OSA and Hypertension:
What You Should Know

1. **Intermittent hypoxia in OSA triggers hemodynamic changes that raise blood pressure through increased fluid retention and vasoconstriction.[1,2]**

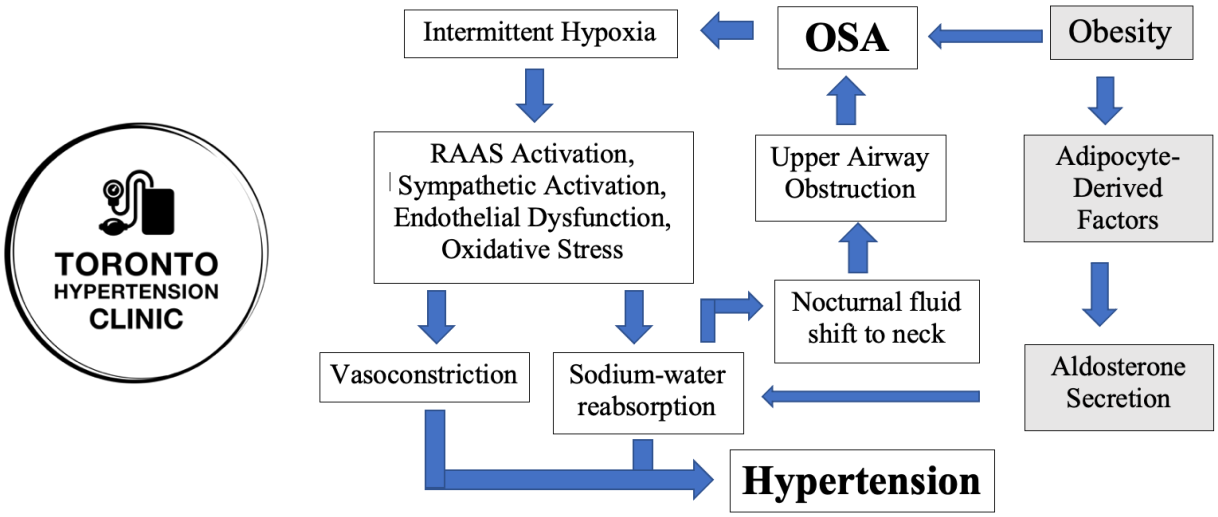

Fig. 1 Hemodynamic effects of OSA that result in hypertension.

2. **Patients with OSA are more likely to be hypertensive, even after controlling for obesity and age. In addition, 71-85% of patients with treatment-resistant hypertension have OSA[3,4].**

   There is a dose-related relationship between OSA severity and blood pressure increase. In one study, those with an Apnea-Hypopnea Index (AHI) of 5-15 had twice the risk of hypertension compared to those with an AHI of 0.[5] On 24hr ABPM, OSA patients are typically "non-dippers" (nocturnal BP decline <10%) or "reverse-dippers" (nocturnal BP higher than daytime BP), often with a diastolic predominance.

3. **Aldosterone excess is common in hypertensive OSA patients, and most patients with primary aldosteronism (PA) have OSA.**

   In addition to hypoxia-driven RAAS activation, it has recently been proposed that aldosterone excess in OSA may result from adipocyte-derived factors that directly induce aldosterone secretion (Fig 1).[6] Patients with PA have a high rate of OSA: in a recent cross-sectional study, 67.6% of patients with PA had OSA.[7]

4. **Treatment of OSA with CPAP has a modest but significant effect on BP reduction.**

   In a 2014 meta-analysis, composed of 30 RCT's and >1900 patients, CPAP therapy reduced systolic BP by a mean of 2.6mmHg, considered significant for reducing cardiovascular events.[8] Ideal therapy of OSA in hypertensive patients is multi-modal, with CPAP therapy, weight loss, and antihypertensive medication. Consider using an aldosterone antagonist earlier on in hypertension treatment to counteract excess circulating aldosterone.

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