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The Vital Prognosis of Subclavian Stenosis

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Objectives This study sought to assess the prognosis of subclavian stenosis (SS) as a potential marker of total and cardiovascular disease (CVD) mortality.

Background Subclavian stenosis, diagnosed by a brachial systolic pressure difference (BSPD) ≥15 mm Hg, is associated with an increased prevalence of CVD risk factors. However, the association between SS and mortality is unknown. We hypothesized that a BSPD ≥15 mm Hg would predict an increased risk of CVD events.

Methods We analyzed baseline and longitudinal data from 3 cohorts. Two were recruited from noninvasive vascular laboratories, and the third was a community-dwelling cohort. Multivariate survival models were used to test for an independent association of SS with total and CVD mortality.

Results Baseline and follow-up data (mean 9.8 years) were complete in 1,778 participants. Subclavian stenosis was found in 157 (8.8%) subjects. Adjusted for age, gender, ethnicity, and cohort of origin, the presence of SS was significantly associated with increased total and CVD mortality (respectively, hazard ratio [HR] 1.42, $p < 0.005$; and HR 1.50, $p = 0.05$). This association persisted after adjustments for CVD risk factors (smoking pack-years, hypertension, diabetes, total/high-density lipoprotein cholesterol ratio, and body mass index) as well as lipid-lowering and antiplatelet therapies (HR 1.40, $p < 0.01$; and HR 1.57, $p < 0.05$ for total and CVD mortality, respectively). When any history of CVD or an ankle-brachial index <0.90 were added to the model, SS remained an independent predictor for total mortality (HR 1.34, $p = 0.02$), with a similar trend for CVD mortality (HR 1.43, $p = 0.09$).

Conclusions The presence of SS, easily diagnosed by comparing systolic pressures in the left and right arm, predicts total and CVD mortality independent of both CVD risk factors and existent CVD at baseline. (J Am Coll Cardiol 2007; 49:1540–5) © 2007 by the American College of Cardiology Foundation

In daily clinical practice, the measurement of systolic blood pressure (SBP) in both arms is recommended for screening for hypertension (1,2). This is to avoid a misdiagnosis in the case of lower SBP in 1 arm. This may typically occur in case of subclavian stenosis (SS), mostly because of atherosclerotic lesions occurring proximally, including lesions of the innominate artery on the right side.

Except for the upper limb ischemia, few other clinical situations require the comparison of both arms' SBPs. In case of dizziness or syncope, especially occurring during arm exertion, a significant brachial systolic pressure difference (BSPD) might reflect the subclavian steal syndrome (3). The presence of a BSPD in the context of acute chest pain should lead one to consider the diagnosis of aortic dissection (4). A BSPD might also be present in case of different types of vasculitis (e.g., Takayasu disease or giant cell arteritis [5]), congenital malformations (e.g., coarctation of the aorta [6]), as well as in thoracic outlet syndrome (7), and sequelae after radiation therapy (8).

Notably, these cases are quite uncommon in daily general practice, and the observation of an asymmetric SBP between both arms does not routinely lead to any specific medical attention other than monitoring the patient's blood pressure on the arm with the highest blood pressure (1,2).

Based on angiographic data as the gold standard, a BSPD ≥15 mm Hg is highly (≥90%) specific for diagnosing SS (9,10). Surprisingly, although SS is mostly related to atherosclerosis (11), it had not received as much attention as

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other patterns of atherosclerosis affecting coronary, carotid, or lower-extremity arteries. Only recently, 2 epidemiologic studies (12,13) reported a significant correlation between SS and major cardiovascular risk factors such as age, smoking, and dyslipidemia. In contrast to the extensive data for peripheral arterial disease (PAD) in lower limbs and incident cardiovascular disease (CVD) events (14,15), the prognostic value of SS, used as a marker of atherosclerotic process, has never been assessed.

In this longitudinal study combining data from 3 cohorts, we sought to assess the prognostic significance of SS for total and cardiovascular mortality. We hypothesized that the presence of a BSPD ≥15 mm Hg used for the diagnosis of SS is associated with increased total and CVD mortality and could then be considered as a cardiovascular prognostic marker.

Methods

We studied baseline and follow-up data of 1,778 subjects (1,093 men and 685 women) of 1,872 individuals participating in 3 distinct cohort studies. We excluded 94 subjects for incomplete data. Two (cohorts A and B) were composed of patients assessed for PAD in medical centers, and the third (cohort C) consisted of a community-dwelling population.

Cohort presentation. Cohort A was composed of 508 patients recruited between 1990 and 1994 among those seen in the prior 10 years for noninvasive lower extremity arterial testing at the San Diego Veterans Administration Center or the University of California, San Diego Medical Center, vascular laboratories (16).

Cohort B consisted of 740 patients in the WALCS (Walking and Leg Circulation Study) cohort, recruited in 3 medical centers in the Chicago area between 1998 and 2000 (17). This cohort included participants identified consecutively from noninvasive vascular laboratories and a large general internal medicine outpatient practice. Because this cohort was assembled with the primary goal of identifying predictors of decline in lower-extremity functioning, exclusion criteria were wheelchair-bound patients, those with leg amputations, nursing home residents, non–English-speaking people, and those with dementia. Additionally, those with an ankle-brachial index (ABI) >1.50 were excluded because of the inability to provide accurate ABI values because of noncompressible leg arteries.

Cohort C was composed of 624 individuals participating in the Lipid Research Clinics protocol between 1978 and 1979 (18). They were initially recruited through postal and telephone contacts. These subjects were predominantly white and from an upper-middle-class community in southern California.

The enrollees of the 3 cohorts gave written informed consent for their participation in the studies. The institution review boards of the University of California, San Diego (cohorts A and C), and of Northwestern University and Catholic Health Partners Hospital (cohort B) approved the study protocols.

Abbreviations and Acronyms

- **ABI** = ankle-brachial index
- **BSPD** = brachial systolic pressure difference
- **CVD** = cardiovascular disease
- **PAD** = peripheral arterial disease
- **SBP** = systolic blood pressure
- **SS** = subclavian stenosis

Blood pressure and ABI measurement. Among the 3 cohorts, the protocols for upper and lower limbs SBP measurements were somewhat different but comparable. Briefly, the participants in cohorts A and C had sequential measurements of brachial and ankle pressures, using 12-cm pneumatic cuffs and either a photoplethysmographic sensor attached to the great toe (cohort A) or a mercury-in-Silastic gauge (cohort C). In these 2 cohorts, arms pressures and then ankles pressures were simultaneously measured twice. The pressure at the site of the cuff was the pressure measured. In these 2 cohorts, the ABI in each leg was calculated by dividing the highest of both ankles' systolic pressures by the highest pressure between both arms. In cohort B, the limbs systolic pressures were measured sequentially using a hand-held Doppler probe (Pocket Dop II, Nicolet Vascular, Golden, Colorado). Each site had 2 sequential measurements taken. In the first set, the sequence of BP measurement was the right arm, right ankle, left ankle, left arm, whereas the reverse sequence was performed during the second set of measurements. The ABI for each leg was calculated by dividing the highest average leg artery systolic pressure (either posterior tibial or dorsalis pedis arteries) by the highest average SBP between both arms. For the estimation of BSPD, the absolute values of the difference between both arms were used. In cohort B, the BSPD was obtained by subtracting the average SBPs of one arm from those of the other.

Because SBPs recorded in cohort C were rounded to the nearest 5 mm Hg, we similarly rounded cohorts A and B SBP results for analysis uniformity.

Data definition. Patient's ethnicity was self-reported. We classified participants as non-Hispanic white, black, Hispanic, and others. Hypertension was defined according to patient's history or use of antihypertensive therapy. Patients were classified as diabetic according to patient's history or use of oral antidiabetic drugs and/or insulin. Clinical CVD included any personal history of myocardial infarction, stroke, transient ischemic attack, or revascularization of the coronary, carotid, or lower-extremity arteries. In addition, subjects were considered to have clinical CVD if the ABI was <0.90.

We defined SS by a BSPD ≥15 mm Hg, according to a previous angiographic study suggesting that this threshold is highly specific (90%) for SS diagnosis (9,10). Additionally, a prior cross-sectional study including these 3 cohorts (13) used a similar threshold and showed a significant relationship between BSPD ≥15 mm Hg and several CVD risk factors as well as PAD. To assess whether SS severity would

correlate with poorer prognosis, we secondarily classified participants' SS into moderate (15 ≤ BSPD <25 mm Hg) or severe SS (BSPD ≥25 mm Hg).

Follow-up. COHORT A. Follow-up data in this cohort were recorded until the end of 2002 by identifying fatal events through Social Security Administration data. Based on death certificates from the state vital statistics offices, cause of death was adjudicated and coded by a certified nosologist.

COHORT B. Follow-up data in this cohort were recorded through the end of 2003 by identifying fatal events through Social Security Administration data. Death certificates were obtained from the State of Illinois and from participants' medical records. Causes of death were adjudicated by a certified nosologist.

COHORT C. The participants were surveyed annually to ascertain fatal events. Reported deaths were confirmed by death certificates, and their causes were adjudicated by a certified nosologist. Follow-up in this cohort was done through the end of October 2002.

Statistical methods. The chi-square test was used for the comparison of SS rates in both genders. Survival curves are presented according to the Kaplan-Meier method, using log-rank tests for statistical significance. For total and CVD mortality, for each cohort as well as for the whole population study, the predictive value of SS for total and CVD mortality was assessed in 3 successive models using the Cox survival analysis. In the first model, SS hazard ratios were adjusted for age and gender. For the whole study population, ethnicity and the cohort of origin were next added in this model. In model 2, CVD risk factors (smoking pack-years, total/high-density lipoprotein cholesterol ratio, history of diabetes, history of hypertension) as well as lipid-lowering and antiplatelet therapies were added to those already present in model 1. Finally, the presence of any CVD was also added in model 3. These successive models were used to present a stepwise control of confounding variables (e.g., CVD risk factors). There were no significant interactions between cohort and any other covariates for SS.

Because the follow-up duration varied significantly among the 3 cohorts, a second set of analyses was performed, limiting the follow-up data to 7 years. This follow-up delay was chosen regarding to the maximal length of the follow-up period recorded in cohort B, which had the shortest mean follow-up duration.

For all tests used, a value of $p \leq 0.05$ was required for statistical significance. Results with a value of $p > 0.05$ but <0.10 were considered borderline. Data were analyzed using Statview 5.0 (SAS Institute Inc., Cary, North Carolina) statistical software.

Role of funding source. The funding organizations played no role in the data collection, study design, data interpretation and analyses, or manuscript writing.

Results

Demographic, risk factor, and mortality data for the 3 cohorts and the overall population study are presented in Table 1. Overall, SS was present in 157 subjects (8.8%),

Table 1 Description of the 3 Cohorts and the Overall Study Population

	Cohort A (n = 505)	Cohort B (n = 662)	Cohort C (n = 611)	Study Population (n = 1,778)
Baseline				
Age (yrs)	68.5 (9.1)	70.8 (8.4)	66.4 (10.3)	68.6 (9.5)
Male gender	445 (88.1%)	372 (56.2%)	276 (45.2%)	1,093 (61.5%)
Ethnicity				
Non-Hispanic white	439 (86.9%)	518 (78.2%)	600 (98.2%)	1,557 (88.2%)
Black	23 (4.6%)	114 (17.2%)	0 (0%)	137 (7.8%)
Hispanic	27 (5.3%)	9 (1.4%)	0 (0%)	36 (2.0%)
Other	16 (3.2%)	21 (3.2%)	11 (1.8%)	35 (2.0%)
Body mass index (kg/m^2)	27.3 (8.0)	27.8 (5.4)	24.8 (3.8)	26.6 (5.9)
Total/high-density lipoprotein cholesterol	4.9 (1.7)	4.8 (1.8)	4.1 (1.7)	4.6 (1.7)
Hypertension	267 (52.3%)	542 (81.9%)	44 (7.2%)	853 (48.0%)
Smoking pack-yrs	48.6 (41.6)	32.2 (33.9)	18.3 (25.4)	32.1 (35.8)
Diabetes	156 (30.9%)	185 (28.0%)	22 (3.6%)	363 (20.4%)
Lipid-lowering therapy	107 (21.2%)	270 (40.8%)	9 (1.5%)	386 (21.7%)
Antiplatelet therapy	349 (69.1%)	385 (58.2%)	55 (9.0%)	789 (44.4%)
Clinical cardiovascular disease	338 (66.9%)	363 (54.8%)	72 (11.8%)	773 (43.5%)
ABI <0.90	286 (56.6%)	399 (60.3%)	87 (14.2%)	772 (43.4%)
Subclavian stenosis	26 (5.2%)	89 (13.4%)	42 (6.9%)	157 (8.8%)
Follow-up				
Mean duration (yrs)	7.0 (3.2)	4.8 (1.6)	16.4 (6.7)	9.4 (6.8)
Total mortality	305 (60.4%)	167 (25.2%)	374 (61.2%)	846 (47.6%)
Cardiovascular disease mortality	175 (34.7%)	50 (7.6%)	74 (12.1%)	299 (16.8%)

ABI = ankle-brachial index.

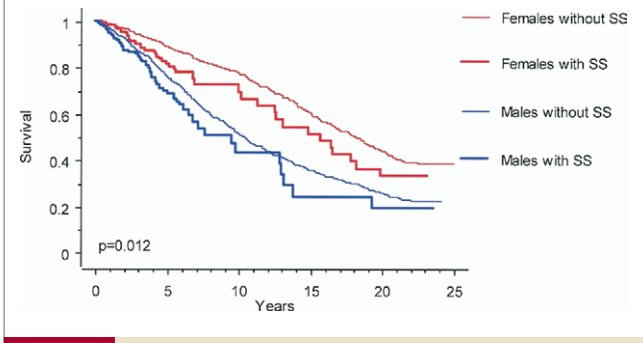

Figure 1 **25-Year Survival According to Gender and Presence of SS**

SS = subclavian stenosis.

more frequent among women than men (11.2% vs. 7.3%, p = 0.006).

The mean follow-up period of the entire study population was at 9.4 ± 6.8 years, with a maximal follow-up duration of 11.4, 6.9, and 24.9 years for cohorts A, B, and C, respectively. During this period, 272 (39.7%) women and 574 (52.6%) men died, including respectively 56 (8.2%) and 243 (22.2%) CVD deaths.

Figure 1 shows the overall survival curves of the entire study population stratified by gender, according to the presence or absence of SS. In both genders, survival rates were lower in the presence of SS. The 7-year survival is shown according to age groups (older or younger than 70 years) and presence of SS in Figure 2. Similar to younger participants, those in the elderly group had a poorer prognosis in the presence of SS.

Figure 3 presents the 7-year survival curves according to the presence or absence of SS in those with and without any CVD (clinical CVD and/or ABI <0.90). Irrespective of CVD prevalence at baseline, those with an SS presented a significantly poorer prognosis. A similar trend (p = 0.08) was found for the 25-year survival analysis (data not shown).

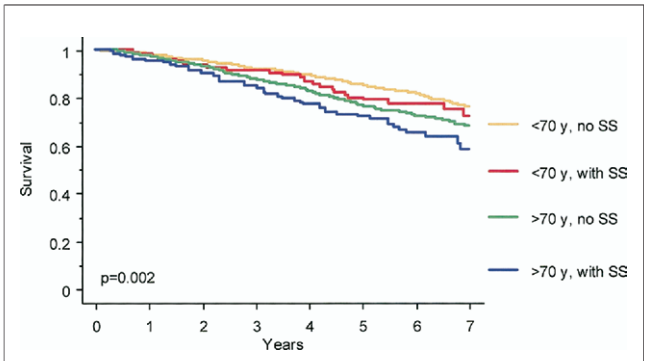

Figure 2 **7-Year Survival According to Age Group and Presence of SS**

SS = subclavian stenosis.

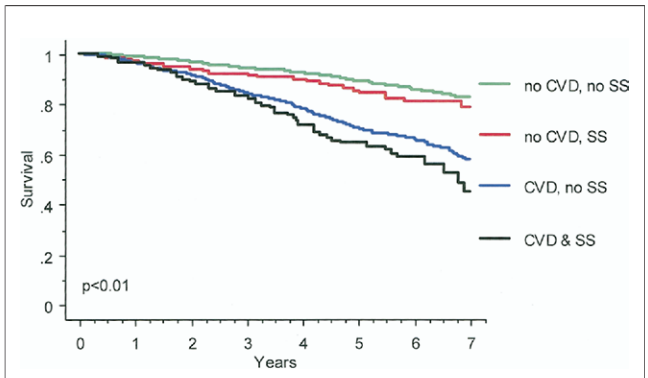

Figure 3 **7-Year Survival According to Presence of Any CVD and Presence of SS**

Any cardiovascular disease (CVD) represents clinical CVD and/or an ankle-brachial index (ABI) <0.90. SS = subclavian stenosis.

Figure 4 shows the 7-year survival curves of the participants classified in 3 categories: no SS, moderate SS, and severe SS. The overall mortality was gradually worse with increasing BSPD categories. Similar significant (p < 0.001) results were found with the 25-year survival analysis.

Table 2 presents the adjusted hazard ratios of SS for total and CVD mortality in the 3 cohorts and the whole population. In the fully adjusted model, a 34% excess total mortality rate was noted in those with SS (p = 0.02), whereas there was a borderline (p = 0.09) 43% CVD mortality excess rate. The results obtained when follow-up was limited to 7 years showed similar trends. For the whole population, the fully adjusted hazard ratios for SS for the 7-year total and CVD mortality were at 1.50 (p = 0.01) and 1.49 (p = 0.12), respectively.

Because a consistent BSPD might lead to missing the diagnosis of hypertension if only one arm's SBP is monitored, one might consider that our results could be related to untreated hypertension rather than an actual predictive value for SS. Indeed, among those with SS, 16% presented

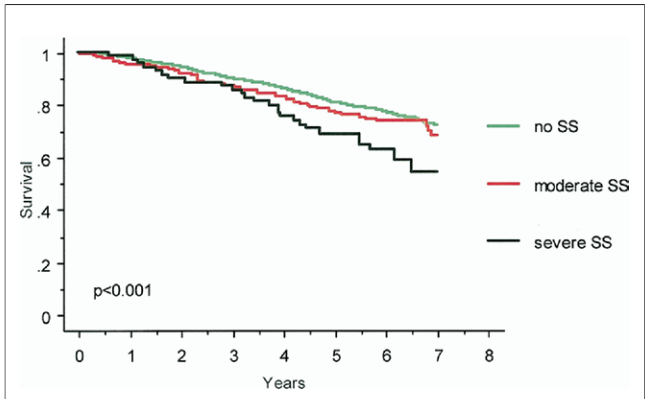

Figure 4 **7-Year Survival According to SS Severity**

SS = subclavian stenosis.

Table 2 Adjusted Hazard Ratios (and 95% Confidence Intervals) of SS (BSPD ≥15) for Total and CVD Mortality (Mean Follow-Up 9.4 yrs)

	Cohort A		Cohort B		Cohort C		Overall*	
Mortality	Total	CVD	Total	CVD	Total	CVD	Total	CVD
Model 1	1.38 (0.84–2.25)	1.26 (0.64–2.48)	1.77* (1.21–2.60)	1.52 (0.74–3.16)	1.28 (0.87–1.90)	1.97 (0.94–4.12)	1.42* (1.12–1.80)	1.50* (1.00–2.52)
Model 2	1.48 (0.88–2.47)	1.48 (0.75–2.93)	1.71* (1.09–2.67)	1.50 (0.66–3.41)	1.14 (0.76–1.70)	2.09 (0.97–4.84)	1.40* (1.09–1.80)	1.57* (1.03–2.40)
Model 3	1.36 (0.81–2.27)	1.31 (0.66–2.60)	1.70* (1.09–2.66)	1.45 (0.63–3.30)	1.10 (0.74–1.65)	2.00 (0.93–4.33)	1.34* (1.04–1.73)	1.43 (0.93–2.18)

Model 1: adjusted for age and gender (plus ethnicity and cohort for the overall population study). Model 2: adjusted as Model 1 + smoking pack-years, total/high-density lipoprotein cholesterol ratio, lipid-lowering therapy, history of diabetes, history of hypertension, body mass index, and antiplatelet therapy. Model 3: adjusted as Model 2 + any CVD (history of clinical CVD and/or ABI <0.9). *No significant interaction between any cohort and SS has been noticed.

ABI = ankle-brachial index; BSPD = brachial systolic pressure difference; CVD = cardiovascular disease; SS = subclavian stenosis.

an elevated (>160 mm Hg) SBP without reported or treated hypertension, compared with 7% among those without SS ($p < 0.001$). Although we consider all these subjects as undiagnosed hypertension (although some might present the "white coat" effect), the introduction of an "undiagnosed hypertension" as an independent variable in our predictive models did not affect hazard ratios and p values of SS, neither for total nor CVD mortality (data not shown). In addition, no significant interaction between the presence of SS and undiagnosed hypertension was found.

Discussion

These results confirm our hypothesis on the predictive value of a brachial systolic pressure asymmetry exceeding 15 mm Hg, commonly used for defining SS in epidemiologic studies.

To our knowledge, this is the first study showing that, similar ABI as a marker of PAD, a significant BSPD can not only be used for the detection of SS, but also is useful as a prognostic marker. The survival of those with SS was similarly affected in the elderly and younger subjects, as well as in those with and without CVD. We also found that the degree of blood pressure asymmetry between both arms was proportional to mortality.

Subclavian stenosis was present in almost 9% of our participants. This prevalence is estimated at 1.9% in a community-dwelling population (2.7% after 70 years) (13). In another population study with a 10-mm Hg threshold, the prevalence of abnormal inter-arm SBP difference was at 9.3% (12). The prevalence of SS is higher in primary care (19) and in hospital (20) patients. By combining 2 vascular laboratory cohorts with a general population cohort, we enriched the sample with SS and increased study power. Table 2 shows no evidence of heterogeneity in hazard ratios across cohorts, indicating the appropriateness of combining cohorts.

Subclavian stenosis is essentially related to atherosclerosis, because other etiologies such as inflammatory diseases, traumatic, or congenital causes compose <5% of cases in laboratory series (11). In a cross-sectional study including our 3 cohorts and another free-living population cohort, a significant association between SS and age, smoking, high-density lipoprotein cholesterol, and the presence of PAD has been reported (13). In the Ohasama population study (12), hypertension, obesity, hypercholesterolemia, diabetes, and low (<1.0) ABI were independent predictive factors for a BSPD >10 mm Hg. In another study on 610 patients of an emergency department, Singer and Hollander (21) found an inter-arm systolic or diastolic difference of more than 20 mm Hg in 19% of cases, with no relation with age, gender, or CV risk factors, but a significantly higher prevalence of known coronary heart disease.

According both to the seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC VII) (1) and the European Society of Hypertension/European Society of Cardiology guidelines (2), blood pressure should be measured in both arms. Unfortunately, these recommendations are not fully respected. In a survey led in the United Kingdom (22), even general practitioners and nurses with an interest in hypertension did not use both arms. Our findings in this study indicate the importance of systematic bilateral measurement of arm blood pressure, not only to avoid missing a hypertension diagnosis, but also to detect easily a typically asymptomatic atherosclerotic disease.

A major finding in our analysis is that similar to other vascular markers (e.g., ABI), the predictive value of SS is independent from the presence of traditional risk factors. Additionally, the association with mortality remained significant even after including data on CVD history and ABI measurement (with borderline results for CVD death). Thus, along with the measurement of ABI, the detection of SS can also be helpful for the estimation of one's CVD prognosis. The comparison of arm SBP for the detection of SS has the major advantage of its feasibility in the physician's office (15). However, this should not be substituted for the ABI measurement, which is of well-documented importance (15). Our results here show that SS detection might provide additional information to ABI, and its presence (irrespective of the ABI) might be considered as a clinical alert sign, with prognostic implications. Similar to PAD, it is plausible that the association between SS and mortality be related to higher rates of concomitant unknown atherosclerotic diseases in other territories, as shown in the study by Shadman et al. (13). The detection of an SS might be considered a finding requiring more aggressive risk factor modification, although there are no published randomized trial data on this question. Other studies, especially in

general practice, are required to confirm our data and to highlight the respective roles of ABI and BSPD measurements in the prognosis determination.

Study limitations. Our study has some limitations. First, rounding SBP results might have affected our ability to detect some borderline cases of SS, but this would not be a systematic bias. Second, the measurements were not performed simultaneously, and the BSPD measured can partially be affected by the beat-to-beat variation. However, in a series of 447 hospitalized patients, Harrison et al. (23) found a mean right minus left SBP at −2 mm Hg when measured sequentially versus +0.5 when measured simultaneously. In another series of 237 subjects primary care, Cassidy and Jones (19) found a mean difference of 4.8 mm Hg between the right and left SBP, compared with 2.9-mm Hg differences between a first and second measurement on the same side. Overall, the difference related to the sequential SBP measurement is substantially below the 15-mm Hg BSPD threshold used to define SS. In addition, blood pressure is usually measured sequentially, and from a practical point of view, our findings can be applied in daily practice. Finally, because the detection of SS was based on the comparison of arm SBPs, we were not able to detect bilateral SS. Although this situation is much less common than unilateral SS, we might have erroneously classified some cases of bilateral SS in the normal group, and this would have reduced the association between SS and mortality observed here. Likewise, a BSPD ≥15 mm Hg is highly specific but poorly sensitive (~50%) for SS diagnosis (9,10). Additional prospective studies are required in the general population to confirm our findings.

Conclusions

In this longitudinal study composed of 3 cohorts, we highlight the vital prognostic information provided by the diagnosis of SS, easily identified by arm SBP asymmetry. The presence of a BSPD ≥15 mm Hg is a marker for significantly reduced survival. Further prospective studies in primary care are needed to assess the magnitude and consequences of our findings in clinical practice.

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