9-1-2020

Self-Reported Sensory Gating and Stress-Related Hypertension

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Background: Increasing evidence views hypertension as a stress-induced disorder. Stressors must be "gated" by the brain before any inflammatory or immune processes that contribute to hypertension are initiated. No studies were found that examined sensory gating in relation to hypertension.

Objectives: The aim of the study was to determine if disturbances in self-reported sensory gating could differentiate normotensive from hypertensive young adults.

Methods: A nonmatched, case–control design was used. We administered an online survey to 163 young adult participants. Participants were predominantly female, in their mid-20s, well educated, and approximately evenly distributed by race and hypertension status. The Sensory Gating Inventory (SGI) measured gating disturbances.

Results: The mean SGI scores were significantly higher among persons diagnosed with hypertension, reflecting a moderate effect size of sensory gating. After adjusting for confounders, however, the normotensive and hypertensive groups were not significantly different on their SGI scores.

Discussion: With an observed moderate effect size of 0.35, but low power, more research is warranted regarding the role of gating disturbances in the development of stress-induced hypertension. Clinically, the SGI may be important for screening patients who would benefit from ambulatory blood pressure monitoring to identify persons with masked hypertension.

Key Words: hypertension • masked hypertension • self-report • sensory gating • Sensory Gating Inventory

Nursing Research, September/October 2020, Vol 69, No 5, 339–346

Over 100 million Americans—nearly half of all adults in the United States—are estimated to have high blood pressure (BP) or hypertension (HTN; Benjamin et al., 2018). The number of adults with HTN has increased with new guidelines that now define Stage 1 HTN as systolic BP between 130 and 139 mm Hg or diastolic between 80 and 89 mm Hg (Whelton et al., 2018). The high prevalence of HTN is a serious public health concern, as high BP is a major risk factor for a number of other diseases (e.g., heart failure, stroke, chronic kidney disease) as well as increased morbidity and mortality. In 2015, HTN was associated with approximately 16% of all deaths (Benjamin et al., 2018). In addition, the rate of HTN increases as one ages. The prevalence of HTN nearly quadruples from the 18–39 years age group to the 40–59 age group (Fryar et al., 2017). Thus, identifying at-risk individuals as early as possible is critical for preventing the onset and sequelae of this potentially deadly disease.

Psychological stress has long been considered to be a risk factor of HTN. However, increasing evidence indicates that stress is actually a causative factor in the development of this disease (Harrison et al., 2011; Marvar & Harrison, 2012; Marvar et al., 2012; Rodriguez-Iturbe et al., 2017). The stress-induced physiological cascade that leads to HTN begins with the central nervous system (CNS) phenomenon of *sensory gating*. Although a great deal of attention has been given to the role of the neural/neuroendocrine systems in the development of HTN, scant research has been done on the role of CNS habituation and sensory gating in the stress response. The purpose of this study, therefore, was to explore if self-reported disturbances in sensory gating were associated with a diagnosis of HTN in young adults 18–30 years of age.

Sensory Gating

Habituation is a decrease in responsiveness to ongoing stressful stimuli and is a major protective mechanism of the CNS (Eisenstein & Eisenstein, 2006). Sensory "gating" is an important aspect of habituation that refers to the brain's ability to selectively regulate its sensitivity to irrelevant sensory stimuli and thus direct processing resources to more important environmental stimuli (Davies et al., 2009). Sensory gating shows substantial heritability (Earls et al., 2016); however, factors in the prenatal and early childhood environment can affect brain development, including gating ability. Neural circuits underlying gating are functional very early in postnatal development

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DOI: 10.1097/NNR.0000000000000440

(1–4 months; Kisley et al., 2003) and continue to mature until about 10 years of age (Brinkman & Stauder, 2007).

Adverse childhood events (e.g., social and economic deprivation, trauma) may lead to "biological embedding" affecting neural circuitry (Hertzman & Boyce, 2010). Such adverse events are associated with gating deficits (McFarlane et al., 2005) and therefore, can influence the child's ability to process environmental stressors later in life. There is growing evidence suggesting that early stress affects the development of prefrontal–hippocampal circuits that integrate past experience and current context to modulate reactivity to stressors (LeDoux, 2012). Hence, the effect of early trauma on the development of gating may result in diminished emotional regulation and increased stress reactivity.

Stress and HTN

Lovallo's (2005) model of cardiovascular reactivity provides a theoretical framework that links sensory gating with central and peripheral nervous system responses that lead to HTN. The basic hypothesis of Lovallo's three-level model is that exaggerated physiological reactivity to stress may begin at multiple levels of the nervous system and in disease-altered tissues. Physiological reactivity to stress may begin in the cerebral cortex and limbic system of the brain. These form a functional unit to detect stressors and then coordinate cognitive–emotional and physiological responses to stressful stimuli. Deficits in sensory gating arising from the caudal orbital/medial frontal cortical regions can affect autonomic responses, such as increased heart rate and BP. They can also trigger a cascade of immune responses, including release of proinflammatory cytokines (e.g., interleukin-6 [IL-6] and tumor necrosis factor-alpha [TNF-α]; Harrison et al., 2011). Increases in IL-6 can then stimulate the release of C-reactive protein (CRP) from the liver and arterial smooth muscle cells (Harrison et al., 2011). CRP, IL-6, and TNF-α can also regulate the expression of other inflammatory cytokines.

Trott and Harrison (2014), in a series of elegant experiments in animal models, found that activation of the CNS activates peripheral T cells that infiltrate the vasculature and kidney where they produce cytokines that stimulate salt and water retention, vasoconstriction, and vascular remodeling. They also provided experimental evidence that peripheral BP elevations "feed forward" to activate the CNS (along with angiotensin II, stress, aldosterone, high dietary sodium) to augment central sympathetic nervous system (SNS) outflow and activation of peripheral T cells. Thus, via its connections to the immune system, SNS activation produces inflammatory cytokines that unleash physiological processes that raise BP, injure the vasculature, and increase the risk of HTN (Calabró et al., 2003; Carnagarin et al., 2019). In addition, angiotensin II does not increase the risk of HTN through sodium balance and vascular tone alone, but also through stimulation of the immune system leading to T-cell activation, increased proinflammatory cytokine release, and vascular inflammation (Harrison et al., 2011; Higaki et al., 2019; Marvar & Harrison, 2012; Marvar et al., 2012; Rodriguez-Iturbe et al., 2017).

Initially viewed as a top-down model of stress responses, research has now demonstrated that, in the face of *chronic* stress, there is a bidirectional relationship as tissue changes and circulating neurohormones affect feedback efficacy and provide bottom-up input that affects CNS processing of stressors (Carnagarin et al., 2019; Ganzel et al., 2010; Marvar & Harrison, 2012; Marvar et al., 2012; McEwen, 2007). Further evidence demonstrates this bidirectional communication between the CNS and immune system, and details how neuroimmune mechanisms contribute to the neurogenic component of HTN (Marvar & Harrison, 2012). Ulrich-Lai and Herman (2009) propose that, although higher order processing in the effector circuits that control stress responses are probably hard-wired, "it is likely that dysfunctions of information processing across these circuits, resulting from environmental adversity and/or genetic factors, lie at the root of maladaptive stress reactions that can culminate in affective disease (e.g., depression, PTSD) and physical infirmities" (p. 406), such as HTN.

Based on this empirical evidence, we hypothesized the following relationships between gating and HTN. First, disturbances in sensory gating affect the perception and evaluation of sensory stimuli (Lovallo's Level I), triggering hypothalamic and brainstem activity. This activity results in SNS responses of increased BP, alterations in immune and inflammatory processes (Level II). These Level II responses contribute to altered vascular function and salt sensitivity (Level III) that are precursors of HTN. Therefore, we hypothesize that sensory gating deficits are associated with increased risk of HTN.

Measuring Sensory Gating

Although disturbances in sensory gating were first described subjectively, in the classic phenomenological study of McGhie and Chapman (1961), much of the recent work in sensory gating has been done using a physiological, paired-click auditory protocol, rather than a self-report measure. During the physiological procedure, gating is assessed by exposing subjects to pairs of identical auditory clicks (S_1, S_2) and measuring the electroencephalogram (EEG) responses (positive and negative peaks in the auditory evoked potential waveforms) at different time points after the stimulus (e.g., P50, N100 msec; Boutros et al., 1999). Gating is measured as the ratio of the amplitude of response to S_2 stimuli by the amplitude of response to S_1 stimuli ($S_2/S_1 \times 100$), with higher ratios reflecting less gating and lower ratios reflecting more intact gating (Boutros et al., 1999). However, using the auditory paired-click, physiological procedure to measure sensory gating is time-consuming, is burdensome to participants, and requires EEG experts to measure and interpret the results. Thus, measuring P50 suppression has little use as a screening tool.

More recent work by Hetrick et al. (2012) has offered a reliable self-report scale to explore the phenomenological dimensions of sensory gating and provides a clinically feasible measure of the gating construct. The Sensory Gating Inventory (SGI) has been found to be highly correlated with physiological measures of sensory gating—specifically reduced P50 suppression (Hetrick et al., 2012; Micoulaud-Franchi et al., 2014; Micoulaud-Franchi, Lopez, et al., 2015; Micoulaud-Franchi, Vaillant, et al., 2015)—and thus provides a valid self-report measure of this neurophysiological process.

In addition to self-reports of sensory gating being validated by their strong association with neurophysiological EEG measures in patient groups as well as healthy young adults (Johannesen et al., 2008; Micoulaud-Franchi et al., 2014), self-reported disturbances have also been strongly linked to disorders associated with HTN. Self-reported disturbances have been associated with neuropsychiatric disorders (e.g., anxiety, depression) as well as with disorders that often begin in childhood, including attention-deficit/hyperactivity disorder (ADHD), posttraumatic stress disorder, and schizophrenia (Abouzeid et al., 2012; Hetrick et al., 2012; Johannesen et al., 2008; Micoulaud-Franchi et al., 2014, 2017; Micoulaud-Franchi, Vaillant, et al., 2015). In adults, each of these conditions is highly comorbid with inflammation, HTN, and lifestyle behaviors associated with HTN (Abouzeid et al., 2012; Baker et al., 2012; Deuschle et al., 2013; Edmondson et al., 2018; Fuemmeler et al., 2011; Mitchell et al., 2013). However, no studies could be found that directly examined the association of sensory gating with HTN.

The aim of this study was to determine if disturbances in self-reported sensory gating could differentiate normotensive from hypertensive patients. We hypothesized that persons diagnosed with stress-related HTN would report more disturbances in sensory gating.

METHODS

Design

A nonmatched, case–control study using an online survey was conducted with young adults receiving primary care within a large, integrated health system within metropolitan Detroit. Intuitional review board approval was obtained from the principal investigator's university as well as from the health system. Following that approval, programmers from the health system identified nearly 2,000 eligible patients (cases and controls) who had a documented health visit within the previous 9 months and had e-mail information on record. The sample was restricted to the age range of 18–30 years in order to identify participants before target organ damage had occurred. E-mails from cases and controls were provided by the programmers. Cases were young adults with a diagnosis of primary HTN (ICD-9 code 401.9) and at least one antihypertensive medication ordered. Controls were young adults receiving care within the institution's primary care clinics, who did not have a recorded diagnosis of HTN, diabetes, kidney disease, cancer, pregnancy, or mental health conditions.

The e-mail addresses were loaded into the Qualtrics Research Suite that is housed on a secure university server. The Qualtrics program then sent e-mails to potential participants providing a brief overview of the study. If the patient was willing to participate, they followed a link to the survey. After reading the consent information and verifying that they were between 18 and 30 years of age, participants completed the survey. Participants could choose to submit their names and contact information by clicking on a link to a different site on the secure university server where they would be eligible for a drawing to receive one of five Visa gift cards worth $100 each.

Recruitment included sending e-mails to 1,923 participants and a follow-up, reminder e-mail 10 days later. Immediate "bounce back" identified 52 e-mails that were no longer valid, and three participants (three men, one woman) asked to be removed from the contact list, resulting in 1,868 potential participants. From that, 200 patients attempted the survey, but 32 people did not complete study questionnaires during the time that the survey link was open. Of the 168 completed responses, five participants were older than the 30-year inclusion criteria and were excluded. Thus, the analytic sample consisted of 163 participants; representing a 9% overall response rate, which is not uncommon for a one-time contact online survey with limited incentives (Hunter et al., 2013).

Measurement and Instrumentation

Demographic data regarding age, gender, race, level of education, and household income were collected. Patients also self-reported as to whether they had ever been diagnosed with potential confounders of anxiety, ADHD, and posttraumatic stress disorder, as *yes/no* response options. Also, self-reported was whether they had experienced childhood trauma, and if yes, the number of traumatic events they experienced.

HTN was based on ICD-9 codes for HTN and documentation of at least one antihypertensive medication prescribed.

The SGI (Hetrick et al., 2012) was used to measure the phenomenological aspects of sensory gating. The SGI is a self-administered instrument containing 36 items that measure the frequency and nature of auditory and visual phenomenology associated with sensory gating. It is a multidimensional instrument with four subscales: perceptual modulation, distractibility, overinclusion, and vulnerability to gating anomalies during periods of stress and fatigue. *Perceptual modulation* assesses participants' ability to modulate stimulus intensity and perceptual inundation. *Distractibility* measures difficulties of focal attention, whereas *overinclusion* assesses anomalies of radial attention as a result of a low threshold of perception. *Vulnerability* reflected perceptual and attentional anomalies during periods of fatigue and stress.

Participants report the frequency with which the items reflect their life experiences using a 6-point Likert scale (0 = *never true* to 5 =*always true*), with a possible total score range of 0–180. Higher scores indicate increased disturbances in sensory gating. Extensive work has been done to establish the reliability and validity of the instrument using healthy young adults (predominantly college students; Hetrick et al., 2012), including demonstrating its strong association with neurophysiological measures of sensory gating (Hetrick et al., 2012; Johannesen et al., 2008; Kisley et al., 2004; Micoulaud-Franchi et al., 2014). In the current study, Cronbach's alpha for the total SGI scale was .97 and ranged between .86 and .93 for the four subscales.

Data Analysis

Statistical analayses were performed using the latest version of SPSS. Analysis began with careful data screening for assessment of outliers, missing data, and testing assumptions of multivariate analysis (Tabachnick & Fidell, 2013). The variable body mass index (BMI) had two outlier cases (BMI > 60) that were managed using group means substitution. All variables were also explored for the extent of a pattern of missing data. There were no missing data on the outcome variable of HTN status; however, some participants had missing data on single SGI items. These missing data were imputed using case mean substitution (Fox-Wasylyshyn & El-Masri, 2005). Multicollinearity among the study variables was not present in our sample.

Descriptive statistics were conducted to describe patient characteristics. Independent-sample *t* test and chi-square comparisons were performed to compare patient characteristics between those with diagnosis of HTN and those without. Multivariate logistic regression was then conducted to compare the HTN and normotensive groups on their SGI scores while adjusting for potential confounders. To maximize parsimony of the resulting regression model, only variables with a *p* value of ≤.25 in the bivariate *t* test and chi-square comparisons were considered in the multivariate logistic regression analysis. Statistical significance was established as $p < .05$ or a 95% confidence interval (CI) that did not include 1.0.

RESULTS

Sample Characteristics

Table 1 displays the demographic characteristics of the 163 participants. Results illustrate that most of the respondents were women, well educated, fairly evenly split by race, and with a wide range of income. Table 1 also illustrates that the average BMI was 30.18 (*SD* = 9.3, *Mdn* = 28.05), and 31 participants (19.0%) were in the extreme obesity category (BMI ≥ 40). Whereas 45% ($n = 73$) of the sample had a diagnosis of HTN, 80.6% of those with extreme obesity had HTN. The most documented comorbid disorders were anxiety (29.4%) and depression (25.2%), but 39 participants (23.9%) also reported childhood trauma. SGI scores ranged from 0 to 4.44 (*M* = 1.49, *SD* = 0.94) on individual items and from 0 to 160 (*M* = 52.55, *SD* = 32.67) for the total SGI score.

Group Comparisons Based on HTN Status

The unadjusted comparisons in Table 1 illustrate that education, $\chi^2(2, N = 136) = 9.68$, $p = .008$, and BMI, $t(161) = 8.49$, $p < .001$, were the only demographic characteristics that were different based on HTN status. Patients with HTN diagnosis had a higher SGI score than those without (*M* = 58.9 and 47.4, respectively), $t(161) = 2.19$, $p = .03$, Cohen's $d = .35$. In addition, the mean fatigue and stress subscale score was different between the two groups, $t(161) = 2.07$, $p = .041$.

Table 2 displays the unadjusted and adjusted logistic regression results. In addition to assessing the total sample, results for women alone are also presented because the sample was predominantly female.

The unadjusted results indicate that, in the total sample, HTN was significantly associated with total SGI (*OR* = 1.01, 95% CI [1.008, 1.02]) and fatigue–stress subscale scores (*OR* = 1.06, 95% CI [1.01, 1.11]). In the unadjusted women-only results, HTN was significantly associated with the total SGI score (*OR* = 1.02, 95% CI [1.01, 1.03]) and subscale scores for perception modulation (*OR* = 1.04, 95% CI [1.01, 1.08]) and overinclusion (*OR* = 1.06, 95% CI [1.01, 1.12]). The adjusted regression results, however, illustrate that neither the SGI nor any of its four subscales was significantly associated with HTN after adjusting for race, education, income, anxiety, and BMI. This result was true for the total sample and for women alone.

DISCUSSION

This is the first study that has examined the relationship of self-reported disturbances in sensory gating and HTN. Other than pilot studies that used a neurophysiological measure to assess cardiovascular reactivity (Peters et al., 2011), no other studies could be found that examined the relationship between processing of environmental stressors and BP outcomes. Three major findings have emerged from this study. First and the major finding, is that the association between self-reported disturbances of sensory gating and HTN warrants further research. This is because, in our study, the SGI mean score was 11 points higher among patients with HTN, with a moderate effect size of 0.35. Second, the weight of participants in our sample was not representative of the general public. Participants in our study were mostly obese, and most of those with HTN were also obese. Although the association of BMI and SGI was weak and nonsignificant, the level of obesity of participants in this study may have attenuated the true relationship between self-reported SGI and HTN. This confounding was evident as the relationship between SGI and HTN went from being significant in the univariate analysis to nonsignificant in the adjusted analysis. Third, results provide beginning support for the use of a

TABLE 1. Sample Characteristics

Variable	Hypertension			χ^2/t	*p*
	No (*n* = 90)	Yes (*n* = 73)	Total (*N* = 163)		
Gender, *n* (%)				0.354	.552
Male	21 (23.3)	20 (27.4)	41 (25.2)		
Female	69 (76.7)	53 (72.6)	122 (74.8)		
Race, *n* (%)				5.37	.068
Caucasian	51 (56.7)	34 (46.6)	85 (52.1)		
African American	27 (30.0)	34 (46.6)	61 (37.4)		
Other	12 (13.3)	5 (6.8)	17 (10.4)		
Age in years, *n* (%)				2.81	.245
18–24	19 (21.1)	11 (15.)	30 (18.4)		
25–28	40 (44.4)	42 (57.5)	82 (50.3)		
29–30	31 (34.4)	20 (27.4)	51 (31.3)		
Education, *n* (%)				9.68	.008
High school or less	5 (5.6)	15 (20.5)	20 (12.3)		
Some college/college	64 (71.9)	49 (67.1)	113 (69.8)		
Graduate	20 (22.5)	9 (12.3)	29 (17.9)		
Annual gross income				5.77	.123
<$25,000	20 (22.5)	25 (34.7)	45 (28.0)		
$25,000 to <$50,000	20 (22.5)	20 (27.8)	20 (24.8)		
$50,000 to <$75,000	19 (21.3)	13 (18.1)	32 (19.9)		
≥$75,000	30 (33.7)	14 (19.4)	44 (27.3)		
ADD diagnosis, *n* (%)	9 (10.0)	10 (13.7)	19 (11.7)	0.54	.464
PTSD diagnosis, *n* (%)	3 (3.3)	5 (6.8)	8 (4.9)	1.07	.301
Anxiety disorder, *n* (%)	23 (25.6)	25 (34.2)	48 (29.4)	1.47	.226
Depression, *n* (%)	20 (22.2)	21 (28.8)	41 (25.2)	0.92	.338
Childhood trauma, *n* (%)	20 (22.2)	19 (26.0)	39 (23.9)	0.32	.571
Childhood poverty, *n* (%)	15 (16.7)	14 (19.2)	29 (17.8)	0.17	.677
BMI, mean (SD)	25.3 (4.13)	36.2 (10.38)	30.18 (9.33)	8.49	<0.001
SGI total score	47.38 (26.61)	58.92 (38.10)	52.55 (32.67)	2.19	.030
Perception modulation	14.3 (10.42)	16.03 (10.32)	15.10 (10.38)	1.05	.296
Distractibility	14.34 (8.48)	16.01 (10.68)	14.72 (9.51)	0.56	.579
Overinclusion	11.78 (7.21)	13.90 (7.89)	12.73 (7.57)	1.80	.077
Fatigue and stress	5.22 (0.55)	6.90 (0.81)	8.75 (6.10)	2.07	.041

Note. P values compare normotensive to hypertensive. ADD = attention-deficit disorder; PTSD = posttraumatic stress disorder; BMI = body mass index; SGI = Sensory Gating Inventory.

self-report instrument as a potential screening tool to identify persons at risk of masked HTN and in need of 24-hour ambulatory monitoring.

Our findings indicated that those with HTN had a mean SGI score that was 11 points higher than that of normotensive patients. Unfortunately, though, this relatively high margin was not significant once we adjusted for BMI in our sample. This was not at all surprising given the fact that our sample trended toward obesity. In our sample, the BMI for the HTN group suggested that they were morbidly obese, whereas the normotensive group was in the overweight category. Thus, our sample was not truly representative of the general population in terms of its BMI scores and how they were distributed across the study groups. In addition, our post hoc power analysis indicated that the actual power of our study based on the reported group means was 60% and that our sample should have been almost doubled ($N = 260$) to achieve the necessary 80% power. Thus, we recommend that our study be replicated on a larger sample that is more representative of the target population in terms of BMI distribution before any conclusions can be made about the association between SGI and HTN.

Further research is also warranted on the relationship of sensory gating and BMI. Although it is well established that BMI is a risk factor for HTN, little is known about the role of gating in the development of obesity. Only one study could be found that examined the relationship of BMI and sensory gating (Tecellioğlu et al., 2015). That study measured sensory gating using the auditory, paired-click physiological method. Results indicated that obese individuals had poorer P50 gating than patients in the control group. That was an observational

TABLE 2. Crude and Adjusted Logistic Regression Results for the Total and Women-Only Samples

	All cases (*N* = 163)						Women only (*n* = 122)					
	Unadjusted			Adjusted			Unadjusted			Adjusted		
Variable	*OR*	95% CI	*p*	*OR*	95% CI	*p*	*OR*	95% CI	*p*	*OR*	95% CI	*p*
SGI total	1.01	[>1.0, 1.02]	.03	1.0	[0.99, 1.02]	.35	1.02	[1.01, 1.03]	.004	1.02	[1.0, 1.03]	.11
Perception modulation	1.02	[0.99, 1.05]	.03	1.01	[0.97, 1.05]	.41	1.04	[1.01, 1.08]	.002	1.03	[0.99, 1.08]	.18
Distractibility	1.00	[0.98, 1.04]	.58	1.0	[0.95, 1.05]	.99	1.02	[0.99, 1.06]	.21	1.02	[0.97, 1.10]	.52
Overinclusion	1.04	[1.00, 1.08]	.08	1.00	[0.94, 1.06]	.97	1.06	[1.01, 1.12]	.018	1.02	[0.95, 1.09]	.65
Fatigue and stress	1.06	[>1.0, 1.11]	.04	1.04	[0.97, 1.12]	.40	1.1	[1.03, 1.18]	.003	1.08	[0.99, 1.18]	.09

Note. Adjusted results controlled for race, education, income, anxiety disorder, and body mass index. SGI = Sensory Gating Inventory.

study, so the temporal relationship between gating deficits and obesity remains unknown. Those results combined with the data from the current study warrant further investigation.

Much of the work in sensory gating has been done using a physiological rather than self-report measure, specifically using a paired-click auditory protocol and then measuring the EEG responses. Using the auditory paired-click, neurophysiological procedure to measure sensory gating is time-consuming, is burdensome to participants, and requires EEG experts to measure and interpret the results. Thus, measuring P50 suppression has little use as a screening tool. However, the self-report measure SGI has been found to be highly correlated with physiological measures of sensory gating—specifically reduced P50 suppression (Hetrick et al., 2012; Micoulaud-Franchi et al., 2014; Micoulaud-Franchi, Lopez, et al., 2015; Micoulaud-Franchi,Vaillant, et al., 2015)—and thus provides a valid self-report measure of this neurophysiological process. Results from the current study are intriguing and suggest that, with more research, the SGI may be a clinically relevant, self-report measure that identifies young people at greatest risk for stress-induced HTN. It may also have the potential to identify patients with masked HTN and identify patients most in need of stress reduction interventions.

Although it was compromised by lack of statistical power, our unadjusted finding, which suggested that for every unit increase in SGI, there is a 2% increase in the likelihood of a woman having HTN, is worth further exploration. This is because with a possible total score range of 0–180, a 10-point increase on SGI could be associated with a 20% increase in HTN. Given that as many as 30% of young adults have masked HTN (i.e., normal clinic reading but elevated ambulatory BP; Cuspidi et al., 2015; Tientcheu et al., 2015) and that the cost of ambulatory BP measurement limits its use as a screening tool, establishing the HTN predictive ability of SGI could aid in making cost-conscious decisions for screening, early diagnosis, and treatment of HTN. In addition to screening for masked HTN, the SGI may provide additional therapeutic benefit to patients with difficult-to-control BP. It may be that patients with sensory gating deficits would especially benefit from stress reduction interventions in order to achieve their BP goals. However, both of these clinical uses of the SGI would require additional specific study.

Further research also is needed to determine if the full SGI is necessary for identifying persons at greatest risk of HTN or with masked HTN. It may be that not all items on the SGI have equal relevance in relation to HTN. In our unadjusted analyses, perceptual modulation over inclusion and fatigue–stress were significantly different between participants with and without HTN. It is possible that a smaller set of questions may prove to be equally valid for discriminating between normotensive and hypertensive patients. A smaller set would be especially important for clinical use. Recently, Micoulaud-Franchi et al. (2017) developed the SGI-16, a shortened version. Initial testing in adults with ADHD revealed strong correlations between the 16- and 36-item SGI scales. Use of the SGI-16 with persons at risk of HTN and its sequelae is warranted.

Limitations

Although the results of this study provide beginning evidence of the relationship of the SGI to HTN, there are limitations to consider. We did not obtain information on medications that patients may have been taking for either HTN or psychosocial distress, which may have attenuated the relationship between HTN and gating deficits. In addition, it is possible that some of the participants in the control group may have undiagnosed HTN, which again would affect the relationship between diagnosis and gating. Self-selection is a potential bias for this study given the large number of persons who received the e-mail recruitment letter versus those who responded to the online survey. The overrepresentation of women and individuals with high BMI scores in our sample may compromise the generalizability of our findings and suggest self-selection bias. The online nature of the survey may yield different results than if participants had been recruited from the clinic sites and completed the survey as a paper-and-pencil instrument. Although the online component may have reduced social desirability

in responses, a mono-method bias cannot be ignored. Furthermore, the self-report nature of the survey may have affected study results. Representativeness is a factor in all online surveys and is most often discussed in terms of demographics. However, one study (Brüggen et al., 2011) found that respondents may also vary based on different response motives, including participant assessment of the relevance of the survey request. For the current study, we suspect that generalizability is affected by the fact that respondents were predominantly well-educated women, whose motivations we are unable to determine. The design of this study is also a limitation. Future studies that prospectively evaluate SGI with incidence of HTN are needed. Despite its limitations, the current study provides important new information for nurses to consider when working with patients at risk or already diagnosed with HTN. Specifically, linking sensory gating with a cardiovascular outcome provides a holistic view of patients, rather than studying them one organ system at a time. Self-reported sensory gating may also provide new insights into the patient–environment interaction as a contributor to health problems.

Conclusion

The current study presents a new, important direction for future research concerning the association between sensory processing of environmental stimuli and the development of HTN. The results suggest the need for more research on larger and more representative samples in terms of gender and BMI distribution. This is especially true given the inherent limitations of the case–control nature of our relatively small study.

Accepted for publication January 20, 2020.

Research reported in this study was supported by a Lily H. Siegert Endowed Faculty Research Award, College of Nursing, Wayne State University, Detroit, MI.

The authors alone are responsible for the content and writing of the paper.

This study was approved by the institutional review boards of Henry Ford Health System and Wayne State University.

The authors have no conflict of interests to report.

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