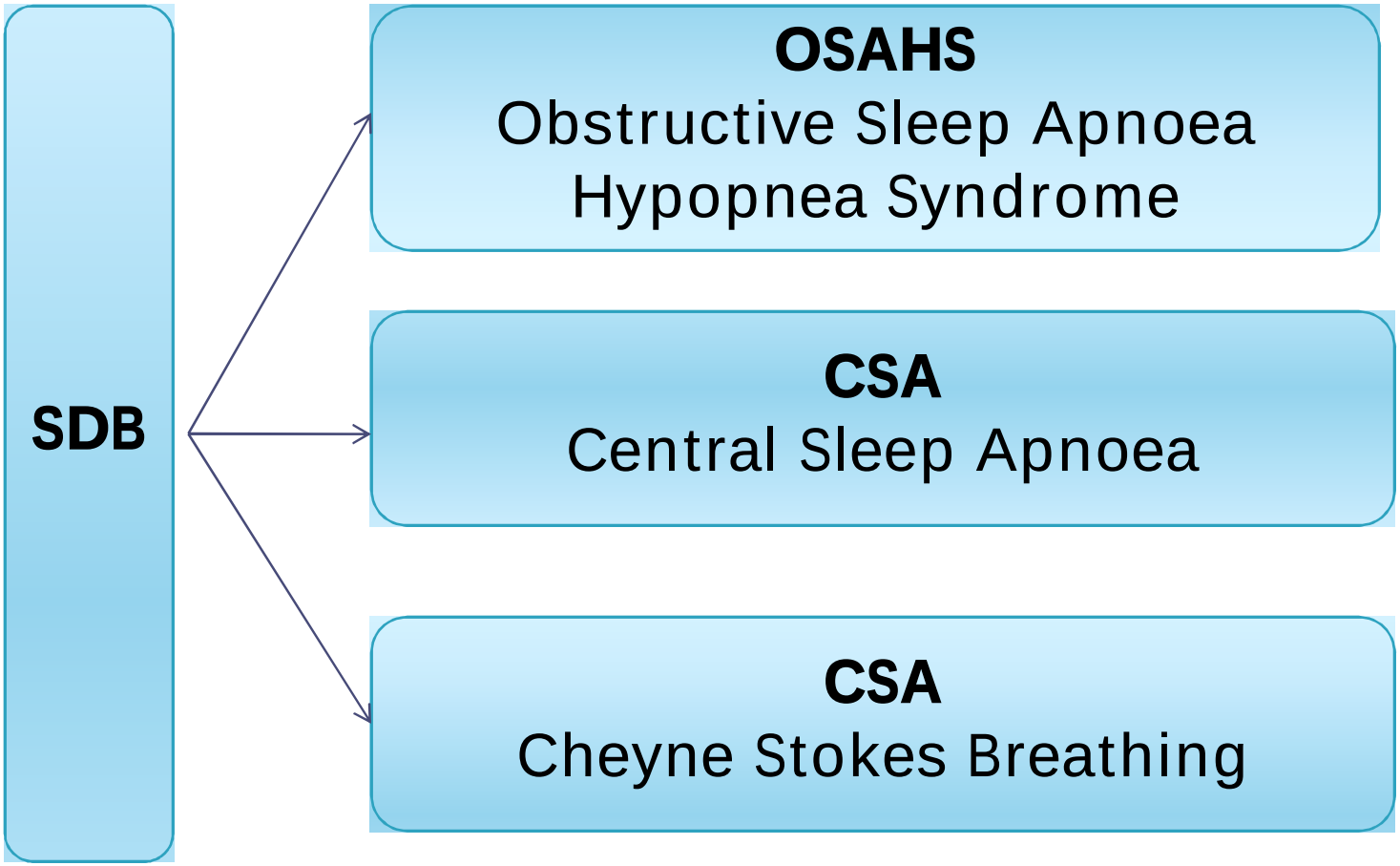
Apollo BRAMWELL HOSPITAL
Medical Excellence

Organization Accredited by Joint Commission International

Wednesday, 2nd October 2013
Burrenchobay Hall
University of Mauritius

- OSA = Obstructive Sleep Apnoea
- CSA = Central Sleep Apnoea
- CSR = Cheyne Stokes Respiration
- SDB = Sleep Disordered Breathing
- EDS = Excessive daytime somnolence
- AHI = Apnoea Hypopnoea Index
- AF = Atrial Fibrillation
- HF = Heart Failure
- PSG = Polysomnography
- CPAP = Continuous Positive Airways Pressure
- ASV = Adaptive Servo Ventilation


SDB
OSAHS
Obstructive Sleep Apnoea
Hypopnea Syndrome
CSA
Central Sleep Apnoea
CSA
Cheyne Stokes Breathing


Frank

- Obese (80%)
- Snorer (± witnessed apnoeas)
- Excessive daytime somnolence (various questionnaires)
- Morning fatigue (unrefreshed)
- Females – Different presentations

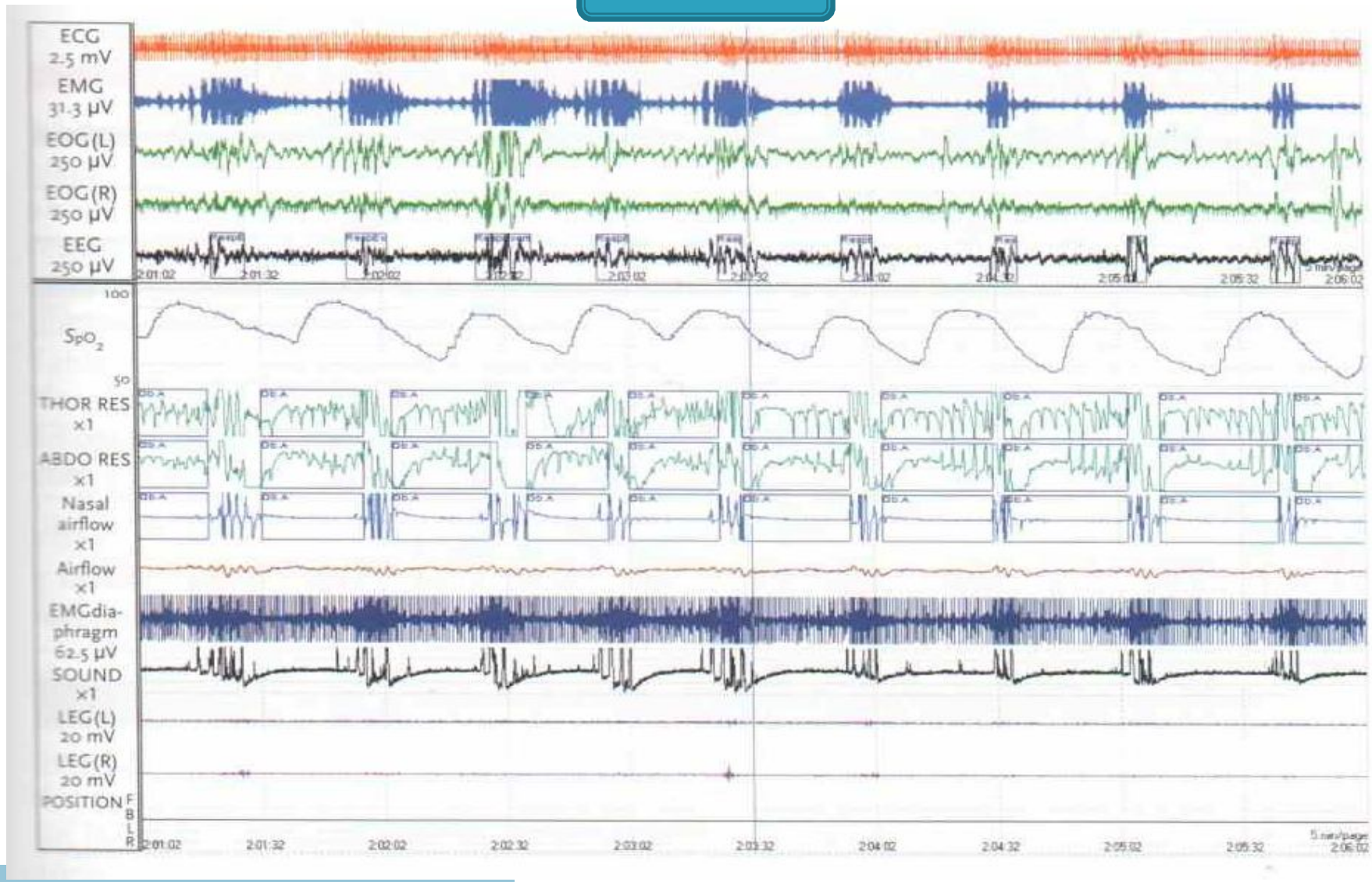
- Obese
- Fat neck (>42cm)
- Mallampati (>2)
- ↑ Waist size (>100cm)

NB: Thin Type (craniofacial)

Site of airway narrowing in patients with the sleep apnoea/ hypopnoea syndrome

- Polygraphy (Sp02, nasal airflow, chest/ abdominal movements)
- Polysomnography (above +EEG, EMG, EOG)
- Simple overnight oximetry (only cases with very high pre test probability)

OSA


ECG
2.5 mV
EMG
31.3 µV
EOG(L)
250 µV
EOG(R)
250 µV
EEG
250 µV
100
SpO2
50
THOR RES
x1
ABDO RES
x1
Nasal
airflow
x1
Airflow
x1
EMGdia-
phragm
62.5 µV
SOUND
x1
LEG(L)
20 mV
LEG(R)
20 mV
POSITION
F
B
L
R
2:01:02
2:01:32
2:02:02
2:02:32
2:03:02
2:03:32
2:04:02
2:04:32
2:05:02
2:05:32
2:06:02

WEINMANN

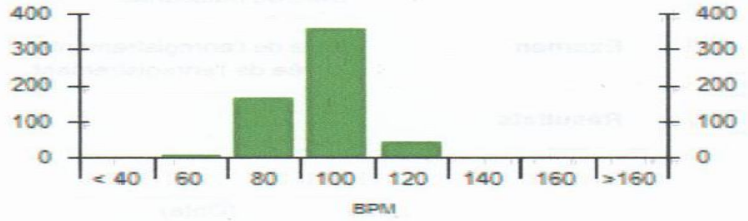
Ronflements

Durée totale des ronflements (PA)	01:53:24 h	Pourcentage des ronflements (PA)	19.9 %

FC/FP

Fréquence cardiaque moyenne (PA)	84.6 bpm
Nombre de variations de la FC (PA)	252
Pourcentage de la FC inférieure à 50 bpm	0.0 %
Pourcentage FC supérieure à 100 bpm	5.4 %

Diagramme de la distribution de la FC



Position

Diagramme de la position

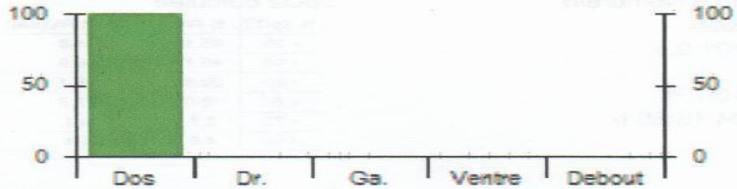
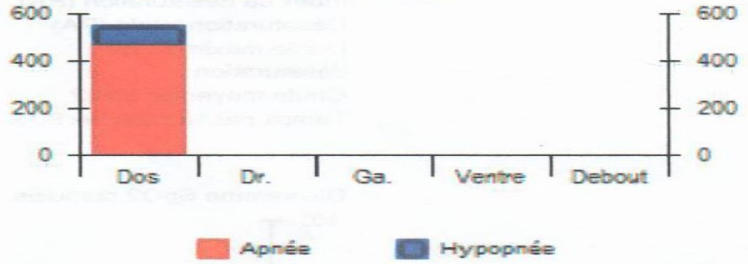
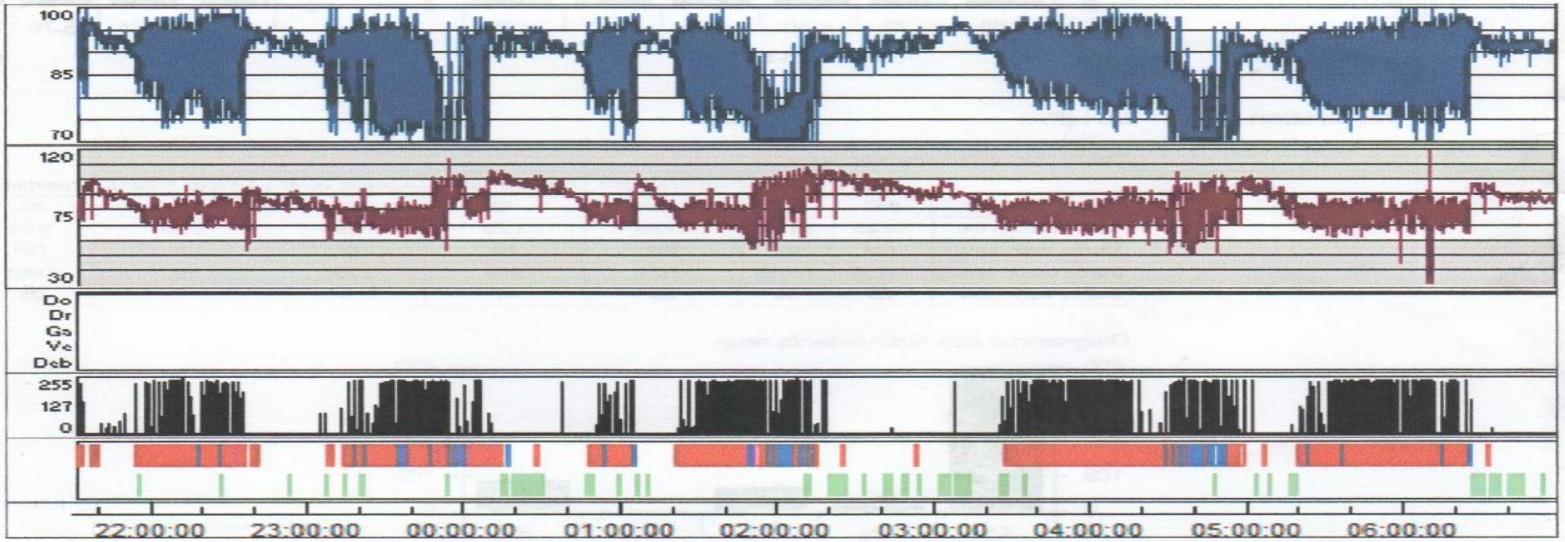


Diagramme événements resp. - position



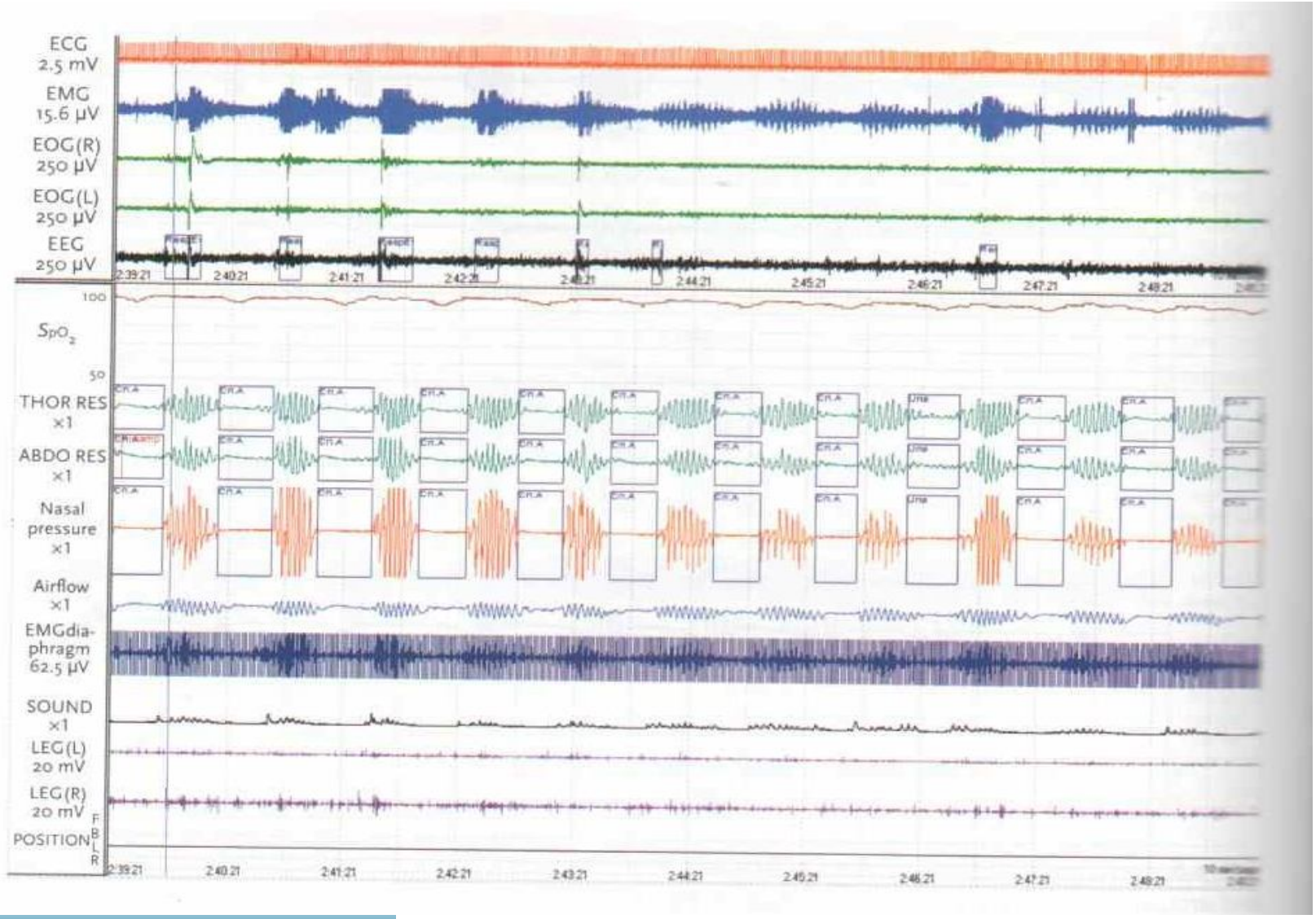
Nombre de changements de position -

Vues d'ensemble



- Severe heart failure
- CVA
- Renal failure
- Opiates

CSA


ECG
2.5 mV
EMG
15.6 µV
EOG(R)
250 µV
EOG(L)
250 µV
EEG
250 µV
100
SpO2
50
THOR RES
×1
ABDO RES
×1
Nasal
pressure
×1
Airflow
×1
EMGdia-
phragm
62.5 µV
SOUND
×1
LEG(L)
20 mV
LEG(R)
20 mV
POSITION
F
B
L
R
2:39:21
2:40:21
2:41:21
2:42:21
2:43:21
2:44:21
2:45:21
2:46:21
2:47:21
2:48:21

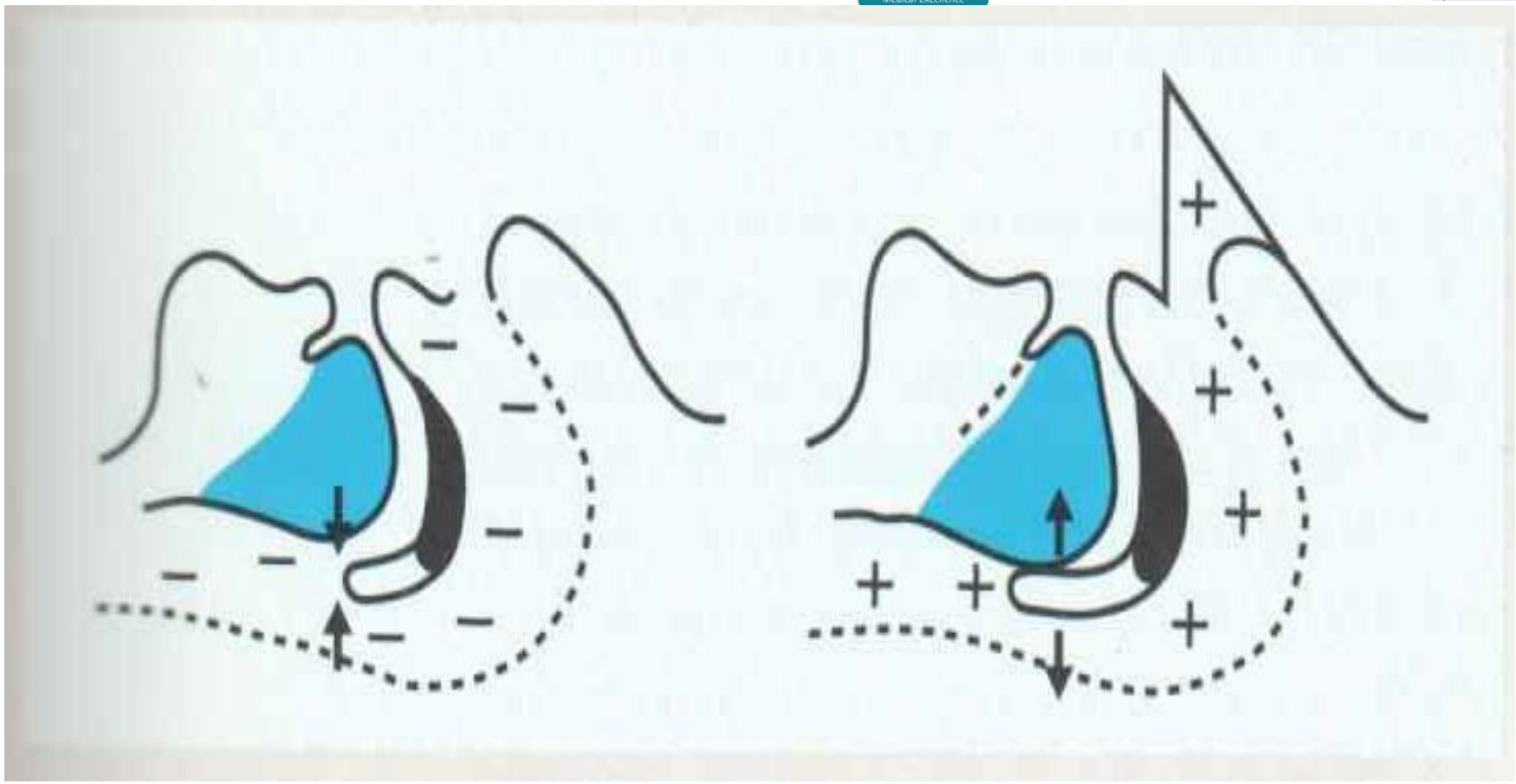
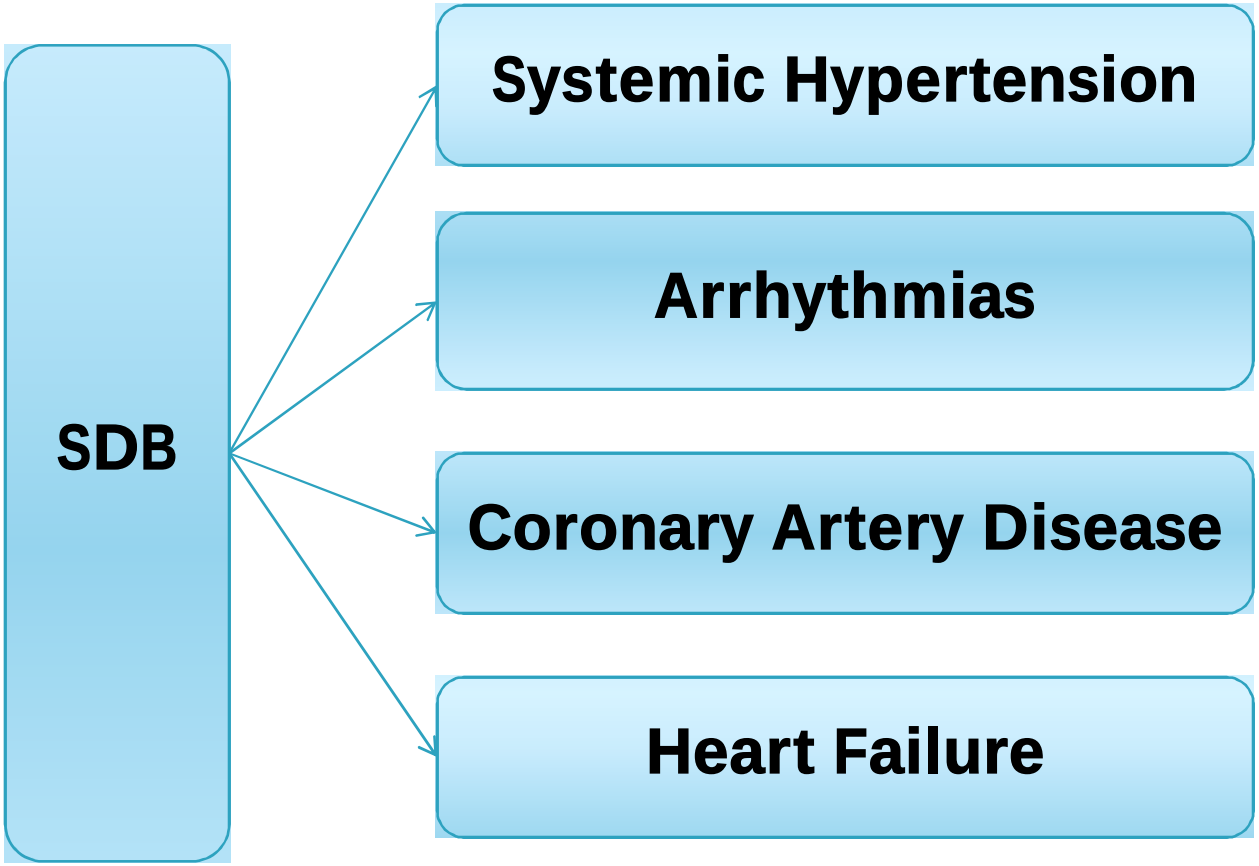


Diagram of pressure within the upper airway, during spontaneous inspiration, showing the tendency for the upper airway to narrow (left), and the effect of treatment with CPAP, which keeps the airway widely patient (right)

Apollo BRAMWELL HOSPITAL
Medical Excellence

Organization Accredited by Joint Commission International

[redacted]


SDB
Systemic Hypertension
Arrhythmias
Coronary Artery Disease
Heart Failure

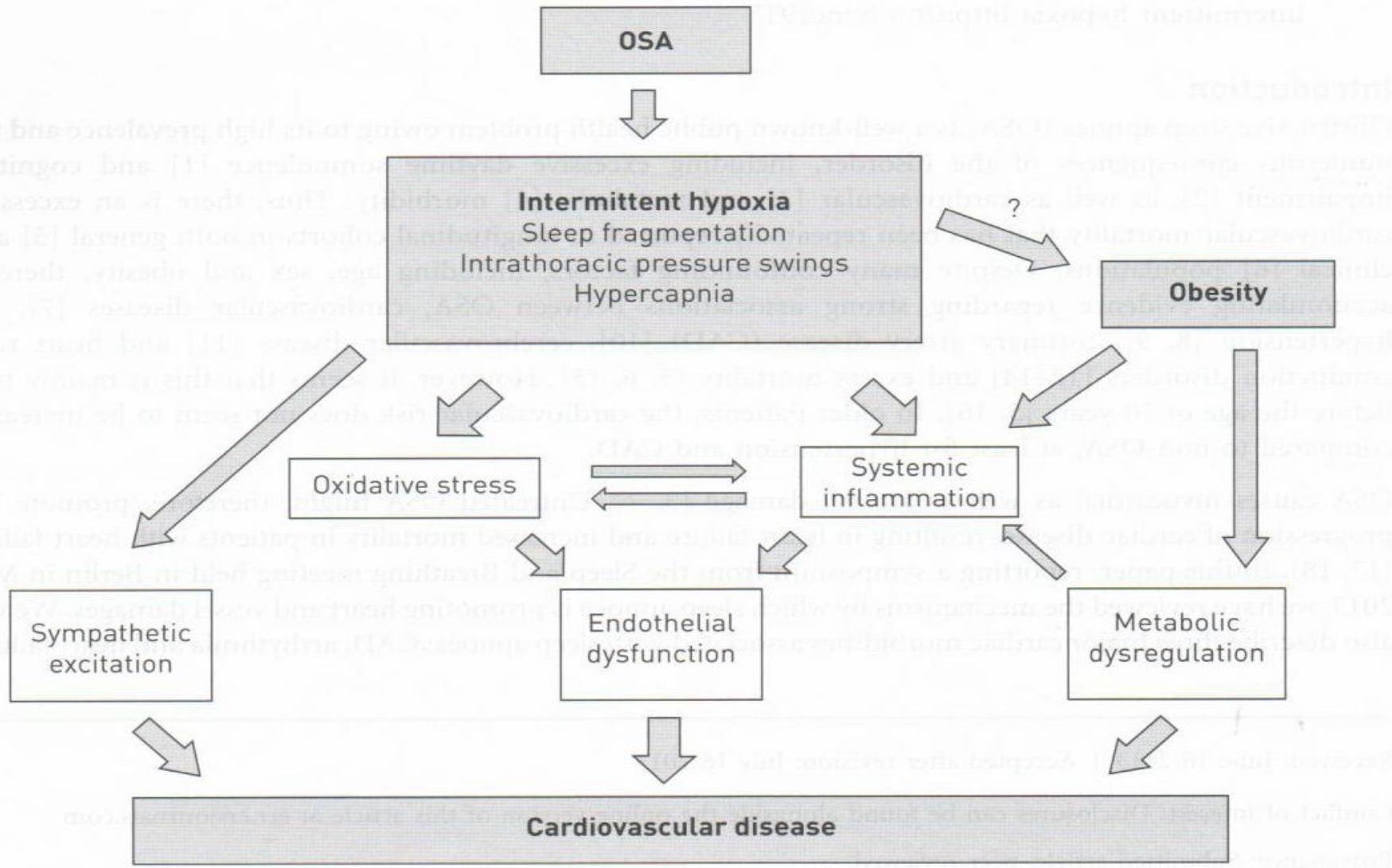


FIGURE 1 Main mechanisms linking obstructive sleep apnoea (OSA), intermittent hypoxia and cardiovascular diseases.

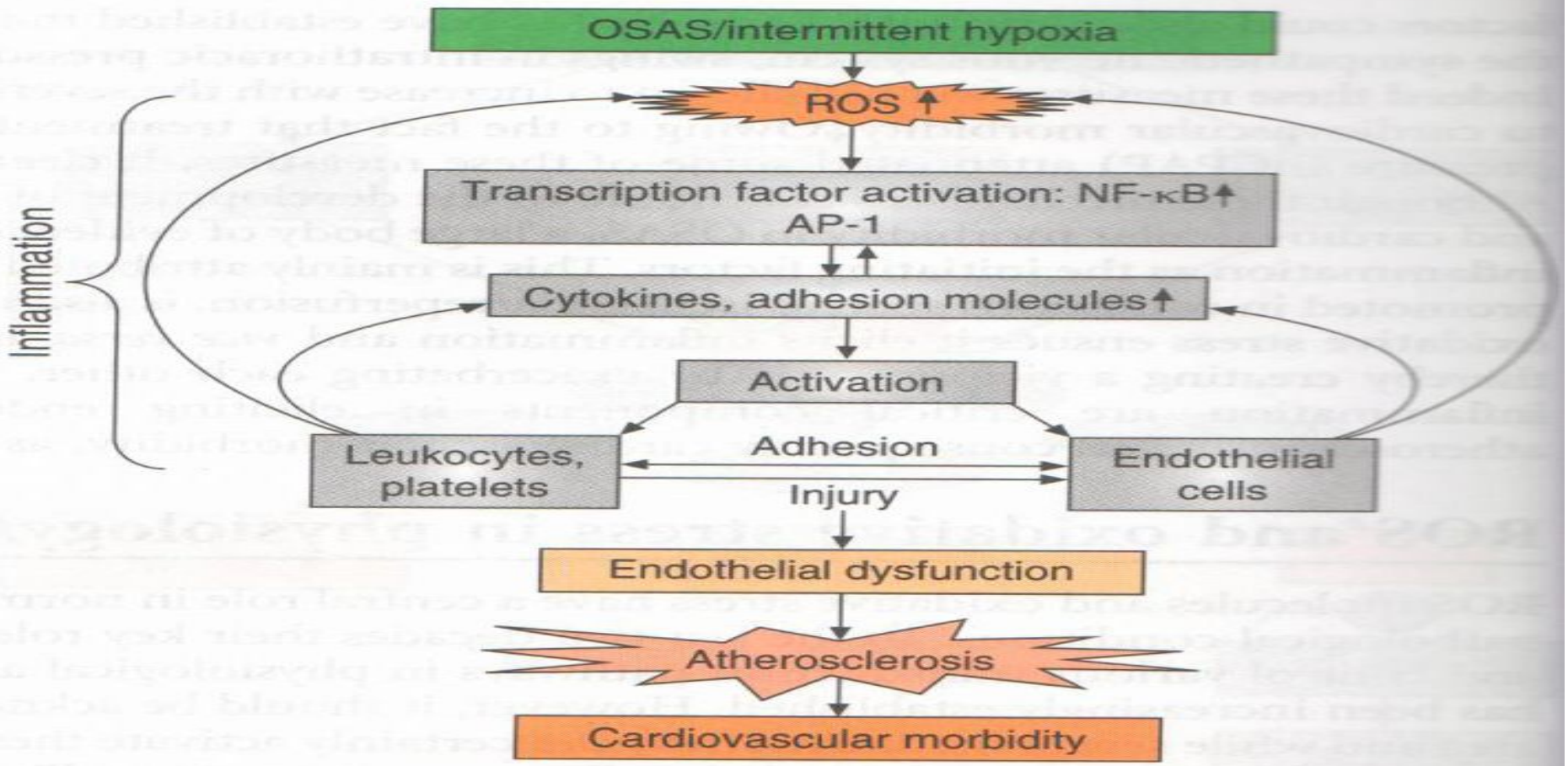


Figure 1. Oxidative stress and inflammation in obstructive sleep apnoea syndrome (OSAS). The intermittent hypoxia induces reactive oxygen species (ROS) formation, which in turn activate an inflammatory cascade *via* activation of transcription factors and downstream genes as inflammatory cytokines and adhesion molecules. These in turn can further activate transcription factors and various blood cells. Activated leukocytes and platelets produce higher amounts of ROS, adhesion molecules and pro-inflammatory cytokines, exacerbating this oxidative-inflammatory cycle and facilitating endothelial dysfunction, which is the prelude to atherosclerosis and cardiovascular morbidity [2]. NF-κB: nuclear factor-κB; AP-1: activator protein 1.

Rule of thirds

- 1/3 Normotensive
- 1/3 Will have “office” hypertension
- 1/3 have “masked” hypertension
- “White Coat Hypertension” rare

- 35% - 80% OSAs have hypertension
- Males
- AHI > 30, >60% hypertensive
- Diastolic hypertension the earliest manifestation
- Commoner in patients with EDS
- May contribute to hypertension in pregnancy

- BP uncontrolled with 3 or more antihypertensives
- 20% of all hypertension is “resistant”
- OSA is present in 60% of patients with resistant hypertension
- CPAP very good at reducing such resistant disease (Spanish Study)

- Normal BP in consulting room
- Abnormal BP on ambulatory BP monitoring

- OSA – An independent risk factor
- 75 % patients admitted for electrical cardioversion have SDB
- Likelihood of maintaining sinus rhythm post AF cardioversion doubled if OSA treated with CPAP
- All treatments for AF work better when OSA is also treated
- NB: Symptoms not very good to detect OSA in AF – Sleep studies are needed

- Both OSA and especially CSA independent risk factors for malignant arrhythmias in patients fitted for cardiac resync + defibrillation

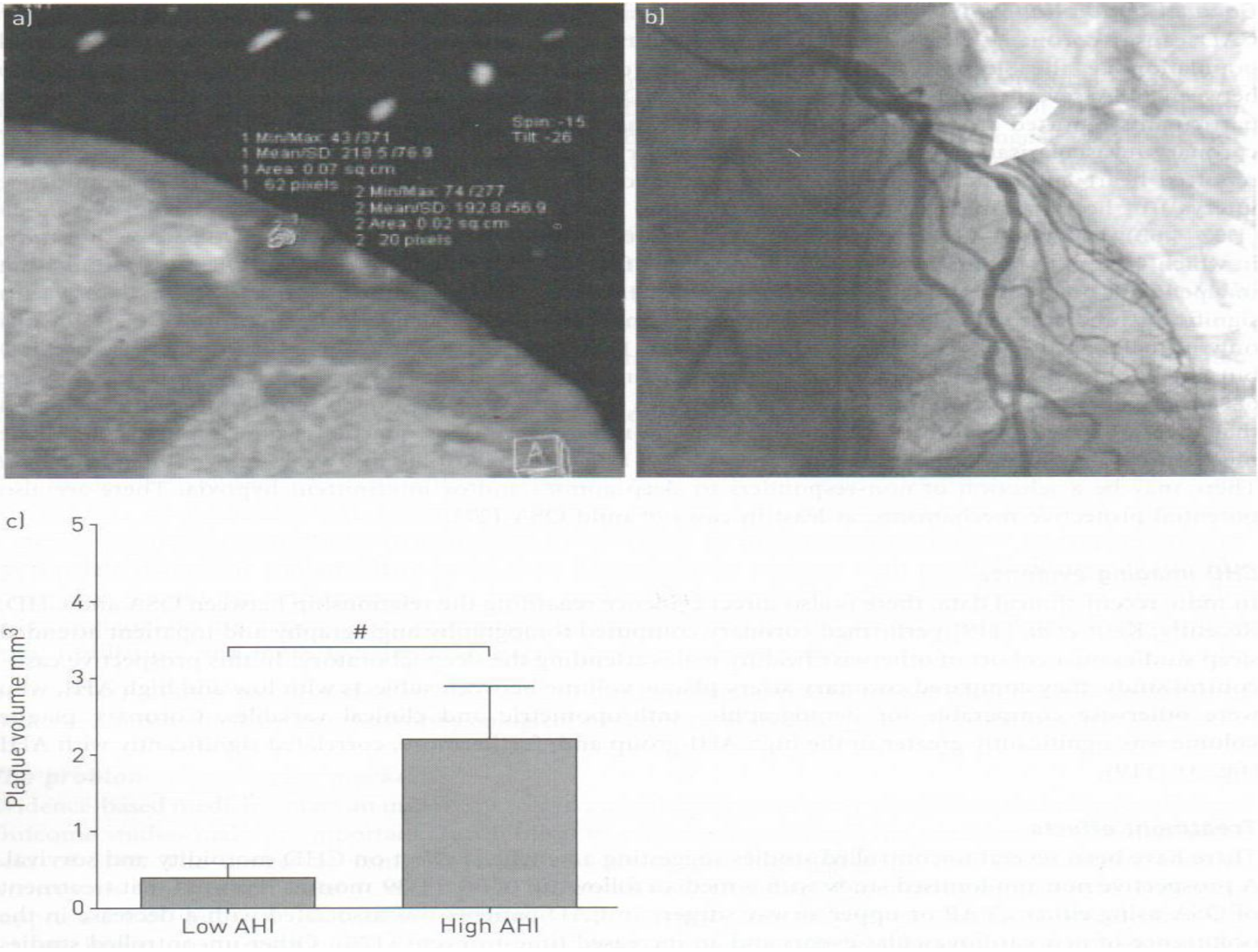
- Treatment of CSA with Adaptive Servo Ventilation (ASV) decreases malignant arrhythmias

- Sleep Heart Health Study (prospective)
  Males, 40 -70 yrs, AHI > 30
  68 % higher risk CAD (cf: AHI < 5)

- ↑ Coronary burden in otherwise healthy males attending sleep laboratory when AHI high

- ↓ Cardiovascular events in some trials when OSA patients used CPAP > 4 hr nocte

- Big prospective trial in progress to evaluate CPAP benefit (SAVE)


a)
Spin -15
Tilt -26
1 Min/Max 43 /371
1 Mean/SD 219.5 /76.9
1 Area 0.07 sq cm
1 62 pixels
2 Min/Max 74 /277
2 Mean/SD 192.8 /56.9
2 Area 0.02 sq cm
2 20 pixels
b)
c)
Plaque volume mm3
5
4
3
2
1
0
#
Low AHI
High AHI

- OSA common in Marfan's (tissue laxity)
- Aortic root dilatation – correlating well with AHI

- Expand X3 faster when OSA present

- Sleep heart health study
- Self reported HF x 2 – 4 ↑
- If AH1 > 30, HF ↑ 58%

- Massive intrathoracic pressure swings during obstructive apnoea (-70 cm H20)
- Hypoxic pulmonary arterial vasoconstriction
- Raised LV afterload
- Myocardial ischaemia
- Sympathetic output +++

- Small studies
- Rx of OSA clearly improves heart failure
- (Low HR, low BP, High LVEF, low urinary norepinephrine)

- Dependent oedema in daytime
- Recumbency cause ROSTRAL FLUID SHIFT
- PERIPHARYNGEAL OEDEMA → Upper airway narrowing

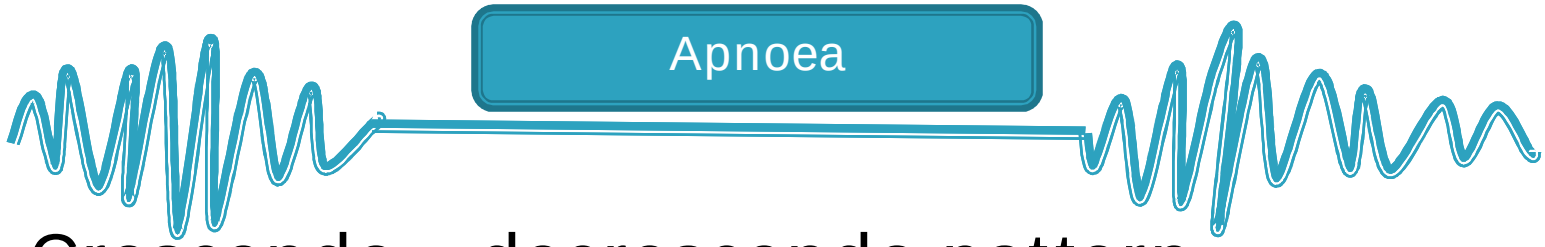
1. Diuretics – Reduced AH1 by 17/hr
   Increased oropharyngeal area

2. Resynchronisation treatment did not improve OSA – may be HF too severe

- Unidirectional
- Heart failure causes CSA
- CSA does not cause heart failure

- Recurrent cessations of airflow
- Simultaneous reduction/ cessation of respiratory effort
- Because no ventilatory impulse from brain stem

- Primary
- Heart Failure
- High altitude
- Brain Stem Lesions
- Opiates

- Periodic breathing
- 

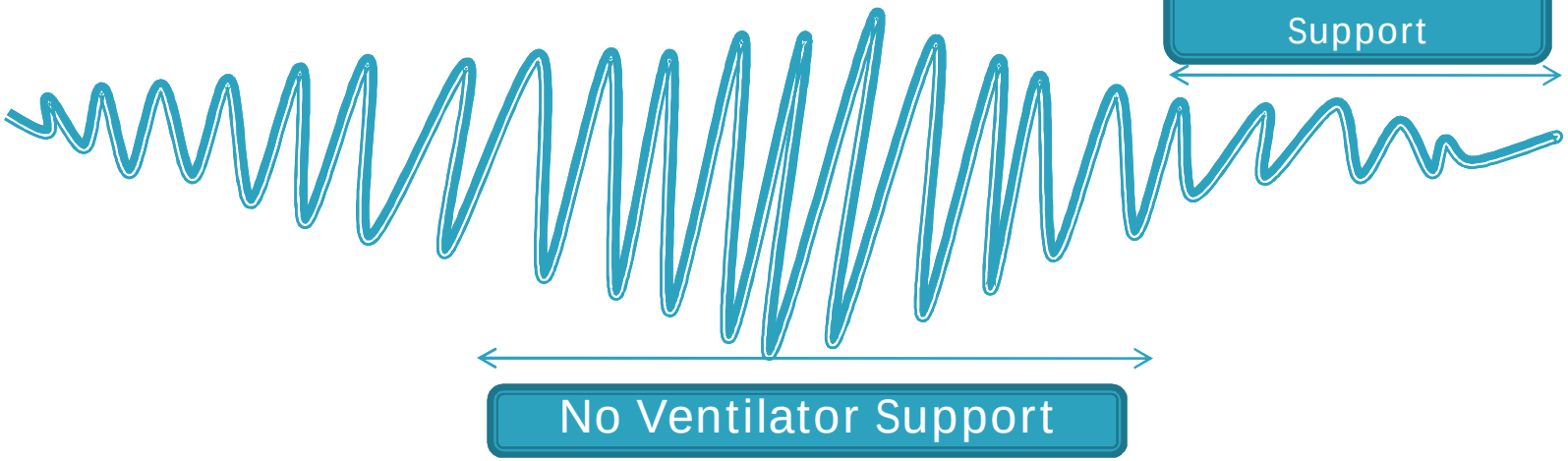
- Crescendo – decrescendo pattern

Associations

- Stable heart failure (51% have SDB)
- Stroke
- AF

- Daytime somnolence not a prominent factor of above in heart failure
- Therefore absence of EDS does not exclude SDB in heart failure
- Proper sleep study, preferably polysomnography is required.

- Rx underlying disease, i.e. Heart Failure
- Do PSG – evaluate presence and severity of apnoeas
- CPAP can be tried first, but results have not been encouraging
- ASV – Adaptive Servo ventilation

- “Anticyclic” Ventilation
- 

- Plus CPAP pressure for associated OSA

CPAP – ASV

- In symptomatic patient
- Refractory heart failure
- Many central apnoeas

- OSA is a common disorder and is a major risk factor for heart disease
- Heart failure is frequently associated with both OSA and CSA
- Treatment of OSA in Heart Failure is mandatory
- CSA should be treated when present in refractory heart failure

- Prevention is better than cure
- Think OSA!
- Think of it as a “NOT TO BE MISSED” diagnosis
- Think of “APNOEAS” in any patient with heart failure
- Refer early