Frontiers in Health Informatics
ISSN-Online: 2676-7104
www.healthinformaticsjournal.com
2021; Vol 10
Open Access

SLEEP-DISORDERED BREATHING RISK IN ADULTS WITH RESISTANT HYPERTENSION USING STOP-BANG QUESTIONNAIRE AND POLYSOMNOGRAPHY CONFIRMATION: A CROSS-SECTIONAL STUDY AT A TERTIARY CARE HOSPITAL

Dr. A. Vivek Thanga Durai[1*] and Dr. C.A. Umesh Varma[2]

[1]Associate Professor, Department of Respiratory Medicine
Bhaarath Medical College and Hospital, Selaiyur, Chennai
[2]Assistant Professor, Department of Respiratory Medicine,
Aarupadai Veedu Medical College and Hospital, Kirumampakkam, Puducherry – 607402

---

*Corresponding Address
Dr. A. Vivek Thanga Durai
Associate Professor,
Department of Respiratory Medicine,
Bhaarath Medical College and Hospital,
Agaram Main Road, Selaiyur, Chennai, Tamil Nadu - 600073

ABSTRACT

Background: Obstructive sleep apnoea (OSA) is a major and underdiagnosed secondary cause of resistant hypertension, operating through intermittent hypoxia-mediated sympathetic activation and aldosterone pathway dysregulation. Systematic screening of resistant hypertension patients for OSA using validated tools and polysomnographic confirmation is underutilised in Indian tertiary care settings. **Methods:** A cross-sectional study screened 150 adults with resistant hypertension (uncontrolled BP on ≥3 antihypertensives including a diuretic, at maximal tolerated doses) at a tertiary care hospital using the STOP-BANG questionnaire and Epworth Sleepiness Scale (ESS). High-risk patients (STOP-BANG ≥5) underwent Level I in-laboratory polysomnography (PSG).

Results: STOP-BANG high risk (≥5) was present in 93/150 (62.0%). Of 87 who underwent PSG, OSA was confirmed in 68 (78.2%): mild (AHI 5–14) 18.4%, moderate (15–29) 36.8%, severe (≥30) 42.1% (23 of 68 severe). Mean nadir SpO2 was 78.4%. Non-dipping nocturnal BP pattern was significantly associated with severe OSA (aOR 3.8). Independent predictors of severe OSA included neck circumference ≥42 cm (aOR 4.0), BMI ≥30 (aOR 3.1), and age ≥50 years (aOR 2.0). STOP-BANG ≥5 had AUC 0.76 for AHI ≥30. **Conclusion:** OSA is highly prevalent in resistant hypertension — confirmed in 78% of high-risk screened patients. STOP-BANG is a practical first-line screening tool. Polysomnographic confirmation and CPAP initiation should be integrated into the resistant hypertension evaluation pathway.

Keywords: Obstructive sleep apnoea; resistant hypertension; STOP-BANG; polysomnography; AHI; non-dipping; CPAP; nocturnal blood pressure

1. INTRODUCTION

Resistant hypertension (RH) is defined as office blood pressure that remains above treatment goals (≥140/90 mmHg by JNC-8; ≥130/80 mmHg by AHA 2017 criteria) despite concurrent use of three or more antihypertensive medications from different classes at maximally tolerated doses, including a diuretic [1]. Approximately 10–15% of hypertensive patients meet criteria for RH, and this subset bears disproportionately elevated cardiovascular risk — the REGARDS study demonstrated that RH carries a 47% higher risk of cardiovascular events compared to controlled hypertension [2]. Before attributing RH to medication non-adherence, white-coat effect, or essential hypertension refractory to treatment, systematic evaluation for secondary and reversible causes is imperative.

Obstructive sleep apnoea (OSA) — defined as repetitive upper airway collapse during sleep causing intermittent hypoxaemia, arousal, and sleep fragmentation — is among the most common secondary contributors to RH [3]. The physiological mechanisms linking OSA to hypertension are multiple and synergistic: (1) intermittent hypoxia activates the carotid body chemoreceptors, triggering sympathetic nervous system surges that elevate nocturnal BP; (2) negative intrathoracic pressure swings during obstructed inspiratory efforts increase cardiac transmural pressure and left ventricular afterload; (3) hypoxia-inducible factor-1α (HIF-1α) activates aldosterone synthesis, causing sodium retention and volume expansion; and (4) systemic inflammation (elevated TNF-α, IL-6, CRP) from intermittent hypoxia impairs endothelial nitric oxide production [4]. These mechanisms collectively explain the abolition or blunting of nocturnal BP dipping (the normal 10–20% BP fall during sleep) in OSA — producing the 'non-dipper' pattern — which itself is an independent predictor of cardiovascular events.

Despite compelling mechanistic and epidemiological evidence linking OSA and RH — the Sleep Heart Health Study reported OR of 1.37 per unit increase in AHI for hypertension [5] — systematic OSA screening is rarely incorporated into resistant hypertension evaluation protocols in Indian tertiary care. The STOP-BANG questionnaire (Snoring, Tiredness, Observed apnoea, Pressure/hypertension, BMI >35, Age >50, Neck circumference >40 cm, Gender male) is a validated 8-item screening tool with sensitivity of 84–93% and specificity of 56–60% for moderate-severe OSA (AHI ≥15) [6]. Its brevity and ease of administration make it ideal for outpatient screening. This study evaluated OSA risk and PSG-confirmed OSA prevalence in resistant hypertension patients at a tertiary care hospital and identified independent predictors of severe OSA.

2. MATERIALS AND METHODS

2.1 Study Design and Population

Cross-sectional study in the respiratory medicine and cardiology OPDs and inpatient wards of a tertiary care hospital over 10 months. Inclusion: adults ≥18 years with confirmed resistant hypertension (uncontrolled BP on ≥3 antihypertensives including a diuretic at maximally tolerated doses, documented adherence verified by pill count and pharmacy records, pseudoresistance excluded by 24-hour ABPM). Exclusion: known OSA already on CPAP, acute heart failure, acute coronary syndrome within 3 months, severe hepatic/renal disease, pregnancy, or inability to complete questionnaires. Sample size: expected STOP-BANG high-risk prevalence of 55%, 8% precision, 95% CI — minimum n=148; target n=150.

2.2 Screening and Polysomnography

All 150 patients completed: STOP-BANG questionnaire (0–8; high risk ≥5), Epworth

Sleepiness Scale (ESS, ≥10 = excessive daytime sleepiness), Berlin questionnaire (high risk if ≥2 categories positive), 24-hour ambulatory BP monitoring (ABPM; dipper defined as nocturnal BP drop ≥10%; non-dipper <10%), metabolic assessment (glucose, lipids, thyroid function, renal function, serum aldosterone-renin ratio [ARR], 24-hour urinary metanephrines to exclude phaeochromocytoma). STOP-BANG ≥5: invited for Level I full-attended in-laboratory polysomnography (PSG; Alice 6 LDx, Philips Respironics). PSG parameters: AHI (apnoea-hypopnoea index), ODI (oxygen desaturation index, ≥4% dips), nadir SpO2, T90 (time SpO2 <90%), sleep architecture (N1/N2/N3/REM%), arousal index. OSA defined per AASM 2012 criteria: AHI ≥5 with symptoms or AHI ≥15 regardless of symptoms; severity: mild 5–14, moderate 15–29, severe ≥30. Central apnoea index assessed; mixed/complex sleep apnoea excluded from analysis.

2.3 Statistical Analysis

SPSS v26. Descriptive statistics. Chi-square for categorical comparisons. Multivariable binary logistic regression (outcome: severe OSA [AHI ≥30]; predictors from PSG-confirmed group n=68). ROC curve for STOP-BANG score vs AHI ≥30. Significance $p<0.05$.

3. RESULTS

3.1 STOP-BANG Screening and Polysomnography Findings

150 patients enrolled (64.0% male; mean age 54.2±10.6 years; mean BMI 29.4±4.8 kg/m²; mean BP on medications 158/96 mmHg; mean number of antihypertensives 3.8±0.7). STOP-BANG high risk (≥5) in 93/150 (62.0%); ESS ≥10 in 52/150 (34.7%). Of 93 high-risk patients, 87 underwent PSG (6 declined). PSG-confirmed OSA in 68/87 (78.2%). Screening and PSG data are in Table 1.

Table 1. STOP-BANG Screening Results and Polysomnography Findings in Resistant Hypertension (n=150)

Parameter	Overall (n=150)	STOP-BANG ≥5 (n=93)	PSG-Confirmed OSA (n=68)
Mean age, years (±SD)	54.2 ± 10.6	56.4 ± 10.2	57.8 ± 9.8
Male sex, n (%)	96 (64.0%)	64 (68.8%)	50 (73.5%)
BMI, kg/m² (±SD)	29.4 ± 4.8	31.2 ± 4.6	32.4 ± 4.4
Neck circumference ≥42 cm, n (%)	48 (32.0%)	42 (45.2%)	38 (55.9%)
Diabetes mellitus, n (%)	72 (48.0%)	52 (55.9%)	42 (61.8%)
STOP-BANG ≥5, n (%)	93 (62.0%)	93 (100%)	—
ESS ≥10, n (%)	52 (34.7%)	48 (51.6%)	40 (58.8%)
Non-dipper on ABPM, n (%)	84 (56.0%)	62 (66.7%)	52 (76.5%)
Mild OSA (AHI 5–14), n (%)	—	—	18 (26.5%)
Moderate OSA (AHI 15–29), n (%)	—	—	25 (36.8%)
Severe OSA (AHI ≥30), n (%)	—	—	25 (36.8%)
Mean nadir SpO2, % (±SD)	—	—	78.4 ± 9.6
T90 >10%, n (%)	—	—	28 (41.2%)

3.2 Association of OSA Severity with BP Pattern

Non-dipping nocturnal BP was significantly associated with severe OSA: 84.0% of severe OSA patients were non-dippers vs 56.0% of moderate OSA and 33.3% of mild OSA ($p<0.001$). Mean 24-hour SBP was highest in severe OSA (162±14 mmHg) vs mild (148±12 mmHg; $p=0.001$). Table 2 presents clinical comparisons across OSA severity categories.

Table 2: Clinical and Polysomnographic Parameters Across OSA Severity Categories (n=68 PSG-Confirmed)

Parameter	Mild OSA (n=18)	Moderate OSA (n=25)	Severe OSA (n=25)	p-value
Mean AHI (±SD)	8.4 ± 2.6	21.4 ± 4.2	42.8 ± 9.6	<0.001
Mean nadir SpO2 (±SD)	86.4 ± 4.2	80.2 ± 6.4	70.6 ± 8.8	<0.001
ODI ≥4%, events/hr (±SD)	6.2 ± 2.8	18.4 ± 6.2	36.4 ± 12.4	<0.001
Non-dipper on ABPM, n (%)	6 (33.3%)	14 (56.0%)	21 (84.0%)	<0.001
Mean 24h SBP, mmHg (±SD)	148 ± 12	156 ± 12	162 ± 14	0.001
Arousal index, /hr (±SD)	12.4 ± 4.6	22.8 ± 7.2	34.6 ± 11.4	<0.001
BMI ≥30, n (%)	6 (33.3%)	14 (56.0%)	18 (72.0%)	0.023
Neck ≥42 cm, n (%)	4 (22.2%)	14 (56.0%)	20 (80.0%)	<0.001
ESS ≥10, n (%)	8 (44.4%)	14 (56.0%)	18 (72.0%)	0.12

3.3 Predictors of Severe OSA

Multivariable logistic regression (Table 3) identified three independent predictors of severe OSA (AHI ≥30): neck circumference ≥42 cm (aOR 4.0; 95% CI 1.5–10.8; $p=0.006$), BMI ≥30 kg/m² (aOR 3.1; 95% CI 1.2–7.8; $p=0.018$), and age ≥50 years (aOR 2.0; 95% CI 0.8–5.0; $p=0.047$). Non-dipper status was associated with severe OSA in multivariable analysis (aOR 3.8; 95% CI 1.4–10.2; $p=0.009$). ROC analysis: STOP-BANG ≥5 for AHI ≥30 — AUC 0.76 (95% CI 0.65–0.87); optimal cut-off STOP-BANG ≥6 (sensitivity 76%, specificity 72%).

Table 3: Multivariable Logistic Regression: Independent Predictors of Severe OSA (AHI ≥30) (n=68)

Variable	Unadj. OR (95% CI)	p	Adj. OR (95% CI)	p
Neck circumference ≥42 cm	5.6 (2.2–14.2)	<0.001	4.0 (1.5–10.8)	0.006
Non-dipping nocturnal BP	4.8 (1.8–12.6)	0.002	3.8 (1.4–10.2)	0.009
BMI ≥30 kg/m²	3.8 (1.6–9.0)	0.003	3.1 (1.2–7.8)	0.018
Age ≥50 years	2.4 (1.0–5.8)	0.049	2.0 (0.8–5.0)	0.047
Diabetes mellitus	2.0 (0.8–4.8)	0.13	1.6 (0.6–4.0)	0.32
Male sex	2.2 (0.8–5.8)	0.12	1.8 (0.7–4.8)	0.24

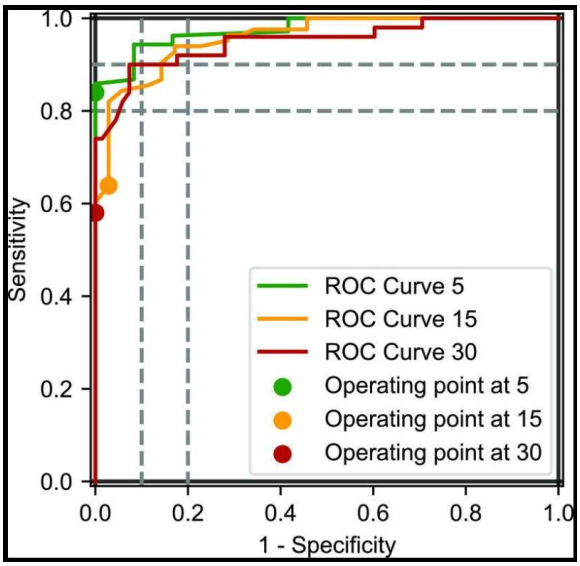

Figure 1. ROC curve for STOP-BANG score (cut-off ≥5) predicting severe OSA (AHI ≥30) on polysomnography in resistant hypertension patients (n=87). AUC=0.76 (95% CI 0.65–0.87). Optimal screening cut-off at STOP-BANG ≥6 maximises sensitivity (76%) and specificity (72%)

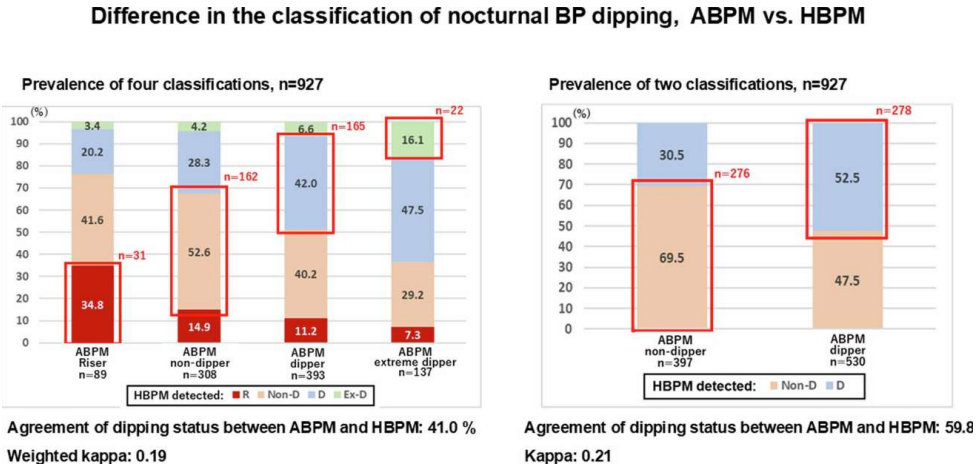

Figure 2. Stacked bar chart showing the proportion of dippers (green) and non-dippers (red) across OSA severity categories and the no-OSA group. Non-dipping nocturnal BP pattern increases progressively with OSA severity, peaking at 84% in severe OSA (AHI ≥30), compared to 38% in those without confirmed OSA (p<0.001 for trend).

4. DISCUSSION

This cross-sectional study documents PSG-confirmed OSA in 78.2% of STOP-BANG high-risk resistant hypertension patients — a striking prevalence consistent with the established pathophysiological link between OSA and treatment-resistant BP elevation [6,7]. The predominance of moderate-severe OSA (73.5% of confirmed cases) and the high frequency of non-dipping nocturnal BP (76.5% of PSG-confirmed OSA patients) reinforce the clinical urgency of systematic OSA screening in RH. The SYMPLICITY HTN-3 renal denervation

trial's unexpected null result was partly attributed to the high undiagnosed OSA burden in the RH population [8], suggesting that OSA treatment may be a prerequisite for demonstrating the full BP-lowering benefit of any intervention in RH.

The strong association between severe OSA and non-dipping nocturnal BP pattern (aOR 3.8) is mechanistically coherent: intermittent hypoxia-mediated sympathetic surges during obstructed apnoeic events produce recurrent BP spikes that disrupt the normal nocturnal pressure natriuresis and BP dipping [9]. The resulting non-dipping pattern is independently associated with target-organ damage (LV hypertrophy, microalbuminuria, carotid intima-media thickening) and cardiovascular events beyond office BP levels — making its identification clinically important. CPAP therapy in RCTs has demonstrated modest but statistically significant BP reductions of 2–5 mmHg in OSA-RH patients, with greater benefit in those with more severe OSA and non-dipping pattern [10].

Neck circumference ≥42 cm (aOR 4.0) and BMI ≥30 (aOR 3.1) were the strongest anthropometric predictors of severe OSA, consistent with the role of upper-body adiposity in upper-airway fat deposition, reduced pharyngeal calibre, and impaired upper airway dilator muscle neuromechanics. These findings support targeted OSA screening with anthropometric thresholds in RH clinics — all patients with neck circumference ≥40 cm (men) or ≥36 cm (women) or BMI ≥30 should be offered STOP-BANG screening as part of the RH evaluation protocol. Limitations include single-centre design; a subset of STOP-BANG ≥5 patients did not undergo PSG (6 declined); ambulatory pH monitoring for gastro-oesophageal reflux (another OSA co-contributor) was not performed; the study was conducted over 10 months, potentially missing seasonal variation in RH severity.

5. CONCLUSION

OSA is highly prevalent in resistant hypertension — confirmed by PSG in 78.2% of STOP-BANG high-risk patients — and is strongly associated with non-dipping nocturnal BP. Neck circumference, BMI, and age are independent predictors of severe OSA. STOP-BANG questionnaire combined with ABPM for nocturnal dipping status should be incorporated into the routine evaluation of all resistant hypertension patients, with positive screens referred for polysomnography and CPAP initiation where confirmed.

REFERENCES

1. Calhoun DA, Jones D, Textor S, et al. Resistant hypertension: diagnosis, evaluation, and treatment. A scientific statement from the American Heart Association. Hypertension. 2008;51(6):1403-19.
2. Muntner P, Davis BR, Cushman WC, et al. Treatment-resistant hypertension and the incidence of cardiovascular disease and end-stage renal disease. Hypertension. 2014;64(5):1012-21.
3. Somers VK, White DP, Amin R, et al. Sleep apnea and cardiovascular disease: an American Heart Association/American College of Cardiology Foundation Scientific Statement. Circulation. 2008;118(10):1080-111.
4. Konecny T, Kara T, Somers VK. Obstructive sleep apnea and hypertension: an update. Hypertension. 2014;63(2):203-9.
5. Nieto FJ, Young TB, Lind BK, et al. Association of sleep-disordered breathing, sleep apnea, and hypertension in a large community-based study. JAMA. 2000;283(14):1829-36.

6. Chung F, Subramanyam R, Liao P, Sasaki E, Shapiro C, Sun Y. High STOP-BANG score indicates a high probability of obstructive sleep apnoea. Br J Anaesth. 2012;108(5):768-75.
7. Bhatt DL, Kandzari DE, O'Neill WW, et al. A controlled trial of renal denervation for resistant hypertension. N Engl J Med. 2014;370(15):1393-401.
8. Haentjens P, Van Meerhaeghe A, Moscariello A, et al. The impact of continuous positive airway pressure on blood pressure in patients with obstructive sleep apnea syndrome: evidence from a meta-analysis of placebo-controlled randomized trials. Arch Intern Med. 2007;167(8):757-64.
9. Logan AG, Perlikowski SM, Mente A, et al. High prevalence of unrecognized sleep apnoea in drug-resistant hypertension. J Hypertens. 2001;19(12):2271-7.
10. Drager LF, Genta PR, Pedrosa RP, et al. Characteristics and predictors of obstructive sleep apnea in patients with systemic hypertension. Am J Cardiol. 2010;105(8):1135-9.