Journal of Biosciences and Medicines, 2024, 12, 236-248
https://www.scirp.org/journal/jbm
ISSN Online: 2327-509X
ISSN Print: 2327-5081

Progress in Research on Primary Snoring and Hypertension

Xiaoling Zhang*, **Yingqing Feng**#

Department of Cardiology, Zhuhai People's Hospital Affiliated to Jinan University, Zhuhai, China
Email: 15186139633@163.com, #651792209@qq.com

How to cite this paper: Zhang, X.L. and Feng, Y.Q. (2024) Progress in Research on Primary Snoring and Hypertension. *Journal of Biosciences and Medicines*, **12**, 236-248.
https://doi.org/10.4236/jbm.2024.1212019

Received: November 5, 2024
Accepted: December 9, 2024
Published: December 12, 2024

Abstract

Research has established a significant association between primary snoring and hypertension, suggesting that primary snoring may be a potential risk factor for developing high blood pressure. This condition can adversely impact the cardiovascular system and metabolic processes, promote vascular smooth muscle hypertrophy, and either contribute to or exacerbate hypertension. Presently, comprehensive management strategies are primarily implemented for the treatment of both primary snoring and hypertension.

Keywords

Primary Snoring, Snoring Measurement, Hypertension, Prognosis of Hypertension, Intervention Measures

1. Introduction

Hypertension is a prevalent chronic condition that affects many individuals and presents challenges for both personal health and society, contributing notably to healthcare costs and economic burdens. It is also a crucial risk factor for cardiovascular diseases, underscoring its importance for our overall health. Similarly, snoring, often associated with sleep disorders, occurs when the muscles in the throat relax during sleep. This relaxation causes the surrounding tissues to vibrate, producing sound [1]. Snoring can be a sign of several conditions, including primary snoring (PS), upper airway resistance syndrome (UARS), and obstructive sleep apnea (OSA), among other sleep disorders. Notably, PS may indicate early signs of narrowing in the upper airway during sleep, potentially signaling the initial stages of upper respiratory issues.

*First author.
#Corresponding author.

2. Definition of PS

Primary snoring is defined as the regular sound produced by the vibration of the soft palate and adjacent tissues during sleep. To ascertain whether snoring behavior within a population indicates the presence of OSA or is classified as PS, a definitive diagnosis necessitates using polysomnography (PSG). This multi-channel sleep monitoring procedure is conducted in a controlled laboratory environment to ensure accurate results. Most current studies define the apnea-hypopnea index (AHI) using a cutoff value of less than 5 - 15 episodes per hour of sleep. Some studies also incorporate BMI [2] [3], peak snoring intensity [4], and absence of daytime symptoms [5] into the definition. The widespread adoption of PSG encounters several challenges. These include a complex and time-consuming operation process, the high costs of the necessary equipment, and the requirement for patients to visit specialized sleep laboratories for testing. Additionally, skilled personnel are needed to monitor and analyze the results. Long waiting periods for appointments further complicate efforts to promote its widespread use. The current definition of PS is still lacking a consistent conclusion. Therefore, epidemiological studies on snoring can increase the possibility of screening for OSA to some extent. In current hypertension management, actively improving patients' adverse sleep behaviors is vital.

3. Snoring Measurement Methods

Objective measurement devices for snoring encompass PSG conducted in laboratory settings, portable PSG, and smartphone application software (**Table 1**) designed for monitoring the duration and peak intensity of snoring. In the subjective realm, questionnaires are frequently employed to collect data regarding the intensity and frequency of snoring episodes. The inherent variability of snoring during nighttime, coupled with the lack of standardized reference values among researchers and clinicians concerning intensity and frequency, contributes to the absence of specific diagnostic criteria for evaluating both acoustic indicators of snoring and the associated

Table 1. Measurement methods for snoring.

Measurement Equipment	Detection Method	Measurement Content	Influencing Factors	Applicable Population
PSG/Portable Sleeping Monitoring Recorder (PSMR)	Microphone [6] [7]	Acoustic analysis	The location of the microphone Position during sleep The intensity threshold of snoring	Patients with a high suspicion of moderate to severe OSA and other sleep-disordered breathing conditions; or those with severe complications or other sleep disorders, such as chronic obstructive pulmonary disease, congestive heart failure, neuromuscular disorders
PSG/PSMR	Piezoelectric sensor [8] [9]/humidity sensor [10]	The vibration caused by snoring is converted into an electrical signal that oscillates in proportion to the change in air pressure/periodic humidity changes during breathing and is monitored to record breathing behavior	Sensor Location/The algorithm for converting vibration frequency/sleep breathing humidity into electrical signals is inconsistent	
PSG	nasal transducer [11]-[13]	Capture pressure airflow signals and audio recordings	Sleeping Positions Sensor placement is affected by changes in sleep posture/pressure differences defining snoring	

Continued

Smartphone applications	Microphone [14] [15]	Acoustic analysis	Sleeping Position of Mobile Phone/Differences in Defining Snoring Parameters/Differences in Approaches to Handling Snoring Sounds	OSA remote management, monitoring, and follow-up have certain application prospects.
Questionnaire survey [16] [17]	"Do you snore when you sleep?" Those who answered "yes" were further asked about their snoring frequency. Participants were divided into "never/unknown," "occasionally," and "often."	The data is typically collected through face-to-face interviews or questionnaires conducted by researchers who have received training	Subjectivity/Privacy/Unobjective	Epidemiological survey of snoring and screening for OSA

non-acoustic and social impacts. Although there are various methods for measuring snoring, the measurement results still show heterogeneity due to individual differences, environment, device accuracy, and the subjective judgments of the assessors.

Estimating snoring parameters at a single nighttime point may not comprehensively reflect the underlying characteristics of snoring or the cumulative exposure risks associated with these characteristics over time. Consequently, it is imperative to integrate snoring assessments into clinical care and the management of sleep disorders. In contrast to relying on PSG, which involves single-night recordings in laboratory environments, the exploration and application of novel methods that detect and evaluate snoring in a simple, objective, and non-invasive manner offer enhanced clinical practicality and convenience.

4. Clinical Features of PS

Craniofacial abnormalities and pharyngeal collapse during sleep are recognized as potential etiological and pathophysiological factors contributing to the progression of OSA. Among these factors, the obstruction theory attributable to the hypertrophy of the uvula and palatal structures is widely accepted. PSG is regarded as the gold standard for differentiating between PS and OSA; however, it cannot pinpoint the specific site of obstruction. Karakoc *et al.* [18] did not reveal significant differences in nasal congestion between patients who snore without OSA and those with OSA characterized by an unspecified AHI.

Nevertheless, noteworthy differences were identified concerning the location of airway obstruction as categorized by the Fujita classification and the Mallampati tongue size classification within the AHI-based grouping. The evaluation of the obstruction site is of paramount importance for informing patient treatment strategies. A study [19] examining the cephalometric characteristics of Turkish male populations revealed that the cephalometric analysis of individuals with PS and OSA exhibited varying degrees of similar traits. These included an increased distance between the mandibular plane and the hyoid bone, a reduced posterior airway space, and an elongated soft palate. In OSA subjects, the posterior airway space and the distance between the mandibular plane and the hyoid bone were notably greater. In contrast, the distance for PS subjects exceeded that of the normal control group. These findings may elucidate the posterior displacement of the tongue and the constriction of the pharyngeal airway, particularly in obesity, which may contribute to neck fat deposition leading to the downward movement of the hyoid bone. The cephalometric analysis of PS and OSA subjects showed varying degrees of similar characteristics, including increased distance between the mandibular plane and hyoid bone, reduced posterior airway space, and increased length of the soft palate. The OSA subjects had higher values for the posterior airway space and the distance between the mandibular plane and hyoid bone than the normal control group. In contrast, the PS subjects had a higher mandibular plane and hyoid bone distance. This may explain the backward movement of the tongue and narrowing of the pharyngeal airway, where obesity leading to neck fat deposition may cause the hyoid bone to move downward. Previous studies have reported that the hyoid bone level of PS subjects is more similar to the normal control group rather than OSA subjects [20].

Numerous studies have demonstrated that OSA is an independent risk factor for hypertension, and some studies consider OSA to be a progressive disease, with PS and patients with mild and moderate OSA experiencing an increase in AHI over time. However, this is affected first by weight gain and then by time [21]. In contrast to pharyngeal muscle hypertrophy in the obstruction theory, the neurogenic hypothesis of OSA postulates that the vibrating action of snoring leads to nerve atrophy, which in turn leads to muscle atrophy [22]. Two theories are often considered mutually exclusive, but they may complement each other. That is, the initial differences in the pharyngeal structure may lead to snoring, which can cause atrophy of some muscles and compensatory hypertrophy of different muscles, exacerbating respiratory problems and anatomical structural changes. The vibrational effects of snoring may have a degenerative impact on the local nerves, creating a self-perpetuating cycle that can lead to progressive nerve damage. This damage may impair the ability of the upper airway muscles to maintain their openness.

Additionally, Gates *et al.* [23] conducted a study on a small group of 11 patients who did not have UARS or cortical arousal. They found that, compared to a control group, snorers showed increased activity in the sympathetic nervous system and decreased activity in the parasympathetic nervous system. The imbalance in autonomic nervous system regulation and heart rate variability alterations are being explored as a potential mechanism through which snoring may contribute to cardiovascular events. This study posits that PS, even in the absence of upper UARS, may indicate a continuum of disease susceptibility that could ultimately progress to severe OSA. Although there are no overt pathological or injurious manifestations associated with PS, its potential neural implications and its role in the development of OSA merit careful examination.

5. The Current Research Status of PS and Hypertension

5.1. PS and Hypertension Risk Factors

Arteriosclerosis (AS) represents an early manifestation of the aging process within the vascular system, characterized by a deterioration in the arterial ability to contract and relax. This condition diminishes the elasticity of the arteries and results in an increase in pulse pressure, which subsequently inflicts direct damage on the cardiovascular system. In the progression of arterial lesions, the deterioration of contraction and relaxation functions typically precedes the onset of structural abnormalities. This deterioration gradually escalates into a series of serious cardiovascular events, including ischemia, infarction, and the potential for arterial rupture. Pulse Wave Velocity (PWV) measurement technology is a straightforward and non-invasive approach for assessing the stiffness of arteries. This methodology effectively evaluates arterial rigidity, providing valuable insights into cardiovascular health. A study has found [24] that 30 children who snore but do not demonstrate apnea or hypoventilation on polysomnography are considered to have primary snoring diagnosed with PS demonstrated significantly elevated blood pressure readings across several parameters compared to the control group. Specifically, the systolic blood pressure was measured at 112 ± 10 mmHg in the PS group, in contrast to 105 ± 8 mmHg in the control group ($P = 0.001$). The diastolic blood pressure was recorded at 60 ± 7 mmHg for the PS group, compared to 53 ± 9 mmHg for the control group ($P = 0.004$). Furthermore, the mean blood pressure in the PS group was 81 ± 7 mmHg, while 71 ± 8 mmHg in the control group ($P < 0.001$). Additionally, the PWV was notably higher in the PS group, registering at 9.7 ± 1.6 m/s as opposed to 7.9 ± 2.0 m/s in the control group ($P = 0.001$). Children with PS show significant daytime systemic blood pressure elevation and reduced arterial compliance. After adjusting for multiple regression analysis, PS was independently confirmed as the only key factor affecting reduced arterial compliance unaffected by body weight. This indicates that PS impacts atherosclerosis in early life, and this impact is not influenced by body weight. However, whether it ultimately progresses to adult hypertension and leads to cardiovascular events remains challenging to assess.

5.2. PS and Hypertension

A cross-sectional study conducted in Taiwan Region reveals that compared to children who are not obese and do not have PS, those who are both obese and suffer from moderate to severe OSA have a significantly increased risk of poor blood pressure conditions by three times [25]. This study enrolled 1689 children, which is a sufficiently large size. While PS may not directly elevate the risk of severe adverse blood pressure, the combination of obesity and more significant sleep-disordered breathing does heighten the likelihood of developing such issues. Furthermore, higher blood pressure can exacerbate sleep apnea problems in patients with PS. A retrospective study conducted in Sichuan revealed that higher baseline diastolic blood pressure independently influenced the development of an increased AHI during subsequent follow-ups with PS patients, regardless of the duration of sleep apnea [26]. In a study conducted in Australia utilizing home monitoring technology to assess snoring and sleep conditions, objective snoring time data were collected from 12,287 participants over several nights. The findings revealed that a higher proportion of snoring time (specifically, 12% at the 75th percentile versus 0.04% at the 5th percentile) was associated with approximately a 1.9-fold increase in the risk of uncontrolled hypertension (OR [95%CI]: 1.87 [1.63, 2.15]). Notably, this association was independent of sleep apnea [6]. Even when the effects of sleep apnea are excluded, frequent snoring still increases the risk of poor blood pressure control. This may be more closely related to the chronic obstruction of breathing itself rather than to the impacts of hypoxia or sleep disorders. However, participants were also predominantly male, which limits generalization and raises the need for caution when interpreting sex difference comparisons. Participants were also self-selected based on their decision to purchase and regularly use the under-the-mattress sleep sensor and blood pressure monitor devices from which these data were derived. Consumer choices could reflect concerns about sleep and blood pressure, potentially contributing to a bias toward overestimating snoring prevalence. Additionally, a clinical trial hypothesized that patients with normal blood pressure exhibit a reduced sensitivity to the baroreflex during wakefulness and non-rapid eye movement (NREM) sleep. In contrast, this decrease in sensitivity is deemed absent in other cases. The intervention utilized nasal continuous positive airway pressure (nCPAP). During the application of nCPAP, baroreflex sensitivity was assessed in both the experimental and control groups. Non-apneic snorers exhibited a reduction in baroreflex sensitivity during sleep, but nCPAP treatment effectively mitigated this decline, restoring sensitivity levels to near those observed during wakefulness. In contrast, the control group showed no significant change in baroreflex sensitivity while asleep, and nCPAP treatment did not have a meaningful impact on their measurements [27]. Although adherence to nCPAP was self-reported by the participants, the measurements observed between the groups during the trial were similar. This suggests that PS may change baroreflex sensitivity at night, potentially occurring before noticeable alterations in other cardiovascular variables. Slow-wave sleep (SWS) is a vital component of the sleep cycle, characterized by high-amplitude, low-frequency synchronized slow waves (δ waves). These waves are crucial in body growth, physical recovery, alleviating fatigue, and maintaining endocrine balance. Research indicates a significant interaction between moderate to severe OSA and the duration of SWS with hypertension ($P = 0.002$). A reduction in SWS is linked to a higher incidence of hypertension in OSA patients, a trend not observed in those with primary sleep disorder [28]. This may relate to OSA affecting hypoxia, activating the sympathetic nervous system, and reducing slow-wave sleep (SWS), all of which affect blood pressure regulation. Therefore, a thorough and individualized assessment of PSG is critical. An objective evaluation of the underlying causes is crucial for effective airway management during sleep. This study used a large sample, improving generalizability. Nonetheless, it is essential to consider that office BP may not be representative of the 24-hour BP profile, And the white-coat phenomenon. Participants underwent only a single nocturnal polysomnography, so nocturnal variability and first-night effects cannot be ruled out. Additionally, research on the relationship between PS and uncontrolled hypertension is limited. Given the unequal distribution of medical resources, this area warrants greater attention in future medical practice.

5.3. Primary Snoring and the Prognosis of Hypertension

5.3.1. Carotid Intima-Media Thickness

Snoring, which arises from oscillating pressure waves in the upper airway, can propagate through the surrounding tissues to the wall of the carotid artery. Considering the short distance between the carotid bifurcation and the lateral wall of the pharynx, it is likely that these vibrations influence the carotid artery. In a prospective paired cohort study with a median follow-up period of five years, the findings indicated that childhood primary snoring exposure was significantly correlated with adverse cardiovascular outcomes. Specifically, there was a noted deterioration in endothelial function, an increase in carotid intima-media thickness (IMT), and elevated 24-hour ambulatory blood pressure measurements at the five-year mark. regardless of obstructive sleep apnea severity [29], similar results were found in adult PS [30]. PS presents a significant risk to cardiovascular health during the early stages of growth and development. Further, longitudinal studies must be conducted to investigate the long-term implications of PS. PS may expedite subclinical alterations in adjacent arterial structures, potentially mediated by mechanisms such as mechanical vibration of the mucosal surface or other pathophysiological processes [31]. The duration of PS significantly impacts vascular sclerosis. A generalized propensity score analysis was conducted to examine the carotid IMT among 180 participants who snored and a control group of non-snoring individuals following PSG. The findings revealed an independent association with subclinical alterations in IMT was present solely in female participants who snored for at least 25% of their sleep duration. Specifically, the average carotid IMT demonstrated a progressive increase correlated with snoring duration: non-snoring individuals exhibited an IMT of 0.707 mm, and mild snorers had an IMT of 0.718 mm. In comparison, moderate to severe snorers showed an IMT of 0.774 mm [32]. Morphological differences between men and women, such as fat distribution in neck and soft tissue volumes in the upper airway, could cause gender differences due to snoring. Although IMT itself is not a direct causal factor in the elevation of blood pressure, the progressive thickening of IMT can lead to a gradual narrowing of the carotid artery lumen. This narrowing results in increased resistance to blood flow. Consequently, the heart must augment its output to sustain adequate cerebral perfusion, contributing to increased blood pressure.

5.3.2. Atherosclerosis of the Arteries

Hypertension can harm the vascular endothelium, encourage lipid deposition, and expedite the development of atherosclerosis. This condition, in turn, decreases vascular elasticity and subsequently raises blood pressure further. However, the influence of snoring on atherosclerosis can differ across various body regions. In a study involving 110 participants without obstructive sleep apnea, severe snoring was found to have a significant association with carotid atherosclerosis, demonstrating an (OR = 10.5; 95%CI: 2.1 - 51.8; $P = 0.004$). In contrast, this association was not found concerning femoral atherosclerosis. [33]. In addition, the study group consisted of patients with relatively mild degrees of nocturnal hypoxia. due to the bias towards the selection of snoring subjects over OSA patients. The study was not designed or powered to test for nocturnal hypoxia as a risk factor for atherosclerosis。 Therefore, the lack of association should not be interpreted as excluding hypoxia as a risk factor for atherosclerosis in other study populations. The vibrations and mechanical stresses caused by snoring can be transmitted to the tissues and blood vessels in the neck, potentially resulting in endothelial dysfunction and worsening atherosclerosis, as well as increasing the risk of carotid plaque rupture. Furthermore, the vibrations associated with snoring may elevate local inflammatory responses in the upper airway tissues, further contributing to atherosclerosis in the neck's blood vessels.

5.3.3. Metabolic Syndrome

Metabolic Syndrome (MetS) is a complex pathological condition defined by the presence of abdominal obesity, dyslipidemia, hypertension, hyperglycemia, and insulin resistance, among other interrelated factors that interact synergistically. These elements are interrelated and function synergistically, notably increasing the risk of cardiovascular and cerebrovascular diseases, as well as diabetes. A cross-sectional study [34] conducted in China revealed that patients with obstructive sleep apnea exhibited more pronounced metabolic disorders and a higher prevalence of metabolic syndrome compared to individuals without snoring. PS was significantly associated with an increased risk of MetS in all participants (OR = 2.328, 95%CI: 1.340 - 4.045) and female participants (OR = 2.382, 95%CI: 1.136 - 4.994). About the components of MetS, PS was significantly associated with an increased likelihood of hypertension (OR = 1.730, 95%CI: 1.130 - 2.650); in terms of the Mets component, PS was significantly associated with an increased chance of hypertension (OR = 1.730, 95%CI: 1.130 - 2.650). Moreover, most cross-sectional studies have investigated the correlation between habitual snoring and MetS and its subclinical-related indicators, suggesting that snoring may precede the occurrence of MetS. It has been suggested that sleep-disordered breathing caused by snoring may affect the hyperactivity of the sympathetic nervous system [35]. Thus, by increasing glycogenolysis and gluconeogenesis, it damages glucose homeostasis and induces insulin resistance [36]. Moreover, hypoxia promotes the release of pro-inflammatory cytokines, including tumor necrosis factor-alpha and interleukin-6, leading to impaired glucose tolerance. However, the assessment of snoring in these studies was mainly based on self-reported snoring frequency, and patients with sleep apnea were not excluded. Large-scale prospective studies are still needed to determine the causal relationship between PS and metabolic abnormalities.

5.3.4. Cardiovascular and Cerebrovascular Events

In the exploration of cardiovascular and cerebrovascular events, akin to investigations of metabolic syndrome, most association studies generally assess the link between snoring and cardiovascular or cerebrovascular diseases by considering factors such as the frequency and intensity of snoring, and the duration of snoring episodes. A significant number of these studies concentrate on middle-aged men, likely due to the higher incidence of both snoring and cardiovascular diseases within this demographic. Presently, there exists a limited number of studies that objectively assess the relationship between snoring and the prevalence of cardiovascular and cerebrovascular diseases. Notably, one community cohort study that quantitatively measured snoring indicated that both in an unadjusted Cox regression model and in a model that accounted for OSA and other confounding risk factors, snoring—whether considered as a categorical or continuous variable—did not demonstrate a statistically significant association with mortality, cardiovascular disease, or stroke [37]. Despite the potential implications of the findings, this cohort is characterized by a limited sample size, necessitating further investigation through a larger cohort study to achieve validation.

6. Intervention

Currently, consistent intervention measures for PS and hypertension are lacking. At the same time, an Australian position statement has proposed comprehensive treatment recommendations for PS [38]. The goal is to promote the effective treatment of primary snoring based on the best available evidence and expert experience, including weight loss, reducing alcohol consumption, avoiding medications that exacerbate snoring, positional therapy, the use of mandibular advancement devices, the application of CPAP devices, and, if necessary, surgical treatment. Individualized assessments should be made according to population characteristics, while hypertension treatment emphasizes improving lifestyle and actively seeking out related factors for poor blood pressure control.

7. Conclusion

Currently, there needs to be more objective methods for measuring snoring, and different research tools are selected according to various research purposes. Whether primary snoring is an early stage of increased upper airway resistance remains controversial. However, the studies mentioned above tend to suggest a significant correlation between PS and the early progression of hypertension, as well as an elevated risk of associated adverse clinical outcomes. Furthermore, there is a dearth of consistent intervention measures specifically targeting PS and hypertension. Given this, early identification and adoption of appropriate management measures are crucial for the health management of hypertensive patients.

Conflicts of Interest

The authors declare that they have no known competing financial interest or personal relationships that could have appeared to influence the work reported in this paper.

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Abbreviations

PS	Primary Snoring
PSG	Polysomnography
PSMR	Portable Sleeping Monitoring Recorder
AHI	Apnea-Hypopnea Index
SS	Simply Snoring
UARS	Upper Airway Resistance Syndrome
AS	Arterio Sclerosis
PWV	Pulse Wave Velocity
NREM	Non-Rapid Eye Movement
SWS	Slow-Wave Sleep
IMT	Intima-Media Thickness
EEG	Electroencephalography