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SCIENTIFIC INVESTIGATIONS

Cerebral Hemodynamics in Sleep Apnea and Actigraphy-Determined Sleep Duration in a Sample of the Hispanic Community Health Study/ Study of Latinos

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Study Objectives: We sought to evaluate cerebral hemodynamics in obstructive sleep apnea (OSA) and actigraphy-defined short sleep duration using transcranial Doppler ultrasound (TCD) blood flow velocity in a subsample of Hispanics/Latinos without stroke and cardiovascular disease.

Methods: The sample consisted of consecutive participants at the Miami site of the Hispanic Community Health Study/Study of Latinos (HCHS/SOL) with overnight home sleep testing and 7 days of wrist actigraphy in the Sueño sleep ancillary study. Ninety-five participants had sleep data and TCD determined cerebral hemodynamics. We evaluated the association between OSA (apnea-hypopnea index [AHI] ≥ 5 events/h) and short sleep duration (< 6.8 hours; sample median) with cerebral blood flow velocities (CBFV) and pulsatility index (PI) for the middle cerebral (MCA) and basilar arteries (BA).

Results: Median age was 48 years (range 20–64) with 71% females. Twenty-eight percent of the sample had OSA (AHI ≥ 5 events/h) with median AHI of 10.0 (range 5.0–51.7) events/h. In unadjusted analyses, participants with OSA had lower median CBFV in the BA (30.5 cm/s [interquartile range:10.2] versus 39.4 cm/s [13.3] $P < .05$), but not the MCA, whereas short sleepers had higher median vascular resistance in the MCA (PI = 0.92 [0.18] versus 0.86 [0.14] $P < .05$) and BA (PI = 1.0 [0.17] versus 0.93 [0.24] $P < .05$). After full adjustment, OSA was associated with decreased CBFV (β [SE] = −5.1 [2.5] $P < .05$) in the BA. Short sleep was associated with increased PI (β [SE] = 0.05 [0.02] $P < .05$) in the MCA.

Conclusions: In this sample of Hispanic/Latinos, OSA was associated with decreased daytime blood flow velocity in the BA, whereas actigraphy-defined short sleep duration was associated with increased cerebrovascular pulsatility in the MCA.

Keywords: cerebral hemodynamics, Hispanic/Latinos, sleep apnea, sleep duration, stroke risk

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BRIEF SUMMARY

Current Knowledge/Study Rationale: Although obstructive sleep apnea is a known independent risk factor for stroke, the mechanisms explaining the risk factor are unclear, particularly in Hispanic/Latinos who have an increased risk of cerebrovascular disease compared to non-Hispanic whites. Therefore, this study sought to evaluate potential associations between obstructive sleep apnea and cerebral hemodynamics using sonography in a cohort of Hispanic/Latino adults with no known history of stroke or cardiovascular disease.

Study Impact: This study reports possible impaired cerebral hemodynamics in Hispanics/Latinos with obstructive sleep apnea or short sleep duration, suggesting possible association between obstructive sleep apnea and stroke risk. Further studies are needed to investigate potential pathophysiologic mediators in which obstructive sleep apnea confers cerebrovascular accident risk.

INTRODUCTION

Obstructive sleep apnea (OSA) is an independent risk marker for stroke.[1–3] Studies have linked OSA to carotid intima thickness, carotid stenosis, and small vessel disease.[4–6] Though pathogenesis is unclear, repeated hypoxemia may contribute to vascular inflammation leading to endothelial dysfunction and atherosclerosis.[1] OSA has also been linked to cardiometabolic disorders such as obesity and diabetes, which also contribute to stroke risk.[7,8] However, there is a paucity of studies evaluating mechanisms that may explain the risk of cerebrovascular disease associated with OSA; particularly in Hispanic/Latinos, who have a twofold increased risk of cerebrovascular disease compared to non-Hispanic whites.[9] Similar to OSA, self-reports of short and long sleep durations are more common in Hispanic/Latinos, and are associated with increased cerebrovascular disease.[10] However, most epidemiological data are based on self-reports of sleep duration without accounting for OSA. Pathologic mechanisms linking sleep duration and cerebrovascular disease are not fully understood, more so in Hispanic/Latinos who have a large burden of cerebrovascular disease.

Transcranial Doppler (TCD) ultrasonography is a rapid and noninvasive technique to measure real-time cerebrovascular hemodynamics of major cerebral arteries.[11] Changes in cerebral blood flow velocity (CBFV) of large intracerebral vessels and microvascular resistance as measured by TCD has been observed in individuals with OSA.[12] We previously showed increased arterial pulsatility index (PI), a measure of cerebral small vessel compliance, in OSA during wakefulness using TCD, supporting its clinical relevance and ease of use in measuring cerebral hemodynamics.[13] Epidemiological studies of OSA focused on race-ethnic disparities are limited, but available data suggest elevated prevalence of OSA in Hispanics/Latinos when compared to whites, as well as delays in diagnosis and treatment.[14]

In this study, we evaluated potential associations between OSA and cerebral hemodynamics using TCD in a cohort of Hispanic/Latino adults with no known history of stroke or cardiovascular disease. We also explored associations between cerebral hemodynamics and actigraphy defined sleep duration.

METHODS

Hispanic Community Health Study/Study of Latinos and Sueño Ancillary Study

The Hispanic Community Health Study/Study of Latinos (HCHS/SOL) is a multicenter community-based cohort study examining prevalence and risk factors of chronic disease among 16,145 Hispanic/Latino adults from four urban areas (Bronx, New York; Miami, Florida; Chicago, Illinois; San Diego, California). The baseline evaluation was from 2008 through 2011, gathering information on demographics, socioeconomic status, lifestyle habits, and medical history.[15,16] The Sueño ancillary study was designed to obtain further information on sleep habits through questionnaires and actigraphy in a sample of 2,087 HCHS/SOL participants aged 18 to 64 years evaluated between 2010 through 2013 with valid actigraphy data.[17] Sueño recruited participants from six Hispanic/Latino backgrounds composed of 13.7 % from Central America, 18.1% Cuban, 12.5% Dominican, 20.7% Puerto Rican, 26.8% Mexican, and 8.7% South American.[18] We evaluated the cerebral hemodynamics of consecutive participants (n = 95) at the HCHS/SOL Miami site from June through December of 2013. Based on parent study design, the HCHS/SOL Miami site recruited mostly participants from Cuban background (55.6%) followed by Central (17.5%) and South American (21.6%) backgrounds. We excluded participants with previous respiratory disease (eg, chronic obstructive pulmonary disease), treated for OSA (ie, positive airway pressure), stroke, coronary artery disease, cardiac arrhythmia, heart failure, carotid or intracranial arterial stenosis, and those on medications that could affect vascular tone (eg, calcium channel blockers). The Hispanic Community Health Study/Study of Latinos and Sueño were approved by the Institutional Review Boards at each of the participating sites and all participants provided written informed consent. Additional Institutional Review board approval at the Miami field site along with informed consent from all participants was obtained for the additional cerebral hemodynamic testing.

Main Outcome: Cerebral Hemodynamics

The TCD was performed by a certified technologist (DC) using a portable microprocessor-controlled TCD system on a laptop (EMS-9U, Delica) according to the American Institute of Ultrasound in Medicine guidelines.[19] A low-frequency (2 MHz) pulse-wave ultrasonic signal is transmitted from the skin surface across the cranial vault to the intracerebral vessels and receives the echoes along the same path. The TCD probe is positioned over "acoustic windows" that are specific regions of the skull where the cranial bone is thin, allowing examination of the middle cerebral artery (MCA) and the terminal portion of the internal carotid artery. The basilar artery (BA) was insonated through the occipital window across the foramen magnum. The ultrasound beam emitted from the TCD probe reflects from the erythrocytes traveling within blood flow of the insonated artery. The transducer receives the signal and converts it into an electric signal. The following TCD variables are then averaged across the arterial segments and analyzed as (1) mean flow velocities (MFV), peak systolic velocities (PSV) and end diastolic velocities (EDV), (2) PIs, and (3) resistance indexes in the MCAs and the BAs. The PI is calculated by the Gosling equation, PI = (PSV − EDV) / MFV and resistance index (RI) by the Pourcelot index, RI = (PSV – EDV) / PSV.[20] TCD measurements were conducted in the morning for all patients.

The breath-holding index (BHI) was used to determine the microvascular vasodilatory reactivity in response to carbon dioxide accumulation. Participants were asked to hold their breath for 30 seconds after a normal inspiration, and then breathe normally again while continuously insonating the MCA. MFV were measured before, during, and 1 minute immediately after breath holding. The BHI was derived as the percentage increase in mean flow velocities during breath-holding from baseline adjusted for the length of breath holding time: BHI = ([MFV breath holding – MFV baseline] / MFV baseline) × (100 / time of breath holding).[21]

Sleep Apnea Assessment

Obstructive sleep apnea was assessed at baseline (2008–2011) using the ARES Unicorder 5.2; B-Alert (Carlsbad, California, United States). This self-applied device measures nasal airflow using a nasal pressure cannula and transducer, and transcutaneous oxygen saturation, position, and snoring. Respiratory events were identified as a 50% or greater reduction in airflow lasting greater than or equal to 10 seconds. The apnea-hypopnea index (AHI) was the number of respiratory events per estimated sleep hour; and the number of respiratory events per estimated sleep hour with associated desaturations of greater than or equal to 3% was labeled AHI3.[16]

Sleep Duration: Actigraphy

Sleep duration was defined with wrist actigraphy. An Actiwatch Spectrum (Philips Respironics, Murrysville, PA, United States) wrist actigraph was placed on the nondominant wrist and participants were asked to wear the device continuously for 7 days. Activity and light data were collected throughout this period in 30-second epochs, along with a sleep diary each day.[18,22]

Table 1—Demographics, vascular factors and actigraphy defined sleep habits across categories of OSA in a sample of Hispanic/Latinos at the HCHS/SOL Miami site.

		OSA	
	Total (n = 95)	AHI ≥ 5, (n = 27)	AHI < 5, (n = 68)
Age, years *	48 (20–64)	54 (22–63)	47 (20–64)
Female, n (%) *	67 (71)	14 (52)	53 (56)
Body mass index, kg/m^2 *	29 (19–53)	31 (22–53)	30 (23–53)
Diabetes mellitus, n (%)	6 (8)	2 (7)	4 (6)
Systolic blood pressure, mmHg	123 (90–171)	126 (97–171)	121 (90–166)
Diastolic blood pressure, mmHg	76 (55–105)	77 (59–93)	74 (55–105)
Epworth Sleepiness Scale score	6 (0–22)	6 (0–22)	5 (0–18)
Sleep duration, hours	6.8 (3.2–8.7)	6.5 (3.2–8.7)	6.8 (4.5–8.6)
Sleep fragmentation index, %	19 (9–47)	20 (12–47)	19 (9–42)
Sleep efficiency, %	87 (67–96)	87 (74–95)	87 (67–95)
AHI3, events/h	1.7 (0.0–51.7)	10.0 (5.0–51.7)	0.8 (0.0–4.9)

Values are median (range) or n (%) as indicated. * = $P < .05$ for the difference between OSA groups. AHI = apnea-hypopnea index, AHI3 = number of respiratory events per estimated sleep hour with associated desaturations of greater than or equal to 3%, OSA = obstructive sleep apnea.

Rest-sleep periods were identified using standardized protocol and a validated scoring algorithm alongside polysomnography on an epoch-by-epoch basis. Briefly, epochs considered to be sleep intervals were identified by event markers, sleep diary, white light intensity, and activity in order of importance respectively to establish sleep times. [17,23]

Actigraphy was analyzed in participants with 5 or more days of valid data. We then assessed sleep habits in the domains of sleep duration (sleep during the primary sleep period) and sleep continuity (sleep efficiency and sleep fragmentation index) as previously defined.[17,23]

Average sleep duration, sleep efficiency, and sleep fragmentation index were calculated by averaging across all nights of valid data. For analytical purposes, sleep duration was initially modeled continuously. We defined short sleep duration as less than 6.8 hours, based on the median sleep duration of the sample (n = 95) and consistent with the mean actigraphic sleep duration for the overall Sueño sample (n = 2,087).[18]

Covariates

Obesity was categorized as a body mass index of ≥ 30.0 kg/m^2. Diabetes mellitus was based on self-reported physician diagnosis. The systolic and diastolic blood pressures were obtained during daytime TCD monitoring by using a mercury sphygmomanometer and used as continuous measures.

Statistical Analysis

We evaluated the distribution of the sleep variables and CBFV across all insonated vessels. We evaluated bivariate correlations for continuous variables and differences in the proportions for categorical variables with the Spearman correlation and Mann-Whitney *U* test respectively. Our sample was obtained from a single HCHS/SOL site (Miami) and was relatively small; therefore, we did not use complex survey procedures to account for cluster sampling stratification and sampling weights as done in other HCHS/SOL analyses. We used linear regression models (PROC GLM) to evaluate differences in MFV, PI, and RI with OSA severity and sleep duration adjusting for age, sex, systolic blood pressure, diastolic blood pressure, and diabetes.

All *P* values were based on two-tailed testing and considered significant at $P < .05$. All analyses were completed using SAS v. 9.4 (SAS Institute, Cary, North Carolina, United States).

RESULTS

Demographics

Table 1 presents the characteristics of the sample. The median age was 48 years, and most of the sample were women. Approximately 20% of the sample met the criteria for OSA, using the cutoff of an AHI ≥ 5 events/h. In our sample, participants with OSA were older, but there were no differences in systolic and diastolic blood pressure, daytime sleepiness, sleep duration, sleep efficiency, or sleep fragmentation index by OSA status. Individuals with short sleep were older (median age 51 years, range 22–64) compared to participants' sleep duration of ≥ 6.8 hours (median age 43 years, range 20–64), $P = .0012$ with no differences in sex, sleepiness, body mass index, systolic blood pressure, and diastolic blood pressure.

Differences of Cerebral Hemodynamics in Sleep Apnea and Short Sleep

Table 2 describes the mean CBFV, PI, and resistivity index across OSA and short sleep categories. We observed that participants with OSA had decreased CBFV in the BA when compared to participants without OSA. Participants with short sleep duration had increased PIs and resistivity indexes in the BA and MCA with no differences in mean blood flow velocities.

The median breath holding index (BHI) was 0.90 with a range of 0.03–1.81. However, no statistical differences where observed in the BHI by OSA status (median [range] 0.88

Table 2—Unadjusted differences in cerebral hemodynamics from participants with OSA and actigraphy defined short sleep duration at the HCHS/SOL Miami site.

	BA			MCA		
	MFV	PI	RI	MFV	PI	RI
Obstructive Sleep Apnea						
Yes	30.5 (16.0–42.7) *	0.96 (0.79–1.45)	0.59 (0.52–0.74)	52.0 (39.3–83.2)	0.89 (0.74–1.10)	0.56 (0.49–0.62)
No	39.4 (16.4–69.8)	0.96 (0.61–1.39)	0.59 (0.43–0.72)	56.1 (36.1–92.0)	0.89 (0.67–1.15)	0.55 (0.46–0.65)
Short Sleep Duration						
Yes	35.1 (18.7–59.7)	1.00 (0.61–1.39) *	0.59 (0.43–0.72) *	54.2 (36.1–91.8)	0.92 (0.68–1.15) *	0.57 (0.46–0.65) *
No	38.0 (16.0–69.8)	0.93 (0.70–1.45)	0.57 (0.48–0.74)	58.0 (39.3–92.0)	0.86 (0.67–1.12)	0.54 (0.46–0.64)

Values are median (range). * = $P < .05$. BA = basilar artery, MCA = middle cerebral artery, MFV = mean flow velocities, OSA = obstructive sleep apnea, PI = pulsatility index, RI = resistivity index.

Table 3—Association between obstructive sleep apnea and actigraphy defined-short sleep duration with cerebral hemodynamics in 95 participants from the HCHS/SOL Miami site.

	BA			MCA		
	MFV	PI	RI	MFV	PI	RI
Continuous Sleep Variables						
AHI3	−0.38 (0.19) **	0.01 (0.003) **	0.002 (0.01)	0.1 (0.17)	0.001 (0.002)	0.0002 (0.001)
Sleep duration, hours	−0.28 (1.2)	−0.04 (0.02) **	−0.01 (0.01)	−0.5 (1.3)	−0.002 (0.01)	−0.001 (0.05)
Categorical Sleep Variables						
Sleep apnea	−5.1 (2.5) **	0.04 (0.04)	0.02 (0.001) **	4.6 (2.8) *	0.03 (0.03)	0.001 (0.01)
AHI < 5 events/h	Reference	Reference	Reference	Reference	Reference	Reference
Short sleep	0.9 (2.2)	0.04 (0.03) *	0.01 (0.01)	0.7 (2.4)	0.05 (0.02) **	0.02 (0.01) **
≥ 6.8 hours	Reference	Reference	Reference	Reference	Reference	Reference

Values are β (standard error). Models adjusted for age, sex, systolic blood pressure, diastolic blood pressure, and diabetes mellitus. * = $P < .1$. ** = $P < .05$. AHI = apnea-hypopnea index, AHI3 = number of respiratory events per estimated sleep hour with associated desaturations of greater than or equal to 3%, BA = basilar artery, MCA = middle cerebral artery, MFV = mean flow velocities, PI = pulsatility index, RI = resistivity index.

[0.23–1.30] versus 0.90 [0.03–1.81]) or short sleep duration (0.91 [0.03–1.13] versus 0.88 [0.23–1.81]). **Table S1** in the supplemental material shows Spearman correlations between the AHI, actigraphy-defined sleep habits, covariates, and cerebral hemodynamics. The AHI had positive associations with the PIs and resistivity indexes in the BA, but not in the MCA. Average sleep duration had inverse associations with PI and resistivity index in the BA. Additionally, sleep efficiency had a positive association with basilar CBFV, whereas sleep fragmentation index was negatively associated with CBFV in the BA.

Associations Between Apnea-Hypopnea Index and Short Sleep With Cerebral Hemodynamics

Table 3 shows adjusted linear regression for the AHI and sleep duration. The AHI3 was associated with lower CBFV in the BA with a trend toward increased pulsatility in the BA, with no association with cerebral hemodynamics in the MCA. Actigraphy-defined sleep duration had an inverse association with increased PI in the BA. In contrast, categorical short sleep duration was associated with increased PI and resistivity index in the MCA. No other associations were observed between the actigraphy-defined sleep habits, cerebral hemodynamics, and breath-holding index in the linear regression models (data not shown).

DISCUSSION

In this exploratory study from a community-based subsample of Hispanic/Latino participants free from known cerebrovascular and cardiopulmonary disease, we observed decreased CBFV in the BA during wakefulness in participants with OSA when compared to participants without OSA. Though existing data are limited, our study supports previous reports of decreased resting daytime cerebral blood flow in patients with OSA when compared to controls.[24,25] Interestingly, in adjusted models, we also observed an association between actigraphy-defined short sleep and increased PI in the MCA.

Further, we observed a trend toward less favorable measures of vascular compliance with OSA in the BA. We previously showed increased arterial PI with OSA during wakefulness using TCD, suggesting possible decrease in cerebrovascular compliance and small vessel disease.[13] In a small sample of patients, severity of OSA, reflected by increasing AHI or hypoxia driven respiratory events, may also be associated with greater impairment in cerebral blood flow and autoregulation.[26] Clinically, OSA has been linked with impaired recovery of cerebrovascular blood pressure after orthostasis.[12] Specifically to the BA, there have been limited reports on associations between OSA and BA hemodynamics, though some support impaired vasoreactivity in patients with OSA.[27] In older patients with

known decreased CBFV, the BA may contribute to different metabolic and small vessel demands when compared to the anterior circulation; therefore, different autoregulatory mechanisms may be at play, though this has not been studied.[27]

We did not observe differences in the breath-holding index as a measure of impaired cerebrovascular function. The negative results might be explained by the relatively mild average levels of AHI in our sample, coupled with a small sample size. In addition, the effect of OSA on cerebral hemodynamics might not be evident in a younger sample. However, other studies suggest resting daytime impairments in cerebral blood flow and impaired cerebrovascular autoregulation, may be worsened with increasing hypoxic events in OSA.[12,26]

Sleep Duration and Cerebral Hemodynamics

Additionally, short sleep duration was associated with decreased CBFVs in both the BA and MCA though not significantly. In the BA, we observed decreased CBFV with increased sleep fragmentation and increased CBFV with sleep efficiency. Further, we observed a statistically significant increase in PI with short sleep duration in adjusted models. Some studies have placed both short and long sleepers at greater risk of incidence and mortality from both coronary heart disease and stroke than those sleeping 7 to 8 hours a night.[28] A recent meta-analysis of 16 prospective studies found a J-shaped relationship between sleep duration and total stroke incidence, with long sleepers associated with total, nonfatal, and fatal strokes, and short sleep durations only significantly related to nonfatal strokes.[29] In our study, actigraphy defined sleep duration was used as a continuous variable and categorized into short sleep based on the median of our sample. Previous studies describe large discrepancies between self-reports and actigraphic sleep duration; hence, we did not define short sleep *a priori,* as most previous studies rely on self-reported data with little information on potential stroke mechanisms.[17,18]

Physiological mediators of cerebrovascular disease risk in short sleep duration are not well understood. Chronic sleep deprivation has been linked to metabolic syndrome and increased risk of progression to diabetes in prediabetic patients.[30–32] Though unclear, inflammation, increased caloric intake and weight gain from appetite-related hormonal dysregulation, and association with unhealthy lifestyle choices may play a role in the pathological effects of short sleep duration by leading to increased adiposity and insulin resistance.[33] Shorter sleep durations may associate with worse cerebral hemodynamics by way of increased sympathetic tone, which could cause increased blood pressure, a known contributor to cerebrovascular disease and impaired hemodynamics.[34,35]

Sleep and Small Vessel Disease

Pathophysiology of OSA has been more extensively studied in the cardiovascular system. Generally, OSA is thought to initiate a cascade of oxidative stress, inflammation, platelet dysfunction, and metabolic dysregulation. The sympathetic nervous system is also activated in OSA due to changes in vascular blood gases and pH, sleep arousals, and sleep deprivation.[36] These cellular and molecular mechanisms likely play a role in cerebrovascular pathology seen with OSA, possibly mediating the change in cerebral hemodynamics we observed. This proposed mechanism is a likely gradual chronic process as most patients can have OSA for many years without recognition or diagnosis.[34] It is not clear if potential mechanisms driving cerebral hemodynamic changes between hypoxic respiratory events in OSA and short sleep duration are the same.

Clinically, reduced blood flow may lead to poor blood supply to the cerebral regions more prone to ischemia and resulting in deficits in memory and attention.[37,38] Postmortem studies suggest vascular pathologies can cause cerebral ischemia through impaired blood flow, leading to neurocognitive decline and dementia.[39,40] Hemodynamic abnormalities measured by TCD may be a useful early clinical marker in neurodegenerative disorders with vascular etiology. [41,42] Although this study did not use neurocognitive assessments, further studies on pathogenesis of hemodynamic changes in OSA in association with stroke and dementia are needed.

Limitations of this study include the cross-sectional design that does not allow for assessment of causality. In addition to our small sample size, all participants were recruited at one site in Miami that has proportionally higher numbers of participants of Cuban descent, potentially introducing selection bias and limitations to generalizability. Participants with OSA were significantly older than those without. Several studies show age-related decrease in cerebral blood flow.[43,44] Further, our TCD parameters are measured only at rest and this study did not include vascular function measures that may explain the relationship between OSA and cerebral hemodynamics changes. Other measures of vasomotor reactivity such as CO_2 inhalation challenge test may provide better yield. In addition, functional TCD testing with cognitive and memory tasks have been shown to affect cerebral hemodynamics.[45] Future studies including measures of vascular function could also help explain associations among OSA, sleep duration, and cerebral hemodynamics. Last, we did not apply positive airway pressure while measuring cerebral hemodynamics with TCD, which could improve cerebral hemodynamics. We defined OSA with home sleep studies that have a reduced number of signals compared to polysomnography and may increase false-negative results. However, home sleep studies are considered a good alternative for the diagnosis and management of OSA in those with high pretest probability of OSA, without severe comorbidities, and if able to set up home equipment properly.[44]

In conclusion, we report decreased daytime cerebral blood flow in Hispanic/Latinos with AHI in OSA. Further, short sleep duration may be linked to impaired cerebral hemodynamics as well. These measures of cerebral hemodynamics by TCD in OSA indicate deviations in cerebral vascular blood flow that necessitate further investigation into the pathophysiologic mediation of OSA to cerebrovascular disease risk.

ABBREVIATIONS

AHI, apnea-hypopnea index
BA, basilar artery
CBFV, cerebral blood flow velocities
EDV, end diastolic velocities

HCHS/SOL, Hispanic Community Health Study/Study of Latinos
MCA, middle cerebral artery
MFV, mean flow velocities
MSV, mean systolic velocities
OSA, obstructive sleep apnea
PI, pulsatility index
PSV, peak systolic velocities
RI, resistivity index
TCD, transcranial Doppler

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