# Obstructive Sleep Apnea and systemic Hypertension

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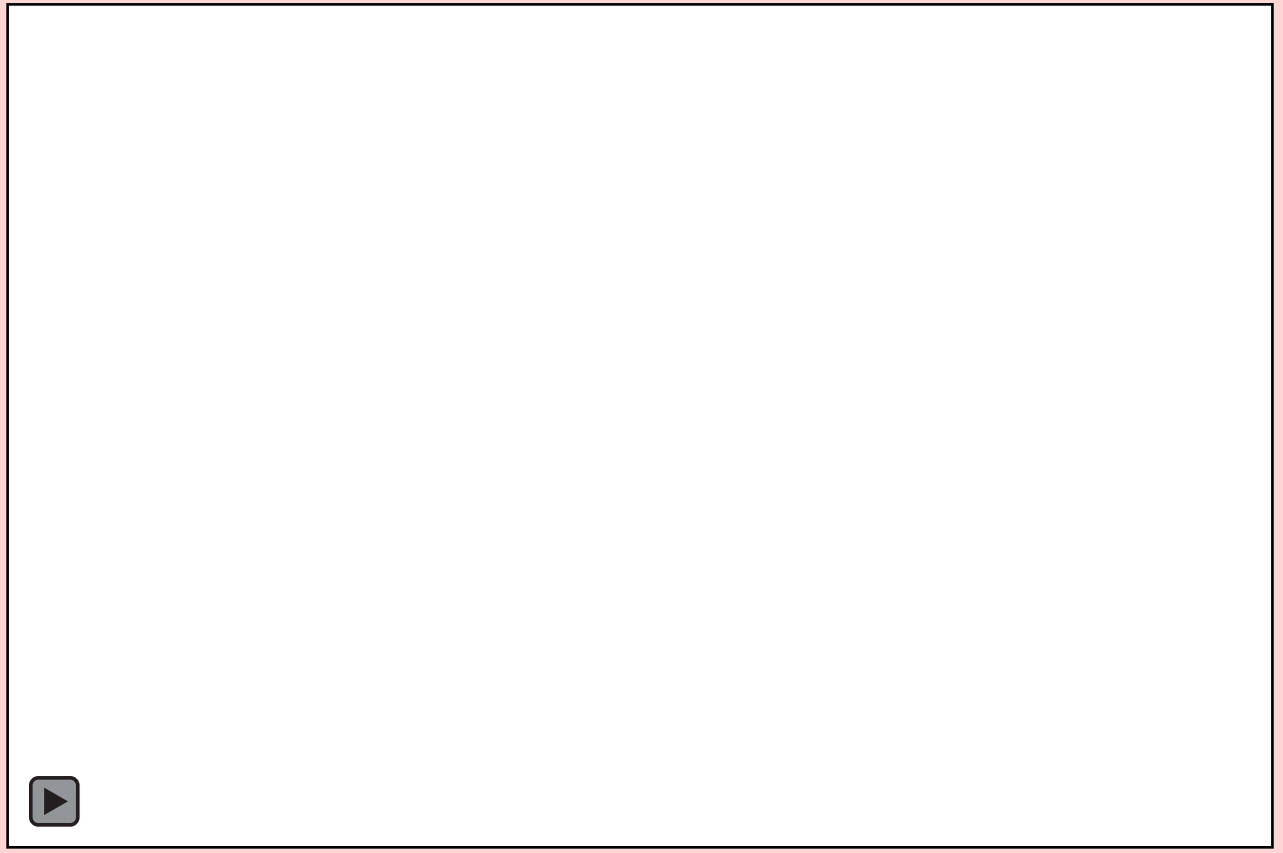
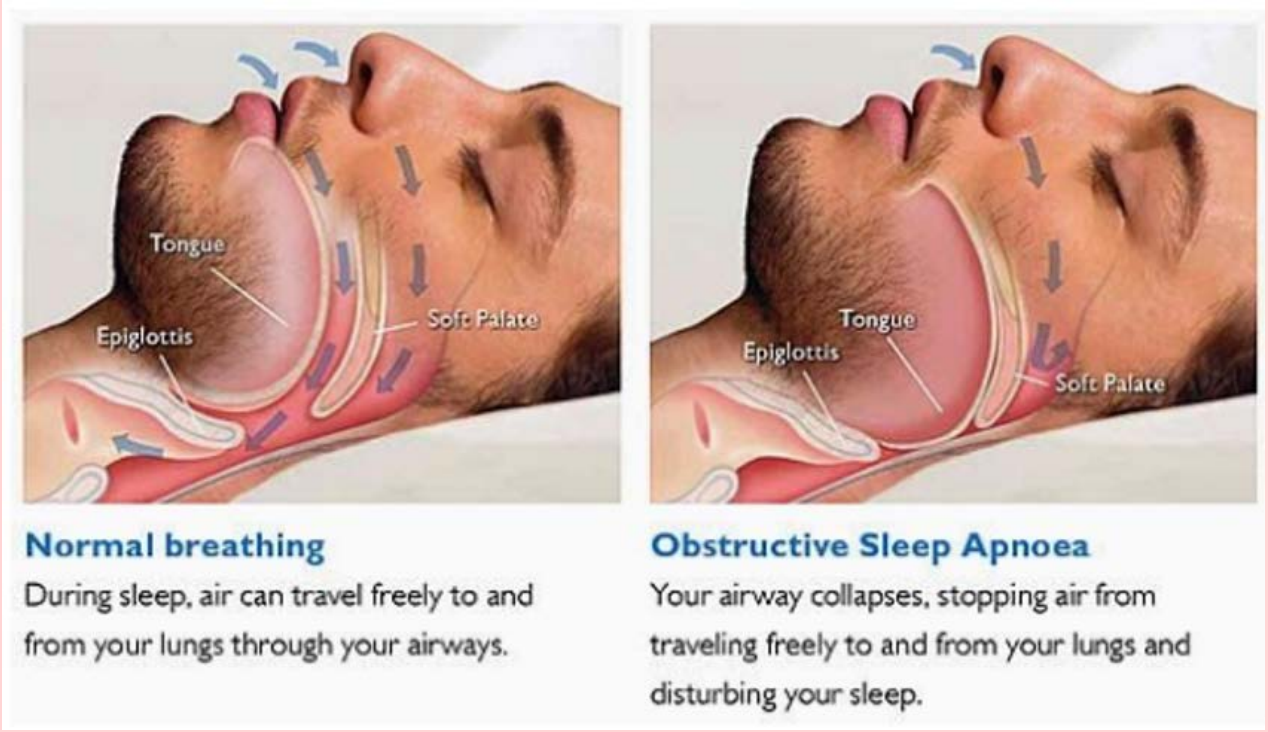


- OSA is a condition described as recurrent episodes of upper airway inspiratory collapse during sleep, ⟹ hypopnea (breathing reduction) or apnea (breathing cessation) episodes.

- The patient waking from sleep terminates the apneic and hypopneic episodes.

- The patient then hyperventilates because of the hypoxemia for a brief period of time.

- This type of apnea occurs when throat muscles intermittently relax and block the airway during sleep.



# Leading risk factors for OSA are:

**Obesity:** BMI>35 kg/m2

**Male sex:** The role of estrogen and progesterone in increasing ventilatory drive

**Old age:** Reduces carotid chemoreceptor sensitivity, decreases lung function efficiency

## Other chronic medical conditions that increase OSA risk include:

- ✓ end-stage renal disease (ESRD),
- ✓ heart failure (CHF),
- ✓ chronic lung disease, and
- ✓ craniofacial abnormalities.

# This results in:

- ✓ Cyclical oxygen desaturation,
- ✓ Reflexive sympathetic hyperactivity,
- ✓ Frequent microarousals,
- ✓ Poor sleep quality,
- ✓ Daytime drowsiness,
- ✓ Decrease quality of life,


- ✓ Increase the risk of daytime and workplace accidents,
- ✓ Chronic daytime fatigue and cravings for energy-dense foods, thus increasing the risk for obesity, dyslipidemia, diabetes, and metabolic syndrome,
- ✓ Higher risk for depression, cognitive delay, and mood lability,
- ✓ Cardiovascular derangements, including coronary artery disease, stroke, arrhythmias, peripheral artery disease, heart failure, and HTN.

**OSA should be suspected in patients with daytime somnolence, poor sleep habits, partner complaints of snoring, obesity, poor quality of life, or failure to achieve *BP* goals despite antihypertensive medication compliance.**

# Diagnosis

- **The diagnosis and severity of OSA are based on the apnea–hypopnea index (*AHI*), which reports the number of apneic and/or hypopneic events during one hour of sleep.**

***** Apneic events obstruct >90% of intrathoracic airflow, whereas hypopneic events obstruct > 30–90% of intrathoracic airflow.**

- **Both types of events last at least 10s and result in oxygen desaturation of 3% or greater.**

| Classification of Obstructive Sleep Apnea | |
|---|---|
| Mild | AHI ≥ 5-15 events per hour |
| Moderate | AHI ≥ 15-30 events per hour |
| Severe | AHI ≥ 30 or more events per hour |

**Screening Surveys:**

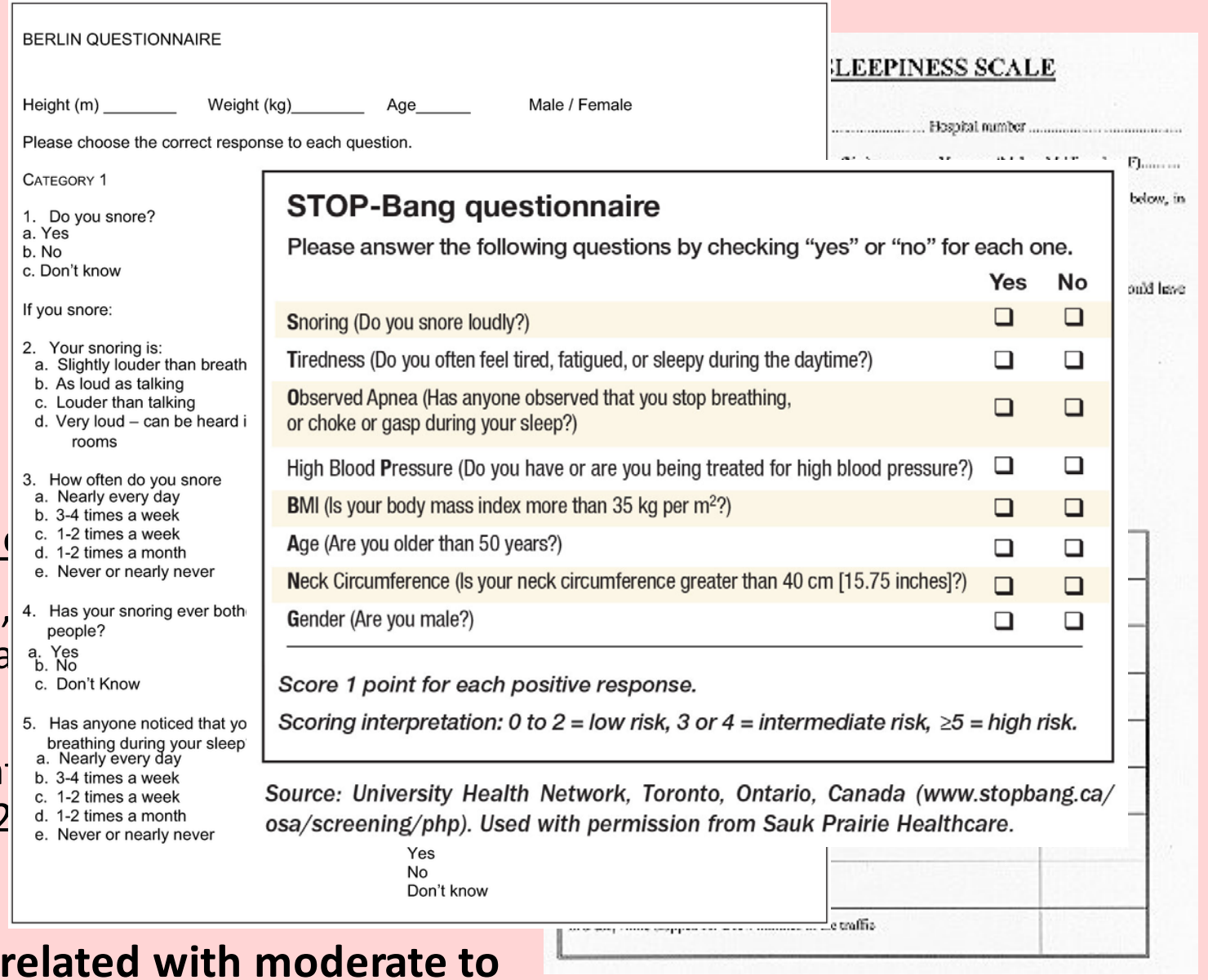
- **Epworth Sleep Scale,**
- **Berlin questionnaire,**
- **STOP-BANG questionnaire.**

❖ Popular among them is the STOP-BANG qu

-Eight questions to gather subjective (snoring, and objective (*BP*, BMI>35 kg/m2, age>50 yea cm, male gender) data.

-The survey's diagnostic sensitivities in patien >15 events/h, and >30 events/h are 83.6%, 92

***** STOP-BANG score ≥5–8, is highly correlated with moderate to severe OSA and resistant HTN.**



BERLIN QUESTIONNAIRE

Height (m) _____ Weight (kg)_____ Age_____ Male / Female

Please choose the correct response to each question.

CATEGORY 1

1. Do you snore?
a. Yes
b. No
c. Don't know

If you snore:

2. Your snoring is:
a. Slightly louder than breath
b. As loud as talking
c. Louder than talking
d. Very loud – can be heard i rooms

3. How often do you snore
a. Nearly every day
b. 3-4 times a week
c. 1-2 times a week
d. 1-2 times a month
e. Never or nearly never

4. Has your snoring ever both people?
a. Yes
b. No
c. Don't Know

5. Has anyone noticed that yo breathing during your sleep
a. Nearly every day
b. 3-4 times a week
c. 1-2 times a week
d. 1-2 times a month
e. Never or nearly never

Yes
No
Don't know

**STOP-Bang questionnaire**

Please answer the following questions by checking "yes" or "no" for each one.

| | Yes | No |
|---|---|---|
| **S**noring (Do you snore loudly?) | ❑ | ❑ |
| **T**iredness (Do you often feel tired, fatigued, or sleepy during the daytime?) | ❑ | ❑ |
| **O**bserved Apnea (Has anyone observed that you stop breathing, or choke or gasp during your sleep?) | ❑ | ❑ |
| High Blood **P**ressure (Do you have or are you being treated for high blood pressure?) | ❑ | ❑ |
| **B**MI (Is your body mass index more than 35 kg per $m^2$?) | ❑ | ❑ |
| **A**ge (Are you older than 50 years?) | ❑ | ❑ |
| **N**eck Circumference (Is your neck circumference greater than 40 cm [15.75 inches]?) | ❑ | ❑ |
| **G**ender (Are you male?) | ❑ | ❑ |

*Score 1 point for each positive response.*

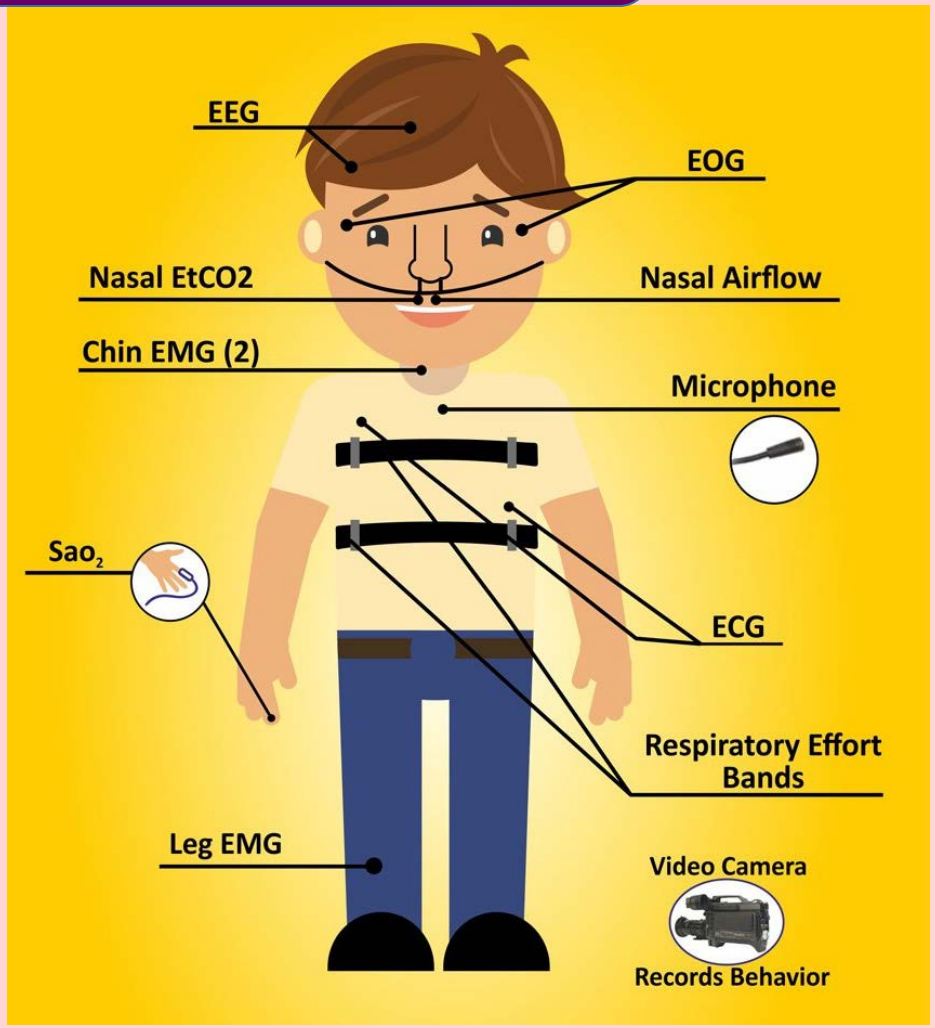
*Scoring interpretation: 0 to 2 = low risk, 3 or 4 = intermediate risk, ≥5 = high risk.*

*Source: University Health Network, Toronto, Ontario, Canada (www.stopbang.ca/osa/screening/php). Used with permission from Sauk Prairie Healthcare.*

LEEPINESS SCALE

Hospital number

❖**Overnight laboratory polysomnography (PSG)** → **Diagnostic Gold Standard**


**!!! A recent European assessment of PSG challenges reported that the percentage of referred patients who arrived for their sleep study declined from 92.5% to only 20% before and after the COVID-19 pandemic, respectively.**

**Home-based sleep tests (HBST)** are increasing in popularity among both prescribers and patients.

HBST are more convenient, **less invasive and nearly half the cost of PSG** and the **diagnostic sensitivities between the two tests are statistically equal.**

# Epidemiology of OSA and HTN

OSA is a highly prevalent sleep disorder that is estimated to affect

**15% to 24% of all adults.**

***But that the number is believed to be incorrect because OSA is still **greatly underdiagnosed** and the prevalence in variable in age and sex related subgroups.

Overlap between patients of obstructive sleep apnea (OSA) and hypertension

↓

the two conditions may have a causal, bidirectional relationship

2003 Joint National Committee (JNC VII) on prevention, detection, evaluation, and treatment of high BP

↓

named OSA as a secondary cause of HTN

2019 American Heart Association (AHA) reported the conclusion of a meta-analysis of 27 cohort studies

↓

Severe OSA (apnea/hypopnea index ≥30) was associated with increased cardiovascular mortality with a hazard ratio of 2.73

OSA → Prevalence of 30% to 50% in HTN patients.

HTN → Prevalence of 30% and 70% in OSA patients.

This is because OSA is under-diagnosed

J Clin Nurs. 2018 May;27(9-10):1901-1912. doi: 10.1111/jocn.14366.

# Factors associated with undiagnosed obstructive sleep apr multisite

Hsiu-Chin Hsu [1]

Affiliations + ex

PMID: 29603807

## Conclusions:

**Nearly 82% of the hypertensive participants were found having undiagnosed obstructive sleep apnoea, and 80% of them were mild or moderate severity.**

**Aims and objectives:** To investigate the distribution and risk factors associated with undiagnosed obstructive sleep apnoea among hypertensive patients.

**Design:** A cross-sectional design.

**Methods:** A total of 215 hypertensive participants were recruited from the cardiovascular outpatients of medical center in northern and middle Taiwan. The Chinese version of Pittsburgh Sleep Quality Index, the Chinese version of the Epworth Sleep Scale and a portable sleep monitoring device were used for data collection. Logistic regression analysis

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ea.

correlated with gender (odds ratio, 0.04; 95% CI, 0.00-0.66), excessive daytime sleepiness (odds ratio, 20.27; 95% CI, 1.58-26.97) and oxygen desaturation index (odds ratio, 4.05; 95% CI, 1.86-8.81).

**Conclusions:** Nearly 82% of the hypertensive participants were found having undiagnosed obstructive sleep apnoea, and 80% of them were mild or moderate severity. Oxygen desaturation index, $SO_2$ and the supine position were found to be major predictors for obstructive sleep apnoea. Remarkably, oxygen desaturation index was the most significant predictor for mild, moderate and severe obstructive sleep apnoea.

**Relevance to clinical practice:** Healthcare providers should enhance their sensitivities to hypertensive patients at a high risk for obstructive sleep apnoea by actively assessing common obstructive sleep apnoea symptoms and providing strategies to alleviate obstructive sleep apnoea symptoms.

Abstract

Daytime sleepiness is a common symptom among hypertensive patients.

The aim of this study was to determine subjective sleepiness assessed by Epworth Sleepiness Scale (ESS) and to asses sleep

normotensives. The study showed that ESS total score is lower in hypertensives than in normotensives with OSA, making the OSA more difficult to suspect. Thus, the low ESS score in hypertensives should not discourage further evaluation.

Journal of the American Society of

A different study **using PSG to observe the effects of HTN on sleep characteristics** in 304 participants who had no prior diagnosis of OSA found that:

**HTN was associated with:**

***Decreased sleep efficiency,**
***Decreased mean and minimum oxygen saturation during apneic episodes,**
***Increased *AHI*, and**
***Increased oxygen desaturation index (ODI), which is defined as the number/hour of apneic events resulting in reductions in oxygen saturation by ≥ 4% from baseline.**

Multicenter Study > J Clin Sleep Med. 2020 Jun 15;16(6):889-898. doi: 10.5664/jcsm.8354.

# Mild obstructive sleep apnea increases hypertension risk,

Izolde Bou
Gianfranco
Paschalis S
European

Affiliations
PMID: 320
Free PMC

**A 2020 study of 4,500 people with OSA identified that merely mild OSA (*AHI* = 11–15 events/h) increased the likelihood of having HTN by 78% when compared to control subjects without OSA (*OR* = 1.779, 95% CI 1.403–2.256).**


**Study objectives:** The association of mild obstructive sleep apnea (OSA) with important clinical outcomes remains unclear. We aimed to investigate the association between mild OSA and systemic arterial hypertension (SAH) in the European Sleep Apnea Database cohort.

**Methods:** In a multicenter sample of 4,732 participants, we analyzed the risk of mild OSA (subclassified into 2 groups: $mild_{AHI\ 5-<11/h}$ (apnea-hypopnea index [AHI], 5 to <11 events/h) and $mild_{AHI\ 11-<15/h}$ (AHI, ≥11 to <15 events/h) compared with nonapneic snorers for prevalent SAH after

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polygraphy (odds ratio, 1.779; 95% CI, 1.403-2.256; P < .001) and polysomnography groups (odds ratio, 1.424; 95% CI, 1.047-1.939; P = .025).

**Conclusions:** Our data suggest a dose-response relationship between mild OSA and SAH risk, starting from 5 events/h in polygraphy recordings and continuing with a further risk increase in the 11- to <150-events/h range. These findings potentially introduce a challenge to traditional thresholds of OSA severity and may help to stratify participants with OSA according to cardiovascular risk.

**Background:** Sleep-disordered breathing is prevalent in the general population and has been linked to chronically elevated blood pressure in cross-sectional epidemiologic studies. We performed a prospective, population-based study of the association between objectively measured sleep-disordered breathing and hypertension (defined as a laboratory- measured ons). tus, and the 4 of these hy for r of nce of ndex at y-mass rettes. ts per hour .42 (95 o 4.9 events l, 1.29 to percent events per

reathing at of known confounding factors. The findings suggest that sleep-disordered breathing is likely to be a risk factor for hypertension and consequent cardiovascular morbidity in the general population.

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PMID: 1080

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The most notable study to characterize this dose–response relationship was published by Peppard et al. in 2000.

In that study, **709 patients with OSA were followed for four years** to **assess the incidence of new onset HTN** among them.

After correction for BMI, neck/weight circumference, age, sex, and alcohol/tobacco use, severity of **OSA positively correlated with incidence of HTN.**

Compared to controls with an *AHI* of 0 events/h, odds ratios for mild OSA (*AHI*=0.1–4.9 events/h), moderate OSA (*AHI* = 5.0–14.9 events/h), and severe OSA (*AHI* ≥ 15 events/h) were 1.42 (95% CI 1.13–1.78), 2.03 (95% CI 1.29–3.17), and 2.89 (95% CI 1.46–5.64), respectively.

Review > J Glob Health. 2018 Jun;8(1):010405. doi: 10.7189/jogh.08.010405.

# Association of obstructive sleep apnea with hypertension: A systematic review and meta-analysis

Haifeng Hou [1 2 3], Weijia Xing [1 3], Wei

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PMID: 29497502 PM

Free PMC article

**The most recent data from a 2018 meta-analysis pooling 26 original studies and over 51,000 participants confirmed a dose–response relationship between HTN and mild OSA (*OR* = 1.184, 95% CI 1.093–1.274, *P* < 0.05), moderate OSA (*OR* = 1.316, 95% CI 1.197–1.433, *P* < 0.05), and severe OSA (*OR* = 1.561, 95% CI 1.287–1.835, *P* < 0.05).**

**Background:** Obstructive sleep apnea (OSA) is a sleep disorder characterized as complete or partial upper airflow cessation during sleep. Although it has been widely accepted that OSA is a risk factor for the development of hypertension, the studies focusing on this topic revealed inconsistent results. We aimed to clarify the association between OSA and hypertension, including essential and medication-resistant hypertension.

**Methods:** The Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) was followed. PubMed and Embase databases

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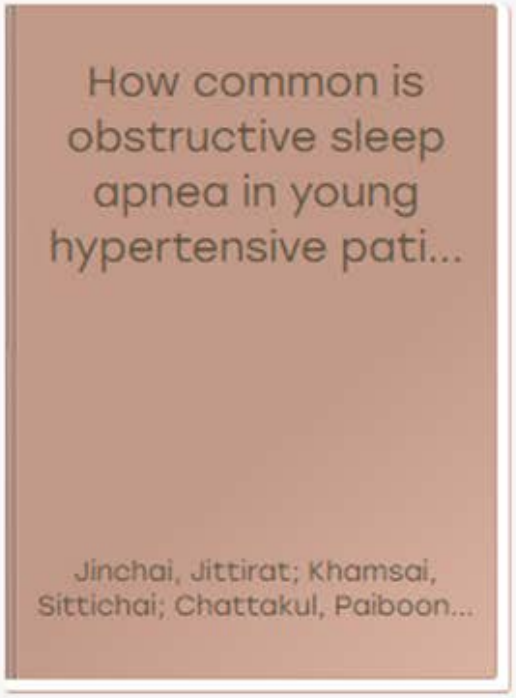
0.05) for mild OSA, 1.316 (95% CI = 1.197-1.433, *P* < 0.05) for moderate OSA and 1.561 (95% CI = 1.287-1.835, *P* < 0.05) for severe OSA.

**Conclusions:** Our findings indicated that OSA is related to an increased risk of resistant hypertension. Mild, moderate and severe OSA are associated essential hypertension, as well a dose-response manner relationship is manifested. The associations are relatively stronger among Caucasians and male OSA patients.

Internal and Emergency Medicine 2020 / 1




How common is obstructive sleep apnea in young hypertensive patients?

Jinchai, Jittirat, Khamsai, Sittichai, Chattakul, Paiboon, Limpawattana, Panita, Chindaprasirt, Jarin, Chotmongkol, Verajit, Silaruks, Songkwan, Senthong, Vichai, Sawanyawisuth, Kittisak

भाषा: English
DOI: 10.1007/s11739-019-02273-3
फ़ाइल: PDF, 530 KB
पत्रिका: Internal and Emergency Medicine
Date: January, 2020

**In a cohort of 593 patients aged 18–35 years who were diagnosed with HTN and screened for secondary causes without diagnostic findings, 88.9% of them had OSA.**

- OSA is the leading cause of **resistant-HTN**, which occurs in 12–15% of all people diagnosed with HTN, and an astounding **70–83% of people with r-HTN also have OSA.**

- **Masked HTN (m-HTN)**

❖ In a 2008 study of 130 newly diagnosed OSA patients, those with OSA were **2.7 times more likely to have m-HTN** when clinic recordings identified *BP* > 125/83 mmHg. Of the 130 patients included in the study,

- **35.4% had essential HTN,**
- **30% had m-HTN, and**
- **3.1% had white coat HTN.**

Collectively, **68.5% of those with OSA had some type of HTN,** which is higher than the ~ 30% prevalence of HTN among the general population.

J Hypertens. 2008 May;26(5):885-92. doi: 10.1097/HJH.0b013e3282f55049.

## Masked hypertension in obstructive sleep apnea syndrome

Jean-Philippe Baguet [1], Patrick Lévy, Gilles Barone-Rochette, Renaud Tamisier, Hélène Pierre, Marie Peeters, Jean-Michel Mallion, Jean-Louis Pépin

Affiliations + expand

PMID: 18398330 DOI: 10.1097/HJH.0b013e3282f55049

## Pathophysiology

- OSA and HTN are **both multifactorial diseases.**
- They **share many of the same risk factors (obesity, male gender, and advancing age).**
- Because of this, and the fact that OSA is the most prevalent secondary cause of HTN, **both also share many pathophysiological mechanisms that link them together.**

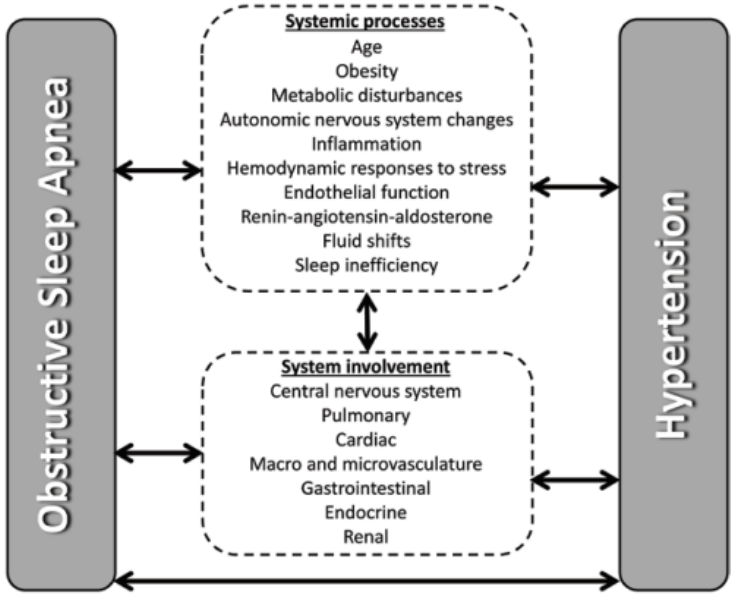
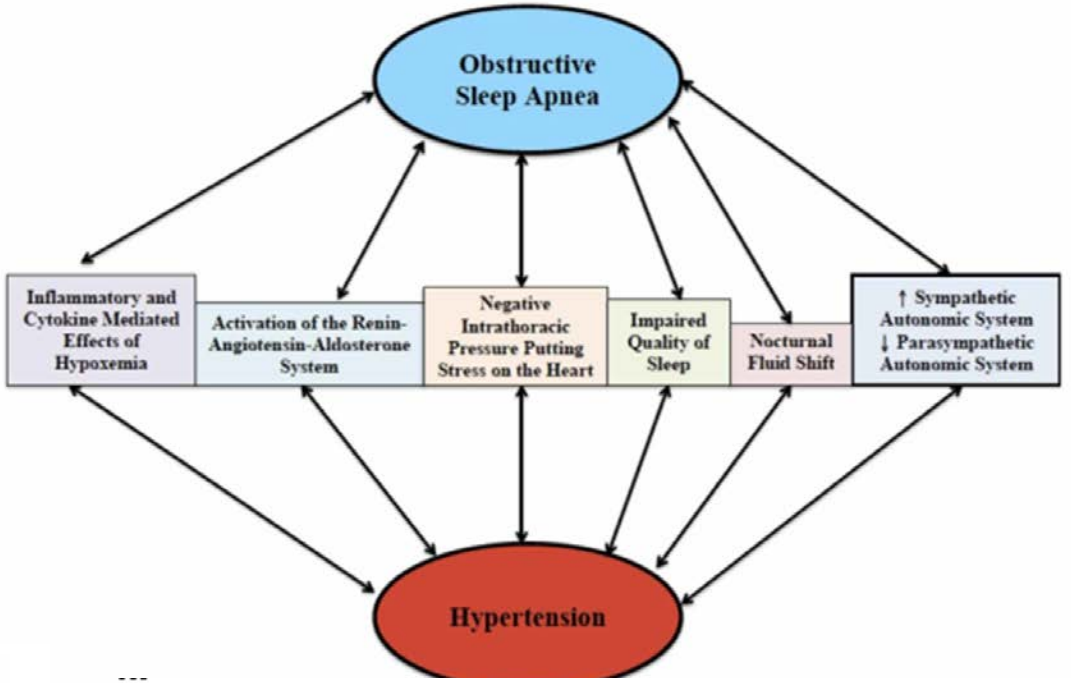



**Figure 3.** Schematic representation of the complex interactions betw



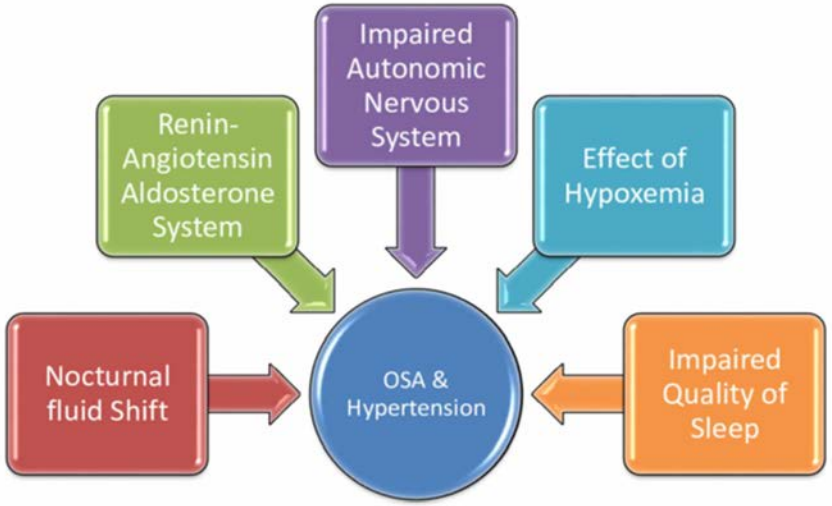




**FIGURE 1: Factors relating hypertension and obstructive sleep apnea (OSA)**

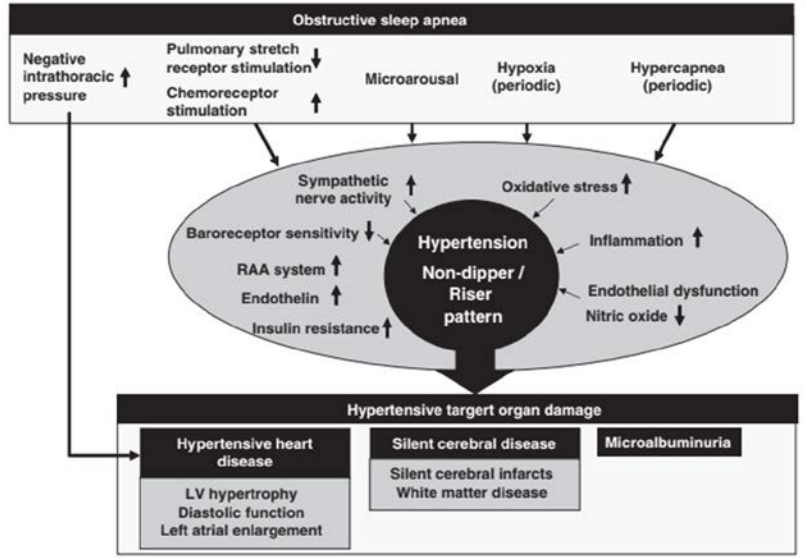



**Figure 1** Mechanism of hypertension and target organ damage in obstructive sleep apnea syndrome. RAA, renin–angiotensin–aldosterone; LV, left ventricular.

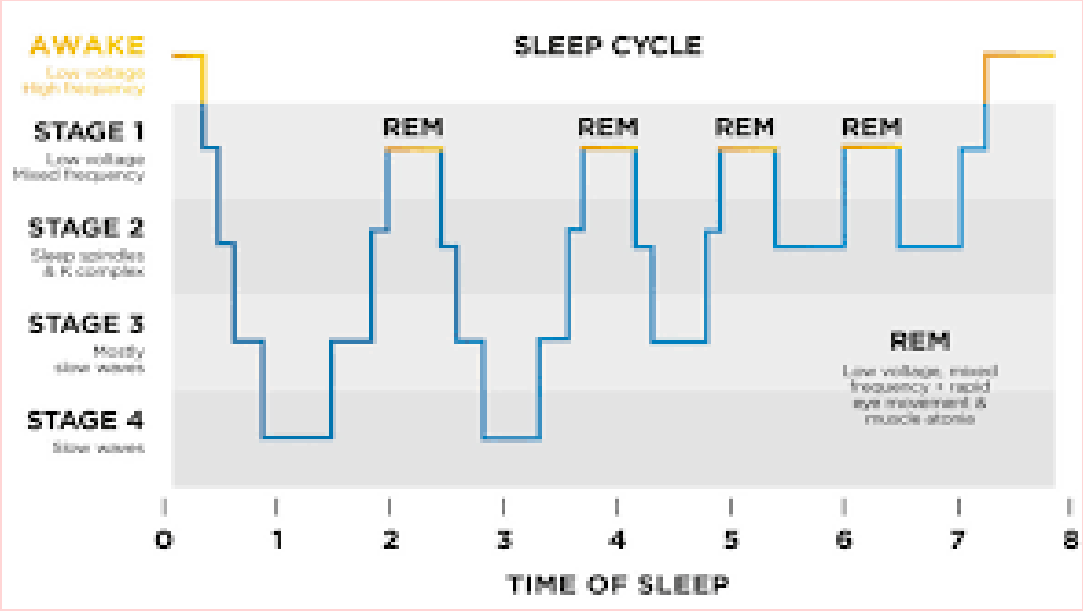
# 1. Sleep Inefficiency Due to OSA

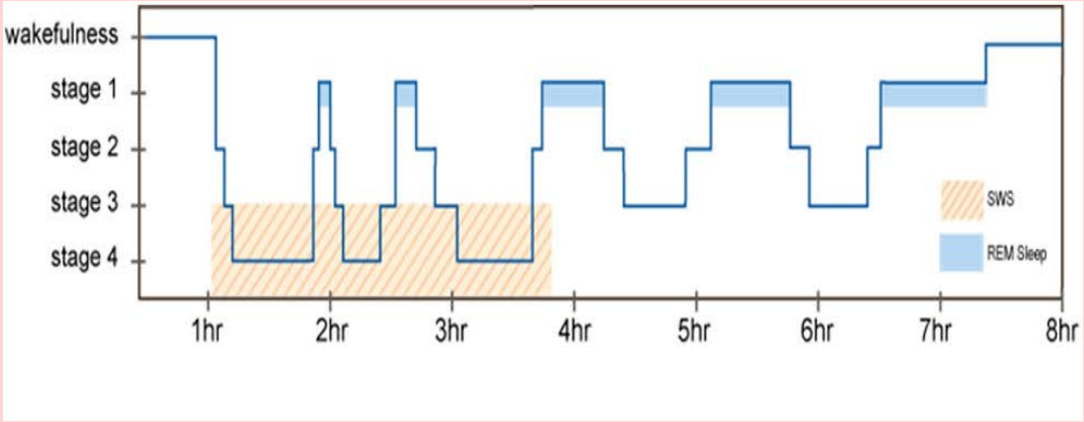
## Non-REM: 80% of sleep

-Decreased sympathetic and increased parasympathetic activity that leads to a "dipping" of both systolic and diastolic BP at night (decreases by 10% to 15%)

## REM:

-Increased sympathetic activity that leads to transient BP surges

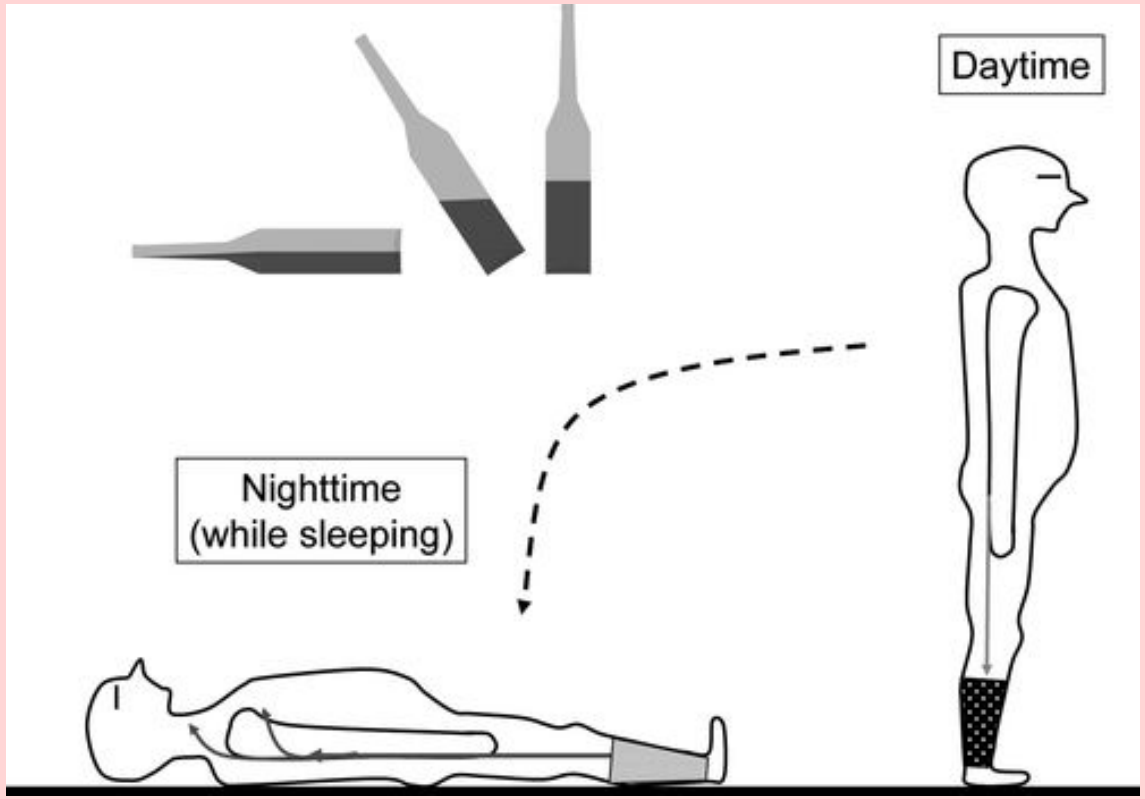


## *2. Nocturnal Fluid Shift in OSA*

During the night, the fluid accumulated in the legs will redistribute to the neck.

- This is especially significant for OSA and HTN patients in that the **reduction of the mean upper airway cross-sectional area can intensify hypopnea/apnea episodes and resultant hypoxia**, which will ultimately lead to transient BP surges.



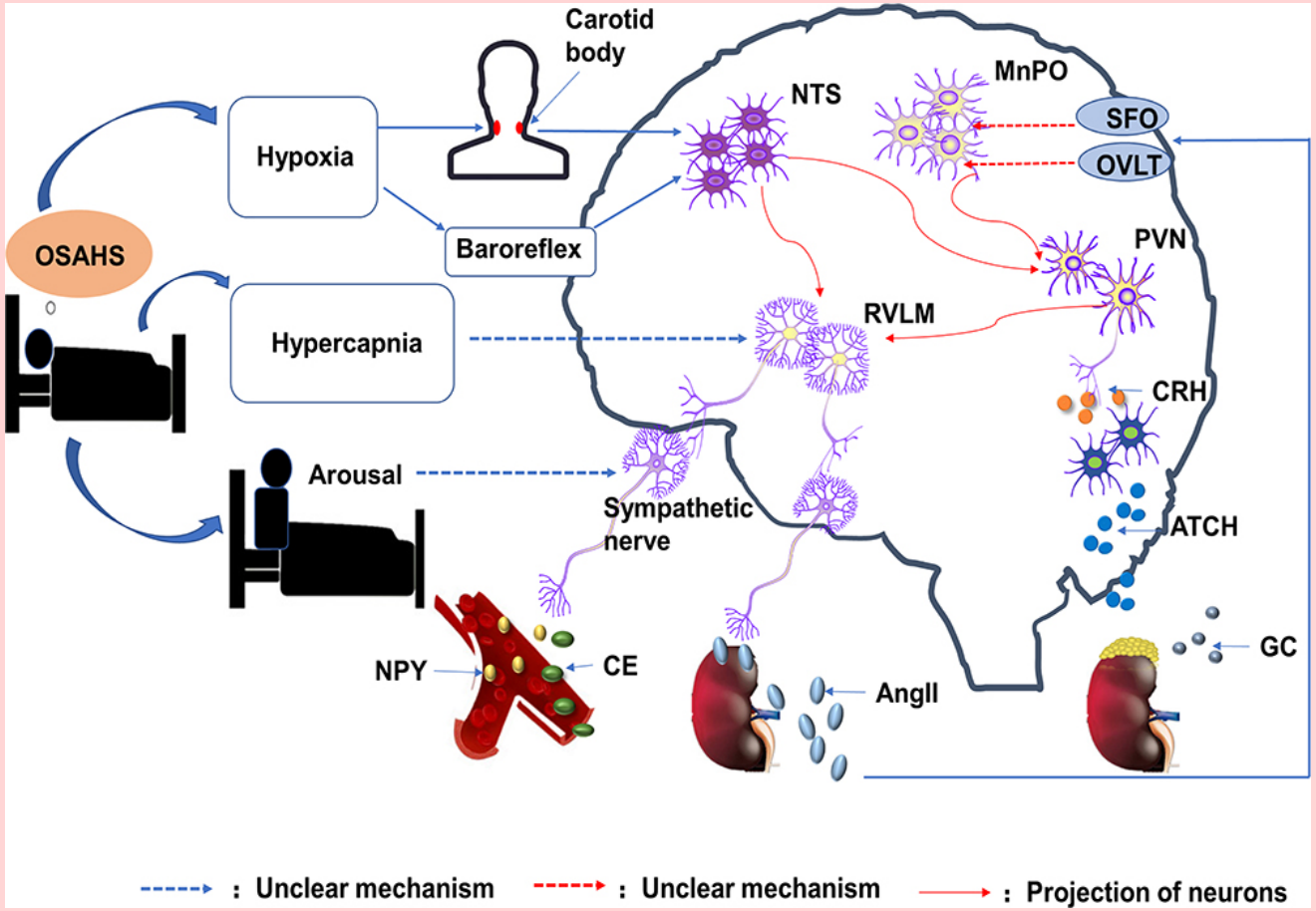
# 3. The Autonomic System Counterregulatory Mechanisms Against Apneic Episodes

- Apneic episodes and transient hypoxemia and hypercapnia **activate the sympathetic autonomic system and down-regulate the parasympathetic autonomic system**.

→ increase in catecholamine levels, causing a rise in heart rate and BP that persists into the next day.

- The **rise is most prominent during post-apneic hyperventilation** going as high as **240/130 mmHg.**

- Over time, this sympathetic stimulation can lead to the development of HTN in an OSA patient.



# 4. The Inflammatory and Cytokine-mediated Effects of Hypoxemia

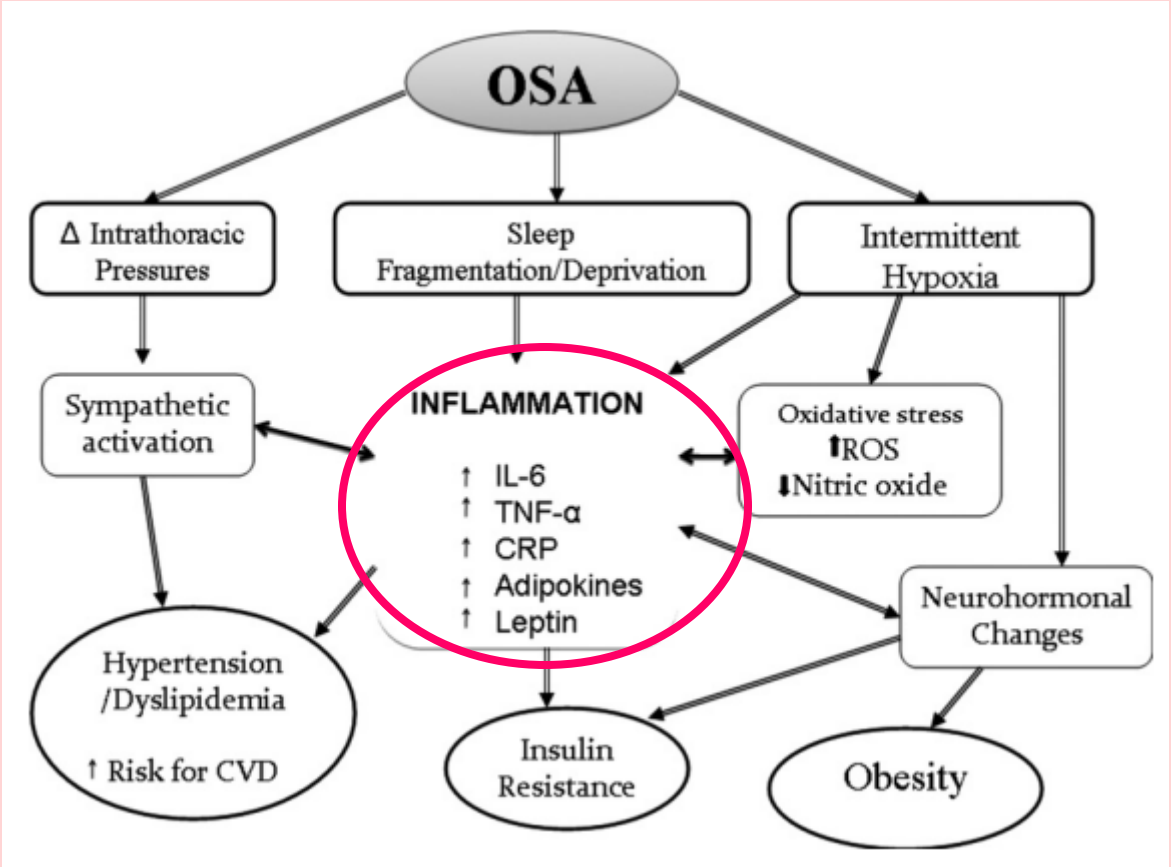
- Intermittent nocturnal hypoxemia and hypercapnia that causes **oxidative stress and inflammation.**

→ Release of reactive inflammatory cytokines (hs-CRP, IL-1, IL-8, IL-6, TNF-α, Rantes, and sICAM), and vasoactive substances.

- This leads to an increase in endothelin,

a decrease in nitric oxide, vasoconstriction,

and endothelial dysfunction.



## *5. The Renin-Angiotensin-Aldosterone System*

**Hypoxemia → Activation of the renin-angiotensin-aldosterone system (RAAS)**

**→ RAAS stimulation increases renin and aldosterone levels**

**→ Fluid retention seen in HTN, which leads to more rostral fluid displacement and an increase in upper airway obstruction.**

- CPAP therapy is associated with a down-regulation of RAAS activity, leading to consequent BP reduction.

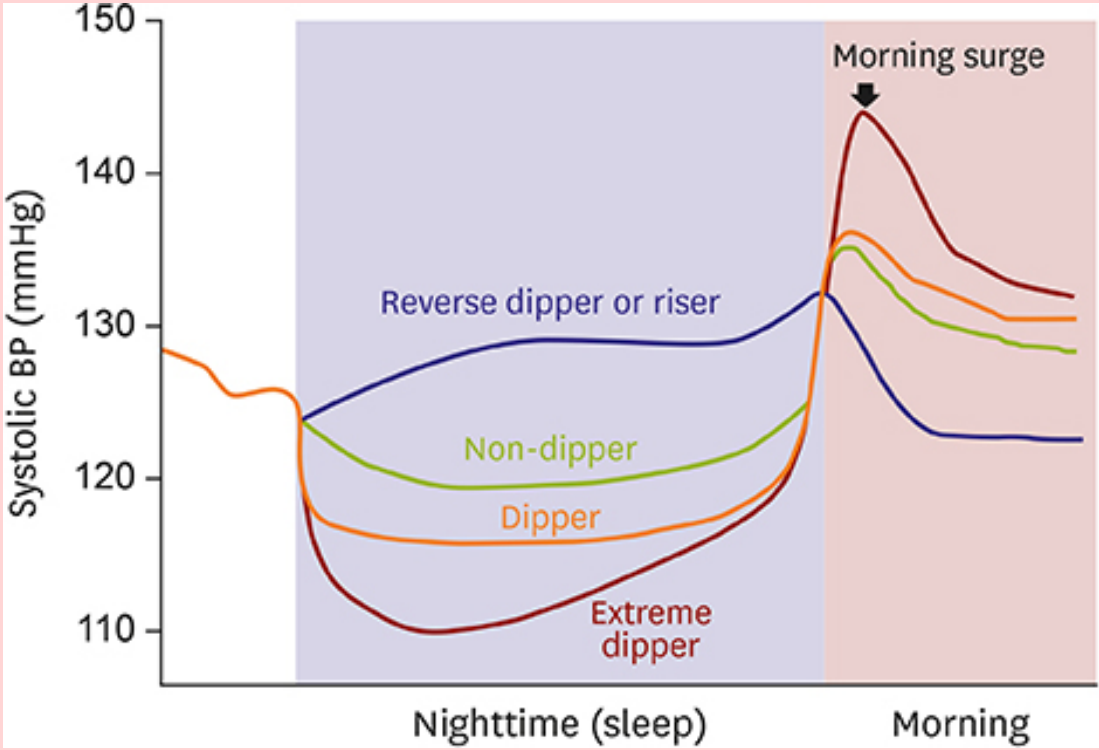
# *6. Non-dipping Phenomenon*

- **SBP and DBP reduce by ~ 10 mmHg (about 10–20%) during slumber, but this dipping phenomenon is reversed in those with OSA.**

- **Patients with OSA have elevated SNA from the obstructed airway, thus antagonizing the natural dipping phenomenon and causing intravascular pressure elevations.**

- **Overtime, the <u>chronic HTN leads to:</u>**

**sclerotic, noncompliant vasculature, decreased endothelial production of vasodilatory nitric oxide, and insensitive baroreceptors—further inhibiting the reflex dipping phenomenon.**



## *7. Sex Steroids*

According to the Wisconsin Sleep Cohort Study, **men are 2–4 times more likely to have OSA compared to women**, and progesterone and estrogen may play an important protective role.

Sex steroids are **neurosteroids** that readily cross the blood–brain barrier, where they **regulate respiratory function by binding to various receptors including GABA**.

# *8. Metabolic Derangements and the Gut Microbiome*

- Chronic OSA **increases energy expenditure**, resulting in **daytime cravings for energy dense foods**. Many of these palatable foods are **high in fat, carbohy**
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**the dietary h**
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**severity.**

> Biomed Pharmacother. 2019 Apr;112:108580. doi: 10.1016/j.biopha.2019.01.041. Epub 2019 Feb 18.

**Lactobacillus rhamnosus GG strain mitigated the development of obstructive sleep apnea-induced hypertension in a high salt diet via regulating TMAO level and CD4 $^{+}$ T cell induced-type I inflammation**

Jing Liu [1], Tianxiang Li [2], Hui Wu [1], Haoze Shi [1], Jinmei Bai [3], Wei Zhao [3], Donghui Jiang [4], Xiufeng Jiang [5]

Affiliations + expand

PMID: 30784906 DOI: 10.1016/j.biopha.2019.01.041

# *9. Hypercortisolism*

- Hypercortisolism, OSA, and obesity are interconnected.
- **Obesity, particularly in the setting of OSA, stimulates cortisol production**.

**When glucocorticoid production exceeds glucocorticoid receptor availability, these steroid hormones begin binding to mineralocorticoid receptors, thus acting as aldosterone agonists and favoring fluid retention**

# Management

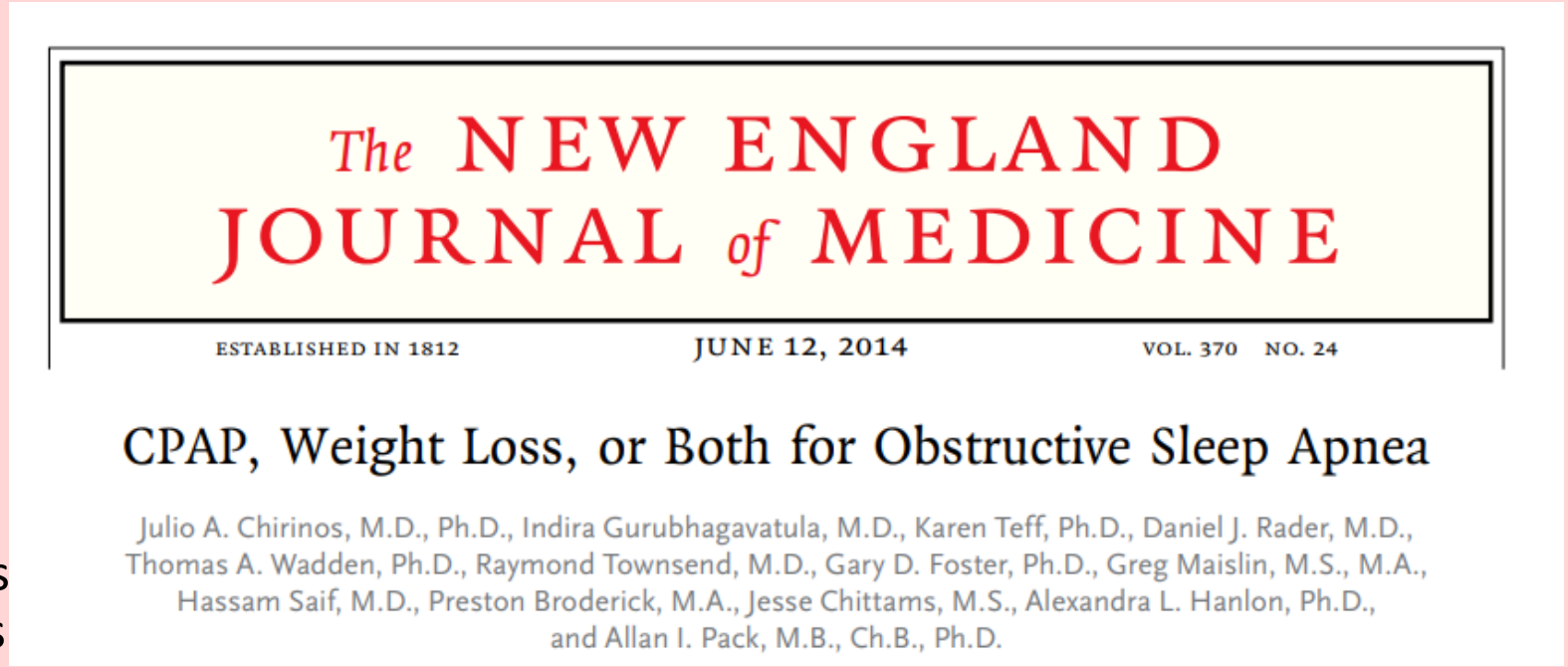
**With OSA and HTN, the goals of initial evaluation are:**

- **To determine the patient's baseline,**
- **Evaluate for target organ damage,**
- **Screen for potentially curable causes,**
- **Identify risk factors that are present,**
- **Determine the prognosis, and**
- **Choose a therapy that is specific to the patient's needs.**
- **A complete history and physical examination should be done.**

**The patient should also undergo extensive laboratory investigations such as:**

Urine analysis, complete blood count, blood chemistry (potassium, sodium, creatinine, fasting glucose, total and high-density lipoprotein or HDL cholesterol), creatinine clearance, 24-hour urinary protein, serum uric acid levels, serum calcium, glycosylated hemoglobin, fasting lipid panel, and plasma renin activity/aldosterone measurements, a 12-lead ECG and an echocardiography

# *Lifestyle Modifications*



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CPAP, Weight Loss, or Both for Obstructive Sleep Apnea

Julio A. Chirinos, M.D., Ph.D., Indira Gurubhagavatula, M.D., Karen Teff, Ph.D., Daniel J. Rader, M.D., Thomas A. Wadden, Ph.D., Raymond Townsend, M.D., Gary D. Foster, Ph.D., Greg Maislin, M.S., M.A., Hassam Saif, M.D., Preston Broderick, M.A., Jesse Chittams, M.S., Alexandra L. Hanlon, Ph.D., and Allan I. Pack, M.B., Ch.B., Ph.D.

- Obesity is one of the few risk factors **in weight** can help reduce the OSA s

In a randomized control trial, to assess the effect of CPAP and weight reduction on OSA patients were divided into groups having CPAP treatment alone, weight reduction or CPAP, and weight reduction together.

A **reduction in CRP, insulin, and triglyceride levels was seen in the group with both interventions**; however, no such reduction in CRP level was noted in patients taking CPAP alone.

- **As deterioration of OSAS occurs with alcohol intake, moderate drinking is to be recommended.**
- **Smokers are advised to quit smoking.**

# *CPAP therapy*

In hypertensive patients with moderate to severe OSAS, **CPAP therapy is attempted as a first-line treatment.**

- **Exerts a BP-lowering effect,**
- **Decreases nocturnal BP surge**
- **Improves cardiovascular prognosis in many OSAS patients**.

**Individual differences in the effect of CPAP** have been observed;

- ✓Higher BP levels,
- ✓Untreated hypertension,
- ✓Nocturnal hypertension
- ✓Resistant hypertension.
- ✓Severe OSAS
- ✓High body mass index, and
- ✓More daytime sleepiness.

**Table 2 Determinants of effective reduction in blood pressure by CPAP therapy in hypertensive patients with obstructive sleep apnea syndrome**

| |
|---|
| *Characteristics* |
| Obesity (increased BMI) |
| *Blood pressure* |
| Higher BP level before treatment |
| Untreated hypertension |
| Nocturnal hypertension |
| Resistant hypertension |
| *OSAS factor* |
| Severe OSAS AHI > 30 |
| OSAS with daytime sleepiness |
| *CPAP factor* |
| Adequate compliance for CPAP use for > 3-h/night |
| Long-term use of CPAP |
| Effectiveness of CPAP (AHI reduction > 50%) |

# CPAP

✓ Delivers **continuous, positively pressurized air into the distal alveoli of the respiratory tree**, which **maintains alveolar patency**.

✓ **Reduces arterial stiffness → reduces HTN**, and

✓ **Improves vascular inflammation in those with OSA**.

** The variable reductions in SBP and DBP range **from - 2 to - 9 mmHg and - 2 to - 7** mmHg, respectively.

➢ It is important to ensure favorable compliance with CPAP,:

**use of CPAP for at least 3h each night, 50% decrease of the AHI and prolonged periods of CPAP use.**

*** **39–50% of patients** prescribed nocturnal CPAP for OSA are **noncompliant with usage**.

*** One study reported that **63% of patients** prescribed CPAP **reported feeling claustrophobic** while using the machine.

Randomized Controlled Trial > Hypertension. 2006 May;47(5):840-5.
doi: 10.1161/01.HYP.0000217128.41284.78. Epub 2006 Apr 3.

# Effects of continuous positive airway pressure versus supplemental oxygen on 24-hour ambulatory blood pressure

Daniel Norman [1], José S Loredo, Richard A Nelesen, Sonia Ancoli-Israel, Paul J Mills, Michael G Ziegler, Joel E Dimsdale

Affiliations + expand

PMID: 16585412 DOI: 10.1161/01.HYP.0000217128.41284.78

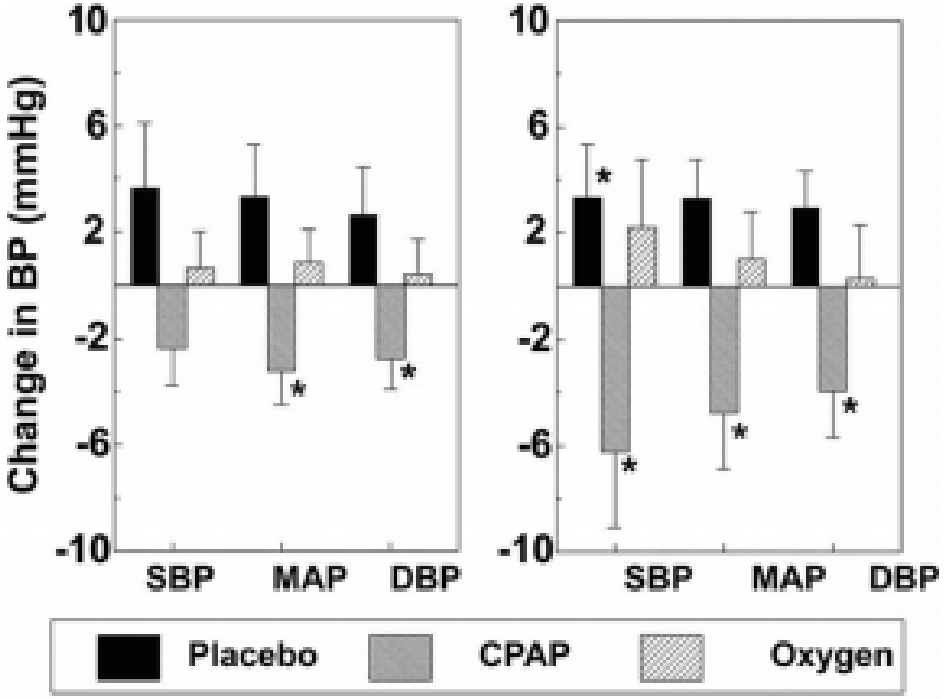



**Figure 1.** Effect of CPAP vs supplemental oxygen on 24-hour ambulatory blood pressure. Use of CPAP was associated with a significant decrease in blood pressure compared with patients treated with placebo or supplemental oxygen. The change was measured from the mean pretreatment minus posttreatment values. *Significant change over time, $P<0.05$. (From Norman et al,[31] copyright 2006. American Heart Association. All rights reserved. Reproduced with permission.)

Review > Hypertension. 2007 Aug;50(2):417-23. doi: 10.1161/HYPERTENSIONAHA.106.085175. Epub 2007 Jun 4.

# Effect of nocturnal nasal continuous positive airway pressure on blood pressure in obstructive sleep apnea

Lydia A Bazzano [1], Zia Khan, Kristi Reynolds, Jiang He

Affiliations + expand

PMID: 17548722 DOI: 10.1161/HYPERTENSIONAHA.106.085175

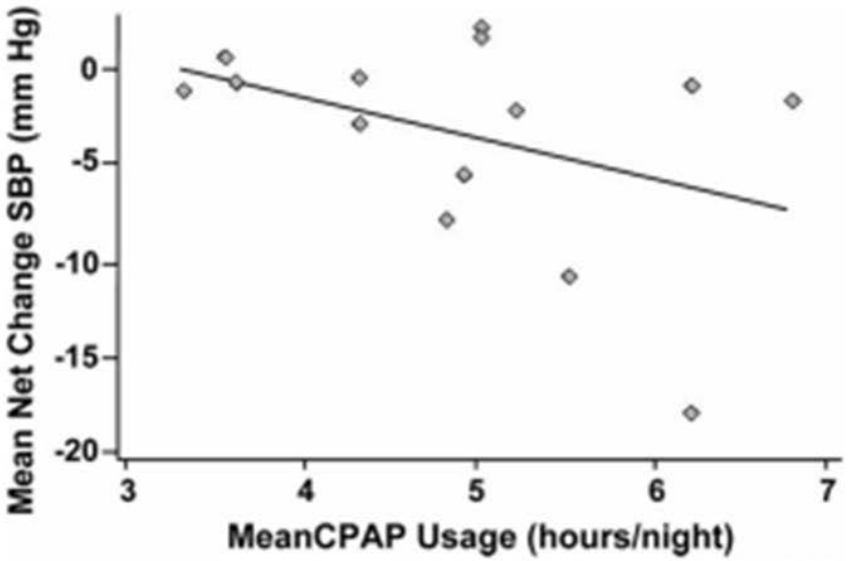
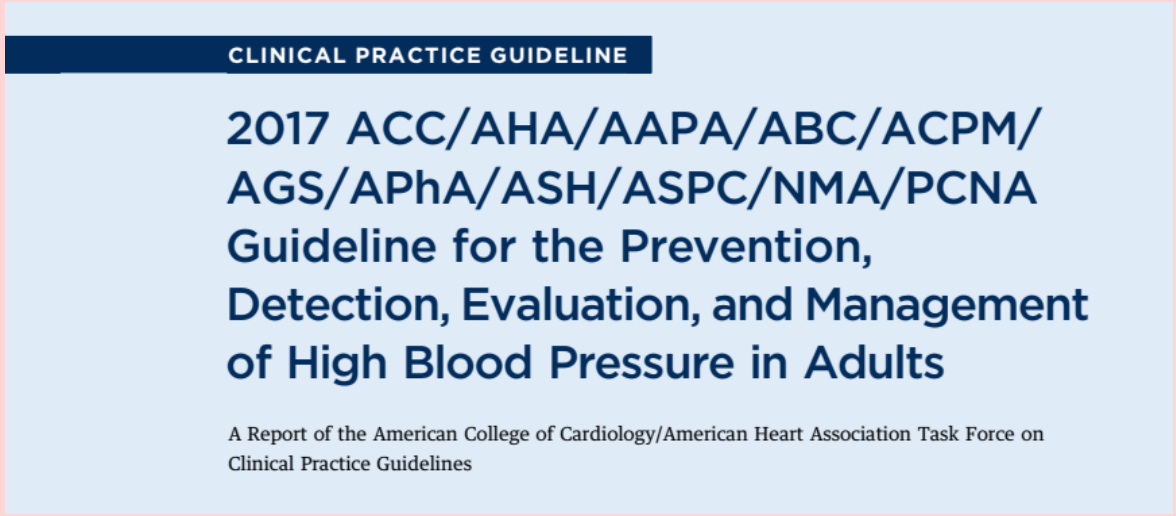



**Figure 2.** Effect of CPAP usage on change in blood pressure. Shown is the relationship between the mean net change in systolic blood pressure (SBP) and the corresponding mean nocturnal CPAP usage based on a meta-analysis of CPAP trials by Bazzano et al.[36] This suggests that longer nightly CPAP use may offer greater benefit in reducing SBP ($r^2$=0.40; $P$=0.13). (From Bazzano et al,[36] copyright 2007. American Heart Association. All rights reserved. Reproduced with permission.)

**Table 1** Summary of meta-analyses of randomized controlled CPAP trials

| Study | Number of trials/patients | BP end point | Minimum CPAP duration | Outcome |
|---|---|---|---|---|
| Alajmi et al. [27] | 10/587 | Office/ambulatory | 4 wk | SBP: −1.38 mm Hg (not significant)<br>DBP: −1.52 mm Hg (not significant)<br>More benefit in more severe OSA; trend for better SBP reduction with better CPAP adherence |
| Bazzano et al. [26] | 16/818 | Office/ambulatory | 2 wk | SBP: −2.46 mm Hg<br>DBP: −1.83 mm Hg<br>More benefit in patients with higher baseline BP, higher BMI, and more severe OSA |
| Haentjens et al. [29] | 12/572 | Ambulatory | 1 wk | 24-h SBP: −1.64 mm Hg<br>24-h DBP: −1.48 mm Hg<br>More benefit in more severe OSA and with better CPAP adherence |
| Mo and He [28] | 7/471 | Ambulatory | 4 wk | 24-h SBP: −0.95 mm Hg (not significant)<br>24-h DBP: −1.78 mm Hg |

*BMI* body mass index, *BP* blood pressure, *CPAP* continuous positive airway pressure, *DBP* diastolic blood pressure, *OSA* obstructive sleep apnea, *SBP* systolic blood pressure




**CPAP is an efficacious treatment for improving obstructive sleep apnea.**

However, studies of the effects of CPAP on BP have demonstrated only small effects on BP (e.g., 2– to 3–mm Hg reductions), with results dependent on patient compliance with CPAP use, severity of obstructive sleep apnea, and presence of daytime sleepiness in study participants.

**Recommendation for Obstructive Sleep Apnea**
**References that support the recommendation are summarized in Online Data Supplement 8.**

| COR | LOE | RECOMMENDATION |
|---|---|---|
| IIb | B-R | 1. In adults with hypertension and obstructive sleep apnea, the effectiveness of continuous positive airway pressure (CPAP) to reduce BP is not well established (S5.4.4-1–S5.4.4-5). |

Stroke

AHA/ASA GUIDELINE

# 2021 Guideline for the Prevention of Stroke in Patients With Stroke a Attack

## A Guideline From the American Heart A

*Reviewed for evidence-based integrity and endorsed by the Congress of Neurological Surgeons.*

*Endorsed by the Society of Vascular and Interventional Neurology*

*The American Academy of Neurology affirms the value of this stat*

## 4.6. Obstructive Sleep Apnea

**Recommendations for Obstructive Sleep Apnea**
**Referenced studies that support recommendations are summarized in online Data Supplements 18 and 19.**

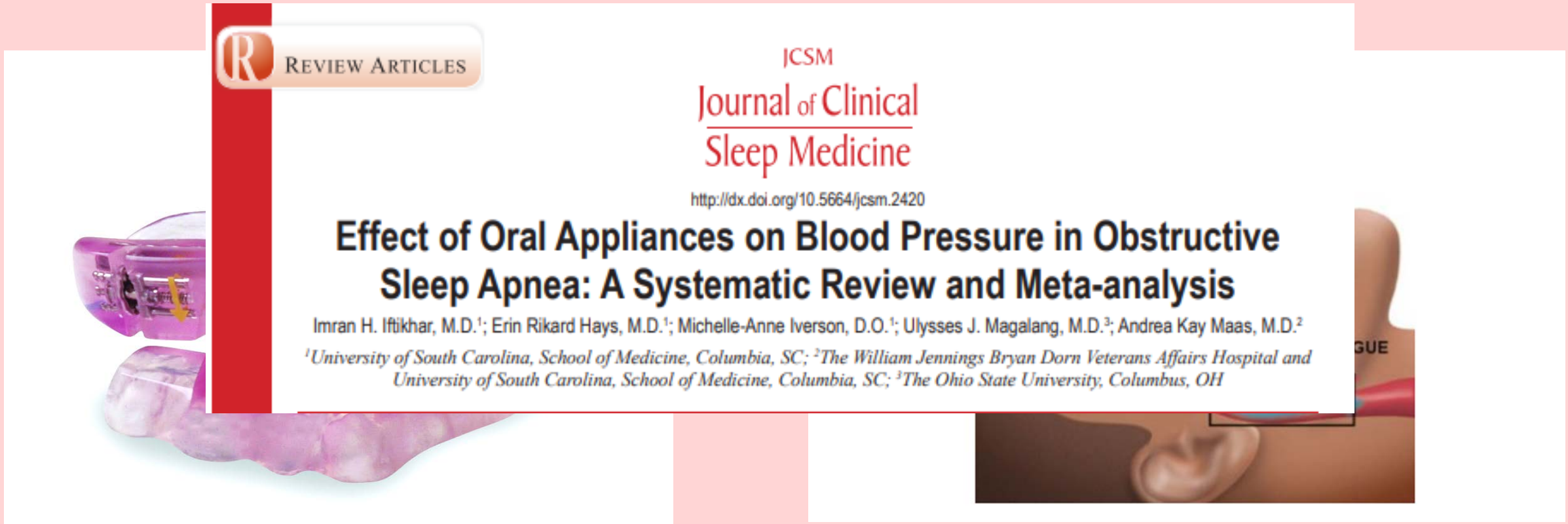
| COR | LOE | Recommendations |
|---|---|---|
| 2a | B-R | 1. In patients with an ischemic stroke or TIA and OSA, treatment with positive airway pressure (eg, continuous positive airway pressure [CPAP]) can be beneficial for improved sleep apnea, BP, sleepiness, and other apnea-related outcomes.[302–314] |
| 2b | B-R | 2. In patients with an ischemic stroke or TIA, an evaluation for OSA may be considered for diagnosing sleep apnea.[302,303,315,316] |

# *Oral Appliances*

In mild to moderate OSA, oral appliances can be recommended as an **alternative treatment to CPAP**.

***A meta-analysis of seven studies (399 OSA patients involved) found that treatment with oral appliances **was more beneficial for BP reduction than CPAP therapy.**

The average drop in the systolic BP and diastolic BP was 2.7 mm Hg.



REVIEW ARTICLES

JCSM
Journal of Clinical Sleep Medicine
http://dx.doi.org/10.5664/jcsm.2420

## Effect of Oral Appliances on Blood Pressure in Obstructive Sleep Apnea: A Systematic Review and Meta-analysis

Imran H. Iftikhar, M.D.[1]; Erin Rikard Hays, M.D.[1]; Michelle-Anne Iverson, D.O.[1]; Ulysses J. Magalang, M.D.[3]; Andrea Kay Maas, M.D.[2]

*[1]University of South Carolina, School of Medicine, Columbia, SC; [2]The William Jennings Bryan Dorn Veterans Affairs Hospital and University of South Carolina, School of Medicine, Columbia, SC; [3]The Ohio State University, Columbus, OH*

# *Antihypertensive drugs*

- There is as yet insufficient evidence to set an optimal target BP level, but it is important to **suppress nocturnal BP at least to the reference levels, that is, 120/70 mmHg.**

- There is also as yet insufficient evidence to suggest whether any specific class of antihypertensive drugs must be used to treat hypertension associated with OSAS.

- **Beta-blockers and aldosterone antagonists** may be the best treatment options as they act on catecholamine release from the activated sympathetic system or from the RAAS system activation.

The aldosterone antagonist **spironolactone** is considered very effective for decreasing the severity of OSA.

- **Diuretic therapy** reduces the intravascular hypervolemia observed in OSA-associated HTN. Diuretics reduce extracellular fluid by 10–12% within a few weeks of treatment initiation.

Clinical Trial > J Hum Hypertens. 2010 Aug;24(8):532-7. doi: 10.1038/jhh.2009.96.
Epub 2009 Dec 17.

# Spironolactone reduces severity of obstructive sleep apnoea in patients with resistant hypertension: a preliminary report

K Gaddam [1], E Pimenta, S J Thomas, S S Cofield, S Oparil, S M Harding, D A Calhoun

Affiliations + expand

PMID: 20016520 PMCID: PMC2891919 DOI: 10.1038/jhh.2009.96

Free PMC article

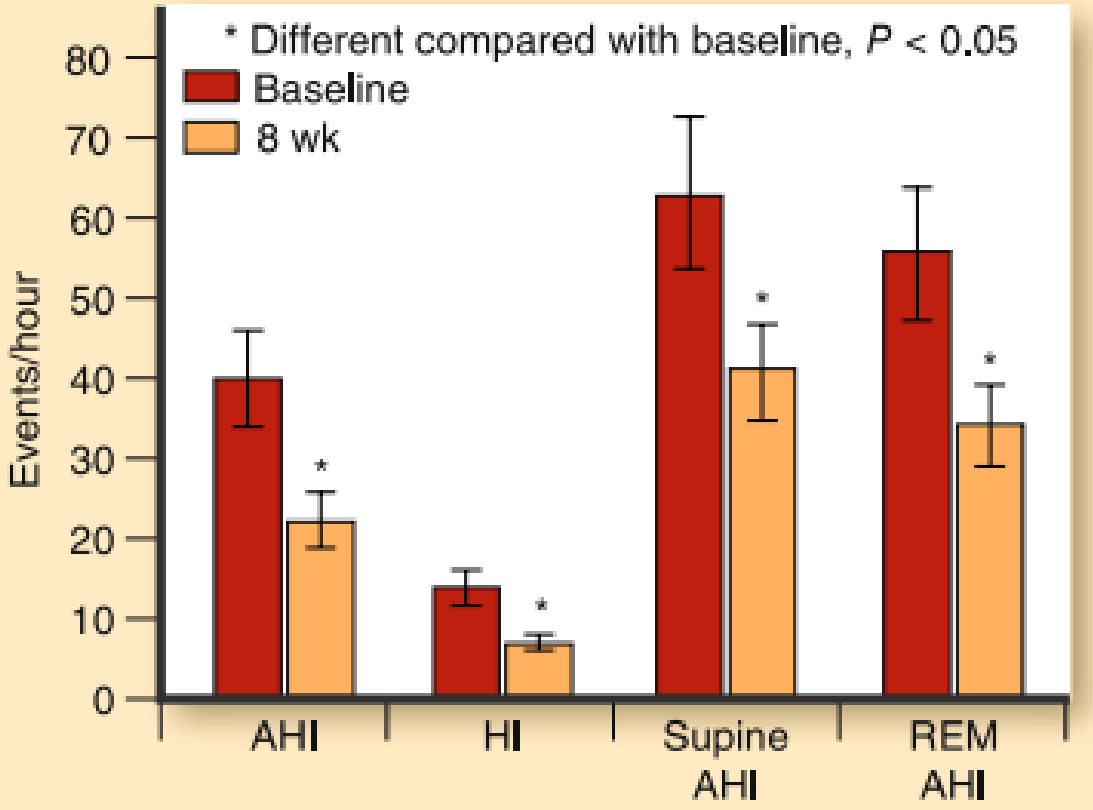



**Fig. 2** Effects of 8 weeks of treatment with spironolactone on apnea-hypopnea index (AHI), hypoxic index (HI), supine AHI, and rapid eye movement sleep (REM) AHI at 8 weeks compared with baseline in patients with resistant hypertension. (*From* Gaddam et al. [47]; with permission)

## Upper ai

- **Tonsillec**
- **Uvulopal**

Randomized Controlled Trial > Sleep Med. 2017 Jun;34:156-161. doi: 10.1016/j.sleep.2017.02.030. Epub 2017 Apr 4.

# Blood pressure after modified uvulopalatopharyngoplasty: results from the SKUP 3 randomized controlled trial

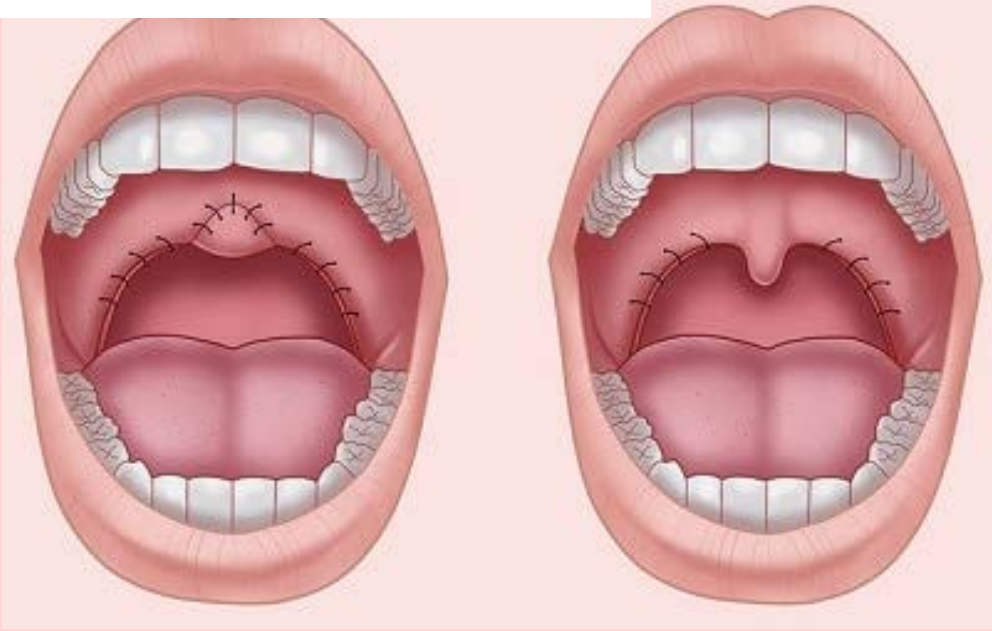
Johan Fehrm [1], Danielle Friberg [2], Johan Bring [3], Nanna Browaldh [2]

Affiliations + expand

PMID: 28522085 DOI: 10.1016/j.sleep.2017.02.030

A 2017 randomized controlled trial determined that modified UPPP **significantly improved sleepiness, nocturnal respirations, and quality of life**.

The trial also determined that the **BP was reduced significantly after surgery** in a select group of patients with moderate to severe OSA.



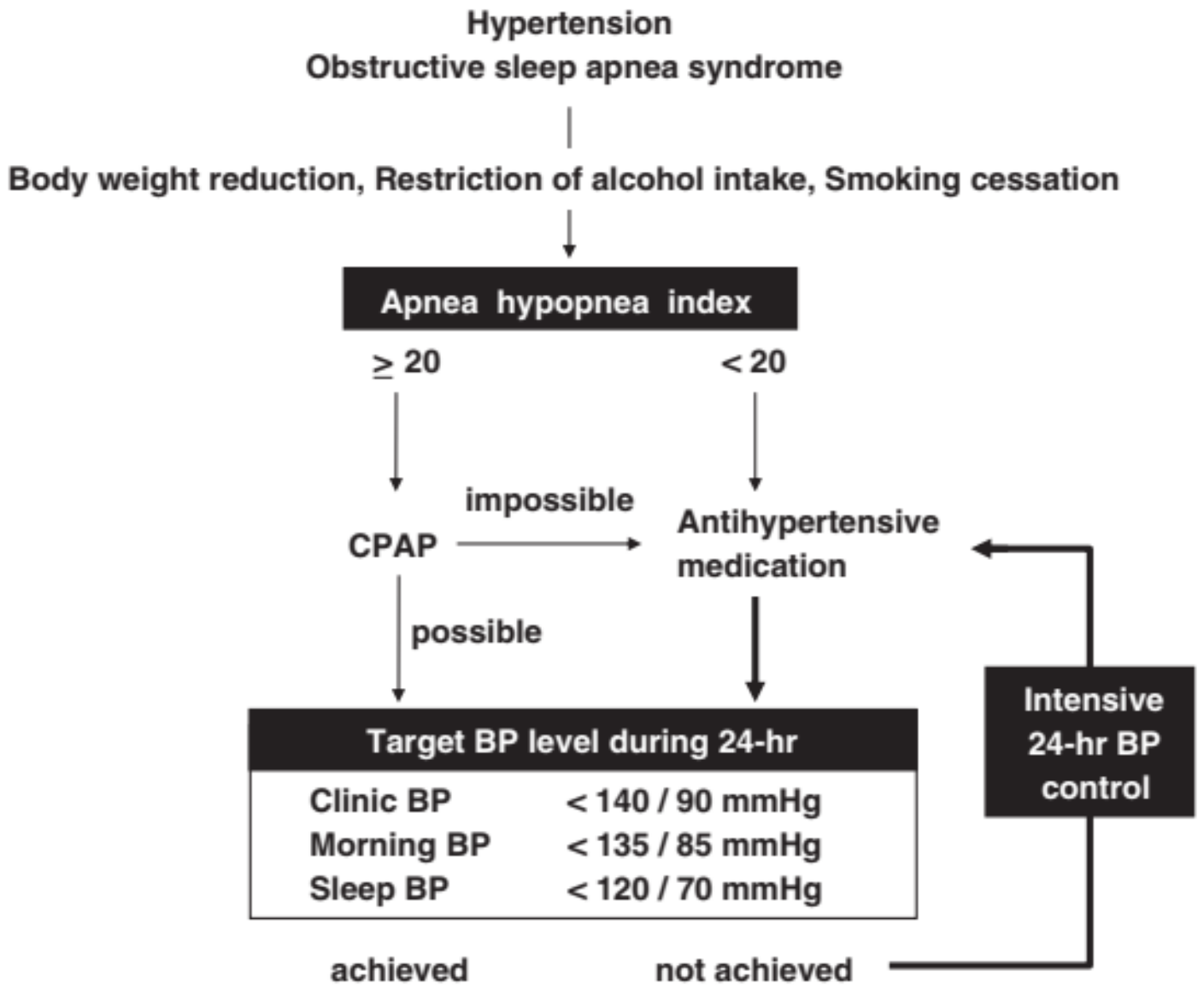
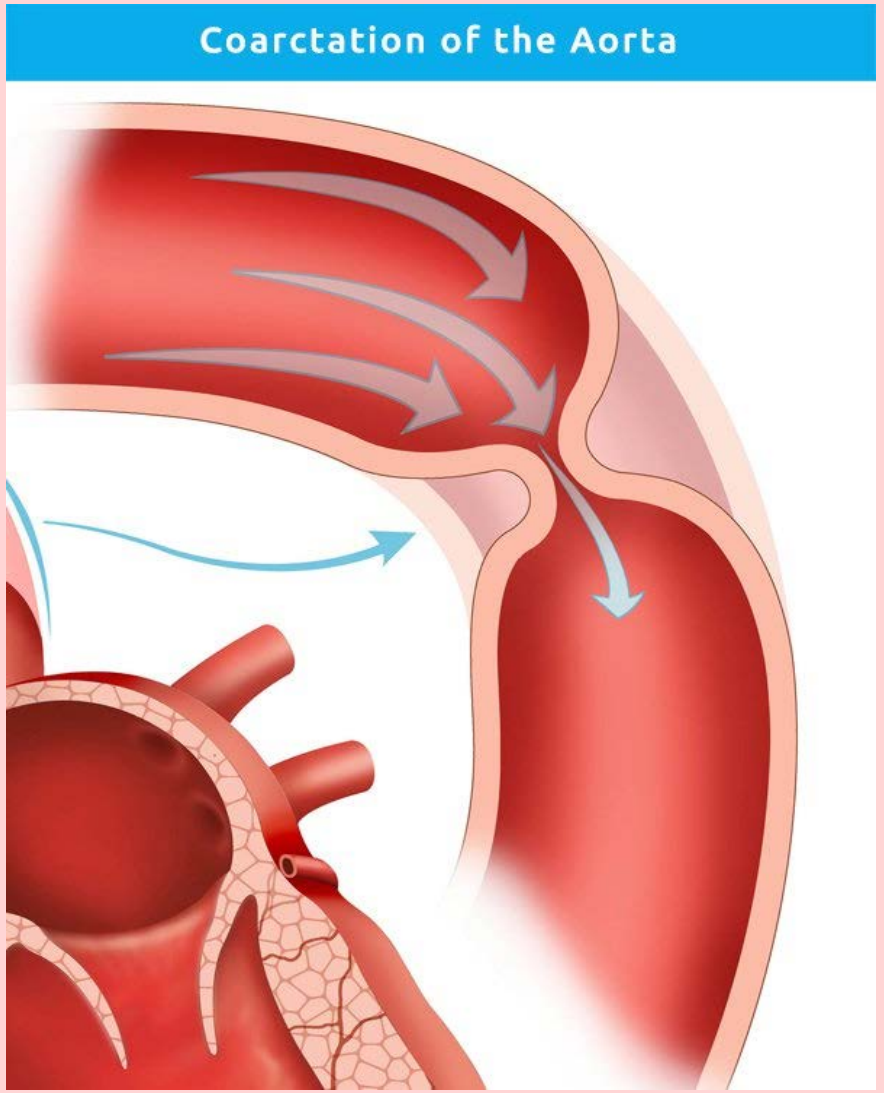


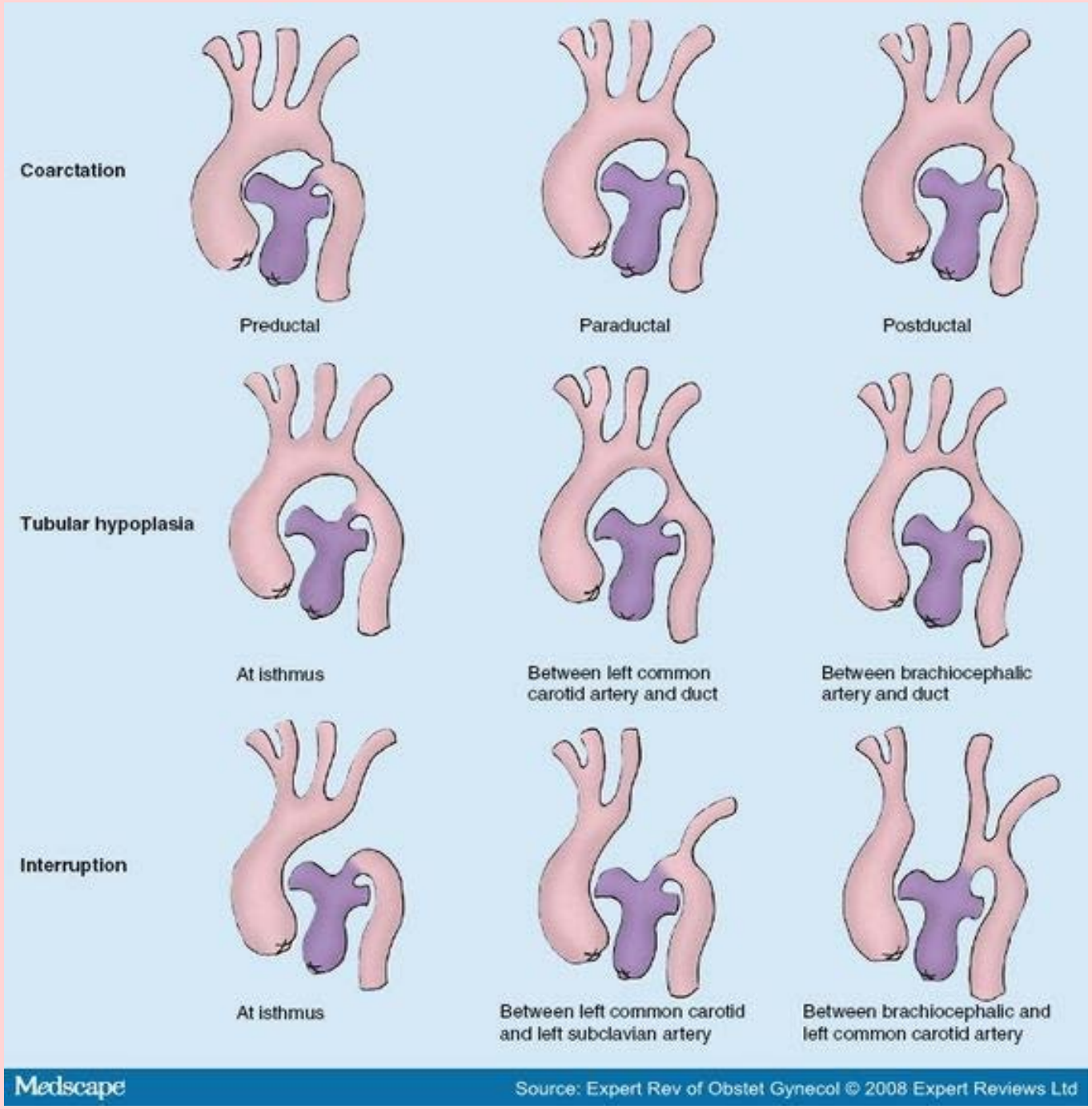

**Figure 3** Management of hypertension with obstructive sleep apnea syndrome.


HYPERTENSION

# Coarctation of Aorta in adults



A spectrum of aortic narrowing from **discrete entity to tubular hypoplasia,** with many variations seen in between these two extremes.



# Epidemiology

- Fifth most common congenital heart defect.
- 6–8% of live births with congenital heart disease, with an estimated incidence of 1 in 2500 births.
- Reported ratio in males to females of between 1.27:1 and 1.74:1
- Average survival age of individuals with unoperated coarctation was approximately 35 years of age, with 75% mortality by 46 years of age.

## ❖ Cardiac association:

**Atrial septal defect (ASD), ventricular septal defect (VSD), atrioventricular canal defect (AVCD), bicuspid aortic valve (BAV), transposition of great arteries (TGA), patent ductus arteriosus (PDA), hypoplastic left heart syndrome.**

## ❖ Noncardiac associations:

**Intracranial aneurysms and CoA**

**5% deaths in patients with aortic coarctation on autopsy review.**
**Most of the aneurysms described are small,**
**and therefore have a low risk of spontaneous rupture.**

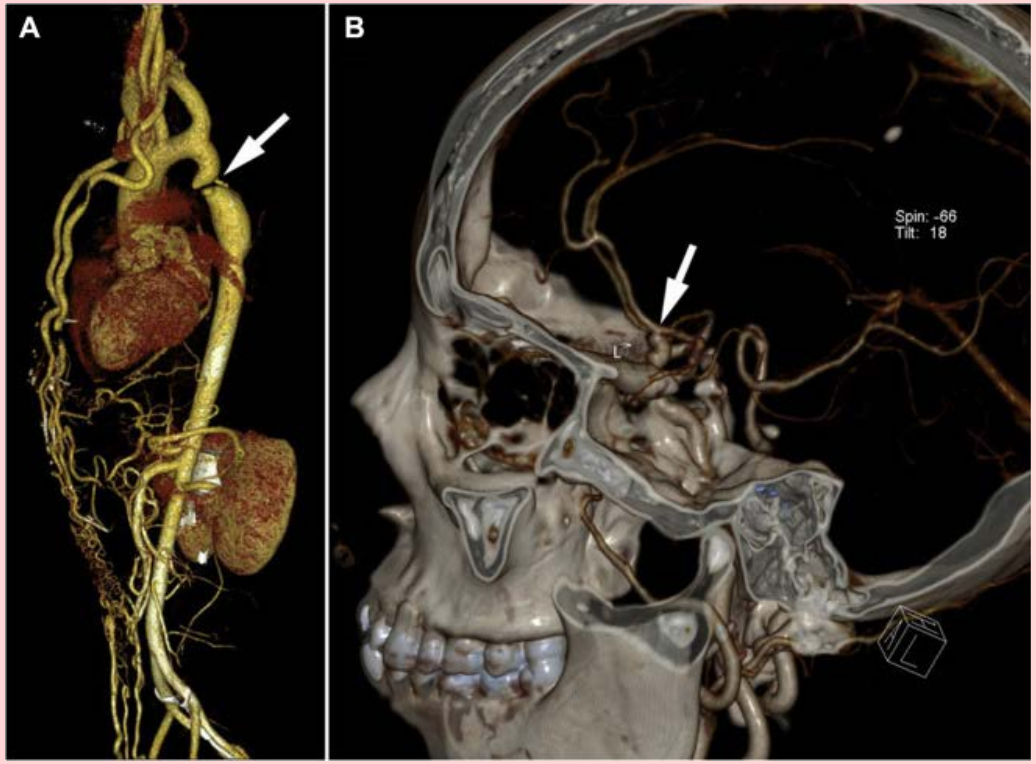


**Table 2** Long-term complications of CoA

| Long-term complications | |
|---|---|
| Local | Recoarctation, aneurysm, dissection, rupture, fistulae, endocarditis |
| Ascending aorta | Aneurysm, dissection, rupture, sinus of Valsalva fistula |
| Aortic valve | (Bicuspid valve), stenosis, regurgitation |
| Left ventricle | Hypertrophy, dilation, systolic dysfunction and/or diastolic dysfunction, heart failure, sudden cardiac death |
| Coronaries | Premature atherosclerosis, ischaemic heart disease |
| Cerebral | Berry aneurysms, intracranial bleeds, atherosclerosis, stroke |
| Systemic | Hypertension, reduced exercise capacity |

**Causes of death:**

- ✓Heart failure,
- ✓Aortic rupture,
- ✓Aortic dissection,
- ✓Endocarditis,
- ✓Intra-cerebral hemorrhage,
- ✓Myocardial infarction.

# Presentation

- Most adults with unrepaired coarctation are **generally asymptomatic.**

- A common presentation of coarctation is **systemic arterial hypertension**.

-In young adults presenting with severe upper extremity hypertension, coarctation should be excluded.

- Patients presenting with severe hypertension may experience symptoms including **angina, headache, epistaxis, and heart failure.**

- **Leg fatigue or claudication** due to post-stenotic hypoperfusion.

# Physical exam

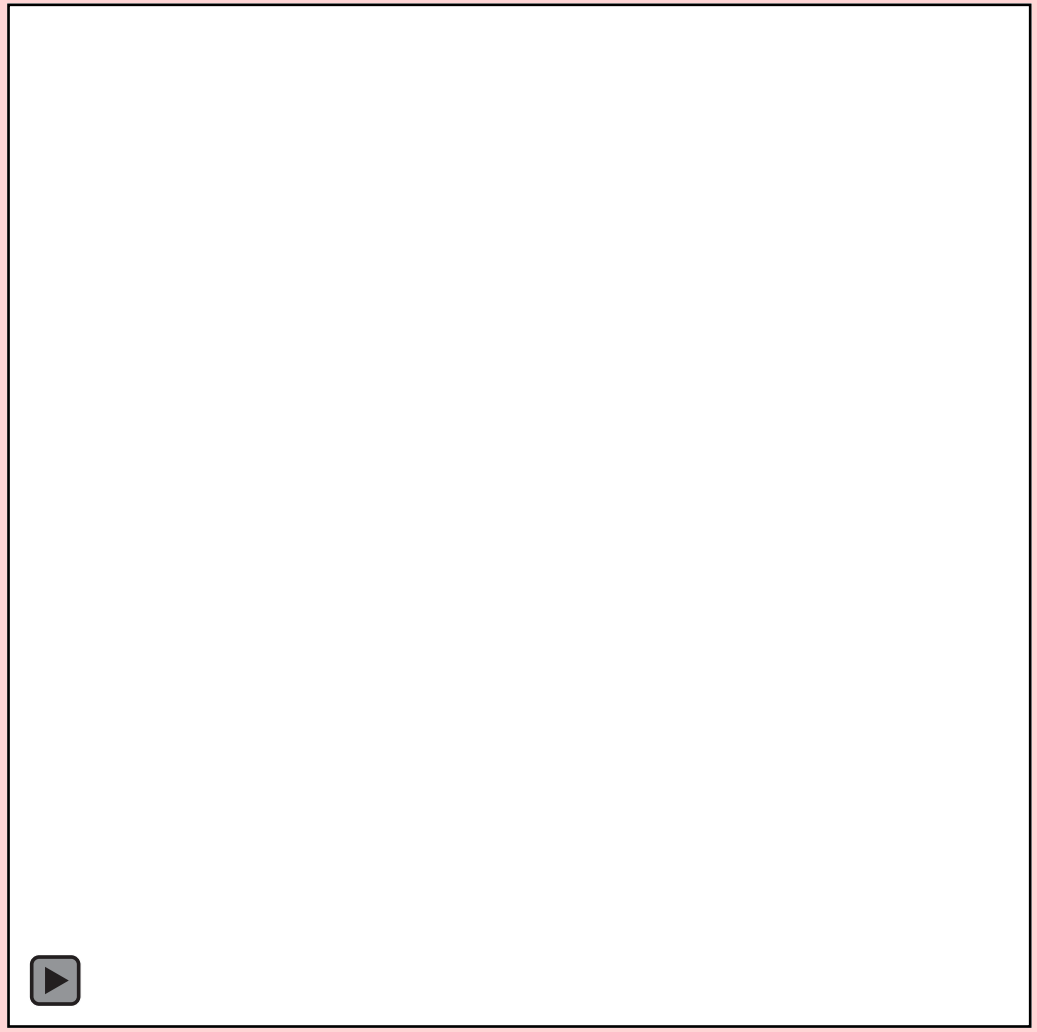
- **Femoral arterial pulses are diminished and usually delayed.**

*****The American Academy of Pediatrics recommends:**

**Simultaneous palpation of the femoral and radial pulses at pre participation sports visits as well as four extremity blood pressure checks.**

- **Auscultation of the left sternal border: Harsh systolic murmur with radiation to the back.**
- **An associated thrill may be palpable in the suprasternal notch.**

- **The finding of a continuous murmur may suggest the presence of arterial collaterals in those with long-standing unrepaired significant coarctation.**



*** If aortic coarctation is suspected **blood pressure should be measured in both arms and legs <u>in supine position</u>.**

- **Normally BP in the lower extremities is 10–20% higher than the upper extremities due to wave amplification.**

- **If BP in the leg is lower than the arm BP by 10 mmHg or more then coarctation should be suspected.**

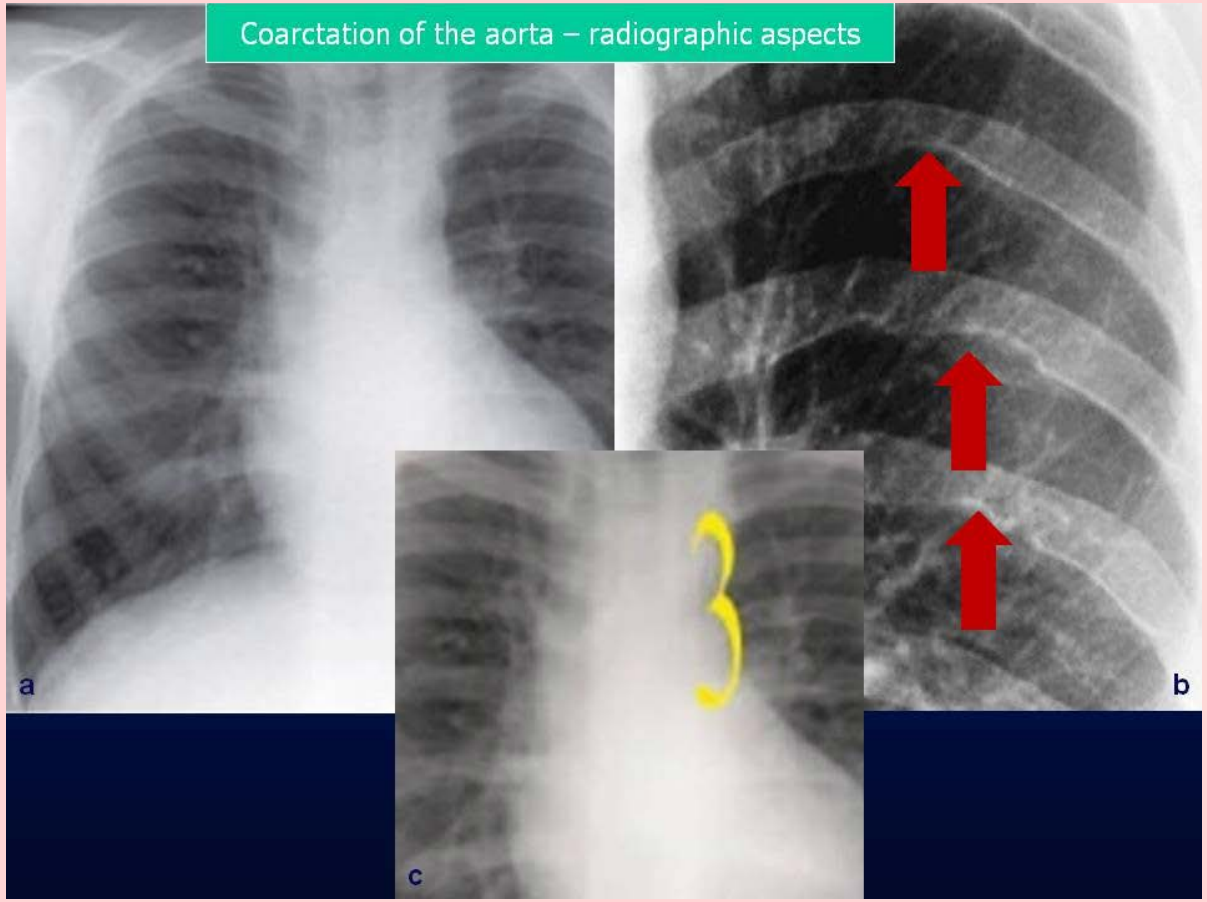
Systolic/ continuous murmur
Weak or absent femoral pulses, radio-femoral delay
Supine arm-leg blood pressure gradient
Hypertension (headache, epistaxis, retinopathy)
Reduced exercise capacity, exercise induced hypertension
Leg fatigue and claudication
Cold feet
Left ventricular hypertrophy, arrhythmia and heart failure
Infective endocarditis
Aortic dissection, rupture
Intracranial haemorrhage

## ECG:

Normal or **evidence of LVH** from chronic left ventricular pressure overload.

## CXR:

- A **"figure of three" sign** formed by the aortic nob, the stenotic segment, and the dilated post stenotic segment of the aorta suggests CoA.
- The heart border can be normal or mildly enlarged.
- **Inferior rib notching** can also be seen in the third to eighth ribs bilaterally caused by the presence of dilated intercostal collateral arteries.



# Echocardiography

- Evidence of left ventricular pressure or volume overload, left ventricular hypertrophy, size, and left ventricular systolic and diastolic dysfunction.

- Associated cardiac defects especially left sided lesions.
-
- The morphology of the aortic valve, and evidence of subvalvular, valvular, and supravalvular aortic stenosis should be interrogated.

- The dimensions of the aortic root and ascending aorta.

# Suprasternal windows:

- **Focal area of narrowing of the thoracic aorta** distal to the takeoff of the left subclavian artery with associated flow turbulence on color flow Doppler.

- The suprasternal notch view is used for obtaining **Doppler gradient**. Systolic velocity in the descending aorta is increased.
- In severe cases there is a gradient during both systole and diastole across the stenosis, which results in the **classic saw tooth pattern.**
- Subcostal imaging is used to **evaluate the distal thoracic and upper abdominal aorta**.

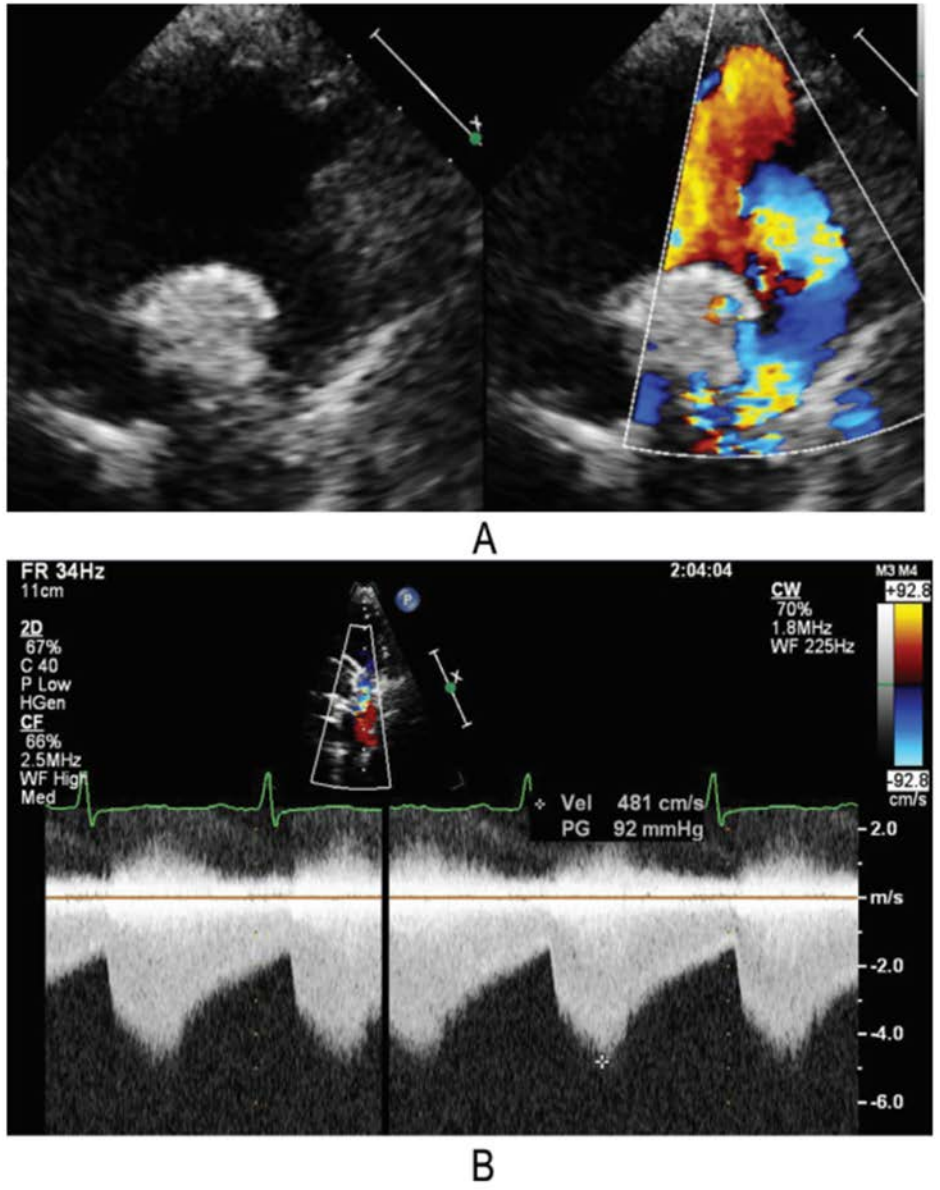
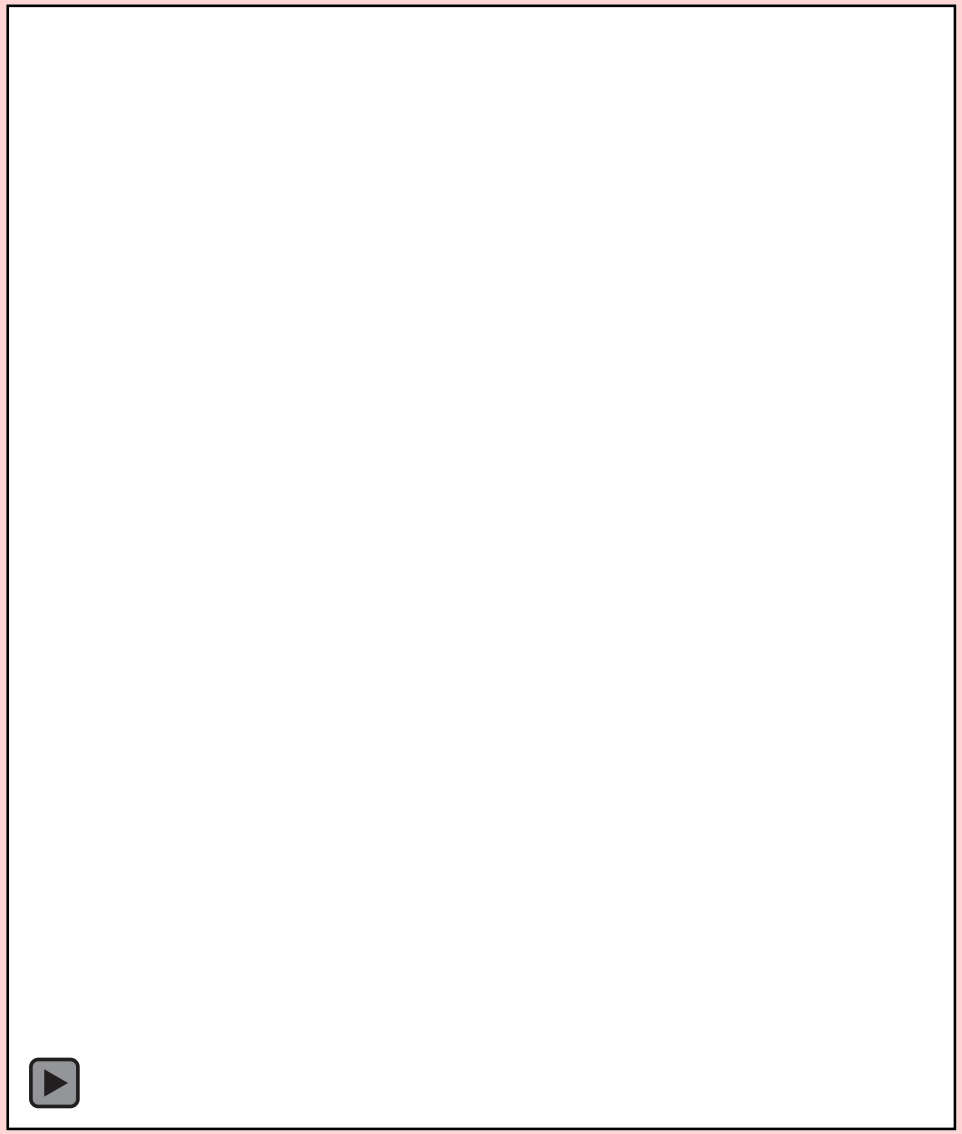


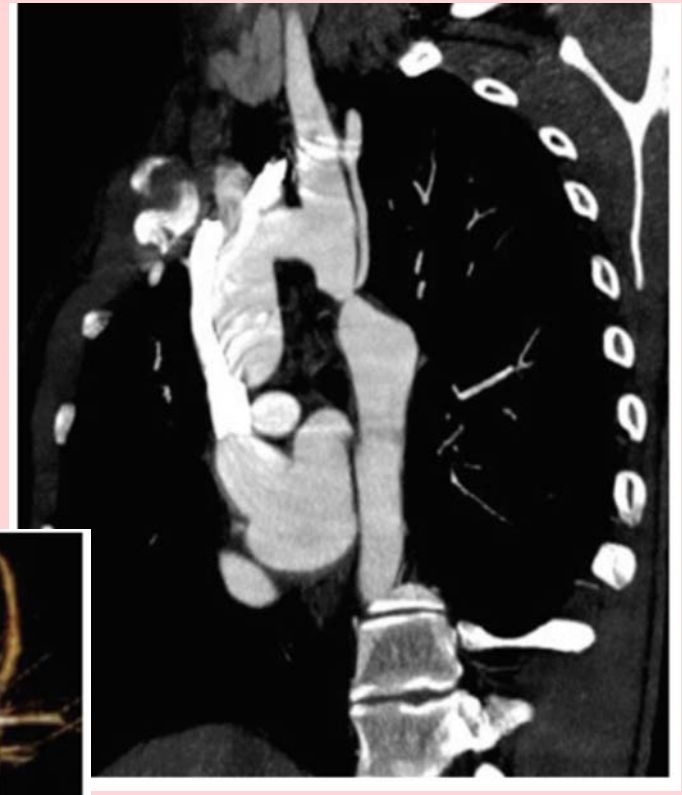

**Figure 3.** (A) 2D echo with color flow doppler showing severe narrowing of the proximal descending aorta with significant turbulence and a peak velocity of 4.8 m/s consistent with severe aortic coarctation. (B) Doppler tracing shows delay in return to baseline in diastole (diastolic drag) and blunting of the abdominal aortic doppler pattern consistent with significant aortic coarctation.

# Magnetic resonance imaging

- Characterize the aortic valve, aortic root, left ventricular size, and function.
- Superior visualization of the aortic arch with precise characterization of the location and extent of coarctation, and assessment of the presence and extent of collateral vessels.
- The measured minimum aortic cross-sectional area.
- Exceptional visualization of the aortic arch and detection of post repair complications including pseudoaneurysms.
- Assessment of post stenotic dilation or aneurysmal formation at the site of a previous repair.
- Left ventricular function and mass.

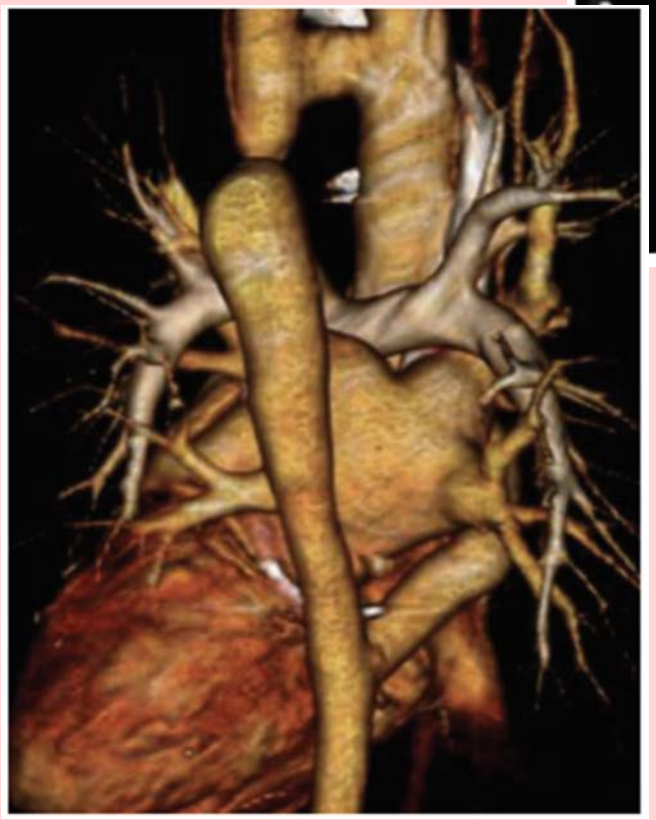


# Computed tomographic angiography (CTA)



## Benefits over MRI:

- Shorter scan time, and
- greater availability and
- less artifacts in transcatheter stents.
- Assess concomitant coronary anomalies
- Patients with pacemakers or implantable cardioverter defibrillators



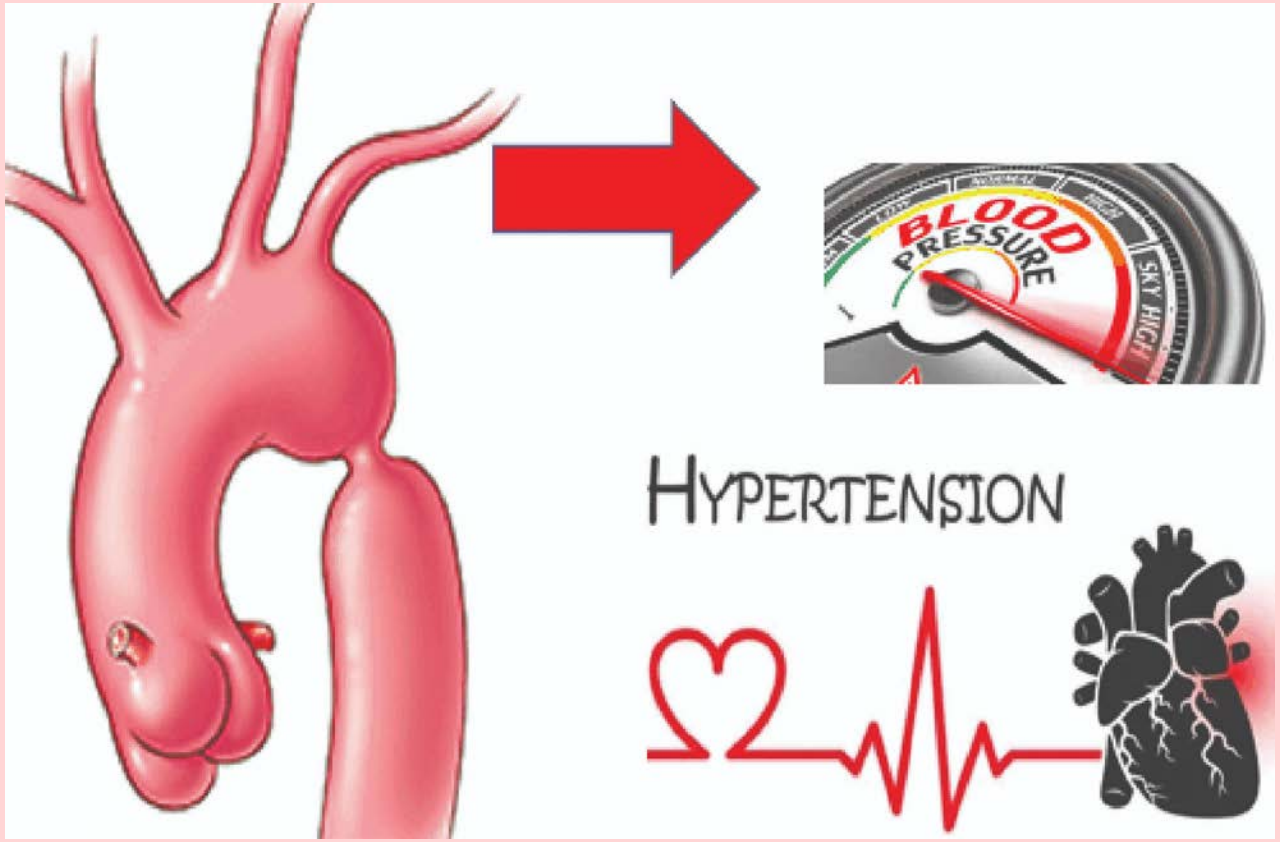
## Can evaluate:

- Coarctation segment,
- Aneurysmal dilation distal to the coarctation segment,
- Recoarctation post repair,
- Hypoplasia of the aortic arch,
- Follow serial aortic dimensions,
- Vascular anomalies such as double superior vena cava or aberrant great vessels,
- Collateral vessel formation.

# Coarctation of Aorta in adults

# &

# Systemic Hypertension



**Age at repair** is an **important determinant of developing late hypertension.**

**Infancy** ⟹ **less than 5% chance of developing hypertension**

**After the age of one** ⟹ **25–33% chance of developing hypertension**

# POTENTIAL MECHANISMS
# INVOLVED IN THE HYPERTENSIVE RESPONSE

# Endothelial dysfunction

***** Some adult studies generally recognize endothelial dysfunction as a consequence rather than a cause of hypertension.**

- **Reduced arterial compliance,**
- **Blunted baroreceptor sensitivity,**
- **Diffuse endothelial dysfunction is also likely to affect peripheral vascular resistance, which has the most profound effects on mean and diastolic blood pressure values rather than systolic values and pulse pressure that are commonly raised in hypertensive CoA patients.**

# Renin-angiotensin system

Early studies examining the impact
in CoA, concentrated on plasma ren
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Comparative Study > Hypertension. 2004 Feb;43(2):317-23.

doi: 10.1161/01.HYP.0000112030.79692.21. Epub 2004 Jan 19.

## Endogenous angiotensin and pressure modulate brain angiotensinogen and AT1A mRNA expression

Carine T Sangaleti [1], Alessandra Crescenzi, Lisete C Michelini

Affiliations + expand

PMID: 14732738 DOI: 10.1161/01.HYP.0000112030.79692.21

Sangaleti et al. have demonstrated that coarctation hypertension in the rat is associated with hyperactivity of the brain renin-angiotensin system as indicated by increased expression of angiotensin II type I receptors mRNA in brainstem areas, known to participate in cardiovascular control.

It is possible that these receptors are involved in the progression of hypertension in post-coarctectomy patients involving the cardiac baroreceptor.

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F B Parker Jr, B Farrell, D H Streeten, M S Blackman, H M Sondheimer, G H Anderson Jr

PMID: 6999245

# abnormal arterial structure and baroreceptor functioning:

**The authors examined fresh resected coarctation tissue and demonstrated:**

*** **Reduced isometric tension induced by potassium, noradrenaline and prostaglandin in the pre-stenotic aortic tissue compared with the post-stenotic area → indicating reduced contractility of the pre-stenotic aorta.**

*** **Increased collagen and reduced smooth muscle content of the pre-stenotic aortic wall.**

## Different reactivity and structure of the prestenotic and poststenotic aorta in human coarctation. Implications for baroreceptor function

J Sehested, U Baandrup, E Mikkelsen

PMID: 7074769 DOI: 10.1161/01.cir.65.6.1060

### Abstract

In eight humans with coarctation, fresh aortic tissue was examined pharmacodynamically. In four of these patients, and in 12 additional patients, the aorta above and below the coarctation was studied morphologically and compared with eight control aortas. By in vitro stimulation with potassium (127 mM), noradrenaline (18 microM), and prostaglandin F2 alpha (28 microM), postcoarctational aortic ring preparations showed a significantly greater contractility than precoarctational rings (p less than 0.05). Volumetric analysis showed significantly more collagen (P less than 0.01) and les smooth muscle mass (p less than 0.01) in the aorta above than below the coarctation. No significant differences were found between sections from the arch and distal to the ligamentum arteriosum in the normal aortas. We conclude that the precoarctational aortic wall is more rigid than the postcoarctational wall. This may influence baroreceptors in the upper vascular bed in such a way as to tolerate a higher pressure. This would explain the preoperative proximal hypertension, the paradoxic hypertension and the frequent lack of normalization of blood pressure postoperatively.

**Vogt et al. measured** local arterial stiffness indices and distensibility in the ascending and descending aortas of pre- and post-operative CoA neonates, and compared these values to matched controls.

**The same group was prospectively re-evaluated at 3 years of age, and persisting impairment of local elastic properties of the ascending aorta was noted in the CoA group, when compared with controls.**

Circulation
Volume 111, Issue 24, 21 June 2005; Pages 3269-3273
https://doi.org/10.1161/CIRCULATIONAHA.104.529792

PEDIATRIC CARDIOLOGY

## Impaired Elastic Properties of the Ascending Newborns Before and Early After Successful Repair

### Proof of a Systemic Vascular Disease of the Prestenotic Arteries?

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ORIGINAL ARTICLE

## Impaired Elastic Properties of the Ascending Aorta Persist Within the First 3 Years After Neonatal Coarctation Repair

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**One third (30-50%) of CoA patients still become hypertensive by adolescence despite early and effective surgical repair.**

**The mechanisms underlying arterial hypertension in corrected coarctation patients:**

- **Re-coarctation,**
- **Structural changes in the wall of peripheral and central vessels,**
- **Reduced baroreceptor sensitivity,**
- **Alterations in the renin–angiotensin system,**
- **Raised plasma concentrations of epinephrine and norepinephrine,**
- **Coexistence of essential hypertension or endothelial dysfunction.**

***It is conceivable that more than one of these systems may be involved.

# “selfish brain” hypothesis

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RESEARCH Open Access

Repaired coarctation of the aorta, persistent arterial hypertension and the selfish brain

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The hypothesis was that:

**VAH with ipCoW (VAH + ipCoW) would be more prevalent in the repaired CoA population developing arterial hypertension compared to normotensive controls and this would predict the development of hypertension after CoA repair**

vertebral artery hypoplasia (VAH)

incomplete posterior circle of Willis (ipCoW)

# Treatment

## Box 1 Indication for treatment of native coarctation of the aorta (CoA) and re-CoA

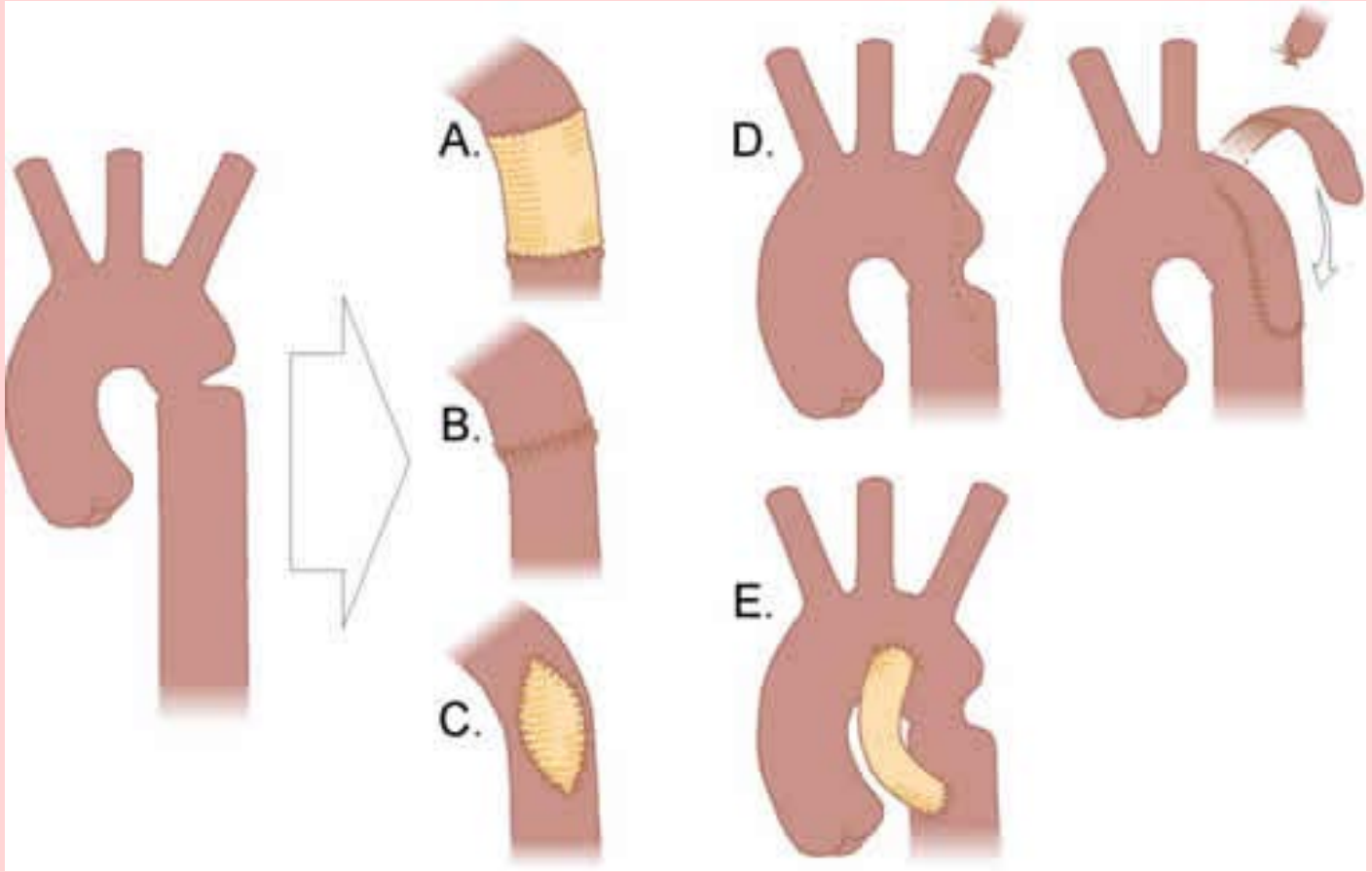
**Indications for treatment:**

- Supine non-invasive pressure gradient >20 mm Hg between upper and lower limbs
- Peak-to-peak coarctation gradient ≥20 mm Hg
- Peak to peak coarctation gradient <20 mm Hg with radiological evidence of significant coarctation with significant collateral flow
- Pathological blood pressure response during exercise
- Significant left ventricular hypertrophy
- Hypertension with ≥50% aortic narrowing relative to the aortic diameter at the level of the diaphragm
- Upper limb hypertension

# Surgical

## Standard procedures:

- Resection with end-to-end anastomosis in short, discrete CoA
- Subclavian flap aortoplasty in long-segment CoA
- Aortic bypass in long-segment CoA
- Prosthetic patch aortoplasty




## Complications

- ✓ Intraoperative spinal cord ischemia
- ✓ Long-term complications include:
- ✓ recurrent CoA (re-CoA) → 5 -10%
- ✓ aortic aneurysm

# Percutaneous intervention

- **Balloon angioplasty** is a percutaneous alternative to surgical repair for older infants and young children (greater than 4 months) with native discrete coarctation.

- However, **stent placement** has replaced balloon angioplasty as the procedure of choice in older children and adults with native coarctation with less recurrent narrowing.

*** Covered stents: less injury to the aortic wall.

## Complications:

- ✓ Unsuccessful intervention (residual pressure gradient ≥20 mmHg),
- ✓ Vascular access site complications (commonly, femoral artery occlusion),
- ✓ Re-CoA,
- ✓ Aortic aneurysm and dissection.

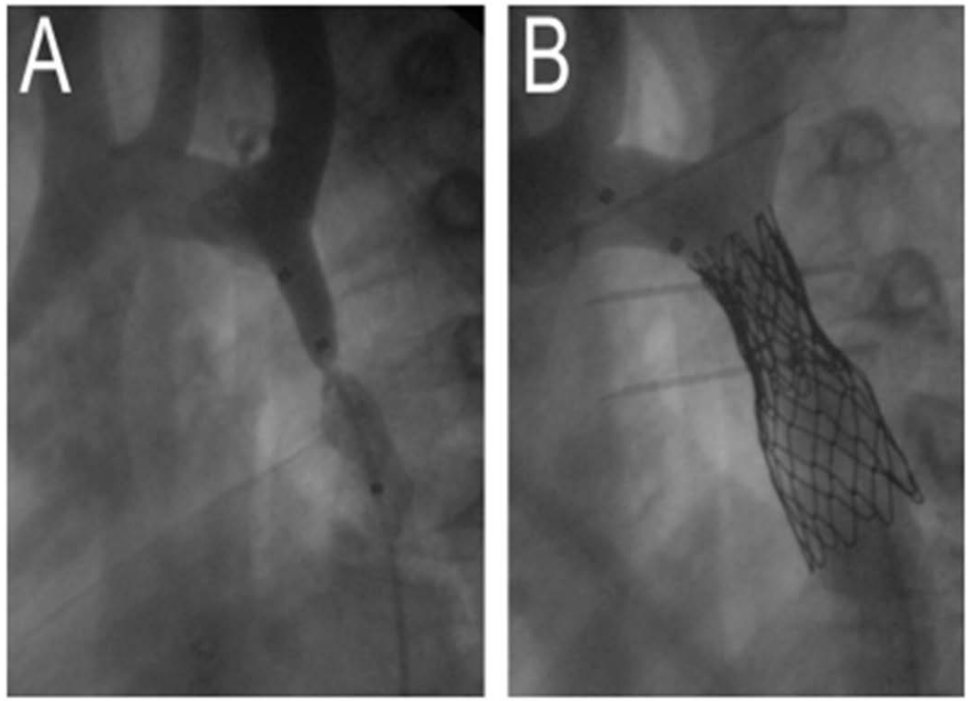



**Figure 4** (A) Sub-atretic coarctation of the aorta in a 5-month-old girl with a gradient of 50 mm Hg across the sub-atretic segment. (B) The aortic arch after stent implantation with complete reduction of the aortic gradient.

# Management of Hypertension in Co.A

- Persistent (unrepaired CoA or repaired after early childhood),
- Recurrent (re-CoA),
- Dynamic (exercise-induced, typically following repair at older age)

**Medical therapy for CoA does not modify the underlying disease process but nonetheless is important to forestall the development of cardiovascular sequelae including coronary artery disease, stroke, aortic aneurysm and dissection, and heart failure.**

# Exercise, recreational sports

**Standard lifestyle modification for hypertension** in children and adults with **both unrepaired and repaired Co.A:**

**weight control,**

**regular aerobic exercise,**

**low-fat and low-sodium diet,**

**smoking cessation,**

**and avoidance of alcohol.**

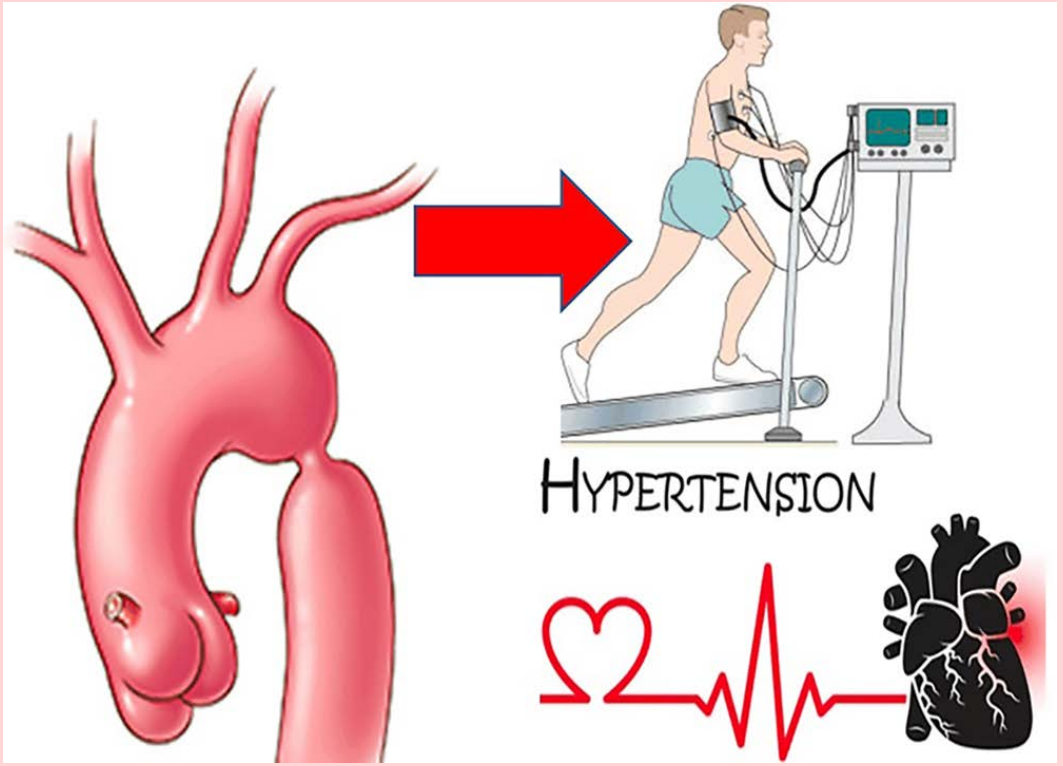
*** **High-intensity static (power lifting), heavy weight lifting, sudden stop-start, or isometric exercises and sports should be avoided** to reduce the risk of strain on the aorta that can lead to aneurysm formation or dissection.

# Competitive sports

Careful, case-by-case evaluation involving **exercise stress testing and echocardiogram**

## Participation in competitive sports without limitations:

- **Mild CoA (absence of significant pressure gradient and significant collateral vessels),**
- **Normal exercise stress test without hypertension (peak systolic blood pressure ≤230 mmHg),**
- **Small pressure gradient at rest (≤20-mmHg differential between upper and lower limbs).**




**Systemic or dynamic hypertension →** **should only engage in low-intensity competitive sports until repair**

**Following repair, athletes should be reevaluated with chest x-ray, electrocardiogram, exercise testing, echocardiogram, and MRI to re-stratify risk prior to engaging in competitive sports.**

## first-line treatments include:

- Beta blockers
- Angiotensin converting enzyme (ACE) inhibitors,
- Angiotensin-receptor blockers.

*** The specific choice of agent is patient-specific and must consider the patient's ascending aortic size (beta blocker preferable) and presence of aortic insufficiency (beta blockers not recommended).

*** Monitoring via 24-h ambulatory blood pressure measurement and exercise stress tests, to follow the rise in blood pressure with physical activity, are also used when considering initiating or uptitrating medical therapy.

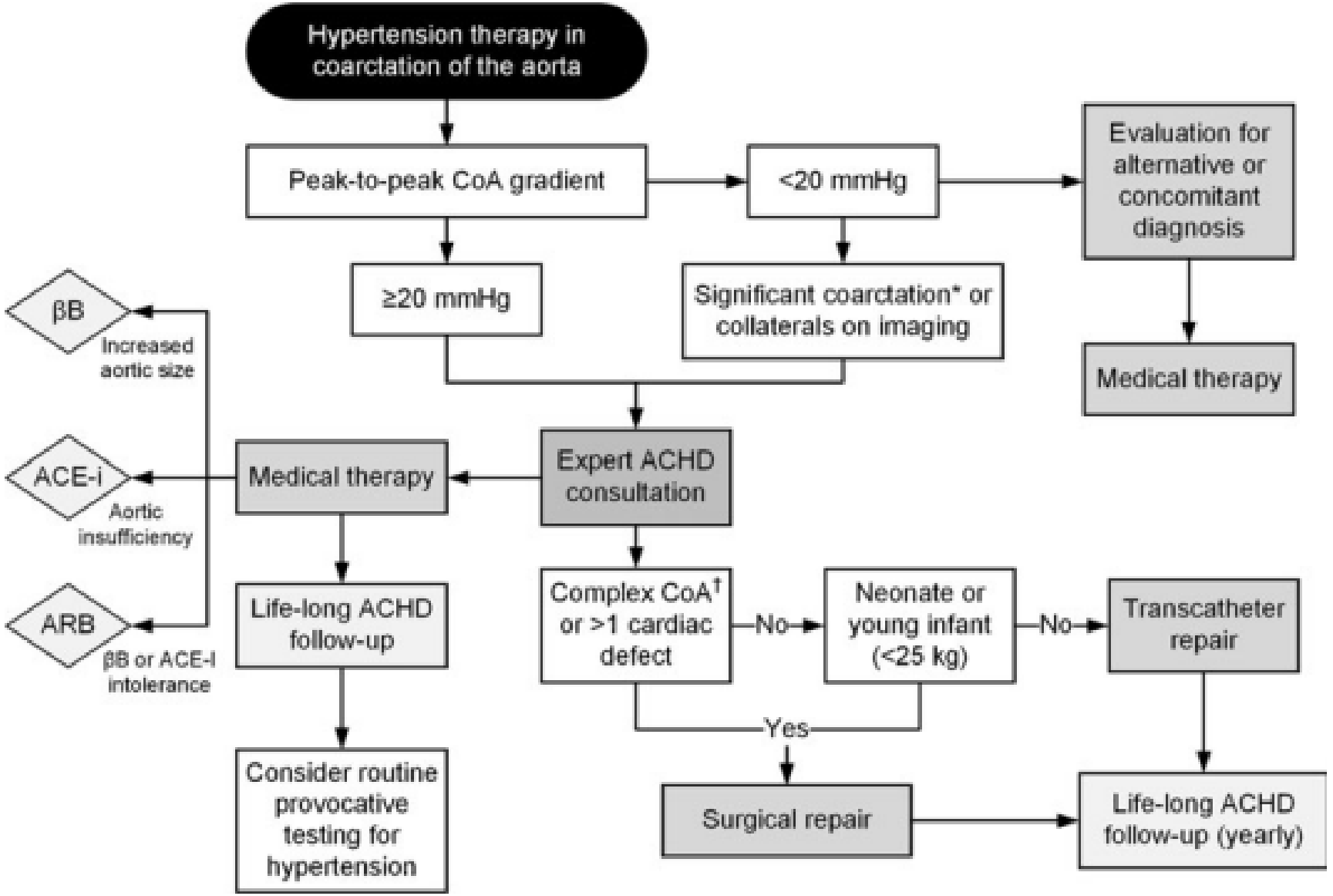



**Fig. 1.** Overview of the treatment of hypertension in the coarctation of the aorta (CoA). *ACHD* adult congenital heart disease, *ACE-i* angiotensin converting enzyme inhibitors, *ARB* angiotensin receptor blockers, *βB* beta blocker.

## Primary prevention of coronary disease

In addition to management of blood pressure,

**treatment of dyslipidemia with both lifestyle modifications, and, if necessary, drug therapy, is important.**

# Endocarditis prophylaxis

- Prior history of infective endocarditis,
- Implantation of prosthetic material, or
- Recent surgical or transcatheter repair in the past 6 months.

*** Uncomplicated CoA, whether unrepaired or repaired, does not require endocarditis prophylaxis.

# Pregnancy

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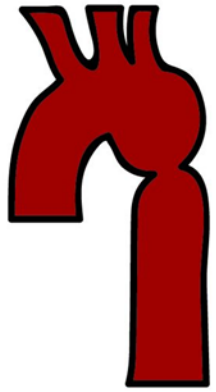
## Aortic coarctation in pregnancy

**303 pregnancies:**

- mean age 30 years
- median pregnancy duration 39 weeks
- 51% nulliparous
- 10% unrepaired CoA
- 27% arterial hypertension



**Pregnancy outcomes:**

- 0% maternal mortality
- 4.3% MACE (3.3% HF, 1% AF)
- 5.3% hypertensive disorders
- 50% caesarean section
- 1.6% fetal or neonatal mortality
- 6.6% low birth weight

## WHO II-III

Mild left ventricular impairment (EF>54%)

Native or tissue valve disease not considered WHO I or IV

Marfan or other HTAD syndrome without aortic dilatation

Aorta <45mm in bicuspid aortic valve

Repaired coarctation

AVSD

ecommended

Vascular Ehlers-Danlos

Severe (re)coarctation

Fontan with any complication

**Follow- up during pregnancy:** Monthly in expert centre

**Delivery**: Expert centre

ing pregnancy: Bimonthly in

t centre

APVR = anomalous pulmonary venous return, ASD = atrial septal defect, AVSD = atrioventricular septal defect, EF = ejection fraction, ESC = European Society of Cardiology, HTAD = hereditary thoracic aorta disease, PDA = persistent ductus arteriosus, VSD = ventricular septal defect, WHO = World health organization

Adapted and modified for congenital heart disease , from the ESC 2018 "Cardiovascular diseases during Pregnancy (management of) Guidelines" Table 3

## Expert cardiology follow-up

**Lifelong follow-up** with an expert in adult congenital heart disease is recommended for CoA, whether unrepaired or repaired.

**Systemic hypertension at rest or induced by exercise** should warrant evaluation for re-CoA.

# Imaging

**Given the likely intrinsic aortopathy associated with CoA, patients, whether unrepaired or repaired, should be monitored closely with CT/MRA performed at intervals of 5 years or less.**

# Thank You for Your Attention