Web Appendix

Supp. Table I. Previous prospective cohort studies of the association between blood pressure and vascular dementia

Study	Size	Age	FU (yrs)	Overall results	Interpretation
Emdin et al. (Current study)	4.2 million individuals/10631 VaD cases	30-90	7.1	Strength of association per 20 mm Hg higher usual SBP diminished with increasing age category, from an HR of 1.62 (95% CI 1.13, 2.35) in the age group 30-50, to an HR of 1.26 (95% CI 1.18, 1.35) in the age group of 51-70, to an HR of 0.97 (95% CI 0.92, 1.03) in the age group 71-90 (p trend =0.006).	Increased SBP associated with increased VaD risk between ages of 30 to 70. No association at age 71-90.
Studies reporting a positive association					
Launer et al. 2001 [1]	3703 men/79 VaD cases	45-68 years	27	High blood pressure associated with OR 10.68 (CI 3.49–32.64) and OR 2.30 (CI 1.08–4.90) risk of VaD among untreated and treated patients	Increased midlife SBP associated with increased VaD risk.
Ninomiya et al. 2011[2]	668 participants/76 VaD cases	65-79 years	17	Late life and midlife blood pressure associated with increased risk of VaD (OR 6.94 CI 2.86, 16.88 for SBP >140 mm Hg at both late and mid life vs. SBP <140 mm Hg at both late and mid life).	Increased midlife and latelife SBP associated with increased VaD risk.
Posner et al. 2002[3]	1259 participants/56 VaD cases	65 years and older	7	History of hypertension associated with increased risk of incident VaD (HR 1.8 CI 1.0, 3.2)	History of hypertension associated with increased VaD risk.
Yamada et al. 2003 [4]	1932 participants/38 VaD cases	Most aged 30-50	25-30	10 mm Hg higher SBP was associated with OR 1.33 (1.14–1.56) for incident VaD.	Increased midlife SBP associated with increased VaD risk.
Yoshitake et al. 1995 [5]	826 participants/ 50 VaD cases	65 years and older	7	23 mm Hg higher SBP associated with RR 1.53 (1.16-2.01) for incidient VaD.	Late life SBP associated with increased VaD risk.
Studies reporting no association or inverse association					
Kuller et al. 2003[6]	2659 participants/128 VaD cases	65 years and older	7-8	Hypertension not associated with risk of incident VaD (HR 1.4 CI 0.96, 2.12)	Elevated blood pressure not associated with risk of VaD.
Verghese et al. 2003 [7]	488 participants/ 28 VaD cases	Older than 75 years	6.7	10 mm Hg lower SBP was associated with a HR 1.01 (CI 0.86, 1.19) for incident VaD.	Increased late life SBP not associated with VaD risk
Ruitenberg et al. 2001 [8]	6668 participants / 46 VaD cases	55 years and older	2.1	10 mm Hg higher SBP associated with a HR 0.93 (CI 0.82, 1.06) for incident VaD. 10 mm Hg higher SBP associated with HR 0.77 (CI 0.61, 0.97) for VaD among antihypertensive users	Increased SBP not associated with VaD risk in general population, inverse association in antihypertensive users
Skoog et al. 1996[9]	94 participants/7 VaD cases	70 years and older	15	No significant difference in baseline SBP or DBP between individuals who developed VaD and who did not	No association of SBP and DBP with incident VaD; underpowered to detect a difference with VaD

Footnote for Table 1: A 2005 systematic review examined the association between blood pressure and risk of dementia.[6] We updated this review, using a modified version of the search strategy in this review, with the substitution of the key word “vascular dementia” for “dementia”, and searching MEDLINE from January 1st, 2005 to 29th of April, 2015. We included all prospective studies of the association between blood pressure and vascular dementia, excluding cross-sectional studies and studies which examined the association between blood pressure and unspecified dementia.

Supp. Table II. Read codes used to identify vascular dementia cases in primary care records.

Read Code	Description
E004.00	Arteriosclerotic dementia
E004.11	Multi infarct dementia
E004000	Uncomplicated arteriosclerotic dementia
E004100	Arteriosclerotic dementia with delirium
E004200	Arteriosclerotic dementia with paranoia
E004300	Arteriosclerotic dementia with depression
E004z00	Arteriosclerotic dementia NOS
Eu01.00	Vascular dementia
Eu01.11	Arteriosclerotic dementia
Eu01000	Vascular dementia of acute onset
Eu01100	Multi-infarct dementia
Eu01111	Predominantly cortical dementia
Eu01200	Subcortical vascular dementia
Eu01300	Mixed cortical and subcortical vascular dementia
Eu01y00	Other vascular dementia
Eu01z00	Vascular dementia; unspecified

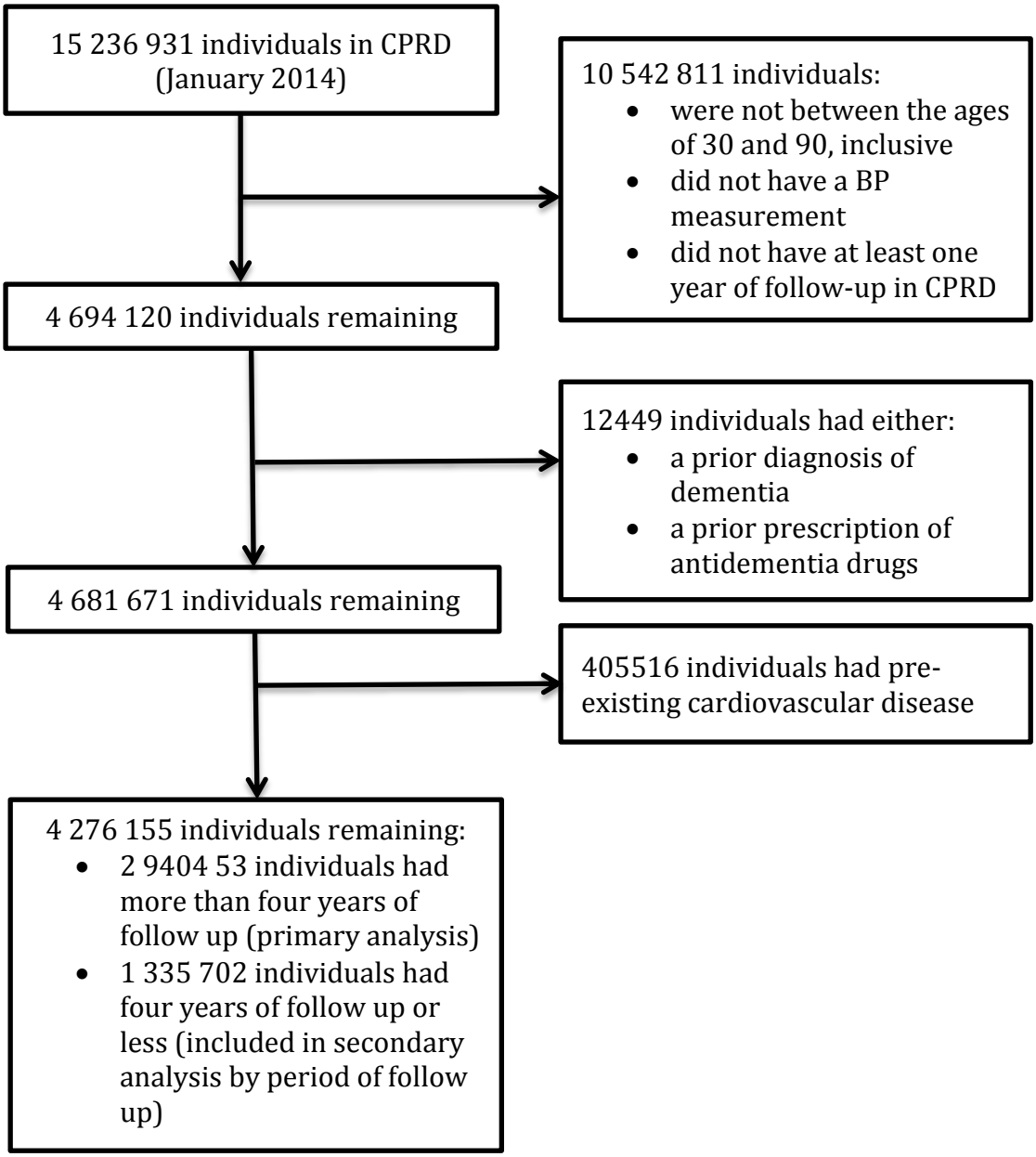
Supp. Table III. Characteristics of individuals included in the primary care cohort.

	≤120 mm Hg	**121-140 mm Hg**	**>140 mm Hg**	**Overall**
N	1 501 575 (35.1%)	1 596 814 (37.3%)	1 177 766 (27.5%)	4 276 155
Age at Baseline (IQ)	39 (33, 48)	47 (37, 58)	59 (48, 70)	46 (36, 59)
Women	996 669 (66.4%)	795 146 (49.8%)	584 795 (49.7%)	2 376 610 (55.6%)
BMI (IQI)	24.3 (21.9, 27.4)	26.3 (23.5, 29.7)	27.4 (24.4, 31.1)	25.8 (23.0, 29.3)
Smoking Status				
Current Smoker	376 132 (30.0%)	368 138 (28.2%)	228 638 (24.9%)	972 908 (28.0%)
Never Smoker	684 032 (54.7%)	702 882 (53.8%)	498 683 (54.3%)	1 885 597 (54.3%)
Ex Smoker	190 874 (15.3%)	234 436 (18.0%)	191 719 (20.9%)	617 029 (17.8%)
Cholesterol				
Total (IQI)	5.2 (4.5, 6.0)	5.4 (4.7, 6.2)	5.6 (4.9, 6.4)	5.5 (4.7, 6.2)
HDL (IQI)	1.1 (1.4, 1.7)	1.3 (1.1, 1.6)	1.4 (1.1, 1.6)	1.4 (1.1, 1.6)
Diabetes	26354 (1.8%)	53406 (3.3%)	57677 (4.9%)	137437 (3.2%)
Therapies				
Antihypertensive at Baseline	57340 (3.8%)	138517 (8.7%)	235591 (20.0%)	431448 (10.1%)
Antihypertensive During Follow-up	201900 (13.4%)	411889 (25.8%)	626585 (53.2%)	1240374 (29.0%)
Lipid lowering at Baseline	13814 (0.9%)	34118 (2.1%)	33343 (2.8%)	81275 (1.9%)
Lipid lowering During Follow-up	110576 (7.4%)	244029 (15.3%)	313696 (26.6%)	668301 (15.6%)

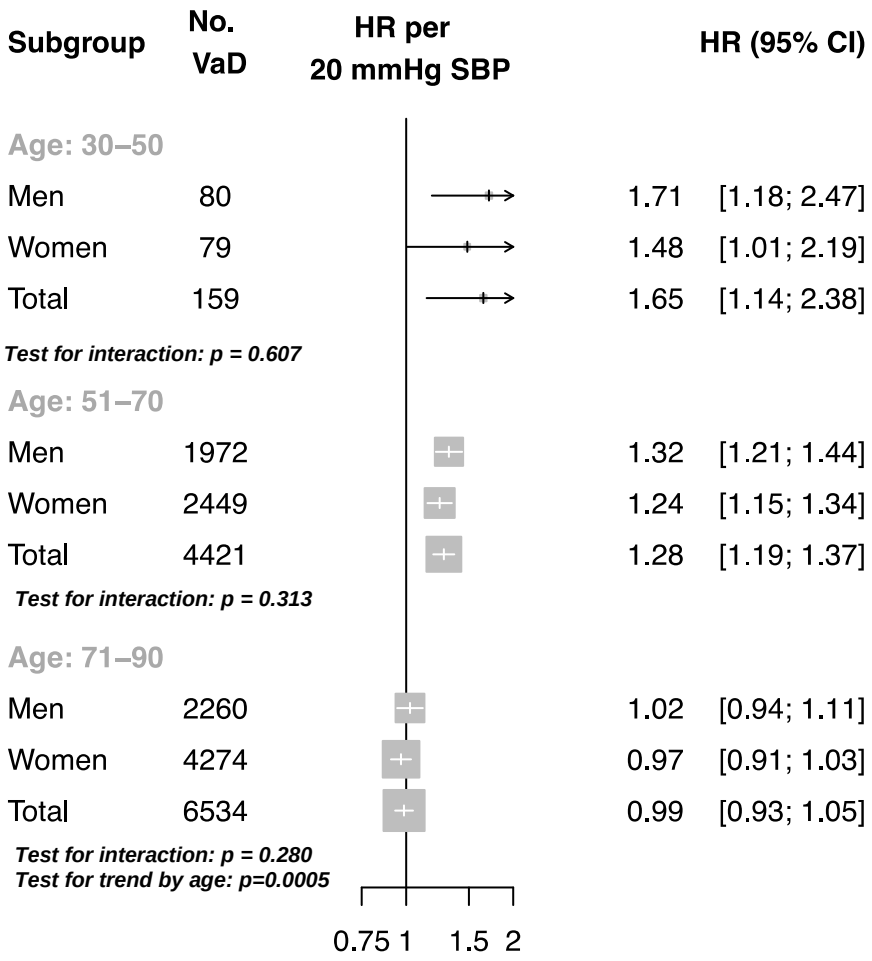
IQI refers to interquartile interval (25th percentile, 75th percentile). Proportion of variables missing: BMI (30.5%), smoking status (18.7%), total cholesterol (72.9%), HDL cholesterol (80.1%).

Supp. Table IV. Risk of dementia during follow-up (excluding premorbid and baseline dementia) in relation to mean pre-morbid blood pressure (hazard ratio per 20/10mmHg increase in mean SBP/DBP) readings in cohort of patients with a first TIA or stroke (OXVASC cohort) stratified by median age (approx. 75 years) and by measurement period (years prior to TIA/stroke) stratified by age, with two sensitivity analyses: 1. excluding patients on BP-lowering medication prior to the first TIA/stroke; 2. excluding dementia with onset after recurrent stroke.

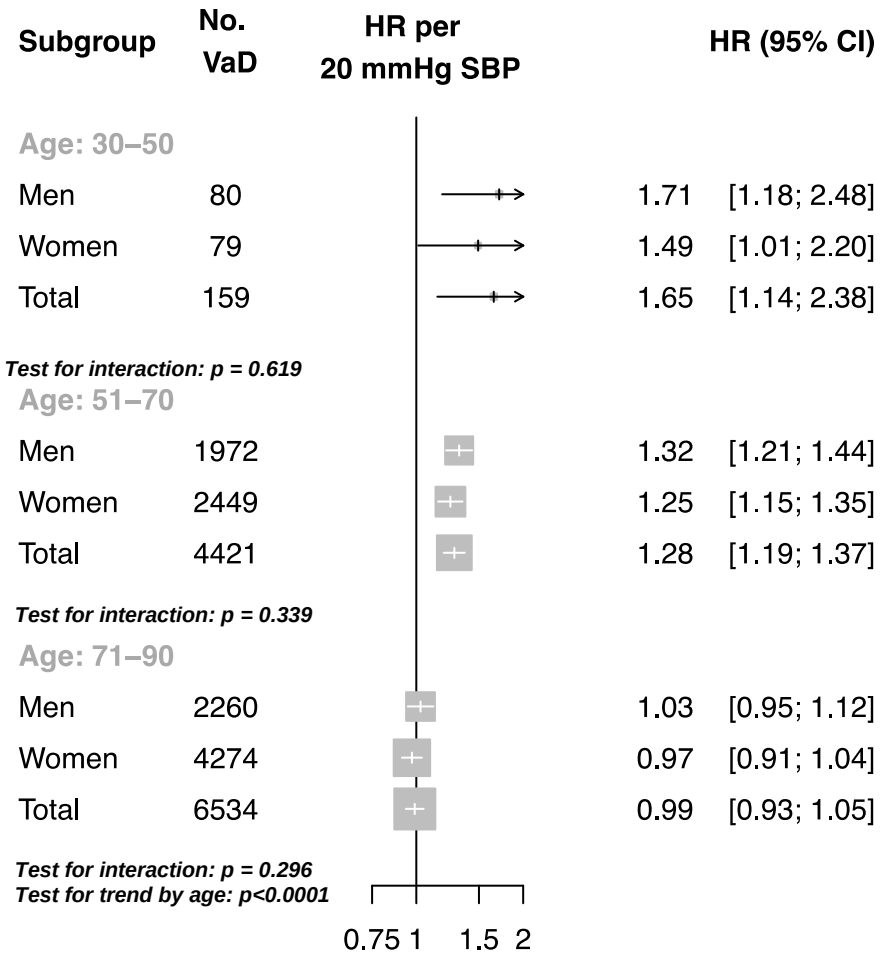
	All patients (n=1680; 314 events)				**Patients not on prior BP-lowering drugs (n=695; 92 events)**				**All patients (censored at recurrent stroke) (n=1680; 287 events)**			
	Unadjusted		Adjusted		Unadjusted		Adjusted		Unadjusted		Adjusted	
	HR (95% CI)	p	HR (95% CI)	p	HR (95% CI)	p	HR (95% CI)	p	HR (95% CI)	p	HR (95% CI)	p
Age<75years												
Mean SBP												
10-20 years ago	1.70 (1.29-2.25)	0.0002	1.51 (1.12-2.05)	0.0075	2.44 (1.40-4.25)	0.0017	2.11 (1.15-3.88)	0.017	1.63 (1.21-2.19)	0.0011	1.45 (1.05-2.00)	0.025
5-9 years ago	1.48 (1.10-2.00)	0.0096	1.26 (0.91-1.74)	0.16	1.47 (0.85-2.54)	0.17	1.23 (0.69-2.19)	0.49	1.41 (1.03-1.93)	0.032	1.21 (0.86-1.69)	0.28
<5 years ago	1.24 (1.02-1.50)	0.029	1.14 (0.95-1.37)	0.16	1.32 (0.97-1.80)	0.073	1.23 (0.92-1.64)	0.17	1.26 (1.02-1.56)	0.029	1.17 (0.95-1.43)	0.13
Most recent	1.32 (1.04-1.68)	0.024	1.19 (0.93-1.53)	0.17	1.79 (1.22-2.63)	0.0032	1.63 (1.09-2.44)	0.018	1.34 (1.04-1.73)	0.025	1.22 (0.94-1.59)	0.14
Mean DBP												
10-20 years ago	1.47 (1.11-1.94)	0.0076	1.32 (0.99-1.77)	0.063	1.87 (1.12-3.10)	0.016	1.67 (0.98-2.85)	0.061	1.41 (1.05-1.89)	0.024	1.27 (0.93-1.73)	0.13
5-9 years ago	1.09 (0.81-1.47)	0.57	1.08 (0.79-1.46)	0.64	0.89 (0.52-1.54)	0.69	0.86 (0.48-1.51)	0.59	1.07 (0.79-1.46)	0.66	1.05 (0.76-1.46)	0.75
<5 years ago	1.08 (0.95-1.22)	0.25	1.05 (0.92-1.20)	0.47	1.15 (0.94-1.41)	0.18	1.12 (0.91-1.39)	0.29	1.09 (0.95-1.26)	0.21	1.07 (0.92-1.25)	0.37
Most recent	1.01 (0.81-1.27)	0.91	1.04 (0.83-1.31)	0.72	1.40 (0.98-1.99)	0.064	1.37 (0.97-1.93)	0.079	1.03 (0.81-1.30)	0.83	1.05 (0.83-1.34)	0.67
Age≥75years												
Mean SBP												
10-20 years ago	1.19 (0.99-1.43)	0.059	1.16 (0.96-1.39)	0.12	1.57 (1.05-2.35)	0.028	1.41 (0.93-2.14)	0.10	1.17 (0.97-1.41)	0.098	1.14 (0.94-1.37)	0.18
5-9 years ago	1.32 (1.11-1.56)	0.0014	1.27 (1.07-1.50)	0.0057	1.38 (0.99-1.93)	0.058	1.22 (0.87-1.73)	0.25	1.30 (1.09-1.55)	0.0035	1.25 (1.05-1.50)	0.012
<5 years ago	1.00 (0.91-1.09)	0.93	1.00 (0.91-1.09)	0.95	1.01 (0.86-1.18)	0.89	1.00 (0.86-1.16)	1.00	1.01 (0.91-1.11)	0.91	1.01 (0.91-1.11)	0.90
Most recent	0.94 (0.83-1.08)	0.38	0.93 (0.82-1.07)	0.31	0.99 (0.74-1.32)	0.96	0.97 (0.74-1.27)	0.82	0.95 (0.83-1.09)	0.48	0.94 (0.82-1.08)	0.41
Mean DBP												
10-20 years ago	1.14 (0.96-1.36)	0.14	1.15 (0.97-1.37)	0.11	1.65 (1.08-2.52)	0.019	1.54 (1.02-2.34)	0.041	1.11 (0.93-1.33)	0.25	1.12 (0.94-1.35)	0.21
5-9 years ago	1.25 (1.04-1.50)	0.017	1.25 (1.04-1.50)	0.015	1.60 (1.09-2.34)	0.015	1.53 (1.04-2.24)	0.031	1.23 (1.01-1.49)	0.035	1.23 (1.02-1.49)	0.033
<5 years ago	1.00 (0.91-1.09)	1.00	1.02 (0.93-1.11)	0.70	1.02 (0.88-1.18)	0.83	1.03 (0.88-1.21)	0.69	1.00 (0.91-1.10)	0.95	1.02 (0.93-1.12)	0.68
Most recent	0.99 (0.88-1.12)	0.90	1.01 (0.89-1.15)	0.85	1.03 (0.79-1.34)	0.83	1.08 (0.83-1.40)	0.58	0.97 (0.86-1.11)	0.70	0.99 (0.87-1.13)	0.93



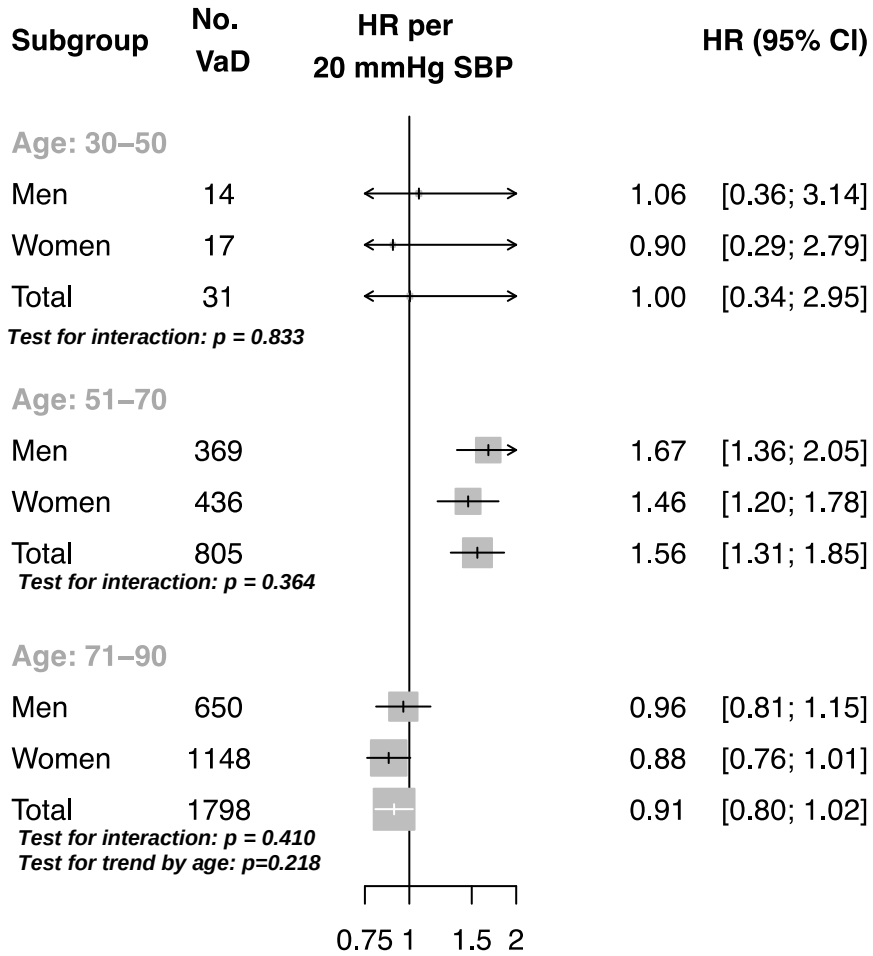
Supp. Figure I Flow chart of identification of individuals for analysis in the primary care cohort.



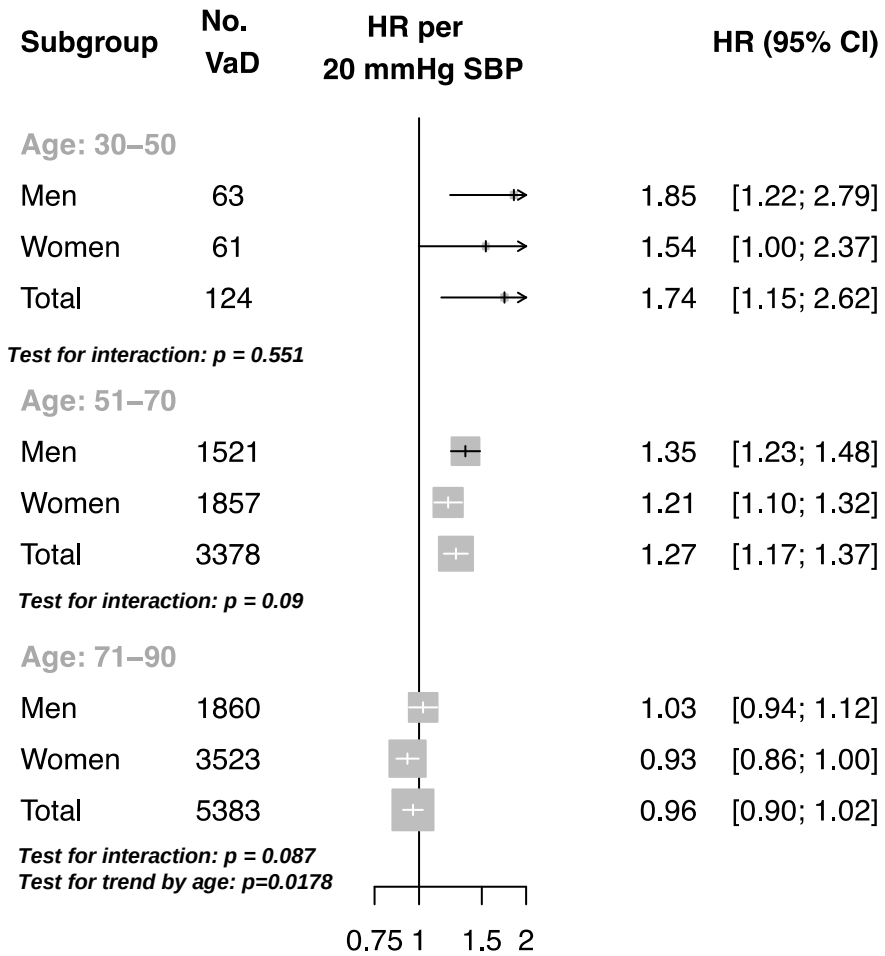
Supp. Figure II. Risk of vascular dementia per 20 mm Hg higher usual systolic blood pressure in the primary care cohort. Models adjusted for BMI, gender, smoking status, age categories, an interaction term of age categories with continuous age, an interaction term of age categories with usual systolic blood pressure (plotted) and total cholesterol, HDL cholesterol and diabetes.



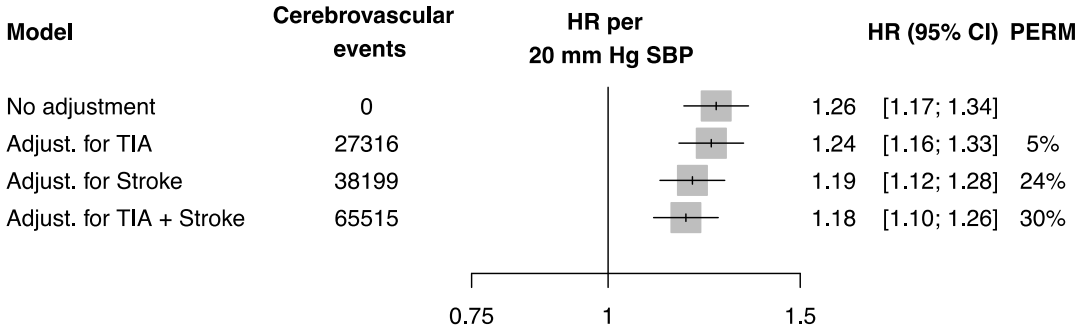
Supp. Figure III. Risk of vascular dementia per 20 mm Hg higher usual systolic blood pressure in the primary care cohort. Models adjusted for BMI, gender, smoking status, age categories, an interaction term of age categories with continuous age, an interaction term of age categories with usual systolic blood pressure (plotted) and total cholesterol, HDL cholesterol, diabetes and baseline period of blood pressure measurement.



Supp. Figure IV. Risk of vascular dementia per 20 mm Hg higher usual systolic blood pressure with exclusion of individuals prescribed antihypertensive therapies or lipid lowering therapies in the primary care cohort. Models adjusted for BMI, gender, smoking status, age categories, an interaction term of age categories with continuous age and an interaction term of age categories with usual systolic blood pressure (plotted).



Supp. Figure V. Risk of vascular dementia per 20 mm Hg higher usual systolic blood pressure with exclusion of individuals diagnosed with vascular dementia and Alzheimer's disease as well as those prescribed antidementia drugs in the primary care cohort. Models adjusted for BMI, gender, smoking status, age categories, an interaction term of age categories with continuous age and an interaction term of age categories with usual systolic blood pressure (plotted).



Supp. Figure VI. Risk of vascular dementia after progressive adjustment for future transient ischemic attack and future stroke among individuals 70 years or less at baseline in the primary care cohort. Models adjusted for blood pressure (plotted), age, sex, smoking status and BMI, and TIA and stroke where labeled.

Supplementary Methods

Regression Dilution Bias

Measurement error and within-individual variation in a risk factor can reduce the magnitude of estimated associations between a given risk factor and future events. In order to correct for this "regression dilution bias", regression dilution ratios were calculated by regressing serial measurements of blood pressure within the median follow-up on baseline values using generalized estimate equations. Calculated hazard ratios for measured blood pressure were then multiplied by the inverse of these ratios (attenuation factor), to estimate the association for usual blood pressure. Attenuation factors of 2.17 and 2.57 were calculated for systolic and diastolic blood pressures respectively. To graph floating absolute risks against usual blood pressure, measured blood pressure was "shrunk" towards the overall mean blood pressure by the calculated regression dilution ratios, as has been performed previously.[10]

Mediating effects of TIA and stroke

To investigate to what extent the potential association between blood pressure and vascular dementia was mediated by stroke or TIA, we adjusted for occurrence of stroke or TIA during follow up (that is, either before censoring for individuals who were not diagnosed with vascular dementia or before diagnosis of vascular dementia for individuals who were diagnosed with vascular dementia during follow-up). A stroke or TIA event was defined from either diagnosis within primary care (defined through 15 Read codes[11]), diagnosis through cause-specific hospitalization or diagnosis through cause-specific mortality (defined through the ICD 10 codes: I60, I61, I63, I64, G46.3-G46.7, I67.2, I67.9 for stroke, and G45.8, G45.9 for TIA). To determine what proportion of the association between usual blood pressure and risk of vascular dementia was mediated by future stroke and transient ischemic attack, we calculated the percentage of excess risk mediated (PERM) using the formula: $PERM = (HR_{(confounder\ adjusted)} - HR_{(confounder\ adjusted\ and\ mediator\ adjusted)})/(1-HR_{(confounder\ adjusted)})$. For the analysis of the mediation of the risk of vascular dementia by stroke or TIA, only the first stroke or TIA was counted (that is, we did not adjust for multiple strokes or TIAs, but adjusted for whether an individual had a stroke or TIA during follow up).

Oxford Vascular Study Methods

Patients with TIA or stroke were prospectively recruited from 1st April 2002-31st March 2007 into the Oxford Vascular Study (OXVASC), a prospective population-based cohort study of all acute vascular events occurring within a defined population of 92 728 covered by nine primary care practices in Oxfordshire, UK.[10,11] The study was approved by the local research ethics committee. Informed written consent (or assent from relatives) was obtained for study interview, for face-to-face and telephone follow-up and for indirect follow-up using primary care physician records, hospital records and death certificate data. Where patients died before first assessment or where assent from a family member could not be obtained in patients lacking capacity, the ethics committee approved review of the patient's medical records.

The study methods have been described in detail elsewhere.[10-13] Patients were ascertained after index TIA or stroke by study clinicians through a combination of hot and cold pursuit.[10-12] TIA and stroke were defined clinically by WHO criteria.[5] Baseline brain and vascular imaging was performed and all cases were reviewed by a senior vascular neurologist (PMR). Patient data were collected at study entry by interview using a standardised form and cross-referenced with primary care

records.[10-12] Functional status was assessed using modified Rankin[15] and Barthel[16] scores.

The lifetime medical record held by the primary care physician was manually reviewed and all recorded premorbid BPs ascertained. All BP readings recorded in primary care records from 1990 onwards were used for determination of premorbid BP, with sensitivity analyses using readings during the last 5 years or from 5-10 years before the event.

Cognitive screening and diagnosis of dementia
Follow-up interviews were done by trained clinical research fellows or nurses at 1 and 6 months and 1 and 5 years either in a hospital clinic or by home visit where hospital visit was not possible. Telephone follow-up was done when face-to-face follow-up was not possible.

Cognitive testing was done at all follow-ups using a combination of MMSE,[17] TICSm[18] and MoCA,[19] all of which we have validated against the National Institute of Neurological Disorders and Stroke-Canadian Stroke Network (NINDS-CSN) Vascular Cognitive Impairment Harmonisation Standards Neuropsychological Battery in the OXVASC population.[20-23] Since we and others have shown that exclusion of patients unavailable for clinic follow-up substantially reduces the measured rate of post-event dementia,[24-27] telephone testing using the TICSm or telephone MoCA (out of 12)[23] was used in patients who were not available for face-to-face assessment (e.g. moved away from study area).

Dementia was defined as pre- or post-event according to whether the diagnosis was made before or after the index event.[13] Post-event dementia diagnosis required MMSE<24[28] and remaining <24 for all subsequent follow-ups or MoCA<20[29] or TICSm<22 or Telephone-MoCA<9.[23] For subjects with an incomplete test (eg dysphasia, visual impairment, inability to use the dominant arm) individual patient study records and primary care information were reviewed. For patients without a direct face-to-face study assessment, post-event dementia was diagnosed by specialist review of all primary and secondary care medical record.[13,27]

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