ORIGINAL ARTICLE

Self-reported Age of Hypertension Onset and Hypertension-Mediated Organ Damage in Middle-Aged Individuals

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BACKGROUND
Objectively defined early onset hypertension, based on repeated blood pressure measurements, is a strong risk factor for cardiovascular disease (CVD). We aimed to assess if also self-reported hypertension onset age is associated with hypertension-mediated organ damage (HMOD). Additionally, we evaluated the agreement between self-reported and objectively defined hypertension onset age.

METHODS
We studied 2,649 participants (50 ± 4 years at the time of outcome assessment, 57% women) of the Coronary Artery Risk Development in Young Adults (CARDIA) study who underwent measurements for echocardiographic left ventricular hypertrophy (LVH), left ventricular diastolic dysfunction (LVDD), coronary calcification, and albuminuria. We divided the participants into groups according to self-reported hypertension onset age (<35 years, 35–44 years, ≥45 years, and no hypertension). We used multivariable-adjusted logistic regression models to assess the relation between self-reported hypertension onset age with the presence of HMOD, with those who did not report hypertension as the referent group.

RESULTS
Compared with individuals without self-reported hypertension, self-reported hypertension onset at <35 years was associated with LVH (odds ratio (OR), 2.38; 95% confidence interval (CI), 1.51–3.76), LVDD (OR, 2.32; 95% CI, 1.28–4.18, coronary calcification (OR, 2.87; 95% CI, 1.50–5.47), and albuminuria (OR, 1.62; 95% CI, 0.81–3.26). Self-reported hypertension onset at ≥45 years was only associated with LVDD (OR, 1.81; 95% CI, 1.06–3.08). The agreement between self-reported and objectively defined hypertension onset age groups was 78–79%.

CONCLUSIONS
Our findings suggest that self-reported hypertension onset age, a pragmatically feasible assessment in clinical practice, is a reasonable method for assessing risk of HMOD and CVD.

Keywords: blood pressure; epidemiology; hypertension; organ damage; risk factors; self-report

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There is an ongoing need for pragmatic and efficient approaches to refining our assessment of hypertension associated risks in clinical practice. Despite some limitations with respect to precision, self- or interviewer-administered questionnaires are known to be accurate and useful for determining hypertension status and other health-related factors in practice as well as in epidemiological research. In prior studies that have evaluated whether use of self-reported hypertension is a reliable method for diagnosing hypertension,[1–5] the observed agreement of self-reported hypertension with medical record- or examination-based hypertension has varied between 69% and 88%. Importantly,

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the specificity of self-reported hypertension has generally been reported to be high, despite the sensitivity being low with a high level of between-study variation.

Hypertension-mediated organ damage (HMOD) increases the risk of overt cardiovascular disease (CVD) considerably.[6–9] A limited number of prior studies have demonstrated that objectively defined early onset of hypertension (i.e., based on repeated, objective measurements) is strongly associated with increased risk of HMOD and CVD death, whereas late onset hypertension is not.[10–12] However, no studies have compared the agreement between self-reported and objectively defined hypertension onset age, or their relation with HMOD. As blood pressure (BP) data spanning decades are not usually available in regular clinical practice, physicians often need to rely on self-report to determine the age of hypertension onset. It is therefore important to investigate the association between self-reported age of hypertension onset and HMOD to understand if self-reported hypertension onset age can be used for improving risk assessment in patients with hypertension.

In this study, we studied a sample of 2,649 Coronary Artery Risk Development in Young Adults (CARDIA) study participants who underwent repeated BP measurements and determination of left ventricular hypertrophy (LVH), left ventricular diastolic dysfunction (LVDD), coronary calcification, and albuminuria. The participants also reported their age at the time of hypertension diagnosis. Our focus was to determine if individuals with self-reported early onset hypertension, a pragmatically feasible assessment in clinical practice, have an increased risk for HMOD compared with individuals with self-reported late onset hypertension. In addition, we aimed to assess the agreement between self-reported and objectively defined hypertension onset age.

METHODS

Study population

We included participants from the multicenter CARDIA study. The original CARDIA cohort involved 5,115 participants, recruited between 1985 and 1986, with mean age at baseline (Year 0) 25 ± 4 years (range 18–30). The participants were selected evenly by sex, race, education, and age groups across all 4 study centers in Birmingham, AL; Chicago, IL; Minneapolis, MN; and Oakland, CA. Details of the CARDIA study protocol have been reported previously.[13] Follow-up exams were conducted 2, 5, 7, 10, 15, 20, and 25 years after baseline. For the current study, we included individuals who participated in the Year 25 exam of the CARDIA study in 2010–2011 (n = 3,499). The final study sample included 2,649 individuals, after exclusion of participants with missing covariate or outcome data (n = 819), or information on self-reported hypertension status (n = 41). The CARDIA study was approved by institutional committees in each participating center and all participants provided informed consent.

Data acquisition

BP was measured 3 times at all follow-up exams between 1985–1986 and 2010–2011 as previously described.[12,14] In order to attain comparable BP values, the oscillometric values obtained during the last 2 exams were calibrated to sphygmomanometer values with a previously introduced formula.[15] At the Year 25 exam, measurements for echocardiographic left ventricular mass and pulse wave recordings for early and late phase peak velocities, coronary artery calcification score and urine albumin–creatinine ratio were conducted on all participants. Standardized protocols across all study centers were used for echocardiographic measurements, performed with a 2-dimensionally guided M-mode and Doppler echocardiography. All echocardiograms were analyzed centrally by trained echocardiographic sonographers and left ventricular mass along with pulse wave Doppler recordings of peak velocity flow in early and late diastole was calculated from the echocardiograms.[16] We defined left ventricular mass index as left ventricular mass divided by body surface area [$0.007184 \times$ weight $(kg)^{0.425} \times$ height $(cm)^{0.725}$]. Urinary creatinine and albumin were measured from single, untimed spot urine samples, which were centrally assayed by standard procedures.[17] Coronary artery calcification score was measured using a cardiac multidetector computed tomography and Agatston score was calculated for each as previously described.[18]

Use of antihypertensive medication and smoking status was collected with self-administrated questionnaires. Use of medications was also documented according to any medications brought on examination site. History of preeclampsia or high BP during pregnancy without other symptoms of preeclampsia was self-reported by women at all follow-up visits. Serum glucose, total cholesterol, and high-density lipoprotein cholesterol were quantified from fasting samples.[13,19]

Definitions and subgrouping

Self-reported hypertension onset age was determined using a self-administered questionnaire at the Year 25 exam from replies to the following questions: "Has a doctor or nurse ever said that you have high blood pressure or hypertension?" and "At what age were you first told this?" Objectively defined hypertension onset age was determined as BP ≥140/90 mm Hg or use of antihypertensive agents at 2 consecutively attended exams (at 0, 2, 5, 7, 10, 15, 20, or 25 years of follow-up). When hypertension onset was based on 2 consecutive exams, the age at the first examination on which the criteria for hypertension were met was considered as the age of hypertension onset, consistent with previous studies.[11,12,20] We also used an alternative definition for objectively defined hypertension onset age which required high BP (≥140/90 mm Hg) or use of antihypertensive agents at only one exam. We formed subgroups based on the participants' self-reported age at hypertension onset (<35 years, 35–44 years, ≥45 years, or no hypertension).[12] We defined albuminuria as urine albumin–creatinine ratio >30 mg/g[21] and coronary calcification as Agatston score ≥100.[22] The presence of LVH was defined as left ventricular mass index >115 g/m^2 in men and left ventricular mass index >95 g/m^2 in women.[23] For LVDD, we used the cutoff values for peak velocity flow ratio between early and late diastole as >2.0 or <0.8.[24] We defined diabetes as use of

antihyperglycemic medication or detected serum fasting glucose of ≥7 mmol/l.

Statistical analyses

We examined the participants' characteristics at the Year 25 exam in the whole study sample and by subgroups based on self-reported hypertension onset age. We also compared the baseline (Year 0) characteristics between participants who were included in the study sample and those who were excluded to evaluate the potential for selection bias. We examined the prevalence of HMODs according to hypertension onset age in the whole study sample and additionally by each race–sex group. We used 1-way analysis of variance for continuous variables and chi-squared test for categorial variables to compare the characteristics between the groups. Urine albumin–creatinine ratio was log-transformed for analyses due to skewed distribution. We examined the relation between hypertension onset age and presence of HMOD in a case–control (presence of HMOD vs. no HMOD) study setting. Participants who did not report having hypertension were considered as the referent group. We used univariable and multivariable logistic regression models to study the relation between self-reported age of hypertension onset groups and HMOD. We also assessed the linear trend between age of hypertension onset strata and HMOD by entering the strata in the models as a continuous variable. We included conventional HMOD risk factors, i.e., age, sex, race, total serum cholesterol, high-density lipoprotein cholesterol, body mass index, diabetes, smoking status, use of antihypertensive medication, and systolic BP as covariates in the multivariable-adjusted analyses. Covariate and HMOD data were drawn from the Year 25 exam. We assessed the agreement between self-reported and objectively defined hypertension using weighted kappa coefficients. The kappa weights were constructed, and the weighted kappa coefficients were computed according to the standard settings of SAS software version 9.4. We performed a sensitivity analysis on the agreement between self-reported and objectively defined hypertension using an alternative definition of hypertension onset. In these analyses, objectively defined hypertension onset was based on high BP (BP ≥140/90 mm Hg or use of antihypertensive medication) on a single exam, instead of on 2 consecutive exams. We also performed a subgroup analysis to determine the agreement between self-reported and objectively defined hypertension onset age among individuals without antihypertensive medication. We performed all statistical analyses with SAS version 9.4 (SAS Institute, Cary, NC). We considered 2-sided P values <0.05 as statistically significant.

RESULTS

The participants' characteristics at the Year 25 exam in subgroups by self-reported hypertension onset age are shown in Table 1. The mean age of the study sample was 50 ± 4 years (range 43–55 years), 57% were women, and 48% were black. Participants were more likely to be women and have diabetes in the early onset than in the late onset hypertension group. There were more black participants in the early than in the late onset group. Of the participants with hypertension onset <35 years, 27% self-reported history of having preeclampsia and 42% of having high BP during pregnancy. In all individuals with hypertension, the corresponding prevalence rates were 16% and 19%, respectively. The baseline characteristics of the study sample and the excluded participants were largely similar (Supplementary Table S1 online). Overall, the mean level and prevalence of LVH, LVDD, coronary calcification, and albuminuria measured at the Year 25 exam statistically significantly differed between the subgroups of hypertension onset age ($P < 0.01$ for all, Table 2). The prevalence of all HMODs was highest in the group with reported hypertension onset under 35 years of age (Table 2). The prevalence of HMODs by

Table 1. Study sample characteristics at Year 25

| Characteristic | All | Self-reported HTN onset age | | | | P value |
		<35 y	35–44 y	≥45 y	No HTN	
N	2,649	194	297	340	1,818	
Age, years (SD)	50.1 (3.6)	49.8 (3.7)	49.0 (3.7)	51.8 (2.7)	50.0 (3.6)	<0.001
No. women (%)	1,509 (57.0)	122 (62.9)	175 (58.9)	191 (56.2)	1,021 (56.2)	0.28
Black (%)	1,262 (47.6)	139 (71.7)	216 (72.7)	201 (59.1)	706 (38.8)	<0.001
BMI, kg/m^2 (SD)	25.2 (5.5)	27.8 (6.1)	27.8 (5.8)	27.0 (5.8)	24.2 (5.0)	<0.001
Current smoker (%)	440 (16.6)	34 (17.5)	57 (19.2)	72 (21.2)	277 (15.2)	0.028
Diabetes (%)	236 (8.9)	43 (22.2)	54 (18.2)	52 (15.3)	87 (4.8)	<0.001
Cholesterol, mmol/l (SD)	5.0 (0.9)	4.9 (1.1)	4.8 (0.9)	4.9 (1.0)	5.1 (0.9)	<0.001
HDL, mmol/l (SD)	1.5 (0.5)	1.5 (0.5)	1.4 (0.4)	1.5 (0.4)	1.6 (0.5)	<0.001
Systolic blood pressure, mm Hg (SD)	118 (15.3)	126 (16.5)	126 (16.4)	126 (16.8)	115 (13.3)	<0.001
Diastolic blood pressure, mm Hg (SD)	73.7 (10.8)	79.2 (12.0)	79.4 (11.0)	78.4 (10.9)	71.3 (9.8)	<0.001
Use of antihypertensive medication (%)	677 (25.6)	150 (77.3)	248 (83.5)	254 (74.7)	25 (1.4)	<0.001

Abbreviations: BMI, body mass index; HDL, high-density lipoprotein; HTN, hypertension; SD, standard deviation.

Table 2. Prevalence of hypertension-mediated organ damage by self-reported hypertension onset age

		Self-reported HTN onset age				
	All	**<35 y**	**35–44 y**	**≥45 y**	**No HTN**	**P value**
N	2,649	194	297	340	1,818	
LVMI, g/m^2 (SD)	85 (21.3)	94 (25.6)	91 (25.1)	90 (21.4)	82 (19.4)	<0.001
LVH, n (%)	437 (16.5)	66 (34.0)	65 (21.9)	75 (22.1)	231 (12.7)	<0.001
E/A ratio (SD)	1.3 (0.4)	1.17 (0.3)	1.24 (0.4)	1.20 (0.3)	1.35 (0.4)	<0.001
LVDD, n (%)	234 (8.8)	29 (15.0)	27 (9.1)	40 (11.8)	138 (7.6)	0.001
CAC-score, AU (SD)	40 (206)	140 (542)	60 (288)	57 (168)	23 (103)	<0.001
Coronary calcification, n (%)	230 (8.7)	40 (20.6)	34 (11.5)	49 (14.4)	107 (5.9)	<0.001
UACR, median (Q1–Q3)	4.8 (3.3–8.4)	6.6 (4.0–16.5)	6.2 (4.0–12.9)	5.4 (3.6–9.7)	4.4 (3.1–7.2)	<0.001
Albuminuria, n (%)	159 (6.0)	26 (13.4)	38 (12.8)	28 (8.2)	67 (3.7)	<0.001

Abbreviations: AU, Agatston units; CAC-score, coronary artery calcification score; E/A ratio, ratio between E wave peak velocity flow in early diastole and A wave peak velocity flow in late diastole; HTN, hypertension; LVDD, left ventricular diastolic dysfunction; LVH, left ventricular hypertrophy; LVMI, left ventricular mass index; Q1, lower quartile; Q3, upper quartile; SD, standard deviation; UACR, urine albumin/creatinine ratio.

hypertension onset age in race–sex subgroups is reported in Supplementary Table S2 online.

Unadjusted odds of HMODs were highest in the group with self-reported hypertension onset at <35 years of age (Table 3). Compared with participants who did not report having hypertension, participants with hypertension onset at <35 years had unadjusted odds ratios of 3.54 (95% confidence interval (95% CI), 2.55–4.92), 2.14 (95% CI, 1.39–3.29), 4.15 (95% CI, 2.79–6.19), and 4.05 (95% CI, 2.50–6.54) for LVH, LVDD, coronary calcification, and albuminuria, respectively. After adjusting for HMOD risk factors apart from systolic BP, the odds of HMOD remained statistically significant in participants with onset at <35 years of age (P < 0.01 for all). After additional adjustment for systolic BP, only hypertension onset at <35 years was statistically significantly associated with LVH, LVDD, and coronary calcification or albuminuria, except for LVDD, for which the association with hypertension onset at ≥45 years retained statistical significance (Table 3).

The cumulative incidence of self-reported and objectively defined hypertension onset by age is visualized in Figure 1. During follow-up, 17.9% and 31.4% of the participants developed objectively defined and self-reported hypertension, respectively. However, when the diagnosis of objectively defined hypertension was based on high BP or use of antihypertensive medication on a single exam, 35.3% of the participants developed hypertension. The level of agreement between self-reported and objectively defined hypertension onset age groups is presented in Table 4. When the diagnosis of objective hypertension was based on 2 exams, the sensitivity and specificity of self-reported hypertension were 95% and 83%, respectively. In contrast, when the diagnosis was based on 1 exam, the sensitivity and specificity were 79% and 95%, respectively. The overall agreement between hypertension onset age groups by self-report and hypertension onset age groups based on either 1 or 2 exams was 78.8% and 77.7%, with kappa coefficients of 0.66 (95% CI 0.63–0.68) and 0.48 (95% CI 0.44–0.51), respectively.

The agreement was lower among individuals who were not using antihypertensive medication (Supplementary Table S3 online).

DISCUSSION

In this study, we demonstrate that self-reported early onset hypertension (onset at <35 years) is strongly associated with increased odds of LVH, LVDD, coronary calcification, and albuminuria in midlife, whereas late onset hypertension is not. These associations appear to be similar to what has been previously reported for objectively defined early onset hypertension that was based on repeated BP measurements.[12] The agreement between self-reported and objectively defined hypertension onset age groups ranged from moderate to substantial,[25] depending on the definition of objectively defined hypertension. Given that HMOD is a strong predictor of CVD outcomes,[6–9,26] our findings could have important clinical implications as self-reported hypertension onset age is feasible to assess in everyday clinical practice unlike many other BP indices that are used for measuring long-term BP exposure.

Several different indices have been previously used to assess the impact of long-term exposure to high BP levels, such as antecedent BP, cumulative BP, and BP trajectories.[27–29] Assessment of hypertension onset age seems to add an alternative, and possibly advantageous, method for evaluating long-term BP exposure. Namely, the other previously mentioned indices require complex calculations and precise data on prior repeated BP measurements. These indices are therefore unlikely to be implemented into daily clinical practice. In the current study, we defined age of onset categories by 10-year age strata as previously. However, no standard definition for early onset hypertension exists.[11,12,30] We observed that the prevalence of all HMODs differed between self-reported hypertension onset age subgroups (Table 2), even though the mean BP levels were similar across the subgroups

Table 3. Odd ratios of hypertension-mediated organ damage according to self-reported hypertension onset age

Self-reported HTN onset age	LVH		LVDD		Coronary calcification		Albuminuria	
	n/N	OR (95% CI)	n/N	OR (95% CI)	n/N	OR (95% CI)	n/N	OR (95% CI)
Unadjusted model								
<35	66/194	3.54 (2.55–4.92)*	29/194	2.14 (1.39–3.29)*	40/194	4.15 (2.79–6.19)*	26/194	4.05 (2.50–6.54)*
35–44	65/297	1.93 (1.42–2.62)*	27/297	1.22 (0.79–1.88)	34/297	2.07 (1.38–3.11)*	38/297	3.83 (2.52–5.83)*
≥45	75/340	1.94 (1.45–2.60)*	40/340	1.62 (1.12–2.36)‡	49/340	2.69 (1.88–3.86)*	28/340	2.35 (1.49–3.71)*
No HTN	231/1,818	1.00	138/1,818	1.00	107/1,818	1.00	67/1,818	1.00
Multivariable-adjusted model								
<35	66/194	3.25 (2.09–5.06)*	29/194	2.24 (1.26–3.97)†	40/194	3.22 (1.71–6.04)*	26/194	2.54 (1.29–5.01)†
35–44	65/297	1.84 (1.18–2.87)†	27/297	1.35 (0.75–2.45)	34/297	1.55 (0.80–3.02)	38/297	2.62 (1.37–5.01)†
≥45	75/340	1.78 (1.19–2.66)†	40/340	1.75 (1.05–2.93)‡	49/340	1.17 (0.63–2.15)	28/340	1.59 (0.84–2.99)
No HTN	231/1,818	1.00	138/1,818	1.00	107/1,818	1.00	67/1,818	1.00
Multivariable + SBP-adjusted model								
<35	66/194	2.38 (1.51–3.76)*	29/194	2.32 (1.28–4.18)†	40/194	2.87 (1.50–5.47)†	26/194	1.62 (0.81–3.26)
35–44	65/297	1.30 (0.82–2.07)	27/297	1.40 (0.76–2.58)	34/297	1.38 (0.69–2.73)	38/297	1.62 (0.82–3.18)
≥45	75/340	1.31 (0.86–1.97)	40/340	1.81 (1.06–3.08)‡	49/340	1.06 (0.57–1.96)	28/340	1.04 (0.54–1.98)
No HTN	231/1,818	1.00	138/1,818	1.00	107/1,818	1.00	67/1,818	1.00

Abbreviations: CI, confidence interval; HDL-cholesterol, high-density lipoprotein cholesterol; HTN, hypertension; LVDD, left ventricular diastolic dysfunction; LVH, Left ventricular hypertrophy; n/N indicates number of individuals with organ damage/number of individuals in category; OR, odds ratio; SBP, systolic blood pressure. Multivariable-adjusted model is adjusted for age, sex, race, diabetes, body mass index, total serum cholesterol, HDL-cholesterol, smoking status, and use of antihypertensive medication. In the multivariable-adjusted models, the P for trend in odds ratios was 0.15, 0.17, 0.18, and 0.47 for LVH, LVDD, coronary calcification, and albuminuria, respectively. The corresponding P values were 0.96, 0.17, 0.11, and 0.75 for the multivariable + SBP-adjusted models, respectively.

*P < 0.001.
†P < 0.01.
‡P < 0.05.

at Year 25 exam (Table 1). This could be a result of the between-group differences in historical BP loads. We also observed that women with early onset hypertension were more likely to have history of preeclampsia or high BP during pregnancy, which might also in part explain our findings. In normal clinical settings, physicians will most likely experience challenges with gaining access to previous medical records that may lack consistently measured and documented BP data. Determining hypertension onset age by self-report, however, could be used as a pragmatically feasible method to add precision in the cardiovascular risk assessment of patients with hypertension. In addition, exposure to high BP in either early or later in life calls for different treatment approaches as current guidelines recommend different BP treatment thresholds and targets for older patients.[9,26,31] Furthermore, given the previously described strong heritability and genetic underpinnings of early onset hypertension,[11,20,30,32] hypertension onset age could be therefore used both as a familial
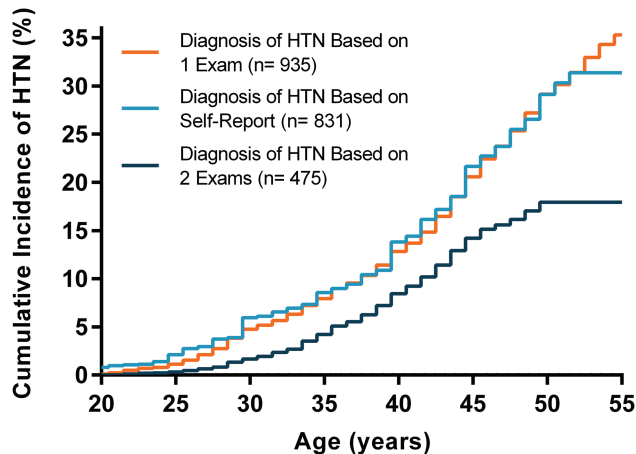

Figure 1. Cumulative incidence of hypertension onset based on self-reported and objectively defined onset age. Self-reported hypertension onset age was based on a self-administered questionnaire at Year 25 exam. Objective hypertension onset age was based on measured BP ≥140/90 mm Hg or use of antihypertensive medication at either 1 or 2 consecutively attended exams between 1985 and 2011. N = 2,649. Abbreviations: BP, blood pressure; HTN, hypertension.

trait when assessing an individual's risk for hypertension and as a specific type of BP trait when estimating risk for CVD outcomes.

In previous studies, the specificity of self-reported hypertension, compared with objectively defined hypertension, has been over 90%. In contrast, sensitivity has varied between 49% and 87%.[1–5] In this study, both specificity and sensitivity of self-reported hypertension were relatively high. We also observed that the agreement between self-reported and objective hypertension onset age, as assessed by the kappa statistic, was substantial when objective hypertension onset based on high BP at 1 exam. However, this agreement was only moderate when high BP on 2 consecutive exams was required for hypertension onset (Table 4 and Figure 1). The optimal definition of objectively defined hypertension onset therefore depends on whether the goal is to achieve maximal correlation between objectively defined hypertension and either self-reported hypertension onset or HMOD. In addition, the correlation between self-reported and objective hypertension onset age will always depend on the diagnostic accuracy of hypertension, the adequacy of patient education, and the clinical patient–physician interaction.

The strengths of this study include a large, diverse, prospective cohort with up to 25 years of follow-up and a high participation rate (68.4% of the original cohort took part in the Year 25 exam). Moreover, information on both self-reported and objectively defined hypertension onset age was available as the participants BP and medication use were serially recorded throughout the study. However, information about the initial source of self-reported hypertension diagnoses was not available in CARDIA study. Yet, self-administrative questionnaires were used which prevent potential social desirability bias.[33,34] In addition, we lacked information on the precise duration or intensity of the participants' antihypertensive treatment. However, we lacked information on the precise duration or intensity of the participants' antihypertensive treatment. We aimed to minimize this effect by accounting use of antihypertensive medication at the Year 25 exam. The mean age of participants during HMOD assessment was 50 years, only 5 years over the lower age threshold of hypertension onset at ≥45 years, which could have

Table 4. Agreement between self-reported and objectively defined age of hypertension onset

| | Objectively defined HTN onset age | | | | | | | |
| | Diagnosis based on high BP at 2 consecutive exams | | | | Diagnosis based on high BP at 1 exam | | | |
	Never, n (%)	<35 y, n (%)	35–44, y n (%)	≥45 y, n (%)	Never, n (%)	<35 y, n (%)	35–44, y n (%)	≥45 y, n (%)
Self-reported HTN onset age								
Never, n (%)	1,794 (67.7)	3 (0.1)	8 (0.3)	13 (0.5)	1,626 (61.4)	42 (1.6)	36 (1.4)	114 (4.3)
<35 y, n (%)	50 (1.9)	65 (2.5)	67 (2.5)	12 (0.5)	18 (0.7)	88 (3.3)	65 (2.5)	23 (0.9)
35–44 y, n (%)	103 (3.9)	20 (0.8)	133 (5.0)	41 (1.6)	23 (0.9)	40 (1.5)	150 (5.7)	84 (3.2)
≥45 y, n (%)	227 (8.6)	5 (0.2)	41 (1.6)	67 (2.5)	47 (1.8)	21 (0.8)	48 (1.8)	224 (8.5)

Abbreviations: BP, blood pressure; HTN, hypertension. Overall agreement between self-reported HTN onset age and objectively defined HTN onset age based on 2 exams was 77.7%, with weighted kappa of 0.48 (95% CI 0.44–0.51). Overall agreement between self-reported HTN onset age and objectively defined HTN onset age based on 1 exam was 78.8%, with weighted kappa of 0.66 (95% CI 0.63–0.68).

some effect on our results. However, the models were adjusted for age at the Year 25 exam to account for this potential bias. Another caveat to the interpretation of our results is that the CARDIA study participants are likely to have increased awareness of their state of health and hypertension status due to regularly attended medical examinations. Our results may not therefore be fully generalizable to the population at large. Future research is therefore warranted to determine whether these findings apply to other similar or different study settings and populations.

In summary, our findings suggest that self-reported age of hypertension onset is a feasible method for assessing risk of HMOD in midlife. More research is warranted to clarify the value of self-reported hypertension onset age in CVD risk prediction in other populations.

SUPPLEMENTARY MATERIAL

Supplementary data are available at *American Journal of Hypertension* online.

DISCLOSURE

The authors declared no conflict of interest.

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