



# Obstructive sleep apnea presenting as pseudopheochromocytoma

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## INTRODUCTION

•The obstructive sleep apnea syndrome (OSAS) has a well documented association with increase cardiovascular morbidity and mortality. The patients with OSAS have a high prevalence of HTA and the OSAS may present similar to a pseudopheochromocytoma (PPH). Indeed, This syndrome can elevate catecholamine secretion and increased sympathetic activity which may mimic the biochemical profile of pheochromocytoma. Treatment of OSAS may normalize the effects of this sympathetic overdrive and resolve excessive catecholamine secretion. We report a case with PPH caused by OSAS; a common medical condition which is less recognized as a cause of raised catecholamines.

## CASE REPORT

A 52-year-old female with a history of poorly controlled resistant HTA was admitted to our hospital with severe HTA. She had a history of fatigue and intermittent episodes of palpitations. Laboratory evaluation was significant for elevated 24-h urinary catecholamine levels (CU=3,5x).

This case was presenting with a clinical and biochemical picture indistinguishable from that of pheochromocytoma. However, neither computed tomography nor MIBG scintigraphy detected any catecholamine-producing tumor in or outside the adrenal glands. Our patient was screened with full polysomnography because of heavy snoring, daytime somnolence and obesity. It revealed severe (OSAS). After 3 months of continuous positive airway pressure therapy, the patient experienced resolution of his presenting symptoms, improved blood pressure control and normalization of his CU.

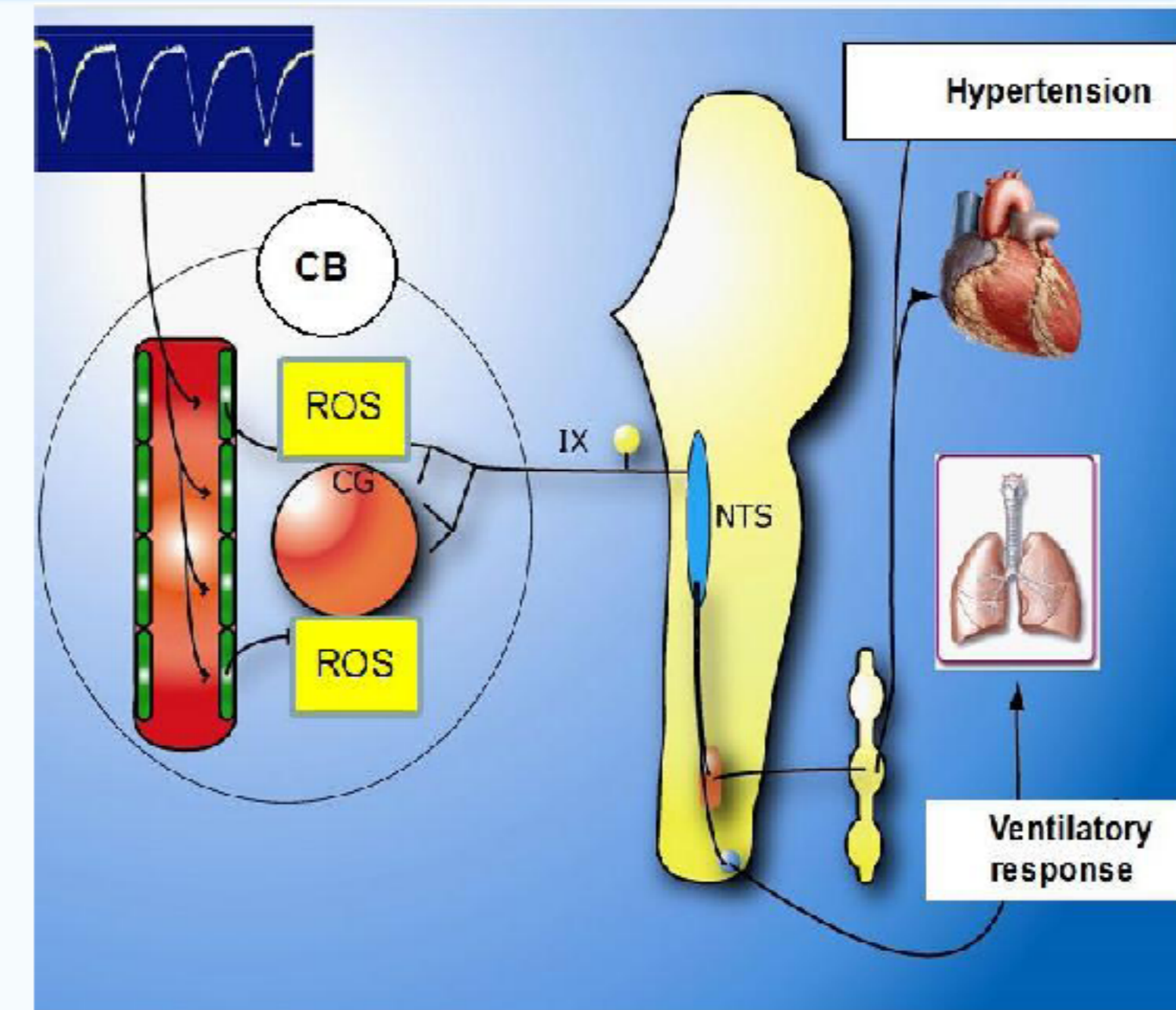


Fig 1: Diagram of the contribution of the carotid body (CB) to the HTA induced by chronic intermittent hypoxia. ROS, reactive oxygen species. NTS, nucleus of the tractus solitarius. CG, chemoreceptor (glomus) cells. [1]

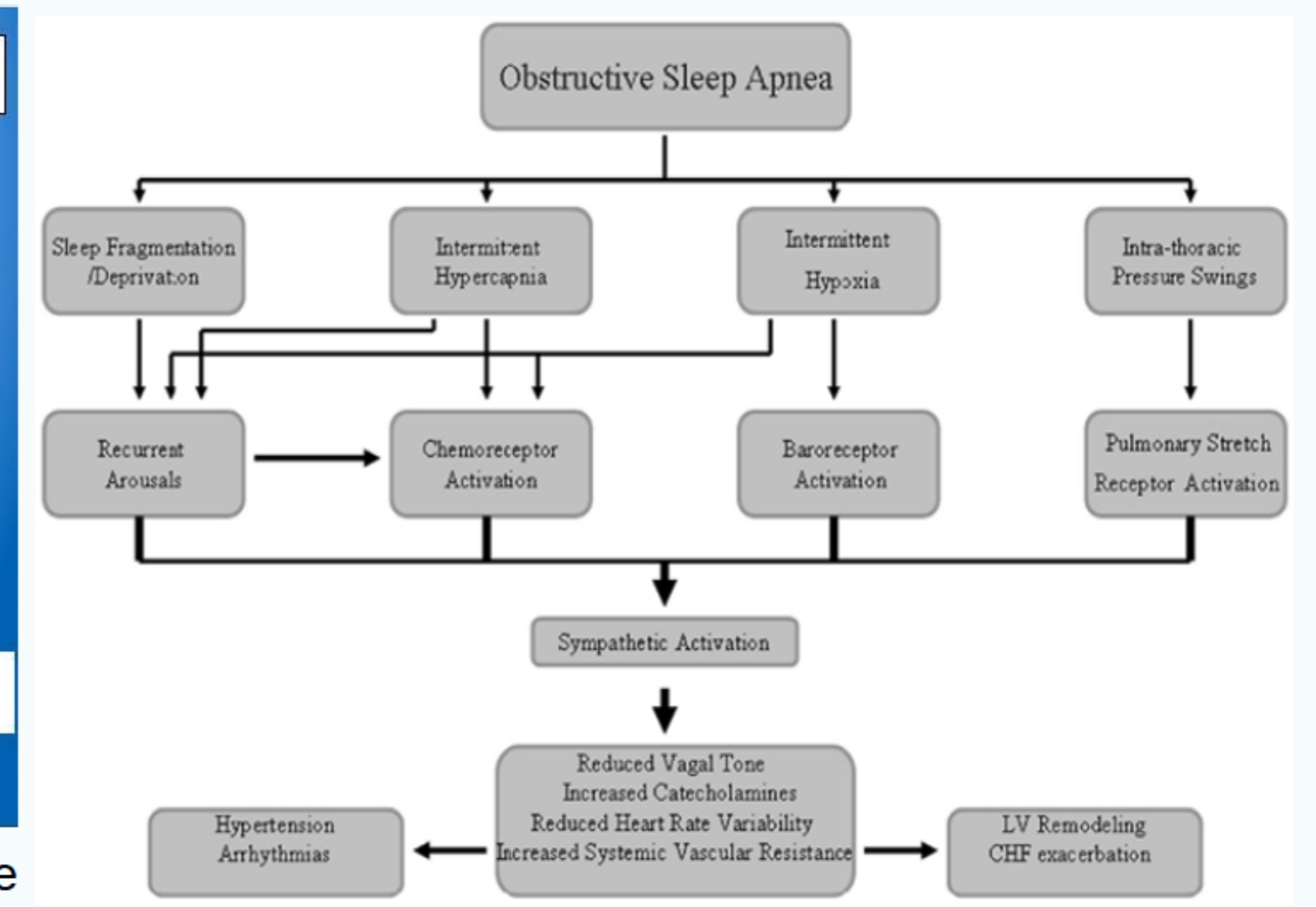


Fig 2: Schematic diagram underlying the sympathetic nerve system activation associated with OSAS that may lead to increased cardiovascular disease risk. [2]

This case highlights sleep disordered breathing as a potentially reversible cause of PPH .

## DISCUSSION

•OSAS is characterized by upper airway occlusion and recurrent cessation of respiratory flow during sleep. Hypoxia ensues, followed by repeated arousal episodes in an attempt to restore airway patency. Patients complain of disturbed sleep and daytime somnolence, and relatives may give a long history of loud snoring. Diagnosis is made by overnight monitoring of arterial oxygenation, chest wall movement, and airflow, proceeding to full polysomnography in equivocal cases.

•Since the 1980s it has been thought that sympathetic activity is up-regulated in patients with sleep-disordered breathing. Twenty-four-hour urinary catecholamine levels were initially found to be increased in patients with untreated sleep apnea compared with those with narcolepsy. Evidence is accumulating to suggest a role for sympathetic overactivity in the pathophysiology of these observations. Indeed a small cohort of male patients with severe sleep apnea and elevated urinary catecholamine levels was found to lose the normal diurnal variation in sympathetic excretion, suggesting increased nocturnal sympathetic activity. OSAS, however, is not the only cause of PPH as they could be showed in other causes, notably physiological stress, antipsychotic drugs, anti-parkinson drugs, tricyclic antidepressants and cocaine use. Panic attack, alcohol withdrawal and carcinoid syndromes are other situations that can explain PPH. None of these conditions were found in our patient who was previously healthy without any medication and denied any alcohol or illicit drug abuse. Other causes of secondary hypertension such as primary hyperaldosteronism, hyperthyroidism, hypercorticism, and endocrine neoplasms were excluded by hormonal explorations.

The exact mechanism of this association is unknown, hypoxia-induced endothelial dysfunction and oxidative stresses are thought to contribute. Further, repetitive arousals in response to anoxic/hypoxic episodes can lead to increased urinary catecholamine and meta-nephrine levels [Fig 1-2].

• Nasal CPAP therapy can reduce this increased sympathetic tone by preventing upper airway closure and mitigating repetitive arousals, as evidenced by normalization of sympathetic markers following treatment. Nasal CPAP therapy is recommended not only to improve HTA and catecholamine hypersecretion but also to distinguish the condition from pheochromocytoma.

## CONCLUSION

• Finally this case represents a patient with untreated OSAS and difficult to control HTA, with clinical and biochemical evidence of increased sympathetic activity mimicking a pheochromocytoma. When imaging fails to reveal the presence of a catecholamine secreting tumor, a diagnosis of OSAS induced PPH should be considered. Recognizing this association is important, as primary treatment for OSAS may lead to a resolution of symptoms and normalization of urinary catecholamine and metanephrine levels.

## References :

1- **Medicine: Sleep and its Disorders Affect Society**, book edited by Chris Idzikowski, ISBN 978-953-51-1725-4, 2014; Contribution of Autonomic Nervous System to the Hypertension Induced by Obstructive Sleep Apnea. By Rodrigo Iturriaga and Juan Idiaquez  
2- Horner, R. L., Brooks, D., Kozar, L. F., Tse, S., and Phillipson, E. A. (1995). Immediate effects of arousal from sleep on cardiac autonomic outflow in the absence of breathing in dogs. *J. Appl. Physiol.* 79, 151-162