



# Obstructive Sleep Apnea in Children

---

Manisha Witmans, MD, FRCPC, FAASM



STOLLERY  
CHILDREN'S  
HOSPITAL



UNIVERSITY OF  
ALBERTA

FACULTY OF MEDICINE  
& DENTISTRY



# Objectives

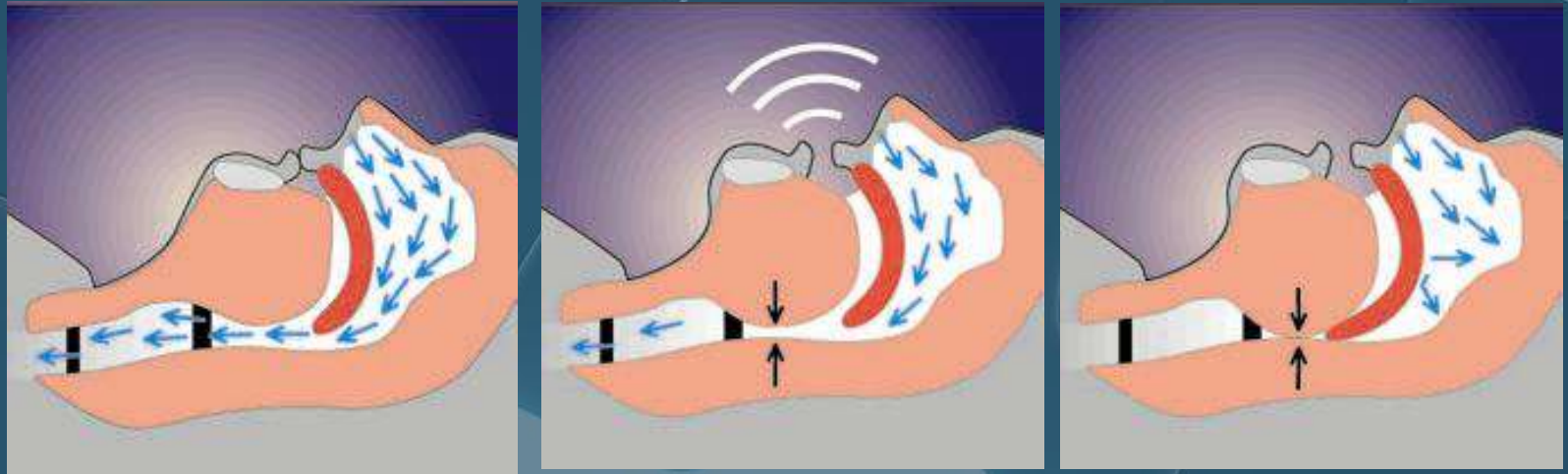
- To challenge the current paradigm of OSA
- Discuss the pathophysiological mechanisms of upper airway dysfunction
- Discuss end-organ dysfunction associated with OSA
- Discuss challenges in diagnosing OSA







# Is This All Sleep Disordered Breathing Is?



# Proposed Phenotypes of OSA

| Symptom              | Type 1 OSA | Type 2 OSA |
|----------------------|------------|------------|
| Daytime Sleepiness   | +          | + + + +    |
| Weight gain/Obesity  | +          | + + + +    |
| Hyperactivity        | + + + +    | -          |
| Lymphoid hyperplasia | + + + +    | + +        |
| Hypertension         | +          | + + + +    |
| LV Dysfunction       | +          | + + + +    |
| Insulin Resistance   | -          | + + + +    |
| Psychiatric Problems | +          | + + +      |

Other proposed types:  
Craniofacial  
Neuromuscular



**Primary or Secondary**

Sensory  
impairment

Neuro-motor  
dysfunction

Upper airway  
dysfunction

Inflammation

Structural  
alteration

**Centrally mediated**

- Hypotonia\*
- Hypertonia\*
- Brainstem dysfunction/compression
- Cervical spinal cord lesion

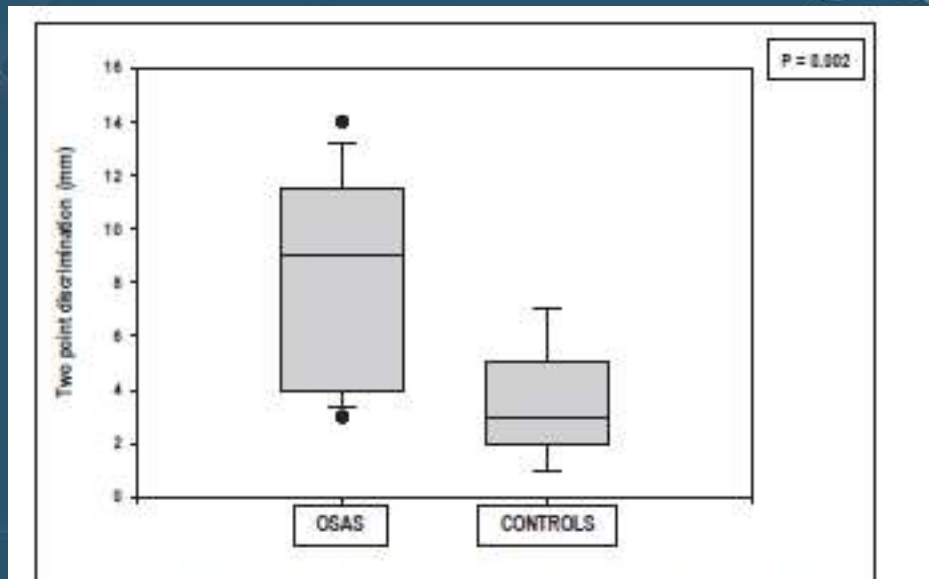
**Peripherally mediated**

- Cranial nerve injury (XII)
- Vocal cord paralysis

→ SDB

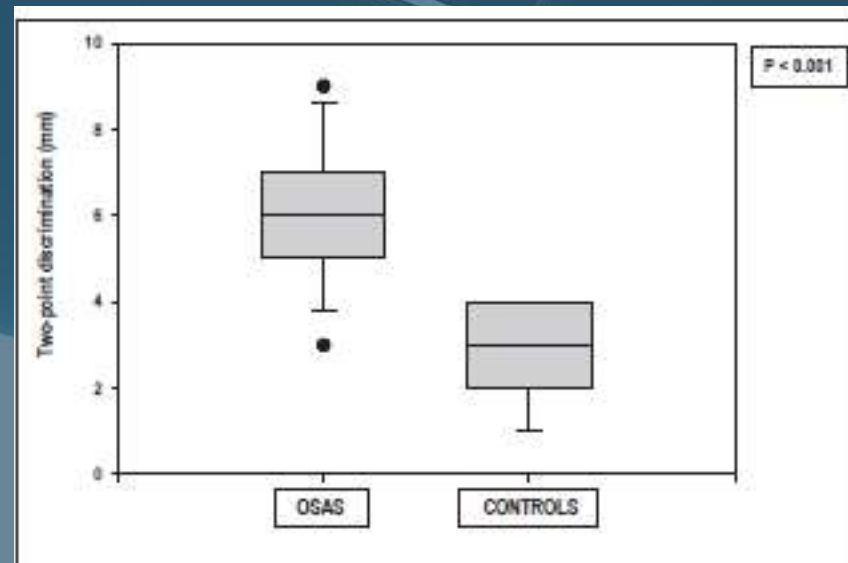
# Sensory Impairment

- Cause or effect?
- Older children



Tongue

Tapia, 2010, Sleep



Anterior Palate



# Hypotonia and SDB







# Endoscopic data

## *Obstruction*

- Deviated nasal septum
- Chronic rhinitis
- Adenoidal
- Tonsillar

## *Collapse*

- Circumferential pharyngeal
- Lateral pharyngeal
- Laryngeal
- Tongue base



# Summary for DS

- Down syndrome children exhibit a collapsing pattern more than other children with SDB
- Lingual collapse is significant but not universal
- Neuro-motor dysfunction overrides structural alterations in this group
- SNP may direct surgery and help avoid unnecessary adeno-tonsillectomies
  
- Fung et al. 2012, Archives of Otolaryngology



# The United Airways Disease

Asthma

Atopy

Obstructive  
Sleep  
Apnea

Common genetic and environmental risk factors



# Inflammation

- SDB
  - Inflammatory markers identified
    - CRP, oxidative species, cytokines, eNO
  - Evidence that treating inflammation improves SDB

## Primary or Secondary

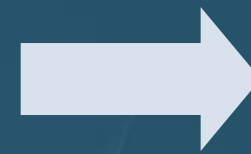
Sensory  
impairment

Neuro-motor  
dysfunction

Upper airway  
dysfunction

Inflammation

Structural  
alteration



# SDB

### Nose

- Deviated septum
- Rhinitis (allergic or non-allergic)
- Choanal atresia
- Nasal polyps

### Nasopharynx

- Midfacial hypoplasia
- Adenoidal hypertrophy\*

### Oropharynx

- Tonsillar hypertrophy\*
- Macroglossia
- Retro/micrognathia
- Infiltration by mucopolysaccharides (Hunter/Hurler syndromes)
- Obesity
- S/P burns
- Acute skull base angle
- Pharyngeal flap
- High arched palate

### Hypopharynx

- Laryngotracheomalacia
- Vocal cord paralysis
- Vascular ring
- Subglottic stenosis
- Hemangioma
- Neurofibroma





# Obesity

- Risk factor for OSAS (OR 4.5)
- Obesity more prevalent in studies evaluating SDB
- Surgery (T&A) does not cure majority of those with OSAS

Redline, AJRCCM, 2005

Bhattacharjee, AJRCCM, 2010

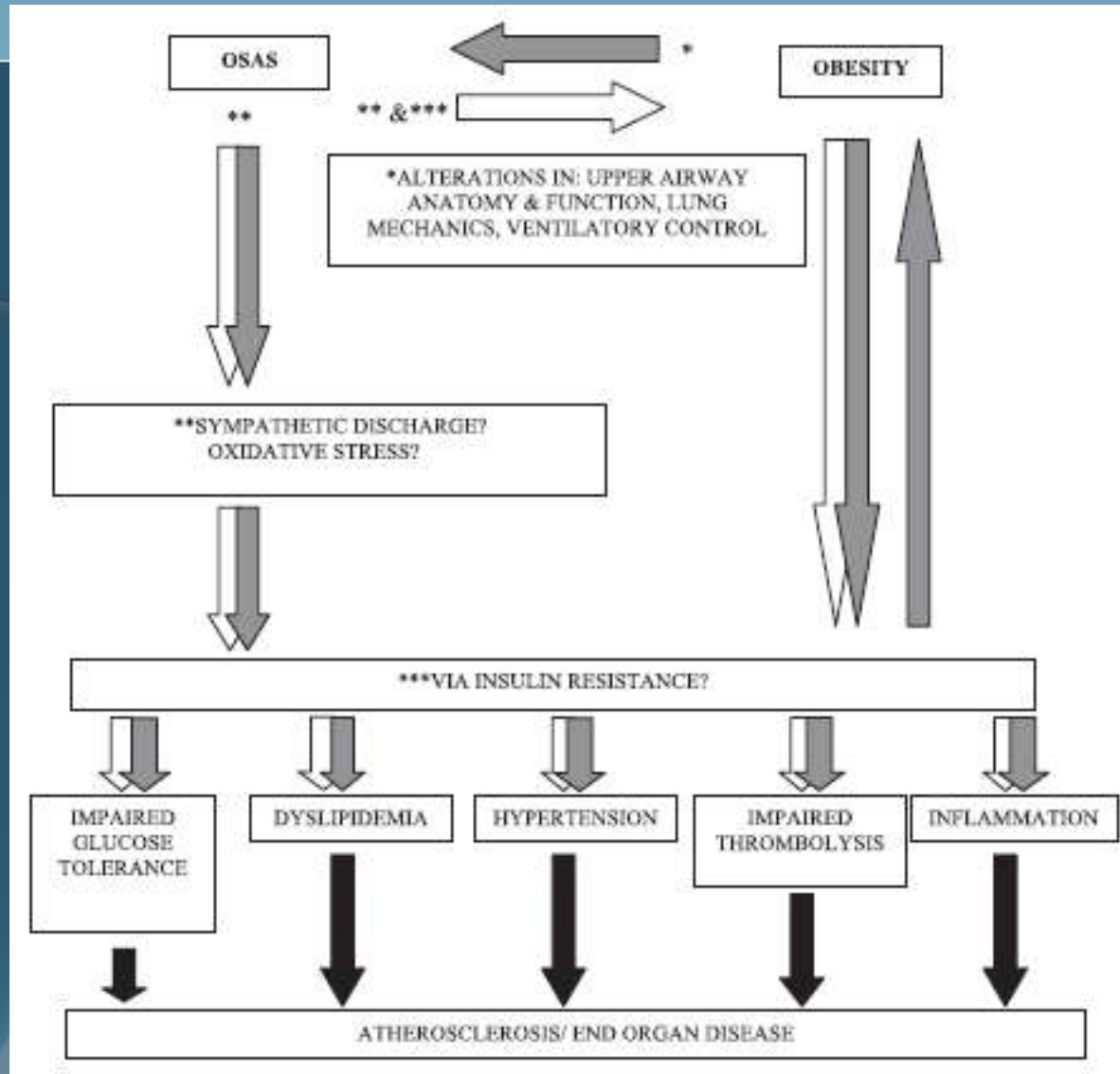
Mitchell, OtoHNS, 2004



# Obesity

- Pathophysiology:
  - Anatomic factors
    - Hormonal, inflammatory
    - Possible soft tissues restricting airway
    - Craniofacial structure
  - Functional factors
    - Increased airway collapsibility
      - Neuromotor tone, increased resistance
      - ? Possible higher Pcrit
  - Chest wall mechanics
  - Altered ventilatory responses

# Childhood Obesity and SDB



Arens,  
2010, J  
Appl Phys.



# End Organ Dysfunction

- Metabolic Derangements
- Cardiovascular dysfunction
- Neurocognitive dysfunction



# SDB and Metabolic Syndrome

- Adjusting for sex, age, race and prematurity, adolescents with OSA
  - Adjusted OR of Met S: 6.49 ( 95% CI, 2.52, 16.70)
  - Degree of AHI and lower minimum SaO<sub>2</sub> increased odds of Met S
- Adjusting for BMI and sex, OSA predisposed to
  - Increased insulin resistance
  - Higher BP
  - Higher LDL

Redline, 2007, AJRCCM





# End Organ Dysfunction

- **Cardiovascular morbidity**
  - Blood pressure regulation
  - Cardiac function
    - End diastolic dysfunction
    - Left ventricular remodelling
  - Endothelial functioning
    - CRP, myeoid related protein 8/14

Amin. AJRCCM, 2002; Amin. Hypertension, 2008

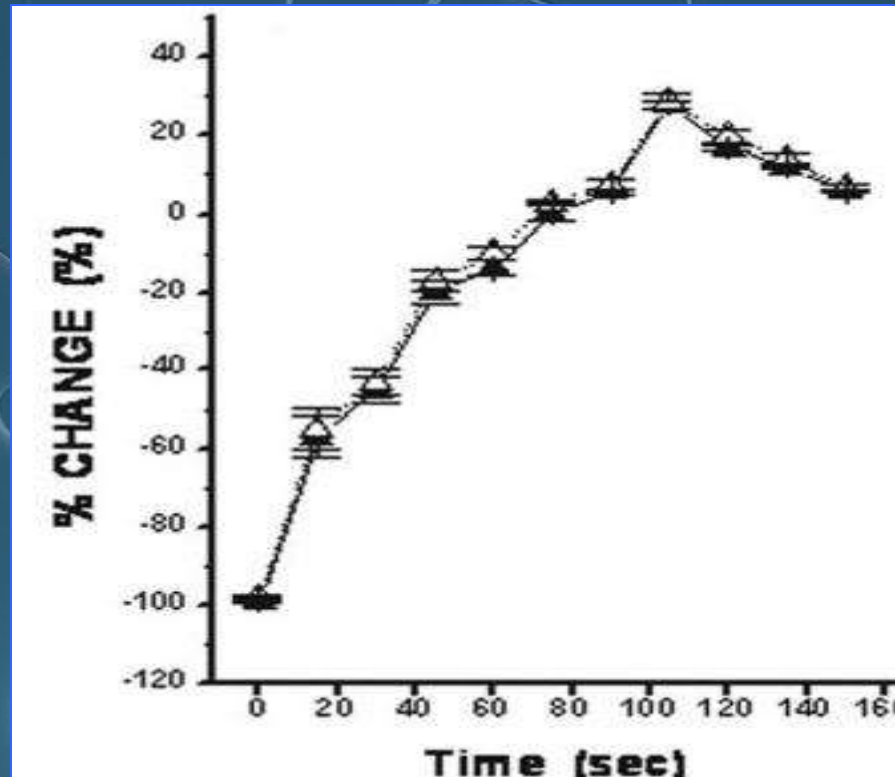
Wang, J Am Coll Cardiol, 2007

Bhattacharjee et al, Prog CV Disease, 2009; Bhattacharjee

Circulation, 2007; Bhattacharjee, Sleep, 2010

# End Organ Dysfunction

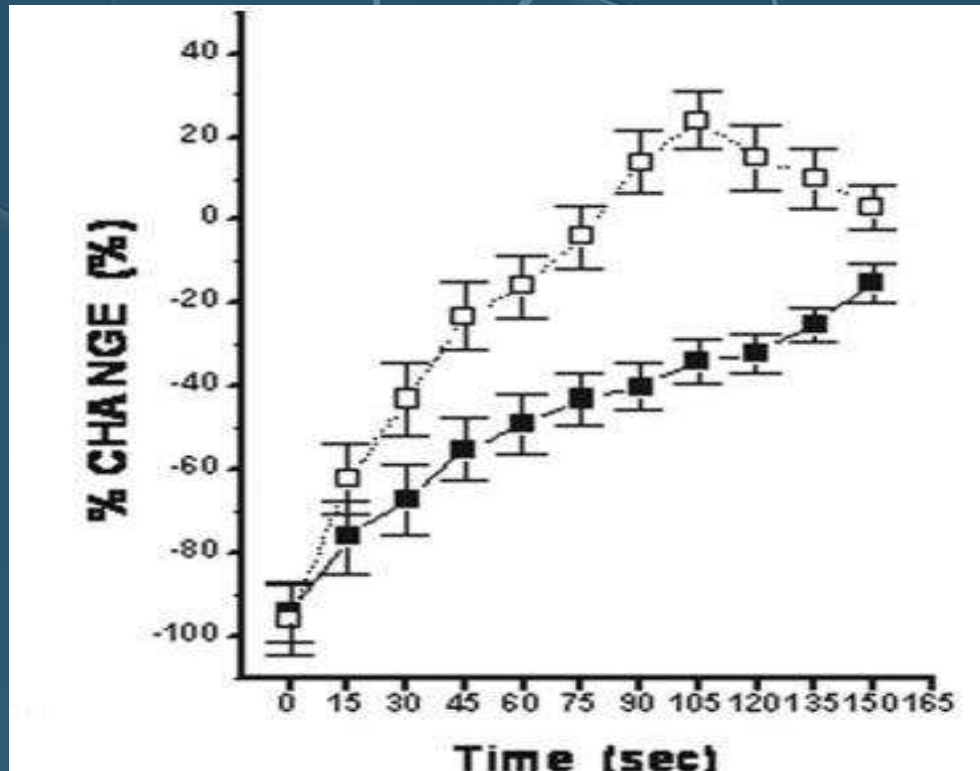
controls



n=8

- Time to reach baseline cutaneous flow was (mean) 69 seconds

# Pre and Post Adenotonsillectomy

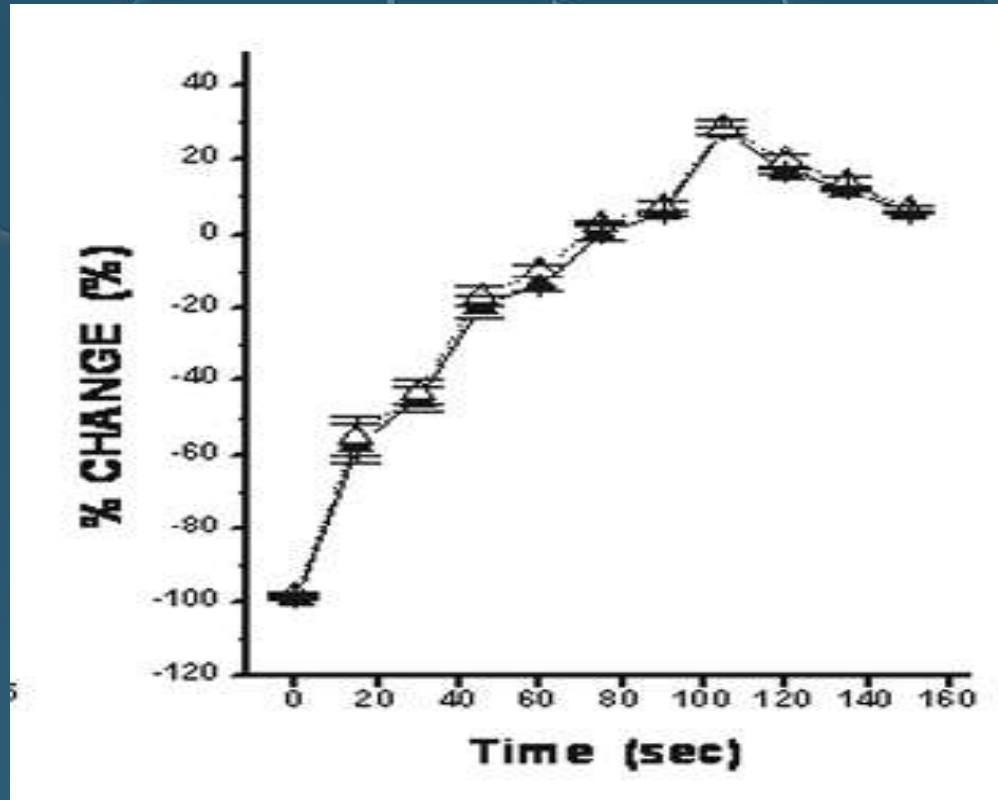


n=26

- Baseline cutaneous flow pre T+A was > 113 seconds
- **Normalized** to 60-80 seconds post surgery

# Pre and Post Adenotonsillectomy

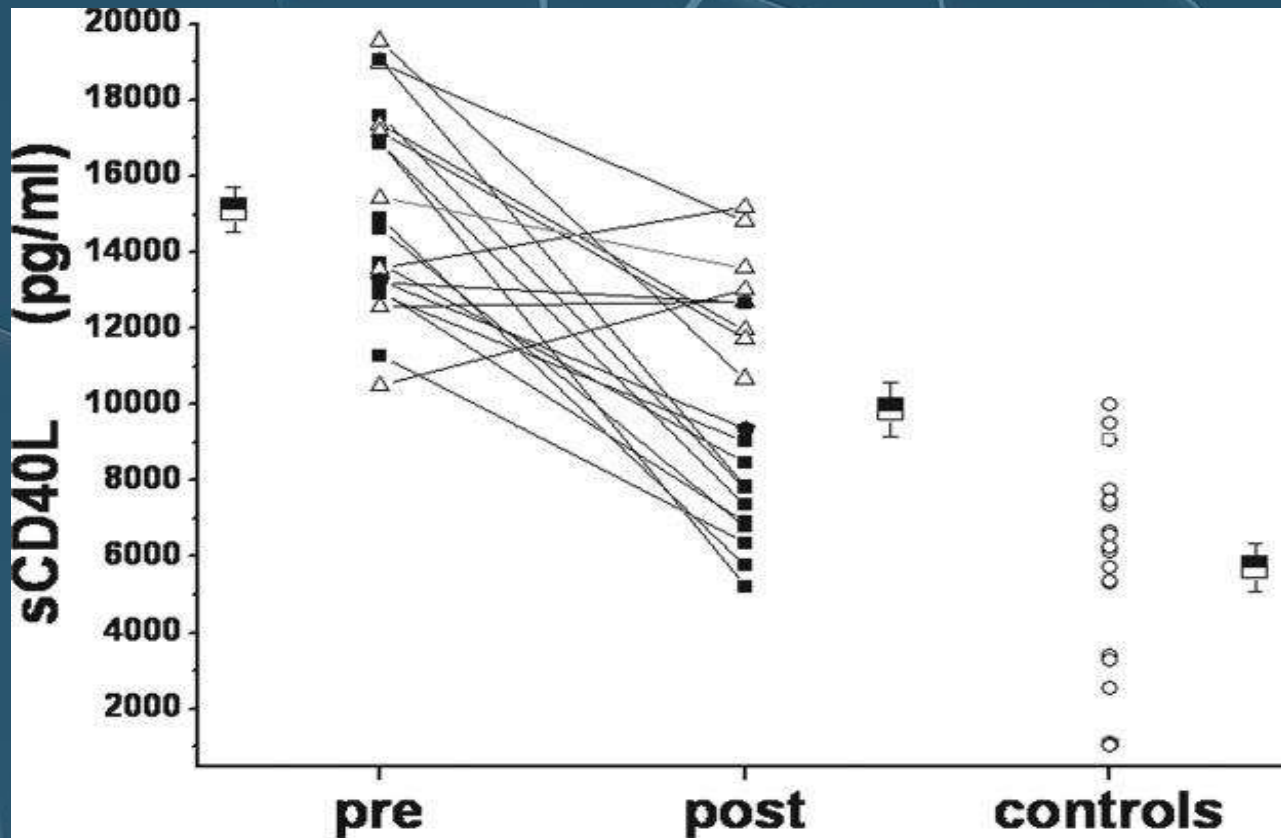
## Family history of CVS Disease



n=6

- Baseline cutaneous flow pre T+A was > 113 seconds
- Did not normalise post surgery

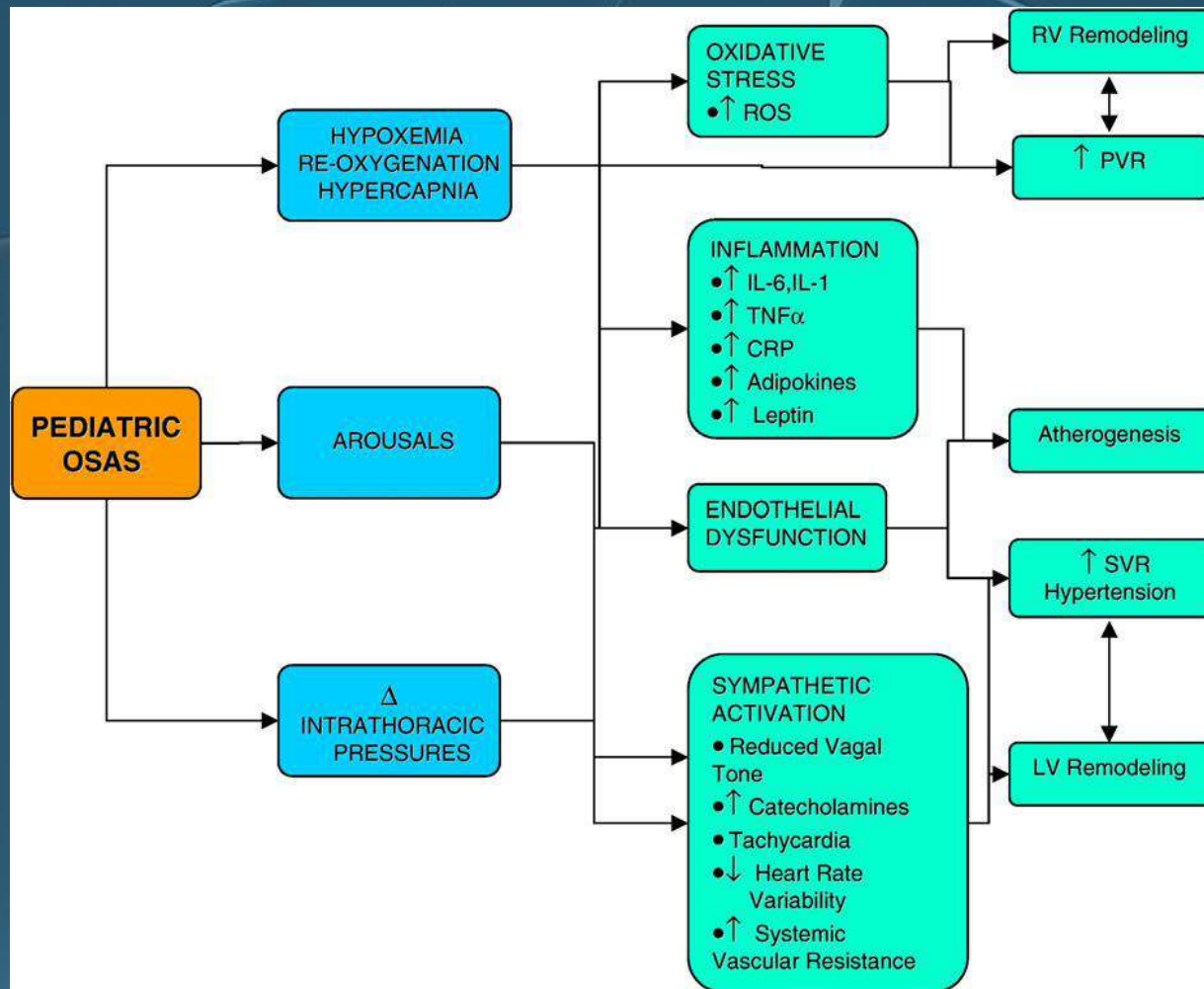
# Inflammatory Marker: sCD40L



- sCD40L high in OSA but significantly reduced post T+A but still higher than controls



# CV Dysfunction and SDB





# OSA and Neurocognition

- Link between habitual snorers with OSA and neurocognition
- Link between habitual snorers without OSA and neurocognition
- Increased risks for neurobehavioural deficits in the context of OSA are
  - Obesity
  - Hypoxemia and sleep fragmentation

**Beebe DW, Sleep 2006**

**Gruber R, Sleep 2007**



# OSA and Neurocognition

| <b>Variable</b>               | <b>No-snoring<br/>(n=87)</b> | <b>No-OSA<br/>(n=112)</b> | <b>OSA<br/>(n=146)</b> |
|-------------------------------|------------------------------|---------------------------|------------------------|
| Age                           | 6.4 ± 0.3                    | 6.4 ± 0.2                 | 6.3 ± 0.3              |
| BMI                           | 16.7 ± .04                   | 16.9 ± 0.5                | 17.0 ± 0.4             |
| AHI                           | 0.0 ± 0.0                    | 0.8 ± 0.3                 | 8.6 ± 2.2 *            |
| Minimum SaO <sub>2</sub>      | 93.1 ± 0.6                   | 90.6 ± 0.7                | 81.6 ± 2.7 *           |
| 2 abnormal NC tests<br>no (%) | 0                            | 3 (2)                     | 16 (11) *              |
| APOE e4 no (%)                | 0                            | 16 (14)                   | 72 (49) *              |

- APOEe4 allele increased among children with cognitive deficits and OSA



# Summary

- OSA is common and can affect many children with physical and neurocognitive consequences
- OSA can be related to adenotonsillar hypertrophy but that is not the only factor to consider
- Evaluating factors that might contribute to the OSA will help target appropriate treatment