within CVD[9–13]; are constrained by examining CVH at middle age[14,15]; or have limited follow-up.[13,15,16] Few have analyzed how CVH relates specifically to stroke in older populations.[17,18] Similarly, there is limited clarity on how transitions in CVH over time can influence stroke incidence at an older age.[19–21] These issues are essential to explore because associations between conventional risk factors and CVD weaken with age due to a selection of survivors.[22–24] Second, despite some shared risk factors, stroke epidemiology and etiology is somewhat distinct from broader CVD outcomes.[7,25] Stroke prevention strategies for older adults may hence require a different focus.

To assess the influence of CVH on incidence of stroke in older age, we used data from the BRHS (British Regional Heart Study), which has been following cardiovascular outcomes in a representative cohort of British men for >40 years. Our specific aims were to (1) compare associations between LS7 metrics, composite CVH scores, and stroke in middle and older age; (2) explore if change in CVH between middle and older age was associated with subsequent stroke incidence; and (3) determine the impact of LS7 metrics on the burden of stroke across middle and older age.

# METHODS

Data supporting the findings of this study are available from the study manager (Ms L Lennon; l.lennon@ucl.ac.uk) upon reasonable request.

The BRHS recruited 7735 men 40 to 59 years, from 24 primary care practices across Britain between 1978 and 1980.[26–28] Participants contributed sociodemographic, health, medication, and lifestyle data through questionnaires and underwent objective and lab-based examinations, including an ECG, at baseline[29,30] and 20-year follow-up (Q20).[27,28] This analysis used information on LS7 metrics collected at both time points together with CVD events and deaths to June 2018. All participants provided written informed consent in accordance with the Declaration of Helsinki. Ethical approval was obtained from relevant local research ethics committees.

## Assessment of CVH

LS7 metrics were measured objectively except for smoking, PA, and diet which were self-reported. Diet was measured at Q20 only.[31] Metrics were categorized as poor, intermediate, and ideal using American Heart Association definitions except smoking, PA and diet, which were classified using BRHS specific cut-offs[29,31–37] (details in Table I in the Data Supplement).

Composite CVH scores were sum of points (0, 1, 2, respectively) assigned to poor, intermediate, and ideal levels of each LS7 metric. CVH scores ranged from 0 to 12 at baseline (dietary information was not available) and 0 to 14 at Q20. Lower scores indicated poorer CVH. CVH categories[38] were derived from CVH score as inadequate (0–4 baseline and Q20), average (5–8 baseline, 5–9 Q20), and optimum (9–12 baseline, 10–14 Q20).

For capturing change in CVH from baseline to Q20, CVH trajectories were derived using CVH scores (exclusive of diet, range 0–12) from each time point. Scores were dichotomized using the median value. A score ≤7 was classed as low and >7 as high CVH; hence each participant belonged to one of 4 CVH trajectory groups: low-low, low-high, high-low, and high-high.

## Ascertainment of Stroke, Myocardial Infarction, and Mortality

Participants were followed up for mortality and nonfatal stroke and myocardial infarction (MI). Deaths were collected through National Health Service Central Registers in Southport (for England and Wales) and Edinburgh (for Scotland), with cause of death coded using the *International Classification of Diseases, Ninth Revision*. Fatal stroke was coded as 430–438 and fatal MI as 410–414.

Nonfatal events were ascertained from ongoing general practitioner reports and biennial reviews of participants' medical records.[27] Nonfatal MI was defined according to World Health Organization criteria[39] and nonfatal stroke as an event producing a neurological deficit for >24 hours. General practitioners were asked to review records of all surviving participants every 2 years and identify any nonfatal stroke on a standard form. In such cases, they were also asked to provide information on clinical presentation, hospital record summaries, and results of specific investigations where available, including brain scans. This material was reviewed by a BRHS clinical assessor, particularly to exclude any nonstroke diagnoses.

Analyses excluded men with prevalent CVD. Prevalent CVD at baseline (stroke, angina, coronary thrombosis, and MI) was determined from self-report of physician diagnosis; and at Q20 if a stroke or MI was noted in record review data before Q20.

### Covariates

Self-reported social class (manual, nonmanual and armed forces; based on longest held occupation) and alcohol intake (none, occasional, light, moderate, and heavy) were recorded at both waves.[28] Atrial fibrillation (AF) was recorded using a 12-lead ECG at Q20.

### Statistical Analyses

Descriptive statistics compared sociodemographic characteristics, LS7 metrics, composite CVH scores and stroke incidence per 1000 person years from baseline and Q20; as well as profiles of CVH trajectories. Cox proportional hazards models estimated hazard ratios (HRs) of stroke for individual LS7 metrics, CVH scores, and trajectory groups. Time to event was calculated from the baseline/Q20 date of examination to a stroke event or death, whichever came first. For participants with neither event, data was censored at the Q20 date for baseline and June 1, 2018 for Q20 analysis, respectively. Adjustments were made for social class at baseline and age and alcohol intake at the respective time point. Proportional hazards assumptions examined using Schoenfeld residuals were found to hold.

Associations were based on available complete cases. However, in sensitivity analyses, we investigated characteristics of men with missing covariates; and robustness of associations by assigning the worst possible LS7 level (poor) to those with missing data on any LS7.

Analyses were conducted using Stata software version 15 (StataCorp LLC, Texas). To explore how useful LS7 metrics were at discriminating between cases and noncases of stroke, we compared Harell's C statistics of multivariate models at baseline and Q20. We also compared the fraction of incident stroke attributable to individual LS7 metrics-population attributable fraction (PAF) at each time point, using the punafcc[40] package for Stata, evaluating the scenario where all participants had the metric in question at the ideal level.

We examined whether excluding men with prevalent heart failure (n=73, 2%) and those with ECG evidence of AF (n=122, 3%) at Q20 affected results; and further explored associations between LS7 metrics, CVH scores, trajectories and a CVD outcome of stroke and MI combined.

## RESULTS

### LS7 and Composite CVH Scores

There were 7274 men (mean age 50 years) without prevalent CVD at baseline. After a median follow-up of 19.8 years, 434 fatal and nonfatal stroke events occurred at a rate of 3.3/1000 person years. Among LS7, glucose (65%) was most and BP (9%) least prevalent at ideal levels. Mean composite CVH score was 6.3 (range, 0–12), with 71% men in the average and only 12% in the optimal CVH category (Figure 1 and Table II in the Data Supplement).

At Q20, there were 3798 men (mean age 69 years) without prevalent CVD. After a median follow-up of 15.7 years, there were 446 stroke events at a rate of 8.7/1000 person years. Among measured LS7, smoking status (84%) was most and BP (7%) least prevalent at ideal levels. Mean composite CVH score was 7.7 (range, 0–14; exclusive of diet: mean 6.8, range 0–12), with 76% of men in the average and 18% in the optimal CVH category (Figure 1 and Table II in the Data Supplement).

Cox regression of individual LS7 metrics (Table 1) revealed that at younger ages, healthier levels of BP, PA, and smoking status were associated with reduced risk of stroke. Compared with poor levels, adjusted HRs (95% CI) for intermediate and ideal levels were, respectively, 0.65 (0.52–0.81) and 0.40 (0.24–0.65) for BP; 0.79 (0.58–1.08) and 0.63 (0.45–0.88) for PA; and 0.69 (0.56–0.86) and 0.59 (0.45–0.78) for smoking. Favourable trends were also seen for better BMI and glucose levels although not statistically significant at a conventional cutoff of $P$=0.05. A unit increase in composite CVH score was associated with 16% reduced risk of stroke, adjusted HR 0.84 (0.79–0.89). Better categories of overall CVH were also protective for stroke: compared with the inadequate category, an average to optimal CVH status was associated with between a 40% to 60% reduction in HRs ($P$ for trend <0.0001).

At Q20, BP was the only LS7 metric showing a clear (but attenuated) association with subsequent stroke. Compared with poor BP, adjusted HRs for intermediate and ideal levels were 0.84 (0.67–1.05) and 0.57 (0.36–0.90), respectively, $P$ for trend 0.0168 (Table 1). Each unit increase in composite CVH score was associated with 5% reduced risk of stroke (adjusted HR, 0.95 [0.90–1.01]). Associations between CVH score categories and stroke similarly became weaker and nonsignificant, $P$ for trend 0.1394.

### Trajectories of CVH Between Baseline and Q20

A fifth of men maintained high CVH from baseline to Q20 (n=641), while more than half had persistently low CVH over the same period (n=1740). Five hundred and sixty three men improved their CVH from low to high, while CVH of 425 men deteriorated from high to low (Table 2). Incidence rates (95% CI) of stroke per 1000 person years were low-low 9.8 (8.6–11.2); low-high 6.2 (4.7–8.2); high-low 7.3 (5.5–9.8); and high-high 7.9 (6.3–9.8). In comparison to low-low, all remaining groups showed reduced probabilities of stroke (Figure 2). Low-high in particular had a 40% reduced stroke risk (adjusted HR, 0.60 [95% CI, 0.44–0.83]).

Excluding men with heart failure and AF at Q20 did not materially affect results of the above analyses.

Less than 3% of the men had missing data at baseline. At Q20, a maximum of 18% of the men had missing covariates. These men were slightly older with a higher incidence of stroke but with similar mean BPs and smoking habits to

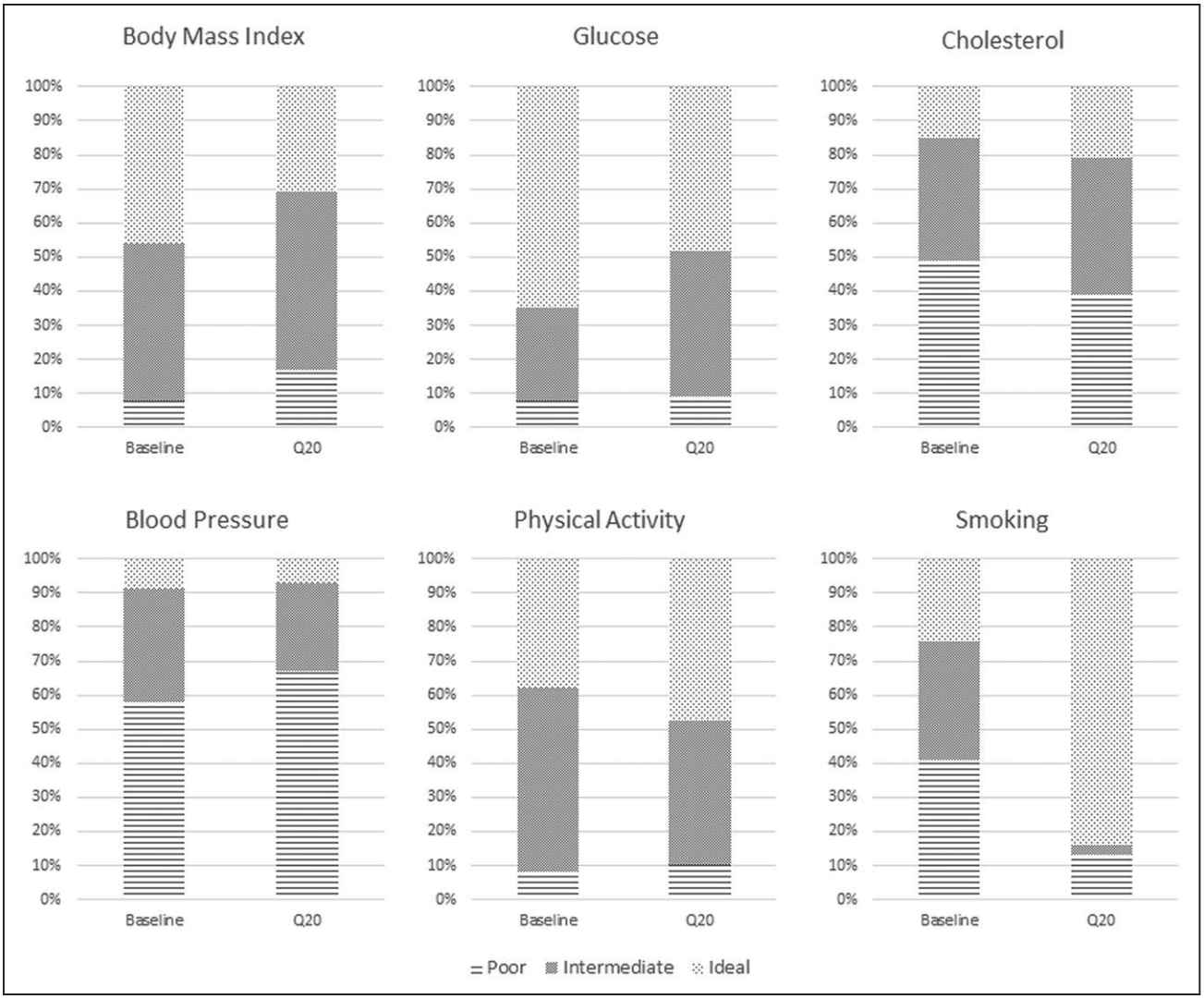


**Figure 1. Proportions of LS7 metrics at baseline and at 20 y follow-up (Q20) among men of the British Regional Heart study free of prevalent cardiovascular disease.**

those with complete data (Table III in the Data Supplement). The men had poorer CVH at baseline. However, associations were robust even in analyses that assumed a poor level for missing LS7 data (Table IV in the Data Supplement).

## Impact of LS7 Across Time

In multivariable models containing all LS7 metrics except diet; and adjusted for age, social class and alcohol intake, Harrell C statistic decreased from 0.7103 at baseline to 0.6548 at Q20. The PAFs of LS7 metrics also decreased over time (Table V in the Data Supplement). Notably, the burden of stroke that could be eliminated by control of high BP reduced from 53% in middle age to 39% in older age.

## CVH and a Combined CVD Outcome

All LS7 metrics measured at Q20, except BMI and cholesterol, showed significant associations in expected directions with MI and stroke combined (Table VI in the Data Supplement). In analysis comparing trajectories to low-low, all groups had significantly reduced risk of a combined CVD outcome with high-high and low-high groups having a risk reduction of similar magnitude.

# DISCUSSION

This prospective analysis assessed the associations and impact of CVH and its component LS7 metrics on stroke burden, during middle and older ages in a general population sample of British men free of CVD.

We noted that BP, PA, and smoking at baseline were associated with stroke in middle age, but only BP maintained a clear (albeit weaker) inverse association with stroke in later life. Others looking at older subjects[16,23,41] have established the influence of BP on stroke across the life-course. Our findings reaffirm its value as a key target in stroke prevention strategies. However, we



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**Table 1. Hazard Ratios [95% CI] for Stroke Among Men in the BRHS, Free of Prevalent CVD at Baseline and at Q20**

| | Baseline 1978–1980* | | Q20 1998–2000† | |
|---|---|---|---|---|
| | Model 1 | Model 2 | Model 1 | Model 2 |
| BMI | N=7273 | N=7256 | N=3783 | N=3717 |
| Poor | 1 | 1 | 1 | 1 |
| Intermediate | 0.79 [0.57–0.10] | 0.81 [0.58–1.13] | 1.20 [0.90–1.60] | 1.22 [0.91–1.62] |
| Ideal | 0.72 [0.52–1.00] | 0.75 [0.54–1.05] | 1.18 [0.87–1.60] | 1.19 [0.87–1.62] |
| *P* for trend | 0.0524 | 0.0950 | 0.2982 | 0.2720 |
| BP | N=7267 | N=7250 | N=3779 | N=3713 |
| Poor | 1 | 1 | 1 | 1 |
| Intermediate | 0.63 [0.51–0.79] | 0.65 [0.52–0.81] | 0.86 [0.69–1.08] | 0.84 [0.67–1.05] |
| Ideal | 0.38 [0.23–0.62] | 0.40 [0.24–0.65] | 0.57 [0.36–0.91] | 0.57 [0.36–0.90] |
| *P* for trend | 0.0001 | 0.0002 | 0.0190 | 0.0168 |
| Glucose | N=7228 | N=7211 | N=3590 | N=3528 |
| Poor | 1 | 1 | 1 | 1 |
| Intermediate | 0.86 [0.62–1.20] | 0.88 [0.63–1.23] | 0.87 [0.62–1.22] | 0.85 [0.61–1.20] |
| Ideal | 0.72 [0.53–0.98] | 0.74 [0.54–1.01] | 0.79 [0.56–1.11] | 0.78 [0.55–1.09] |
| *P* for trend | 0.0397 | 0.0617 | 0.1762 | 0.1467 |
| Cholesterol | N=7232 | N=7215 | N=3618 | N=3556 |
| Poor | 1 | 1 | 1 | 1 |
| Intermediate | 1.04 [0.85–1.27] | 1.03 [0.84–1.26] | 0.87 [0.70–1.08] | 0.85 [0.69–1.06] |
| Ideal | 0.87 [0.65–1.17] | 0.84 [0.63–1.14] | 1.07 [0.84–1.38] | 1.09 [0.85–1.40] |
| *P* for trend | 0.3625 | 0.2659 | 0.5800 | 0.4981 |
| Physical activity | N=7178 | N=7163 | N=3665 | N=3616 |
| Poor | 1 | 1 | 1 | 1 |
| Intermediate | 0.78 [0.57–1.06] | 0.79 [0.58–1.08] | 1.01 [0.71–1.44] | 1.02 [0.71–1.45] |
| Ideal | 0.59 [0.42–0.82] | 0.63 [0.45–0.88] | 0.86 [0.60–1.22] | 0.88 [0.62–1.26] |
| *P* for trend | 0.0018 | 0.0066 | 0.3900 | 0.4988 |
| Smoking | N=7260 | N=7244 | N=3792 | N=3727 |
| Poor | 1 | 1 | 1 | 1 |
| Intermediate | 0.66 [0.54–0.82] | 0.69 [0.56–0.86] | 0.57 [0.25–1.34] | 0.57 [0.25–1.34] |
| Ideal | 0.54 [0.41–0.71] | 0.59 [0.45–0.78] | 0.86 [0.64–1.14] | 0.86 [0.64–1.16] |
| *P* for trend | <0.0001 | 0.0002 | 0.2933 | 0.3266 |
| Elderly diet index | | | N=3512 | N=3452 |
| Poor | … | … | 1 | 1 |
| Intermediate | … | … | 1.08 [0.86–1.35] | 1.13 [0.89–1.42] |
| Ideal | … | … | 1.01 [0.78–1.30] | 1.06 [0.82–1.37] |
| *P* for trend | … | … | 0.9413 | 0.6780 |
| Composite CVH score | N=7112 | N=7097 | N=3177 | N=3135 |
| Per unit increase | 0.83 [0.78–0.87] | 0.84 [0.79–0.89] | 0.95 [0.90–1.00] | 0.95 [0.90–1.01] |
| CVH score categories‡ | | | | |
| Inadequate | 1 | 1 | 1 | 1 |
| Average | 0.56 [0.45–0.70] | 0.59 [0.47–0.73] | 0.71 [0.45–1.10] | 0.73 [0.46–1.15] |
| Optimal | 0.35 [0.23–0.54] | 0.39 [0.25–0.61] | 0.65 [0.40–1.07] | 0.68 [0.41–1.13] |
| *P* for trend | <0.0001 | <0.0001 | 0.0924 | 0.1394 |

Model 1 adjusted for age. Model 2 adjusted additionally for social class and alcohol intake at baseline/Q20. BMI indicates body mass index; BP, blood pressure; BRHS, British Regional Heart Study; CVD, cardiovascular disease; CVH, cardiovascular health; and Q20, 20y follow-up.

*Followed from baseline to Q20.

†Followed from Q20 to June 2018.

‡Inadequate: 0–4 baseline and Q20, average: 5–8 baseline/5–9 Q20, and optimal: 9–12 baseline/10–14 Q20.

**Table 2. Profiles of CVH Trajectories and Hazard Ratios [95% CI] for stroke Among Men in the BRHS, Free of Prevalent CVD at Q20**

| | CVH Trajectories Over 20 Years* | | | |
|---|---|---|---|---|
| | Low-Low (N=1740) | Low-High (N=563) | High-Low (N=425) | High-High (N=641) |
| Stroke events (n) | 217 | 48 | 46 | 78 |
| Incidence rate per 1000 PY (95% CI) | 9.8 (8.6–11.2) | 6.2 (4.7–8.2) | 7.3 (5.5–9.8) | 7.9 (6.3–9.8) |
| Age, y† | 69 (5.4) | 69 (5.5) | 67 (5.2) | 67 (5.4) |
| Social class | | | | |
| Nonmanual | 41 | 46 | 56 | 59 |
| Manual | 56 | 51 | 42 | 39 |
| Armed forces | 3 | 3 | 2 | 2 |
| BMI, kg/m$^2$† | 27.9 (3.8) | 25.6 (3.1) | 26.7 (3.0) | 24.8 (2.6) |
| Poor | 25 | 4 | 13 | 2 |
| Intermediate | 56 | 50 | 63 | 39 |
| Ideal | 19 | 46 | 23 | 58 |
| BP, mm Hg | | | | |
| Sitting systolic† | 156 (23) | 144 (23) | 152 (21) | 138 (24) |
| Sitting diastolic† | 87 (11) | 83 (10) | 88 (10) | 83 (11) |
| Poor | 79 | 51 | 77 | 44 |
| Intermediate | 19 | 37 | 21 | 39 |
| Ideal | 2 | 12 | 2 | 17 |
| Glucose, mmol/L† | 6.4 (2.2) | 5.5 (0.9) | 5.9 (1.0) | 5.5 (0.7) |
| Poor | 16 | 1 | 6 | 1 |
| Intermediate | 49 | 30 | 55 | 31 |
| Ideal | 36 | 69 | 40 | 68 |
| Cholesterol, mmol/L† | 6.3 (1.1) | 5.6 (0.9) | 6.2 (1.0) | 5.5 (0.9) |
| Poor | 52 | 15 | 53 | 17 |
| Intermediate | 36 | 50 | 36 | 43 |
| Ideal | 12 | 35 | 11 | 40 |
| Physical activity | | | | |
| Poor | 15 | 2 | 11 | 3 |
| Intermediate | 50 | 28 | 49 | 28 |
| Ideal | 35 | 70 | 40 | 70 |
| Smoking | | | | |
| Poor | 20 | 4 | 10 | 3 |
| Intermediate | 5 | 1 | 1 | 1 |
| Ideal | 75 | 95 | 88 | 97 |
| Composite CVH score†‡ | 5.7 (1.2) | 8.5 (0.7) | 6.3 (0.9) | 8.8 (0.9) |
| Hazard ratio (95% CI)§ | 1 | 0.60 (0.44–0.83) | 0.83 (0.60–1.15) | 0.86 (0.66–1.12) |

Profiles are as at Q20. Numbers are rounded percentages unless indicated. BMI indicates body mass index; BP, blood pressure; BRHS, British Regional Heart Study; CVD, cardiovascular disease; CVH, cardiovascular health; PY, person year; and Q20, 20y follow-up.

*N=3369, stroke events=389 for men followed from 1998/2000 to June 2018.

†Mean (SD).

‡Range 0–12 excluding diet.

§Adjusted for social class at baseline, age, and alcohol intake at Q20, N=3323.

highlight that the burden of stroke which can potentially be eliminated by achieving ideal BP control decreases with increasing age. Our PAF (39%) among older men (versus 53% at baseline), of BP higher than the ideal (of untreated 120/80 mm Hg) is similar to that estimated by the Rotterdam study[42] among men of a similar mean age (69y) as BRHS; and to the PAF of hypertension calculated (with a higher cutoff) among European participants of the INTERSTROKE study[43] (which also noted hypertension as a stronger risk factor in those <55 years). It is likely that even this (39%) is an overestimate, since in reality all men of older age are unlikely to attain ideal BP as defined by the American Heart Association. It has in fact been observed that among those ≥80

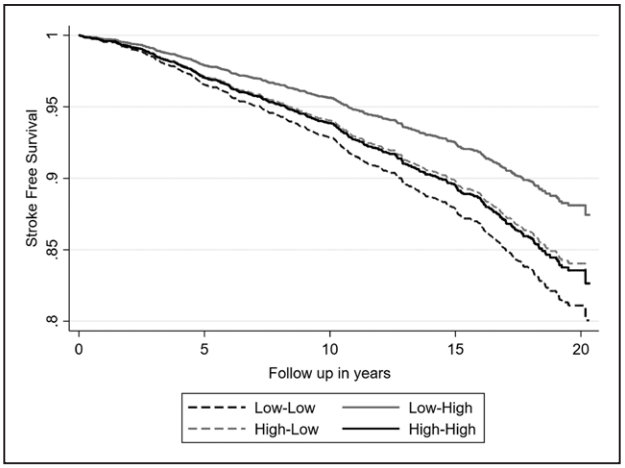


**Figure 2. Stroke free probability curves in cardiovascular health trajectory groups among men free of prevalent cardiovascular disease at 20 y of follow-up; adjusted for age, social class, and alcohol intake.**

years, the PAF for stroke due to hypertension becomes insignificant.[44]

Apart from BP, no other LS7 metrics individually influenced stroke in older men. Accordingly, higher (healthier) composite CVH scores at older ages offered weaker protection against stroke. The C statistic for our multivariate Q20 model, similar to that recorded by Dong et al[17] among participants of a comparable age, also reflects the weak ability of these metrics to jointly, correctly classify stroke events from nonevents at older age.

These findings underscore the need to optimize the detection and management of wider conditions known to increase the risk of stroke in older populations. Research from primary care in the United Kingdom indicates that both screening of AF among those >65 years[45] and its anticoagulation management among those >85 years[46] can be improved. More recently, aging related atrial cardiopathy has been linked to stroke independent of AF.[47] Other risk factors to direct prevention strategies towards include subclinical cardiac dysfunction[48] and impaired kidney function, which has been recently highlighted to increase in impact with increasing life span.[49]

Our analysis using a composite end point of MI and stroke observed that among older men, most individual LS7 metrics as well as CVH score categories exhibited clear expected associations with combined CVD. This may reflect greater influence of conventional factors in old age on coronary outcomes as opposed to stroke, and has also been noted among Swedish men.[41] It further suggests that health promotion targeting conventional factors such as LS7 among older adults would be less likely to reduce the burden of stroke in contrast to MI.

Nevertheless, the trajectory analysis indicated that the importance of adopting and maintaining a healthy lifestyle even in later life cannot be undermined. In previous work analyzing associations of change in CVH status with broad CVD outcomes, Enserro et al[21] using data from the Framingham Offspring study concluded that irrespective of whether they improved or not, people starting with low CVH status had higher rates of composite CVD compared with those maintaining high CVH throughout the study period. Analysis of Whitehall II data using a more precise categorization of CVH trajectories (9 groups) failed to show consistent associations with incident CVD.[19] In comparison, older BRHS men who improved CVH from low to high had reduced risk of stroke and MI combined, of a magnitude similar to that offered by maintaining high CVH throughout life. This suggests that later life CVH has a greater bearing on subsequent CVD. We further noted this benefit, although not as large, for stroke alone. Yang et al[50] do identify a similar protective trend against stroke from positive changes in CVH but among a younger Chinese cohort and over a shorter transition period. We must point out, however, that we cannot identify the exact time between baseline and Q20 when men may have transitioned in CVH status, or indeed, if there was >1 transition. Exposure durations may hence be variable and hazards may not accurately reflect this. Moreover, although similar to the creation of trajectory groups by others,[21] our binary CVH score cutoff is arbitrarily based on the median for both baseline and Q20.

Our study is novel in its exploration of CVH and its association with stroke as a specific outcome during both middle and older age within the same population; with near complete follow-up, over an extended period. Stroke capture has been reliable—the incidence rates of stroke during both middle and older age observed using the study protocol have been comparable with national data.[51,52] Furthermore, we based our analyses on the full range of CVH score (0–12/14) as opposed to only an aggregate of ideal LS7 metrics (0–6/7). This takes into account intermediate levels of a metric and may be more realistic for older ages when drug therapies for diabetes mellitus, dyslipidemia, and hypertension preclude ideal levels of these metrics. It is worth noting here that less than a fifth of our older men attained an optimal composite CVH score needing ≥5 metrics at the ideal level.

Men participating in the BRHS are of predominantly White ethnicity so generalizability of our findings to women and wider British population groups will be limited; however, findings are still relevant to a large section of the contemporary older population in the United Kingdom. Additionally, we lacked a measure of diet at baseline and in deriving CVH trajectories. However, the utility of an overall dietary score may be less consistent with respect to stroke/CVD.[14,16,18] Finally, we were unable to classify stroke into its subtypes and acknowledge that observed associations may not apply equally to ischemic and hemorrhagic stroke.

## CONCLUSIONS

With the exception of BP, CVH is weakly associated with stroke at older ages. Stroke prevention strategies should

ORIGINAL CONTRIBUTION

prioritize control of BP and energize efforts beyond traditional risk factors towards better detection and management of wider causes, including AF. Research into stroke prevention in older adults should also consider potential subclinical conditions such as cardiac and kidney dysfunction that can influence stroke burden.

## ARTICLE INFORMATION

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### Disclosures

None.

### Supplemental Materials

Expanded Materials and Methods
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