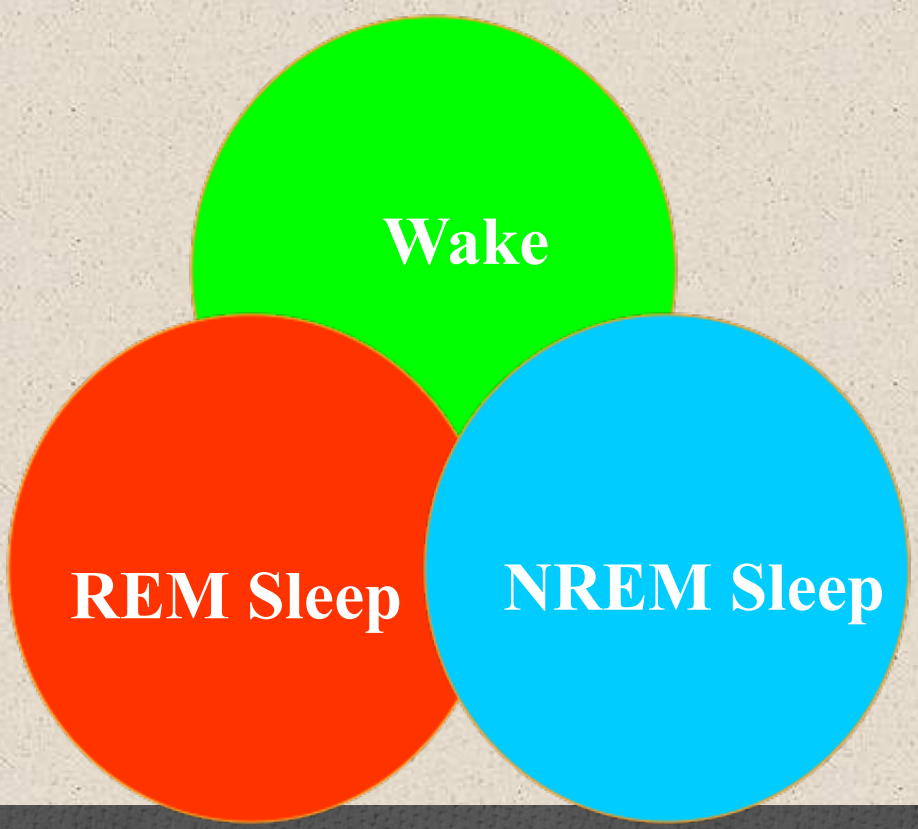
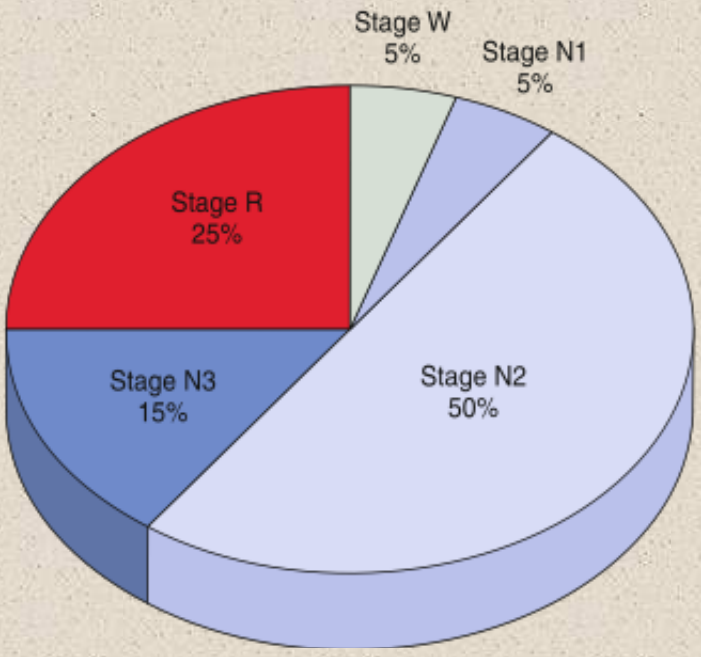

In the name
of GOD



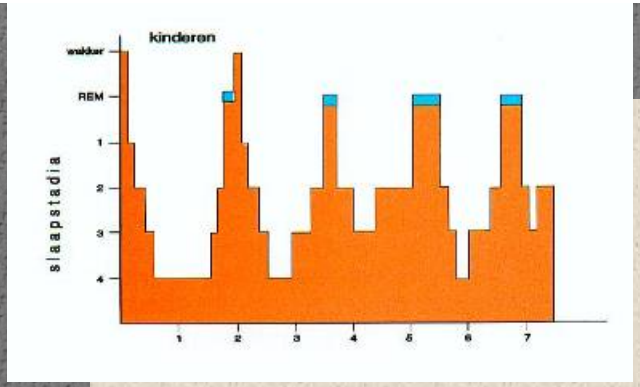
Dr B.Rahimi
pulmonologist
Sleep fellowship

States of being

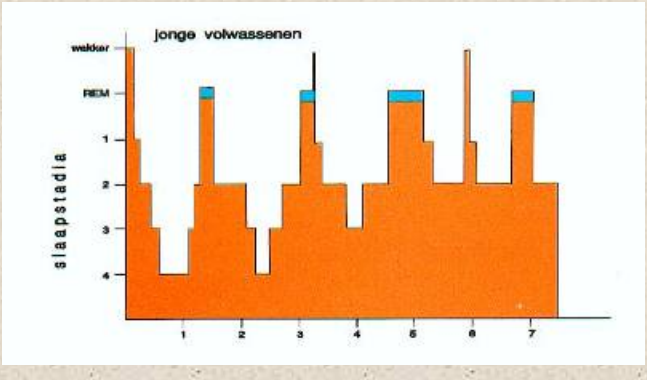



Stage W
5%
Stage N1
5%
Stage R
25%
Stage N3
15%
Stage N2
50%

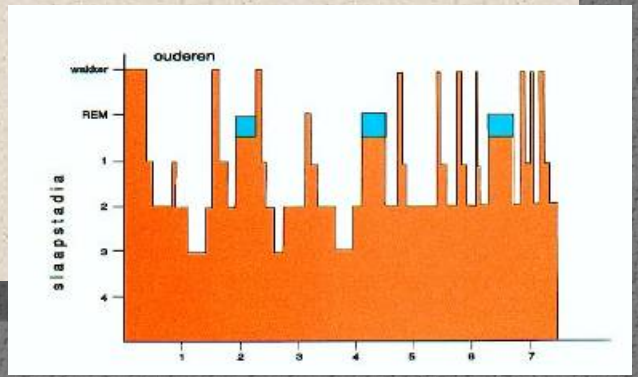
Result of scoring process: Sleep pattern according to age



Children



Adults



Elderly

Normal Sleep: Adults

- Sleep latency: <20 minutes
- Total Sleep Time(TST) 7-7.5 hrs
- Sleep efficiency: >90%
- Arousals: <5/hr
- REM: 20-25% TST
- Apnea/Hypopnea Index (AHI)<5/hr
- Stage 1 & 2 55-60% TST
- Stage 3 (SWS) 15-20% TST

Normal Individuals

- During NREM sleep, CO2 production falls but alveolar ventilation falls proportionately more and PaCO2 increases slightly .
- The fall in ventilation is due to a loss of the wakefulness stimulus to breathe, decreased chemosensitivity to hypoxia and hypercapnia, and increased upper airway resistance.
- The increase in PaCO2 results in a mild decrease in the PaO2.

Clinical Spectrum of SDB

Increasing Airway Resistance/Obstruction

NL Snorer UARS OSA

Chemoreceptor Dysfunction

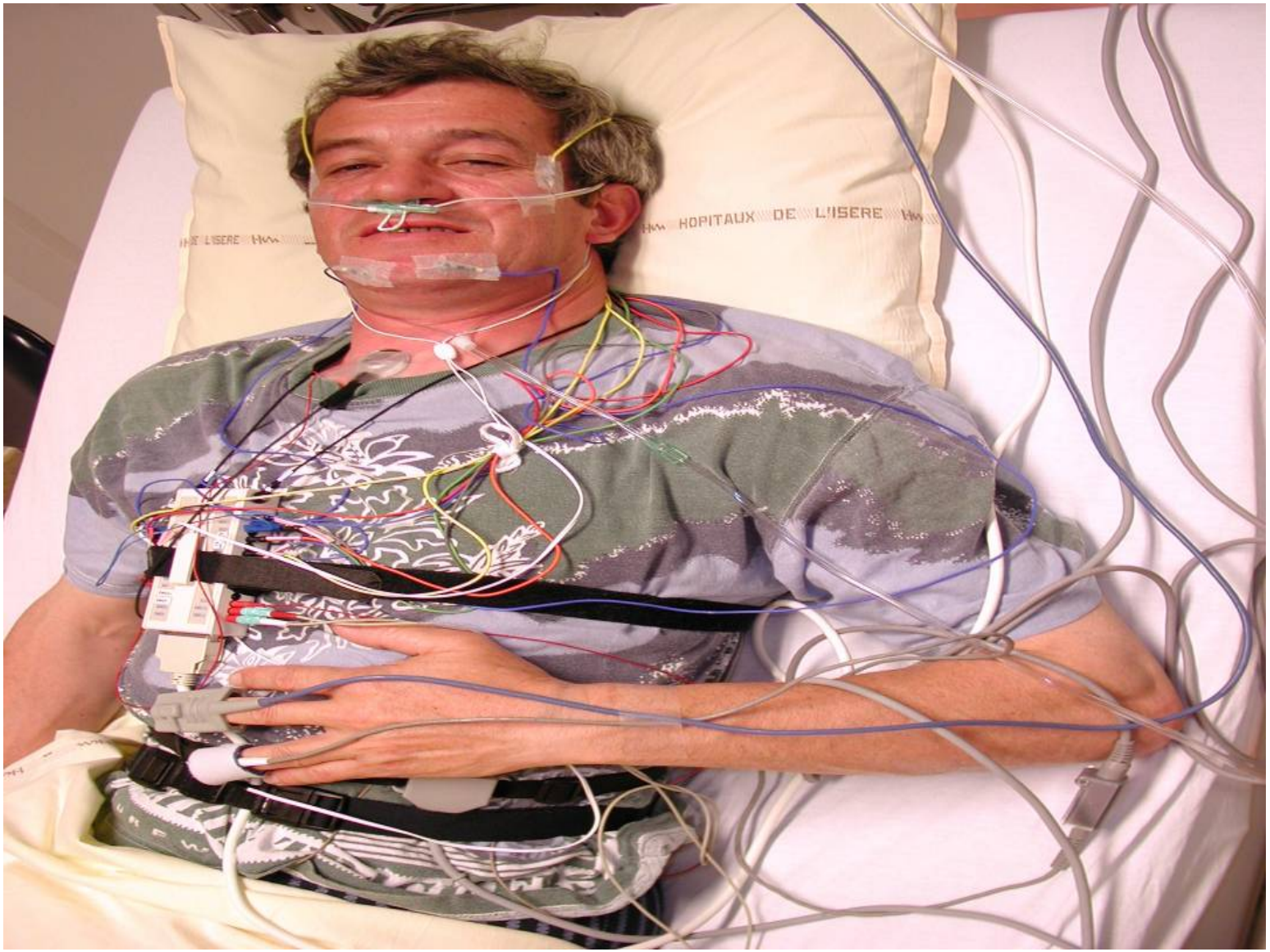
Low NL High

OHS CSA Cheyne-Stoke

Sleep related hypoventilation

- The most important initial step in the evaluation of a patient with sleep complaints is a detailed and focused inclinic interview and physical examination by an experienced provider, which helps generate a differential diagnosis.

- In selected patients the diagnostic polysomnogram (PSG) then plays a pivotal role in confirming the clinician's suspicions and helps guide further management


HOPITAUX DE L'ISERE

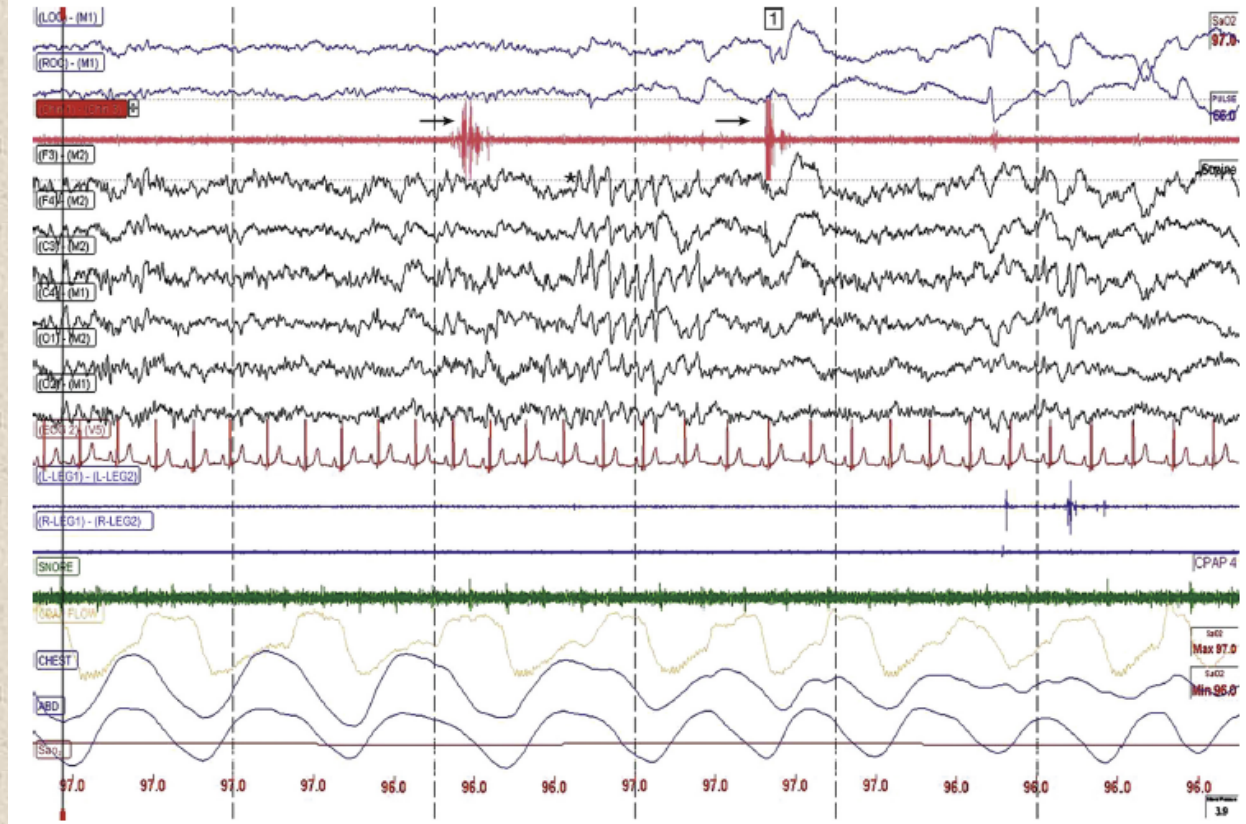


FIGURE 3.15 Thirty-second epoch of stage R sleep displaying sawtooth waves *(*)* and rapid eye movements *(1)*, with transient muscle activity in the chin electromyogram lead *(arrows)*. Montage depicted is according to American Academy of Sleep Medicine scoring criteria.

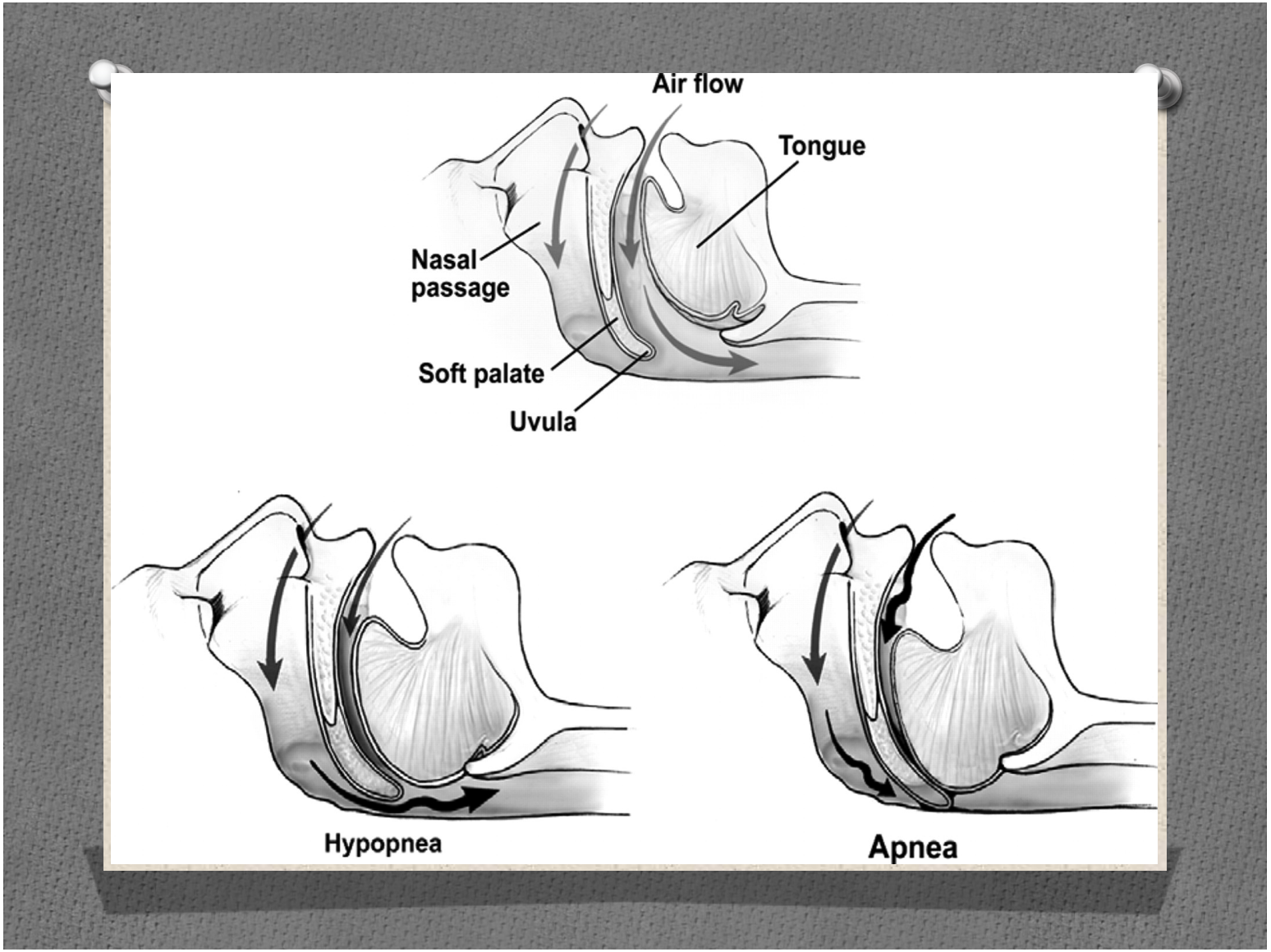
Obstractive sleep apnea

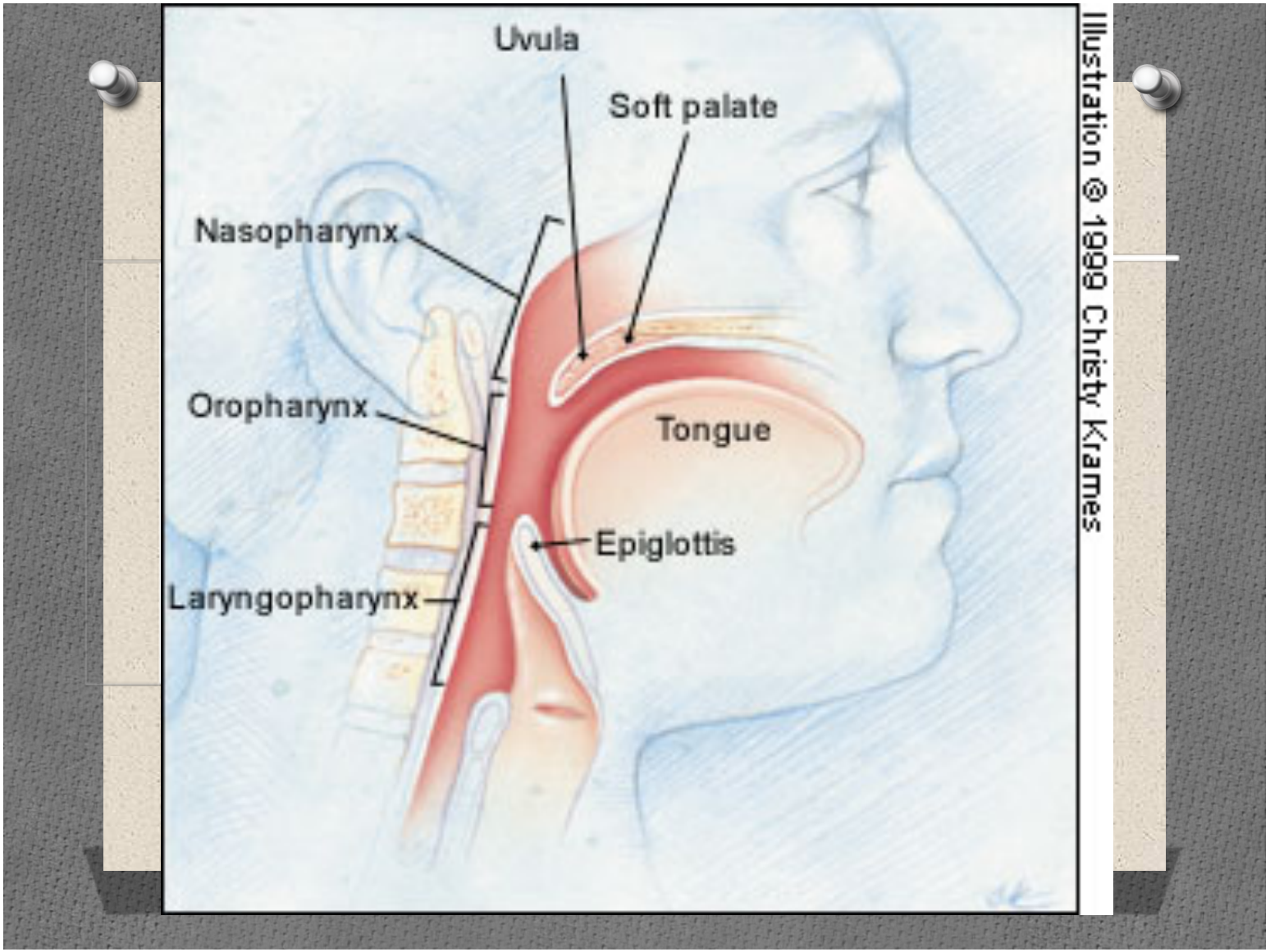
- OSA is characterized by repetitive interruption of ventilation during sleep caused by collapse of the pharyngeal airway .
- A diagnosis of OSA syndrome is accepted when a patient has an apnea-hypopnea index (AHI; number of apneas and hypopneas per hour of sleep) >5 and symptoms of excessive daytime sleepiness .

- An obstructive apnea is a ≥10-second pause in respiration associated with ongoing ventilatory effort.

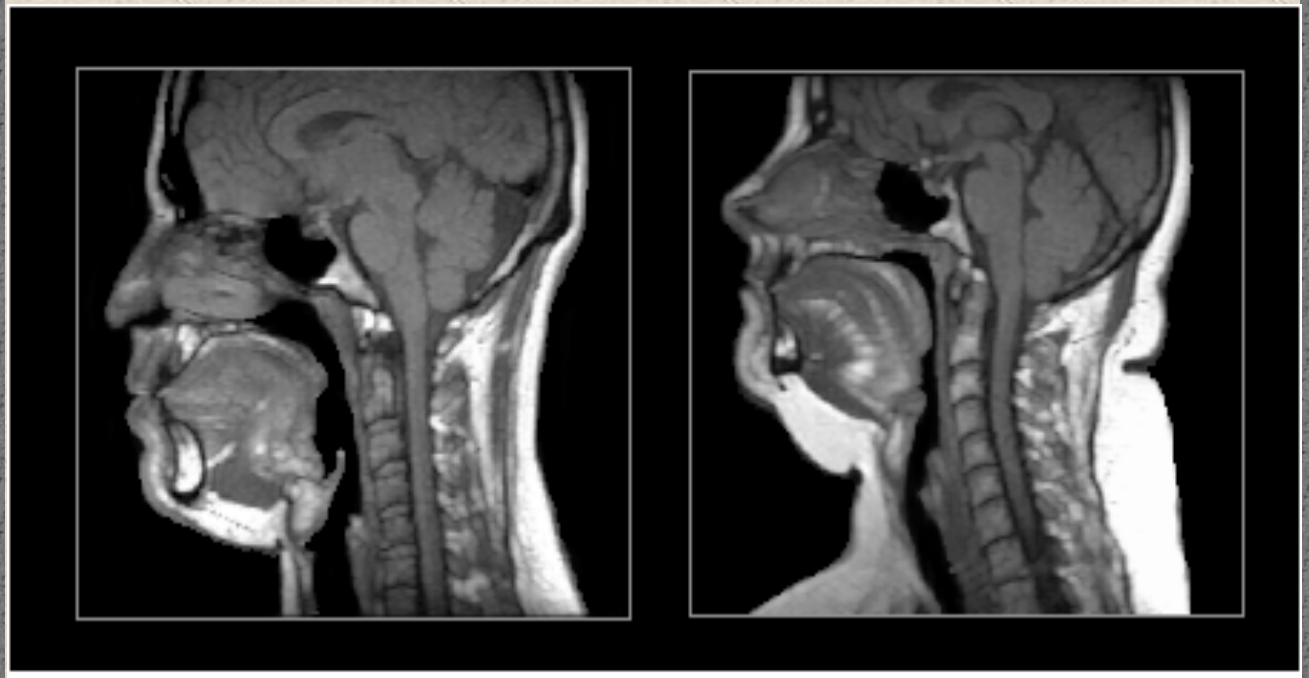
- Obstructive hypopneas are decreases in, but not complete cessation of, ventilation, with an associated fall in oxygen saturation or arousal.

- mild sleep apnea is defined as an AHI of 5 to 15, moderate asanAHIof15to30,andsevereasanAHI > 30 events per hour of sleep.


Air flow
Tongue
Nasal passage
Soft palate
Uvula
Hypopnea
Apnea

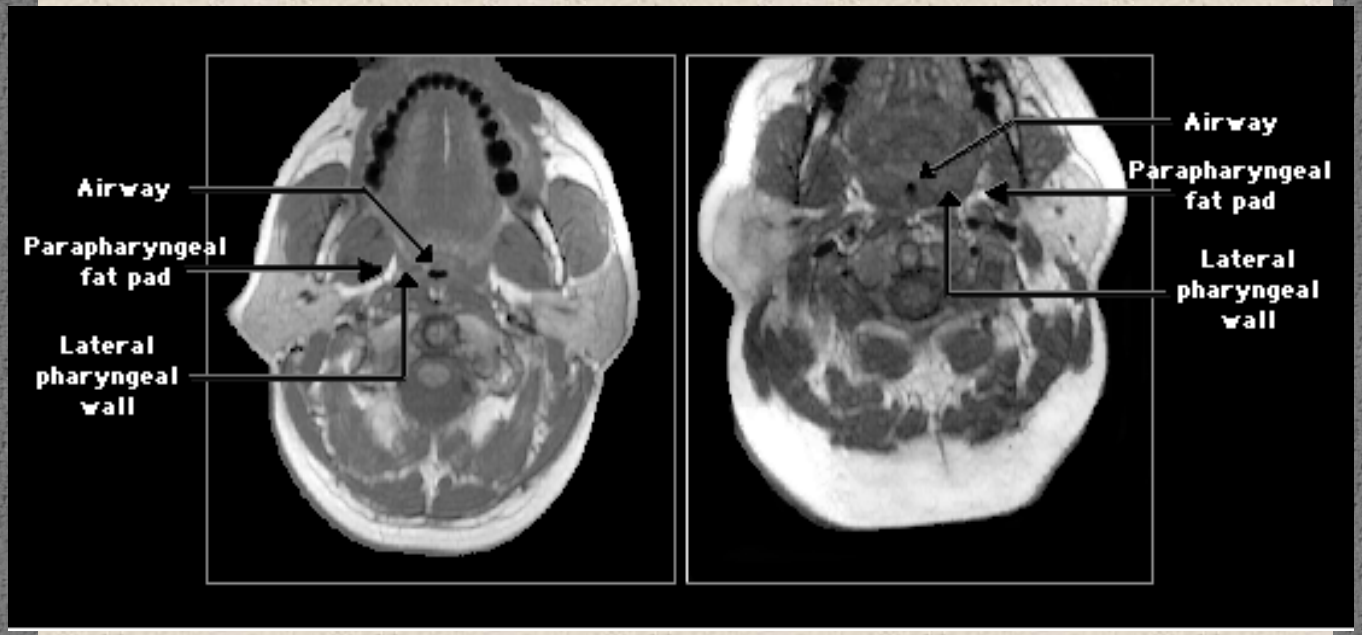

Uvula
Soft palate
Nasopharynx
Oropharynx
Tongue
Epiglottis
Laryngopharynx
Illustration @ 1999 Christy Krames

Airway Architecture



Reduced upper airway size in obstructive sleep apnea

Airway Architecture



- Pharyngeal collapse in patients with OSA generally occurs posterior to the tongue, uvula, and soft palate or some combination of these structures.

Risk Factors

- The major risk factors for OSAHS are obesity and male sex.
- mandibular retrognathia and micrognathia,
- a positive family history of OSAHS, genetic syndromes that reduce upper airway patency
- adenotonsillar hypertrophy (especially in children)
- , menopause (in women),
- various endocrine syndromes (e.g., acromegaly, hypothyroidism).

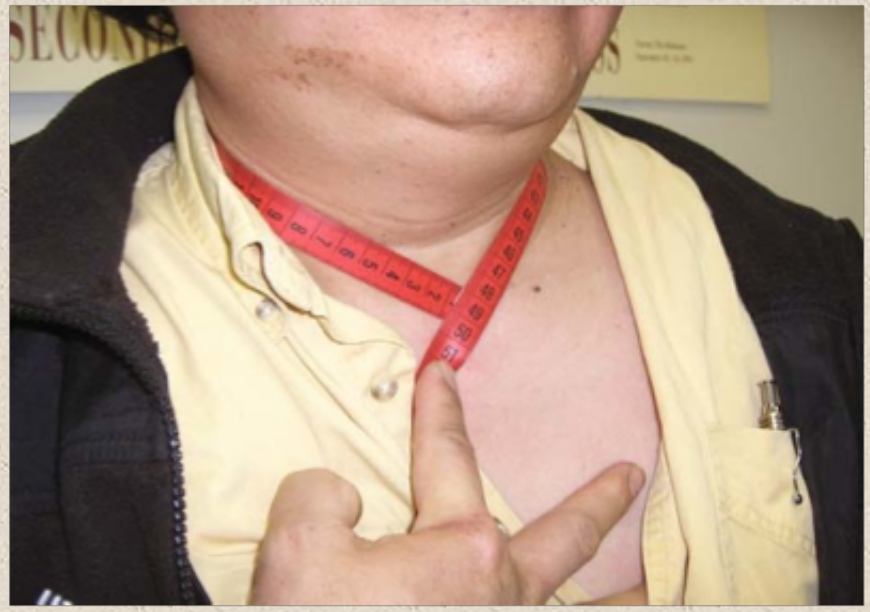
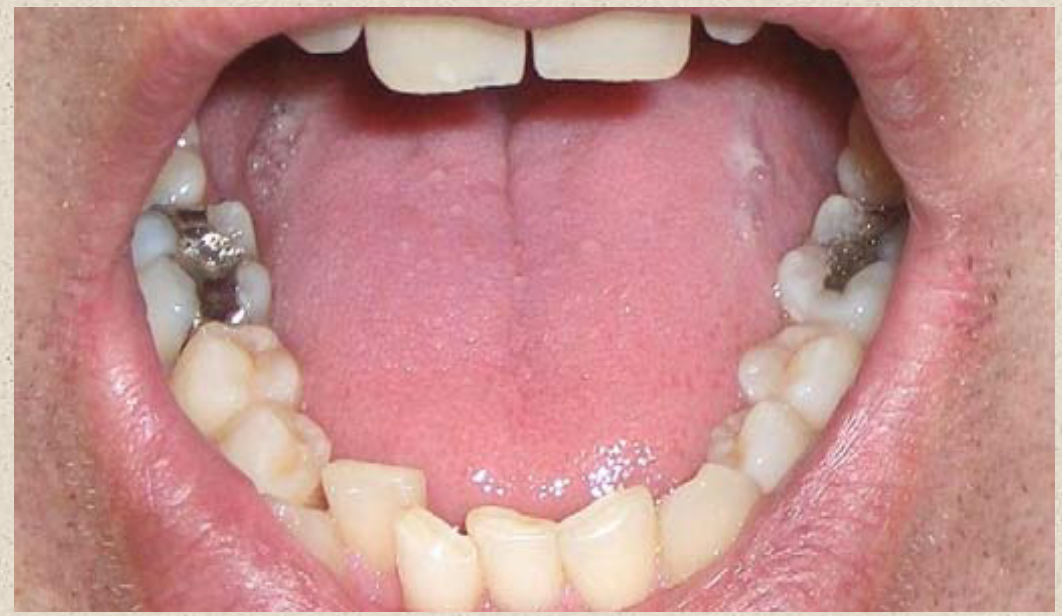
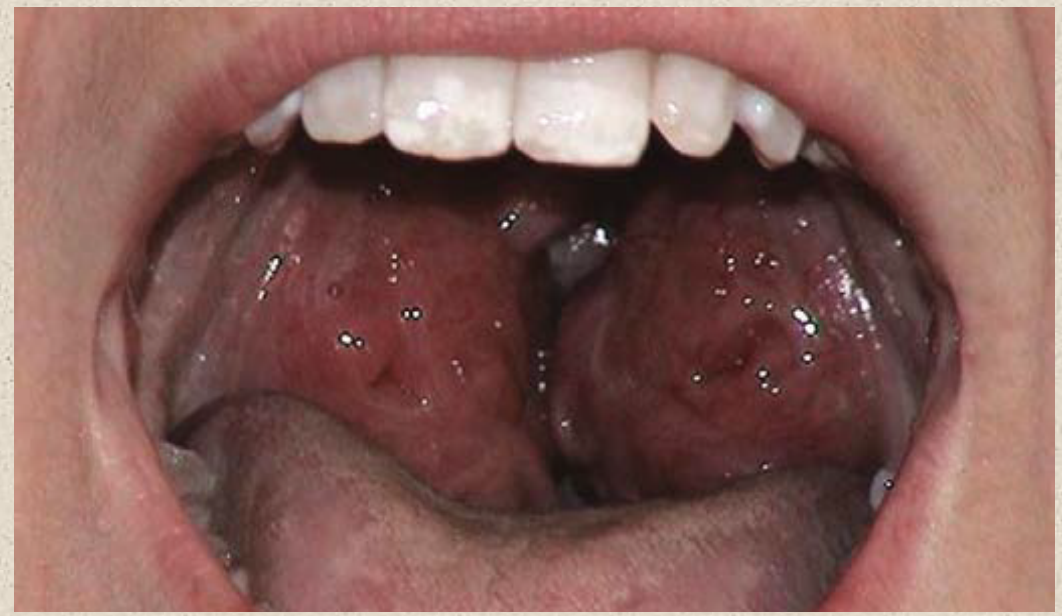


Figure 11.1-56.
Measuring neck collar size.

Small mandible



Tonsils grade 4



Asymmetric mandibular arch

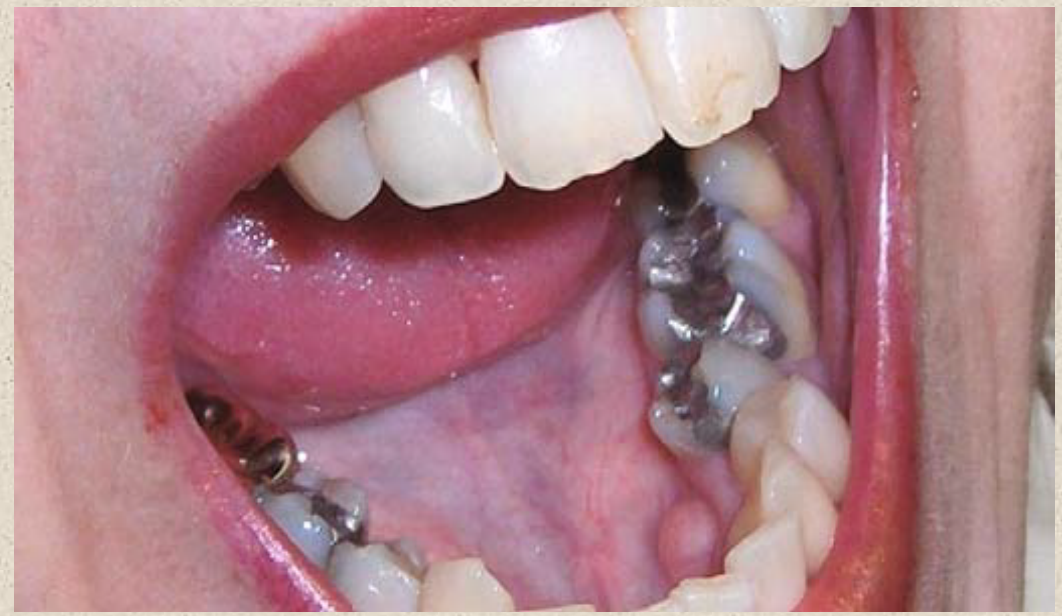


Figure 11.1-30.
Facial reconstruction after a gunshot wound.

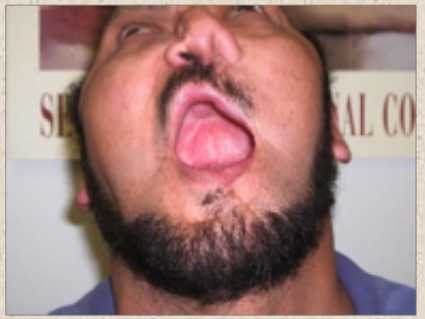


Figure 11.1-31.
Nasal reconstruction led to total obstruction.

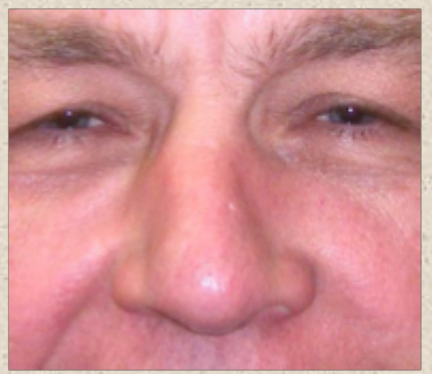
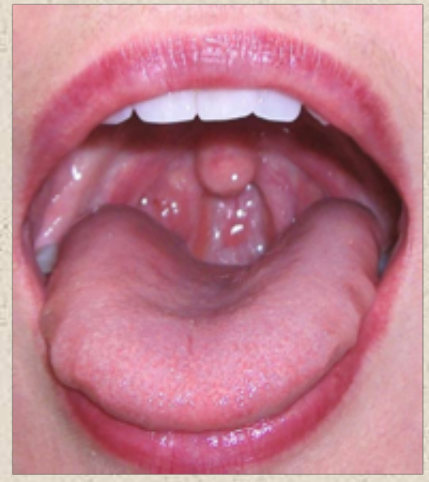
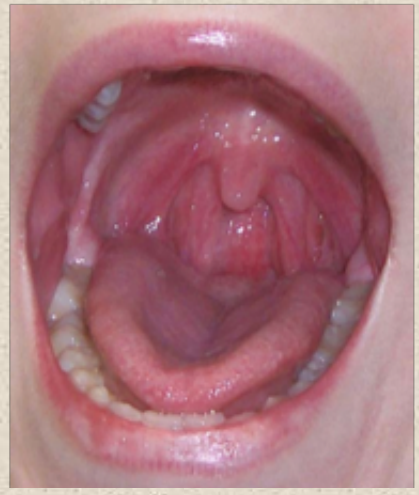


Figure 11.1-32.
Broken nose.

MALLAMPATI





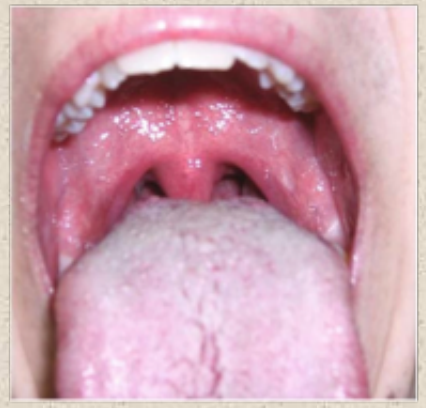


Figure 11.7-38
Mallampati Class II. Posterior pillars and much of uvula not visible.

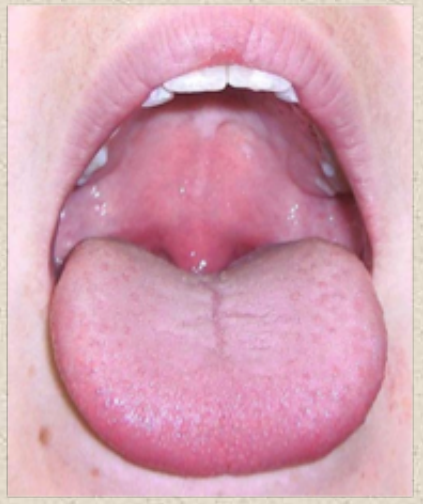


Figure 13.1-40.
Mallampati Class II. Only base of uvula seen.

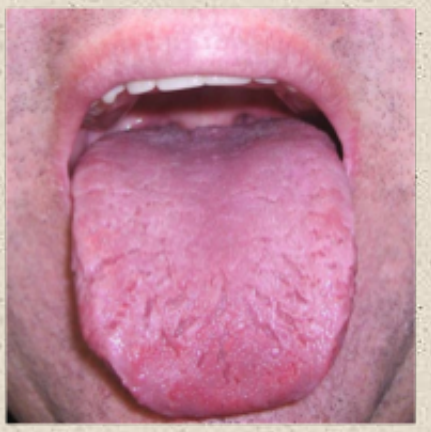
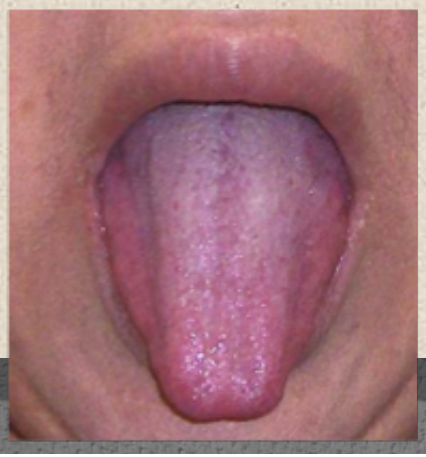
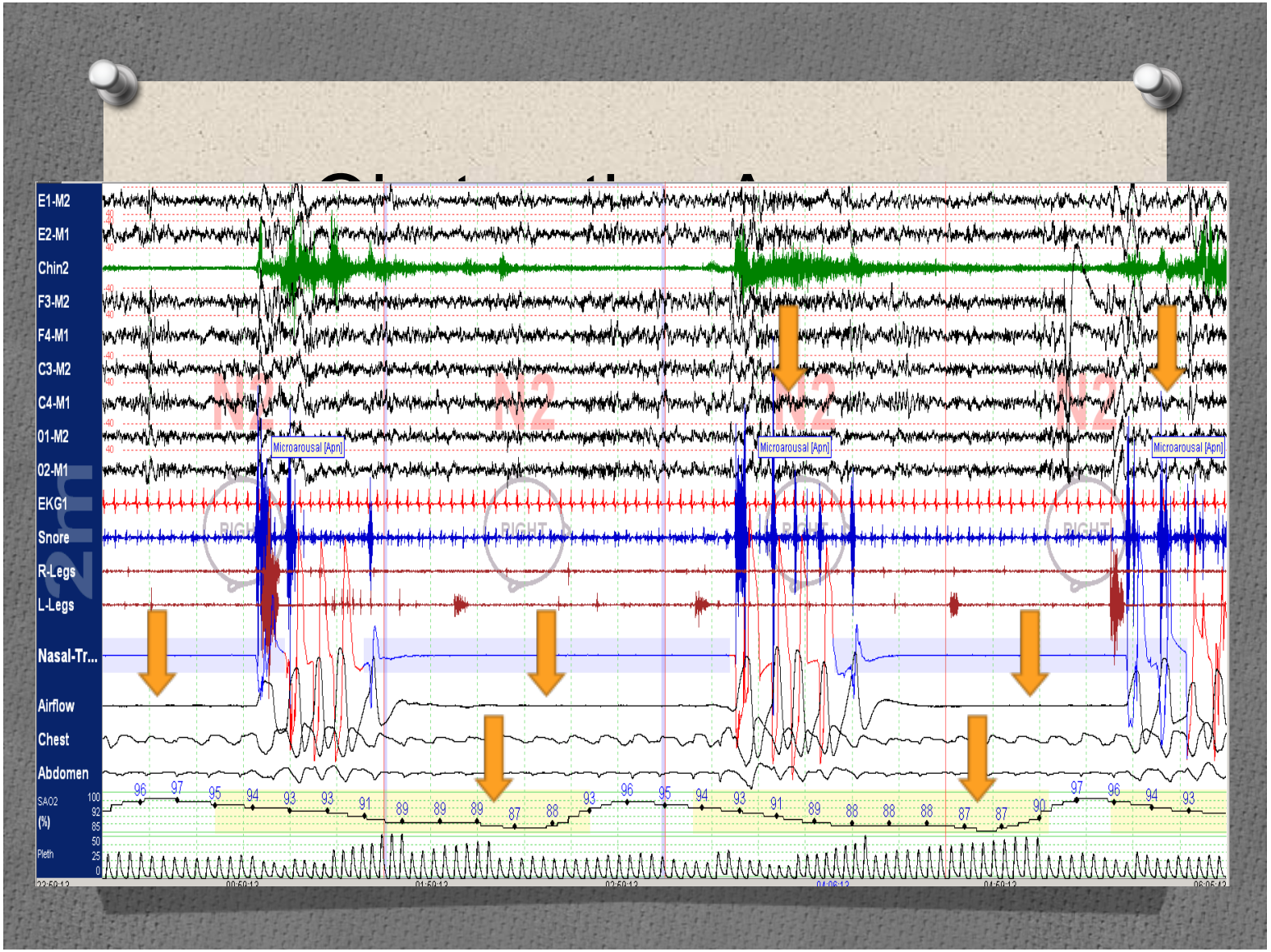
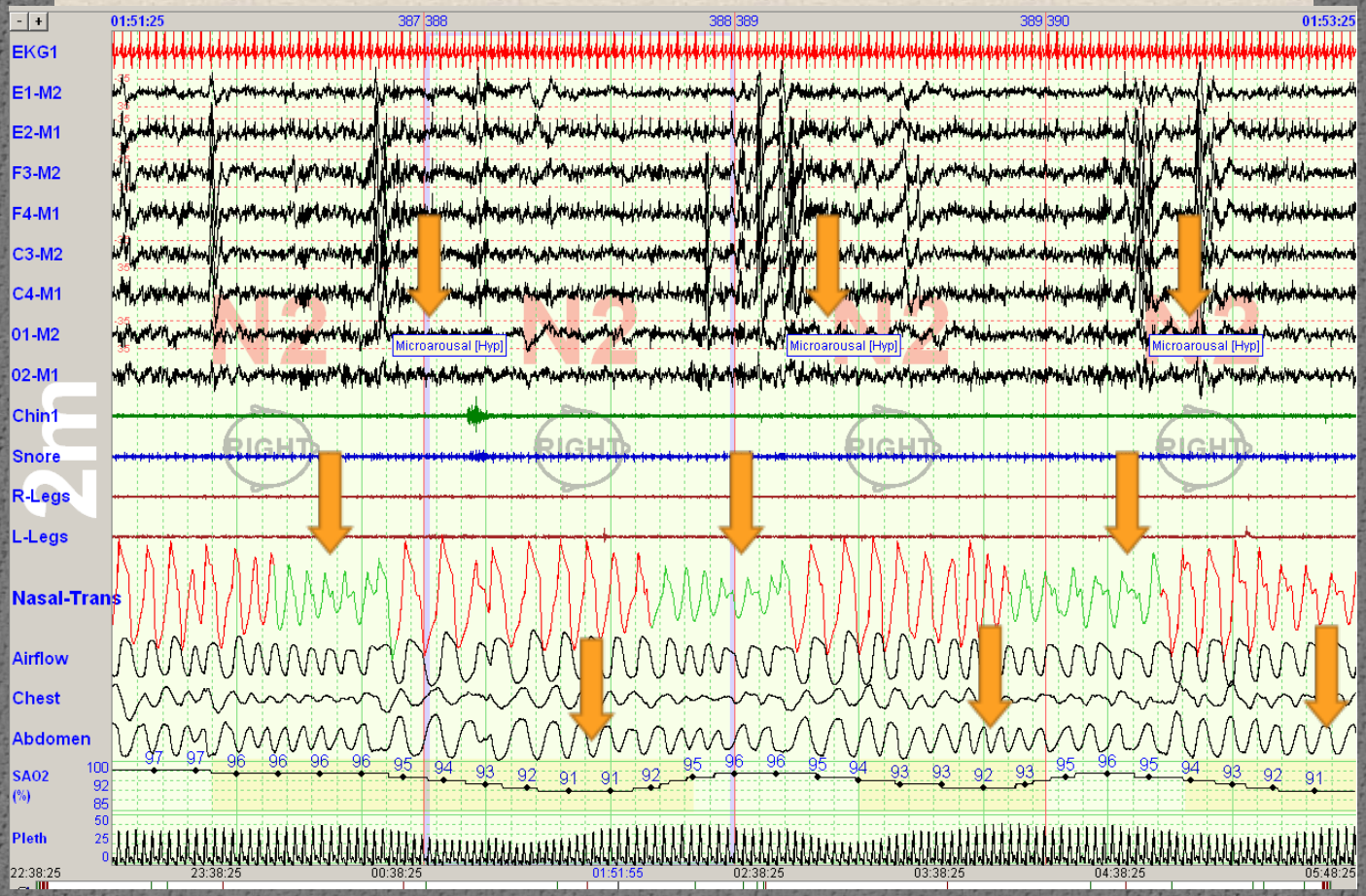


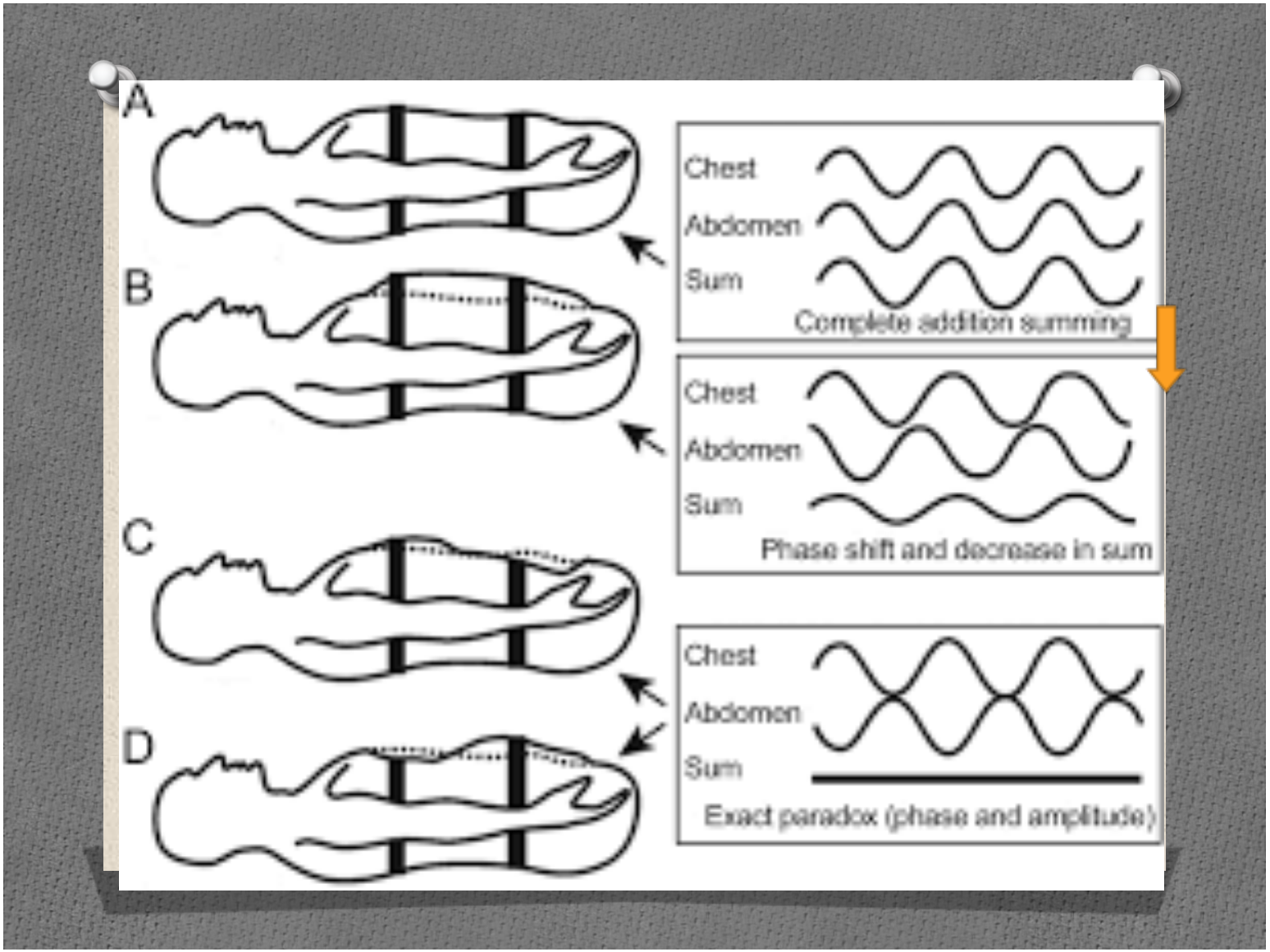
Figure 13.1-41.
Mallampati Class III. Base of uvula barely visible.



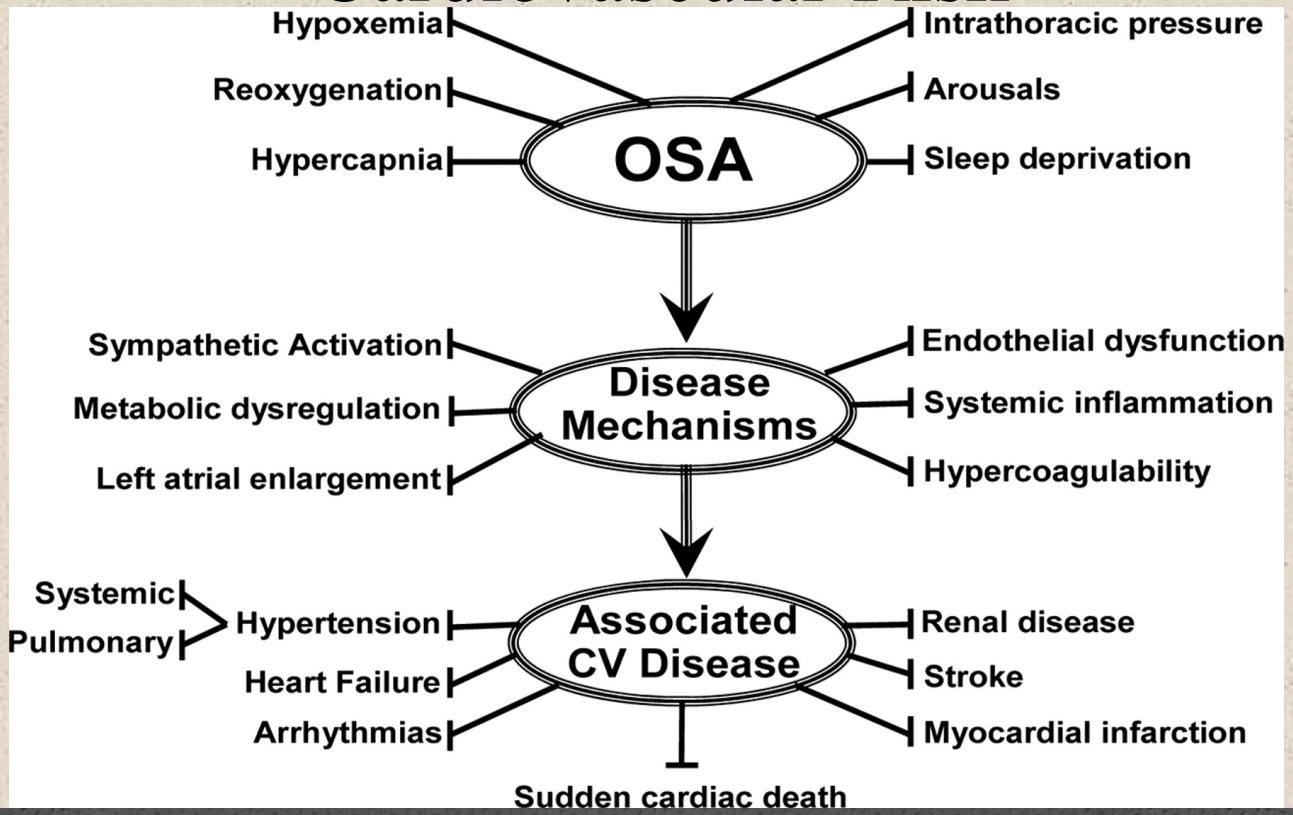

E1-M2
E2-M1
Chin2
F3-M2
F4-M1
C3-M2
C4-M1
01-M2
02-M1
EKG1
Snore
R-Legs
L-Legs
Nasal-Tr...
Airflow
Chest
Abdomen
SAO2 (%)
Pleth
N2
Microarousal [Apn]
RIGHT

Hypopneas


01:51:25
387 388
388 389
389 390
01:53:25
EKG1
E1-M2
E2-M1
F3-M2
F4-M1
C3-M2
C4-M1
01-M2
02-M1
Chin1
Snore
R-Legs
L-Legs
Nasal-Trans
Airflow
Chest
Abdomen
SA02 (%)
Pleth
N2
Microarousal [Hyp]
RIGHT
22:38:25
23:38:25
00:38:25
01:51:55
02:38:25
03:38:25
04:38:25
05:48:25


A
B
C
D
Chest
Abdomen
Sum
Complete addition summing
Chest
Abdomen
Sum
Phase shift and decrease in sum
Chest
Abdomen
Sum
Exact paradox (phase and amplitude)

Mechanisms of Disease and Associated Cardiovascular Risk



Cardiovascular Diseases and OSA Hypertension

OSA and the Origin and Progression of Hypertension

- Animal studies in rats and dog have identified possible mechanisms by which OSA might lead to hypertension such as
- intermittent hypoxemia
- chemoreceptor stimulation
- Sympathetic activation
- renin-angiotensin system

Which patients?

- patients with drug-resistant hypertension, defined as a clinic BP of ≥140/90 mm Hg while taking a combination of ≥3 antihypertensive drugs titrated to maximally recommended doses.
- an independent predictor of uncontrolled hypertension in patients <50 years of age .
- Nondipper HTN

- These data also suggest that continuous positive airway pressure may reduce nocturnal and daytime systolic blood pressure chronically. Randomised trials are needed to confirm the latter results.

- Bazzano LA, Khan Z, Reynolds K, He J. Effect of treatment with nocturnal nasal continuous positive airway pressure on blood pressure in patients with obstructive sleep apnea. Hypertension. 2007; 50: 417–423.

Heart Failure

OSA and the Origin and Progression of Heart Failure

- Hypertension (especially in night)
- Nocturnal oxygen desaturation
- increased BMI
- catecholamines, endothelin, and other growth factors produced in OSA
- reduce myocardial contractility

Arrhythmias

ethiology

- Hypoxemia
- sympathetic activation
- pressure surges
- transmural pressure changes
- systemic inflammation

Myocardial Ischemia and Infarction

Evidence That OSA Contributes to the Origin and Progression of Coronary Artery Disease

- Severe intermittent hypoxemia
- acidosis
- increased BP
- sympathetic vasoconstriction
- changes in intrathoracic and cardiac transmural pressures
- endothelial dysfunction
- systemic inflammation,

Insulin Resistance

- Increased catecholamines, sleep deprivation, and other pathophysiological characteristics of OSA may be associated with insulin resistance.

Pulmonary Arterial Hypertension

OSA and the Origin and Progression of Pulmonary Arterial Hypertension

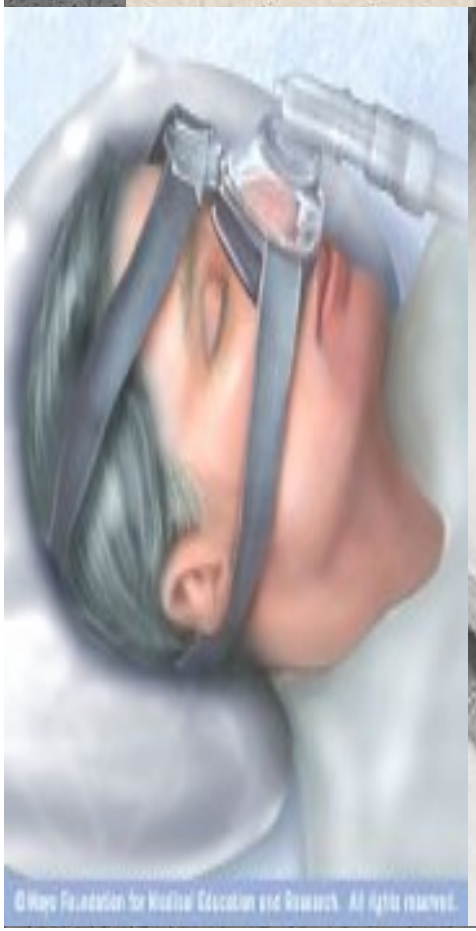
- An unresolved issue is whether nocturnal hypoxemia can lead to daytime pulmonary arterial hypertension or whether daytime hypoxemia, which is commonly observed in these patients, also is required
- . Two confounding factors that may contribute to daytime hypoxemia are severe obesity (obesity-hypoventilation syndrome) and chronic obstructive pulmonary disorder in association with OSA (the so-called overlap syndrome)

Treatment Options in OSA

- Weight loss
- behavioral techniques (positional apnea)
- CPAP
- oral appliances
- BIPAP
- Surgery
- Tracheostomy

How is the pressure applied non-invasively?

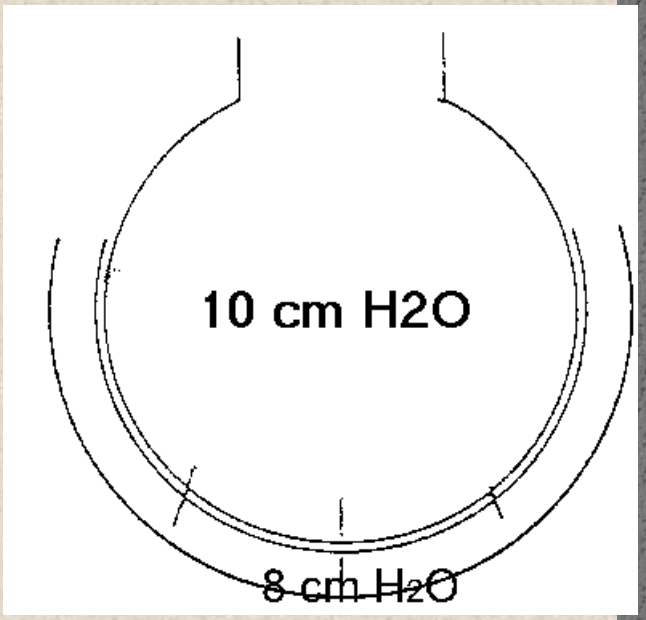
The nasal mask (left) and nasal pillows (middle) and full face mask (left)



Physiology of CPAP

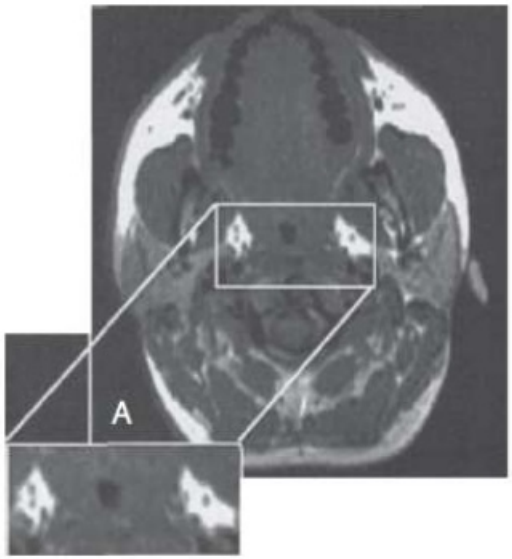
Physiology of CPAP : CPAP Mechanism

- Increases pressure within airway.
- Airways at risk for collapse from excess fluid are stented open.
- Gas exchange is maintained
- Increased work of breathing is minimized
- Splint of oropharangeal

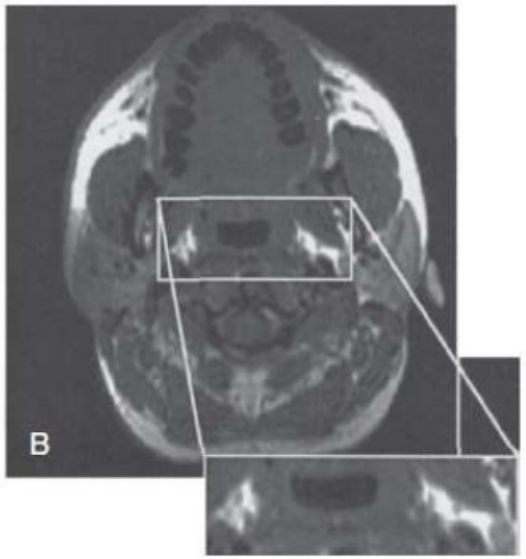


- The occurrence of upper airway obstruction indicates the development of a collapsing transmural pressure across a highly compliant pharyngeal wall.

Mechanism of action of PAP



After starting CPAP

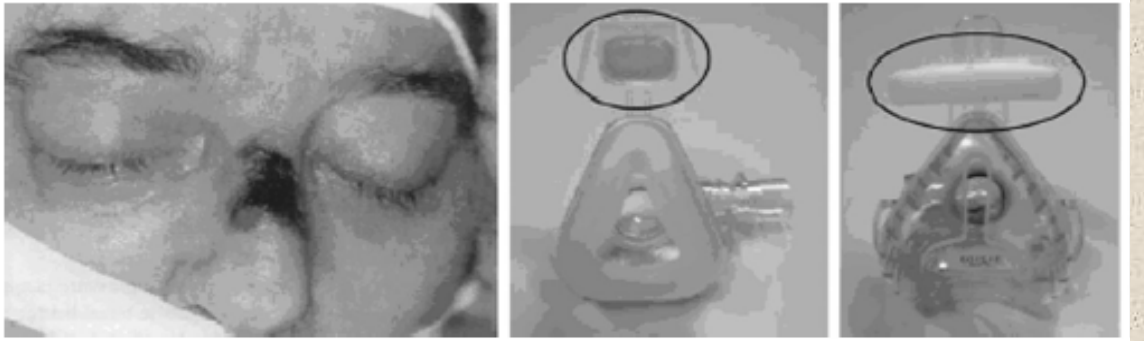


CPAP titration

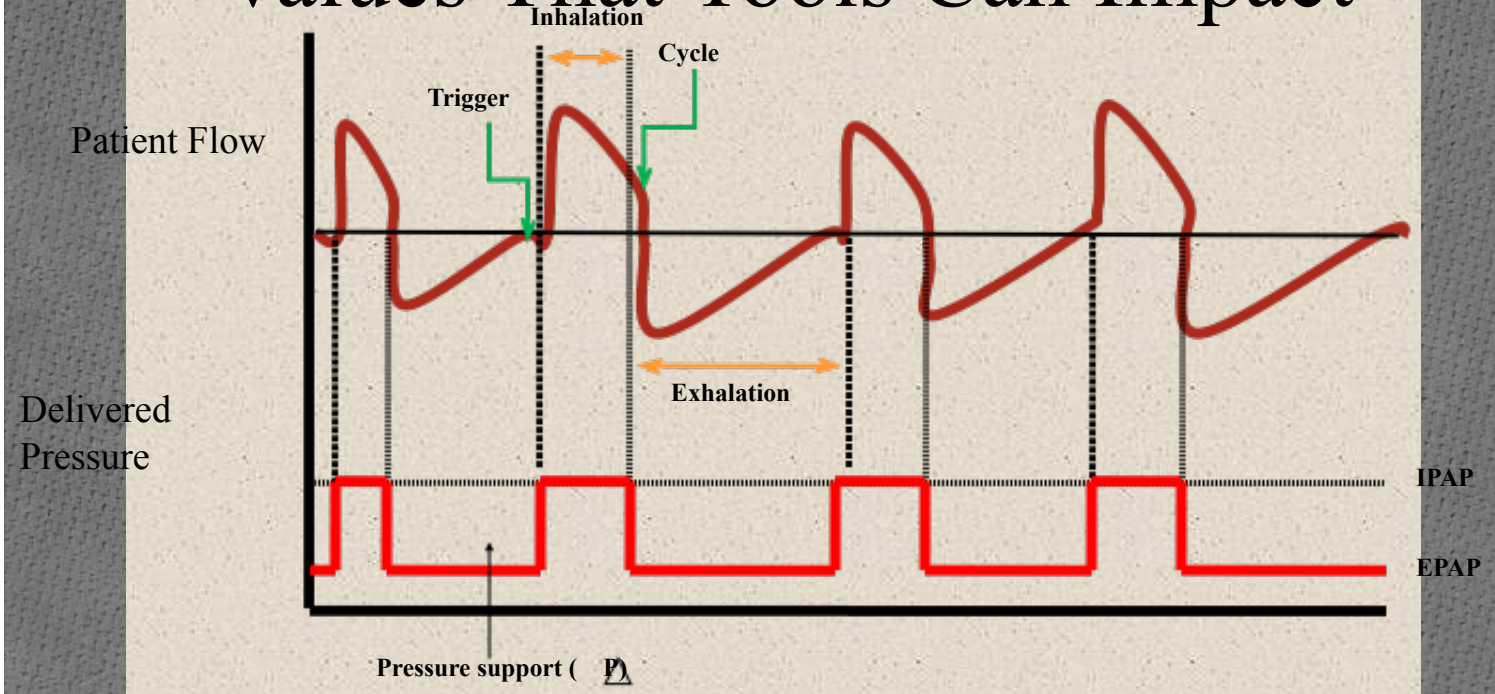
- If all events are abolished then increase pressure further by 1-2cm increment upto 4 cm
- If treatment emergent central apneas appear then reduce the pressure further till central apneas disappear
- If central apneas appear then decrease pressure till they disappear

Complications

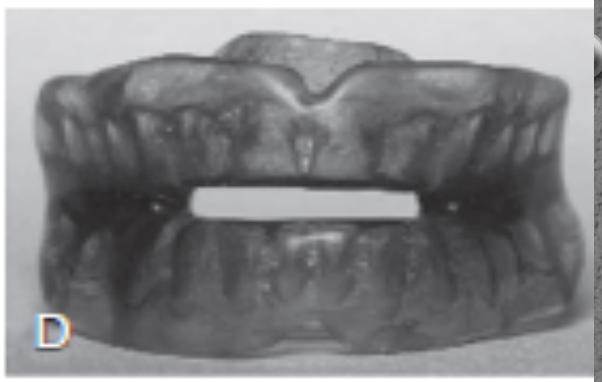
- CPAP may drop BP due to increased intrathoracic pressure.
  - A patient must have a systolic BP of at least 90mmHg to be a candidate for CPAP
  - Increased Intrathoracic pressure means decreased ventricular filling and increased afterload, thus decreasing cardiac output and blood pressure. .
- Risk of pneumothorax
  - Increased intrathoracic pressure = increased risk
  - Higher in Asthmatics and COPD
- Gastric Distention, and vomiting
- Risk of corneal drying
  - High volumes of air blowing at eyes
  - Nose skin irritation



Bilevel Breath Cycle Values That Tools Can Impact




C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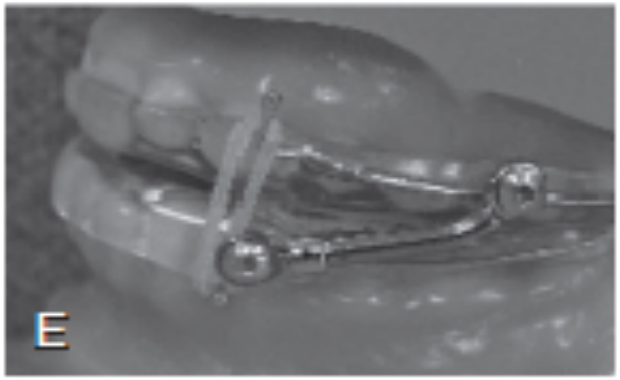
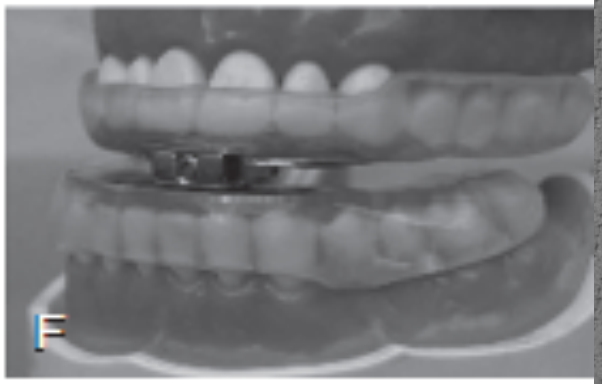

E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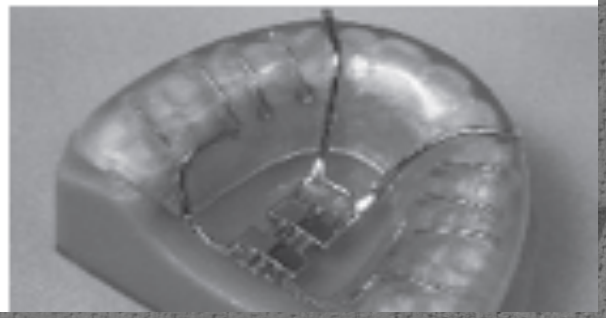
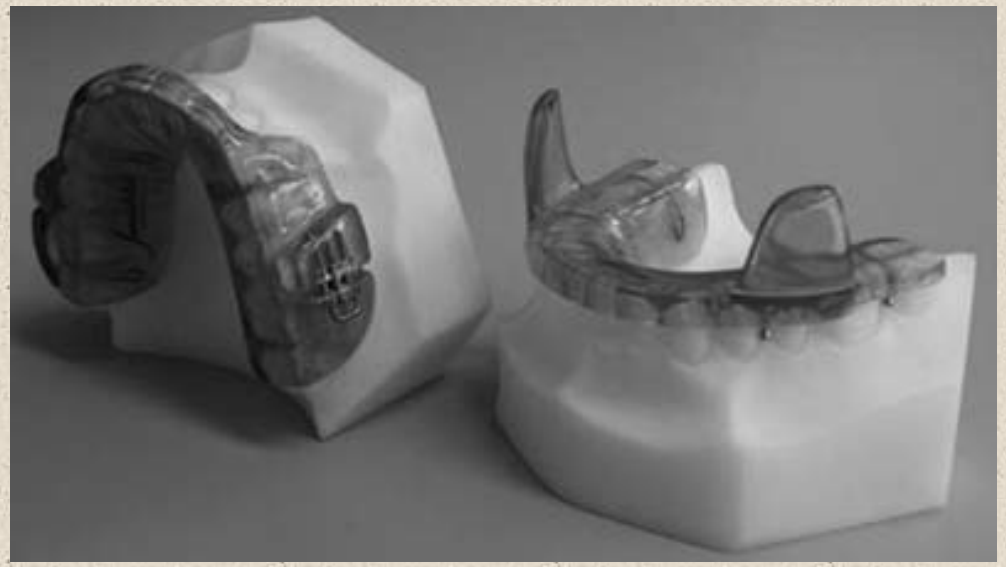
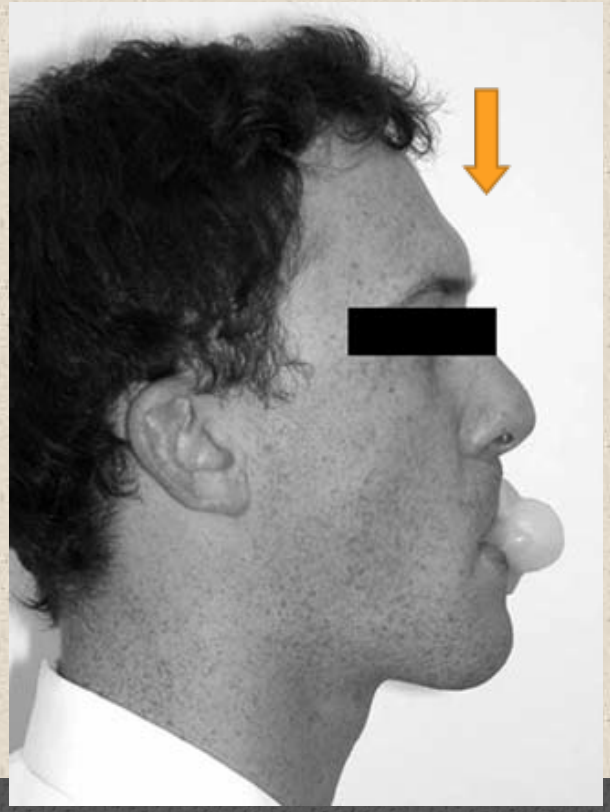


Oral appliance

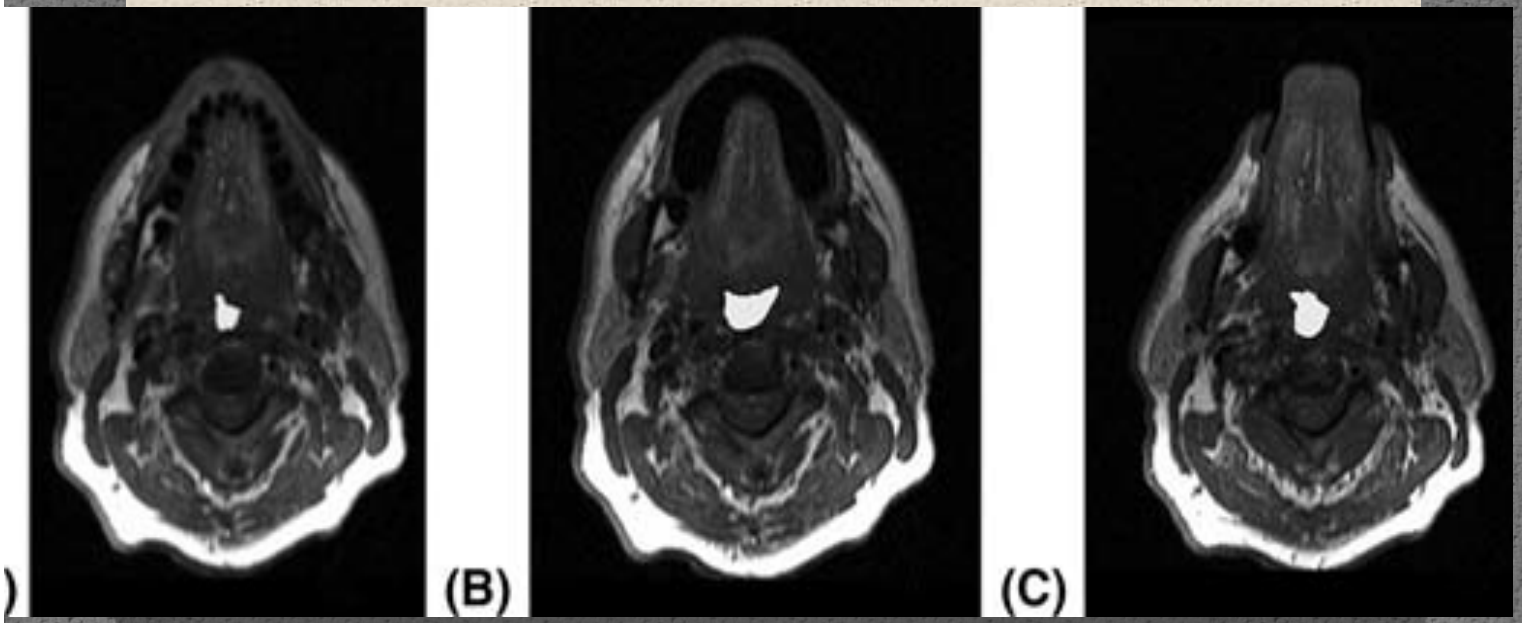
mandibular advancement splints (MAS)

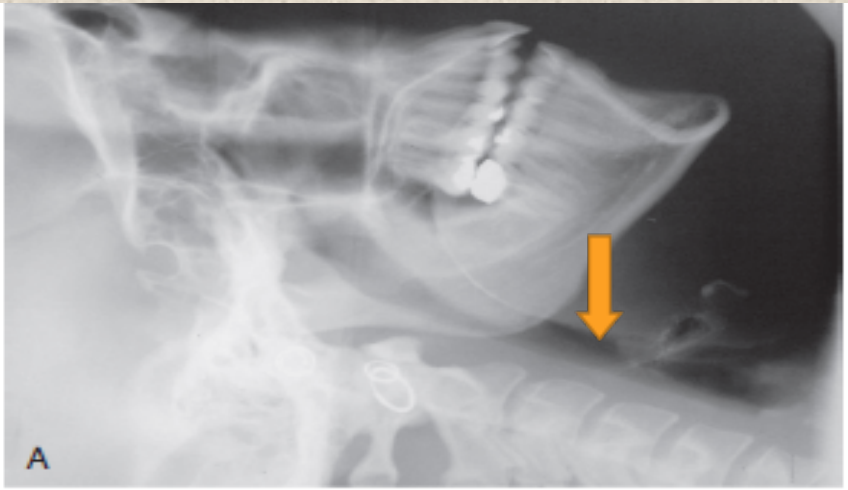


tongue retaining devices (TRD).

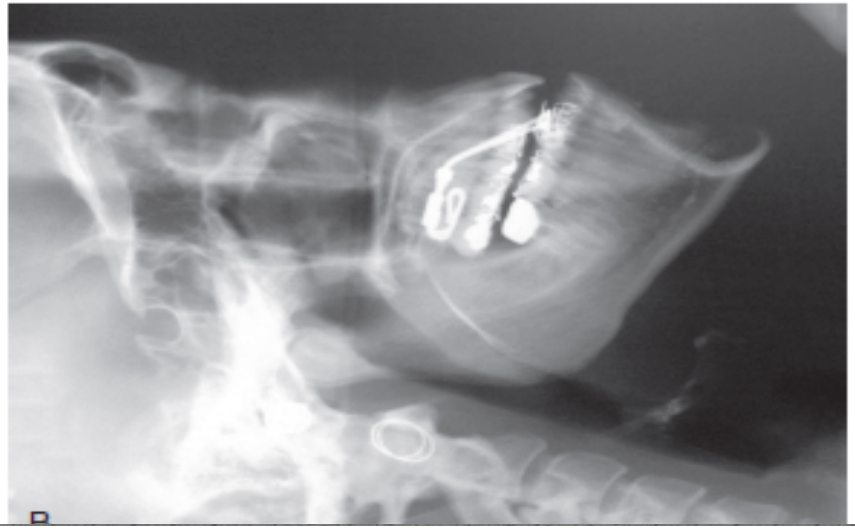


Note the difference in airway changes

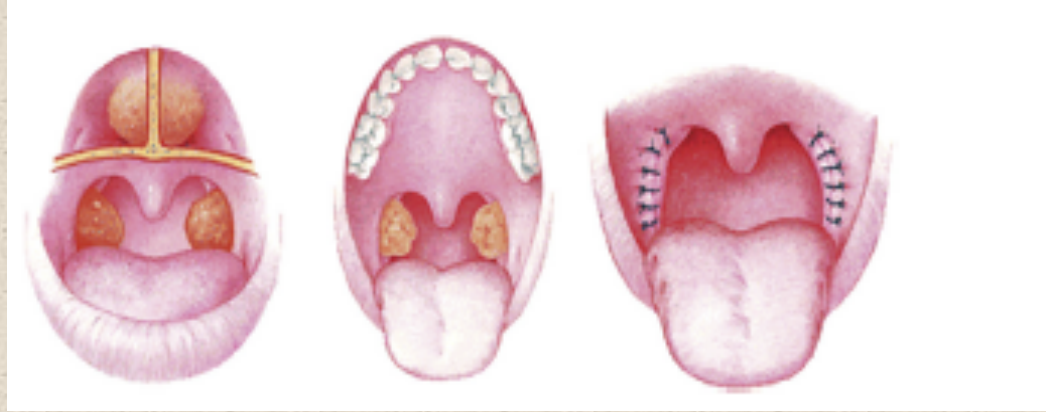




A



B



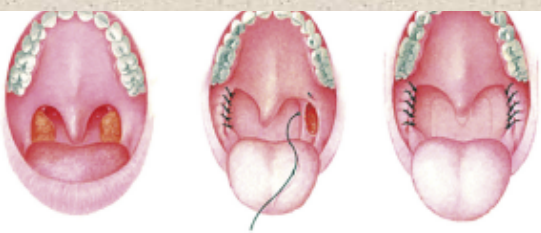


FIGURE 16.6 Uvulopalatopharyngoplasty reduces upper airway obstruction by shortening the uvula, trimming the soft palate, and suturing back the anterior and posterior pharyngeal pillars. Tonsillectomy is performed at the same time if tonsils are found to be enlarged. *(Reprinted with permission of the American Thoracic Society. Copyright © 2011 American Thoracic Society. From Won CH, Li KK, Guilleminault C. Surgical treatment of obstructive sleep apnea: upper airway and maxillomandibular surgery.* Proc Am Thorac Soc. *2008;5[2]:193-199.)*

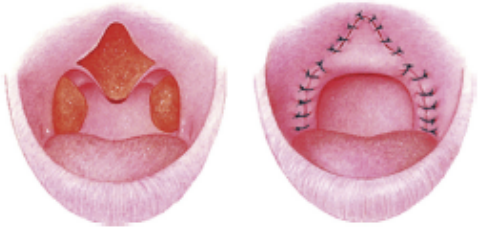
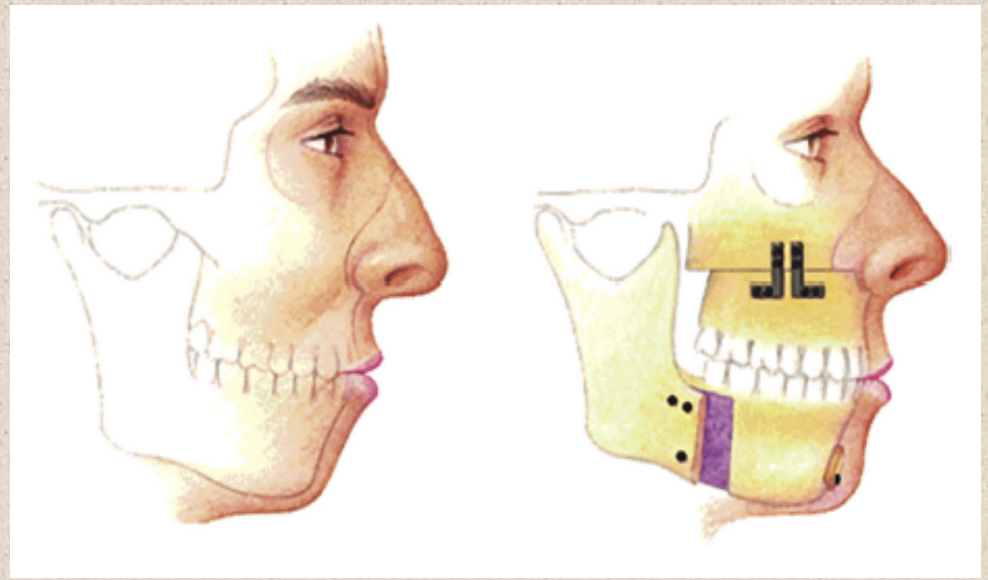


FIGURE 16.7 Uvulopalatal flap is a modification of uvulopharyngoplasty. Instead of removing the uvula and soft palate, the uvula is retracted and tucked superiorly under the soft palate. The pharyngeal pillars are sutured back and tonsils are removed in this procedure as well. *(Reprinted with permission of the American Thoracic Society. Copyright © 2011 American Thoracic Society. From Won CH, Li KK, Guilleminault*

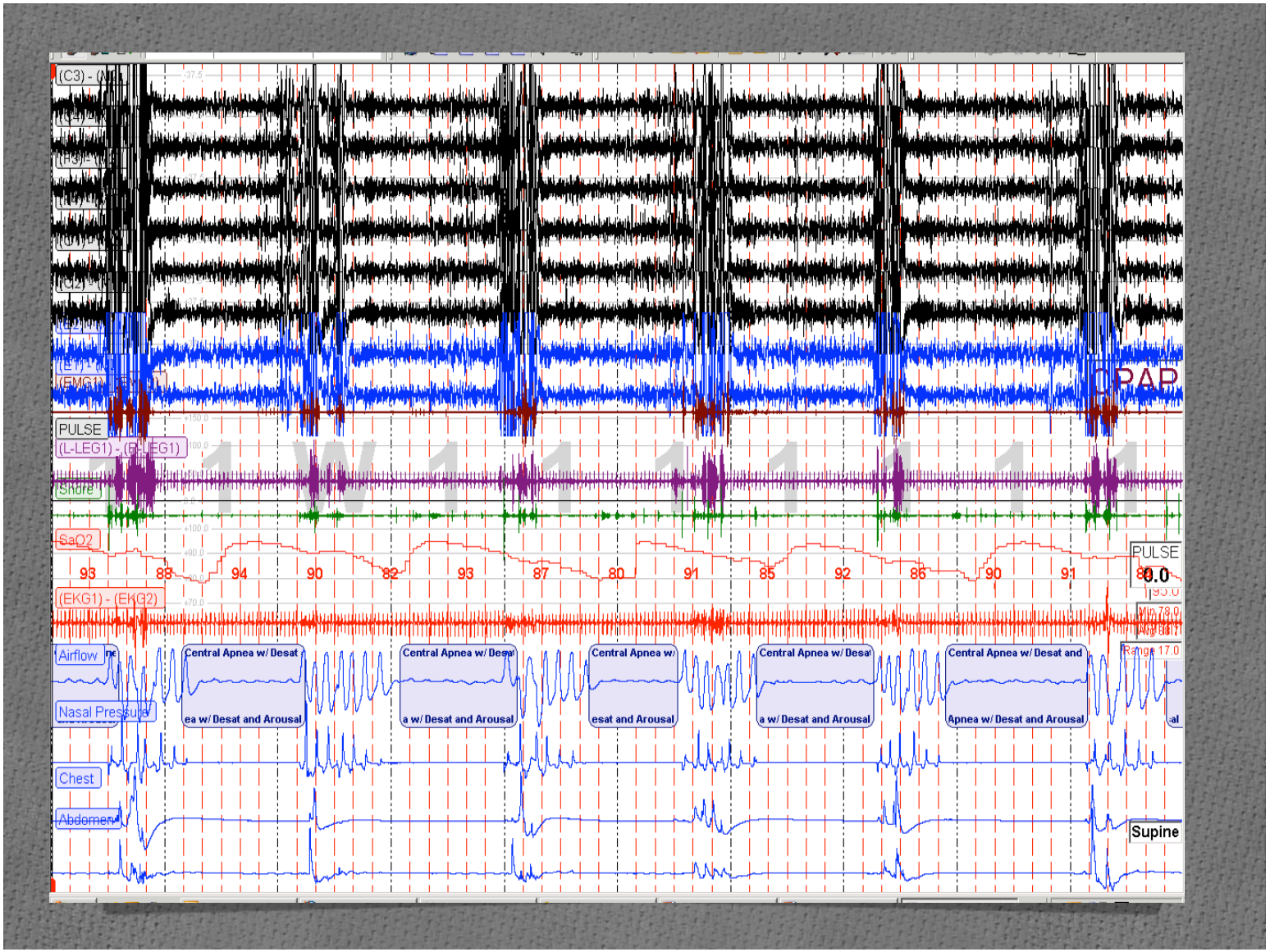


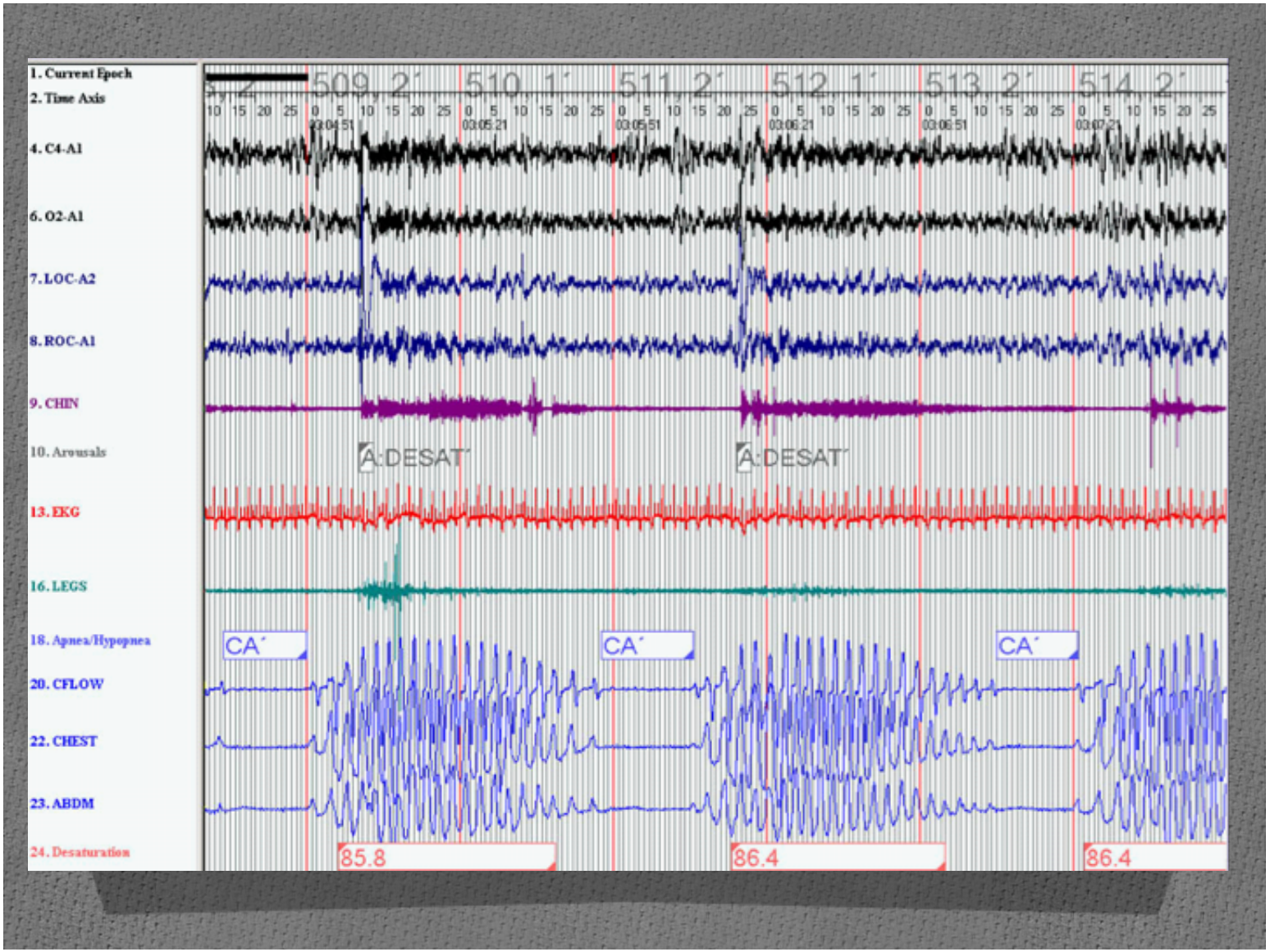
- Bariatric surgery has variable long-term efficacy in treating OSA

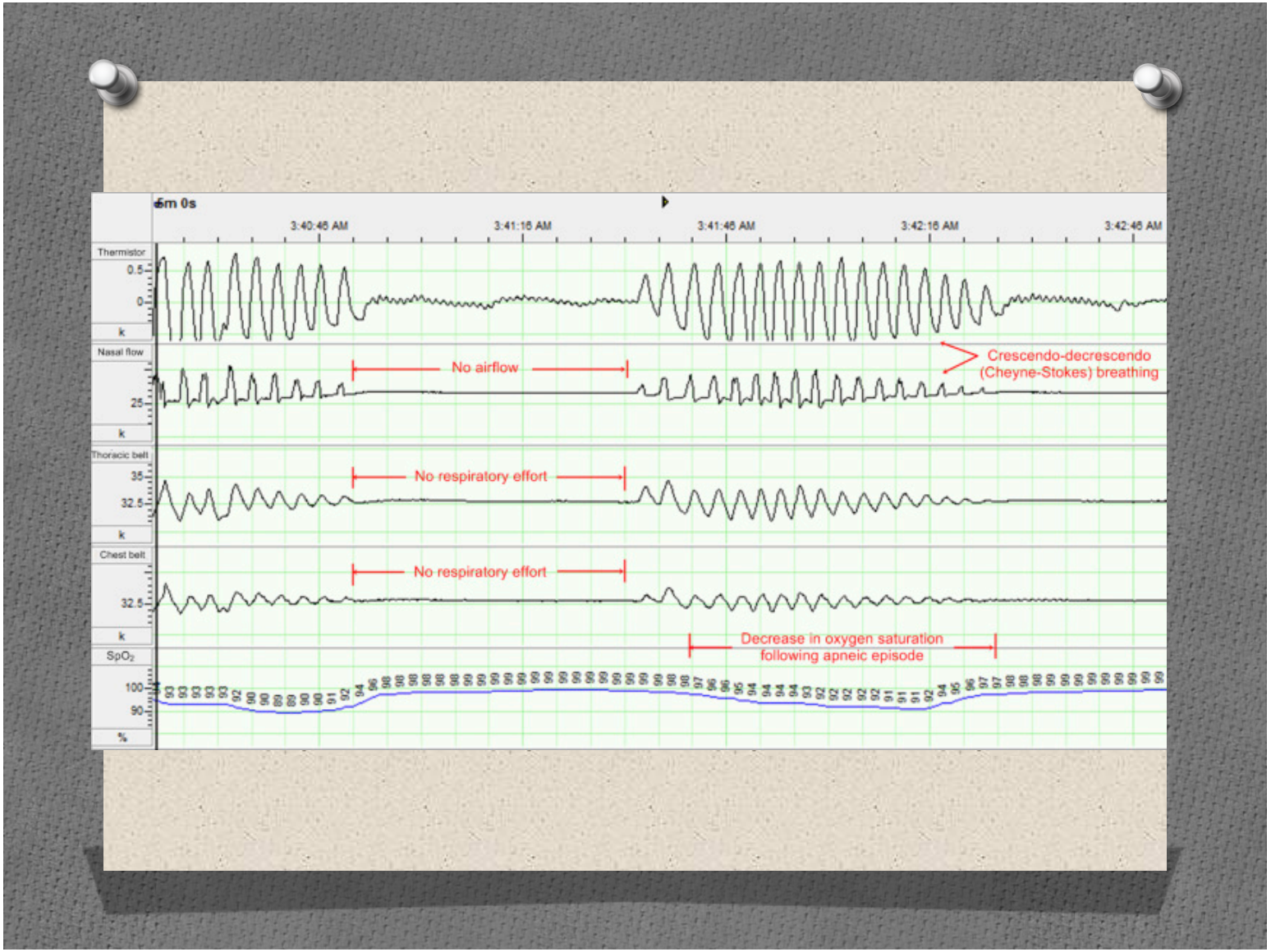
CENTRAL SLEEP APNEA

- The central sleep apnea are characterized by sleep disordered breathing associated with diminished or absent respiratory effort

- coupled with the presence of symptoms including excessive daytime sleepiness, frequent nocturnal awakenings, or both.


(C3) - (M2)
CPAP
PULSE
(L-LEG1) - (R-LEG1)
Snore
SaO2
93
88
94
90
82
93
87
80
91
85
92
86
90
91
PULSE
0.0
(EKG1) - (EKG2)
Airflow
Central Apnea w/ Desat
Central Apnea w/ Desat
Central Apnea w/
Central Apnea w/ Desat
Central Apnea w/ Desat and
ea w/ Desat and Arousal
a w/ Desat and Arousal
esat and Arousal
a w/ Desat and Arousal
Apnea w/ Desat and Arousal
Range 17.0
Nasal Pressure
Chest
Abdomen
Supine


1. Current Epoch
2. Time Axis
4. C4-A1
6. O2-A1
7. LOC-A2
8. ROC-A1
9. CHIN
10. Arousals
13. EKG
16. LEGS
18. Apnea/Hypopnea
20. CFLOW
22. CHEST
23. ABDM
24. Desaturation
509 2'
510 1'
511 2'
512 1'
513 2'
514 2'
A:DESAT
A:DESAT
CA'
CA'
CA'
85.8
86.4
86.4


6m 0s
3:40:46 AM
3:41:16 AM
3:41:46 AM
3:42:16 AM
3:42:46 AM
Thermistor
0.5
0
k
Nasal flow
No airflow
Crescendo-decrescendo
(Cheyne-Stokes) breathing
25
k
Thoracic belt
No respiratory effort
35
32.5
k
Chest belt
No respiratory effort
32.5
k
Decrease in oxygen saturation
following apneic episode
SpO2
100
90
%

- ICSD-3 identifies 6 central sleep apnea syndromes that can affect the adult population.
- (1) Primary CSAS
- (2) Cheyne-Stokes breathing pattern
- (3) Highaltitude periodic breathing
- (4) CSAS due to medical condition not Cheyne Stokes
- (5) CSAS due to drug or substance.
- (6)Treatment emergent CSA

Table 89-2 Classification of Central Sleep Apnea

Hypercapnic ($PCO_2 > 45$) (Decreased respiratory drive)	***Central alveolar hypoventilation***
	Secondary
	Brain stem tumors, infarcts
	Bulbar polio
	Encephalitis
	Primary
	Respiratory neuromyopathy
	Neuromyopathies
	Myotonic dystrophy
	Muscular dystrophy
	Myasthenia gravis
	Amyotrophic lateral sclerosis
	Postpolio syndrome
	Diaphragm paralysis
Nonhypercapnic ($PCO_2 \leq 45$) (Normal or increased respiratory drive)	**Secondary**
	Congestive heart failure (Cheyne-Stokes respiration)
	Brain lesions
	Renal failure
	Acromegaly
	Cerebrovascular disease
	Atrial fibrillation
	High-altitude periodic breathing
	Opioid related
	Complex sleep apnea
	Primary
	Idiopathic central sleep apnea

- “Central sleep apnea is more difficult to treat than the more common obstructive sleep apnea because CPAP and BiPAP machines aren’t as effective
- Because these patients’ diaphragms aren’t receiving signals to breathe, air pressure alone can’t completely relieve their symptoms.”

Oxygen

- Oxygen has been used as a therapeutic intervention in patients with central sleep apnea secondary to heart failure and high altitude CSA .
- The mechanisms underlying the effect of oxygen on ventilatory control during sleep remain elusive

Acetazolamide

- **Acetazolamide has limited supporting evidence but may be considered for the treatment of primaryand other CSA.**

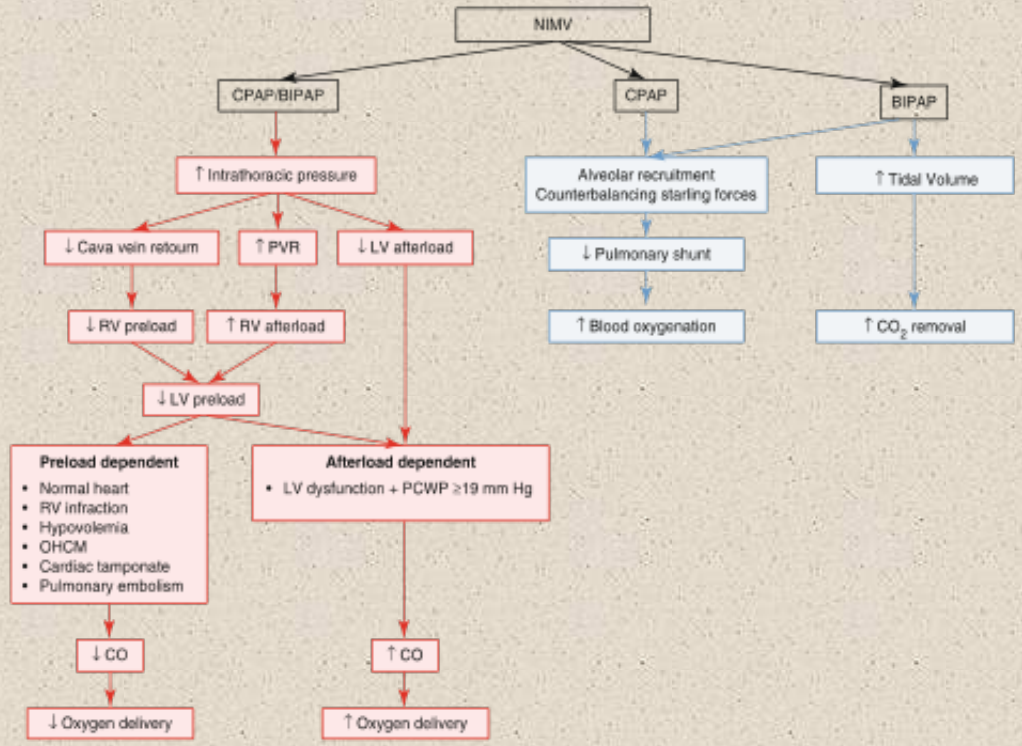
long-term efficacy or potential side effects

ARE LOW

- **The use of zolpidem and triazolam may be considered for the treatment of primary CSA only if the patient does not have underlying risk factors for respiratory depression.**

Continuous Positive Airway Pressure (CPAP)

- Despite optimal therapy of underlying disorders (e.g., heart failure), CSA persists in many patients. Treatment for CSA has used existing approaches for obstructive sleep apnea, most notably continuous positive airway pressure (CPAP) therapy.


NIMV
CPAP/BIPAP
CPAP
BIPAP
↑ Intrathoracic pressure
Alveolar recruitment
Counterbalancing starling forces
↑ Tidal Volume
↓ Cava vein retourn
↑ PVR
↓ LV afterload
↓ Pulmonary shunt
↓ RV preload
↑ RV afterload
↑ Blood oxygenation
↑ CO_2 removal
↓ LV preload
Preload dependent
• Normal heart
• RV infraction
• Hypovolemia
• OHCM
• Cardiac tamponate
• Pulmonary embolism
Afterload dependent
• LV dysfunction + PCWP ≥19 mm Hg
↓ CO
↑ CO
↓ Oxygen delivery
↑ Oxygen delivery

- BPAP in the spontaneous mode may aggravate central apnea caused by hyperventilation. Further investigations are necessary to more definitively characterize the association between BPAP-S and the outcomes of interest

S/T Mode

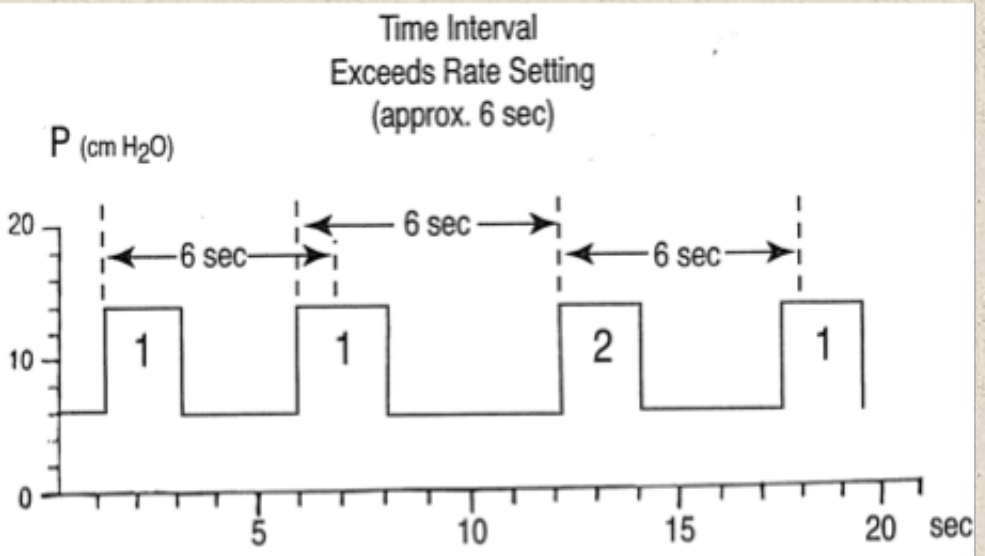
Example:

IPAP = 14 cm H_2O

EPAP = 6 cm H_2O

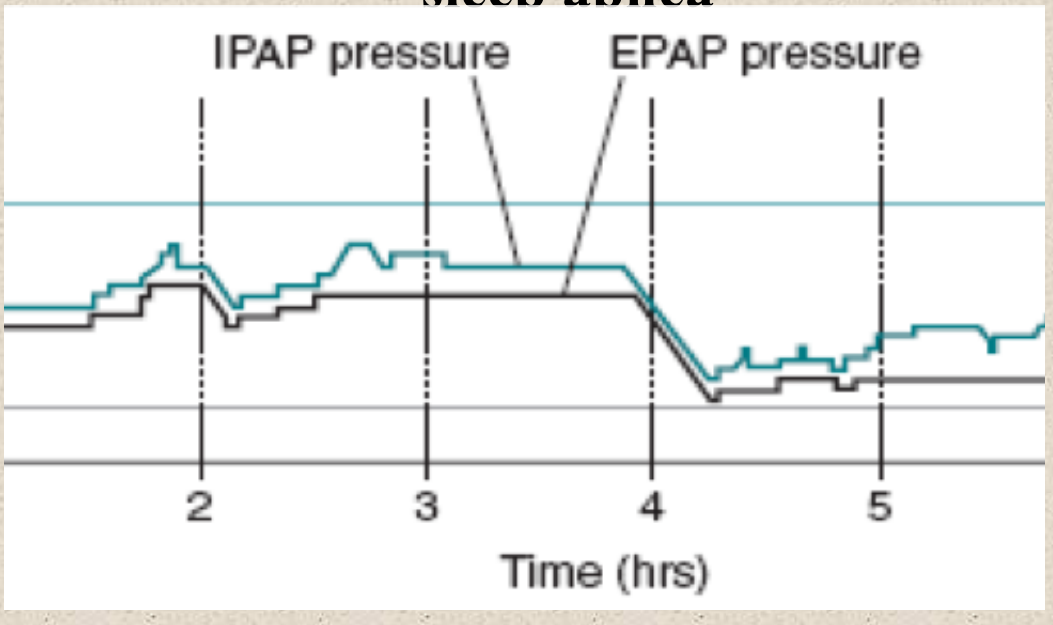
Rate = 10 BPM

PS = 8 cm H_2O

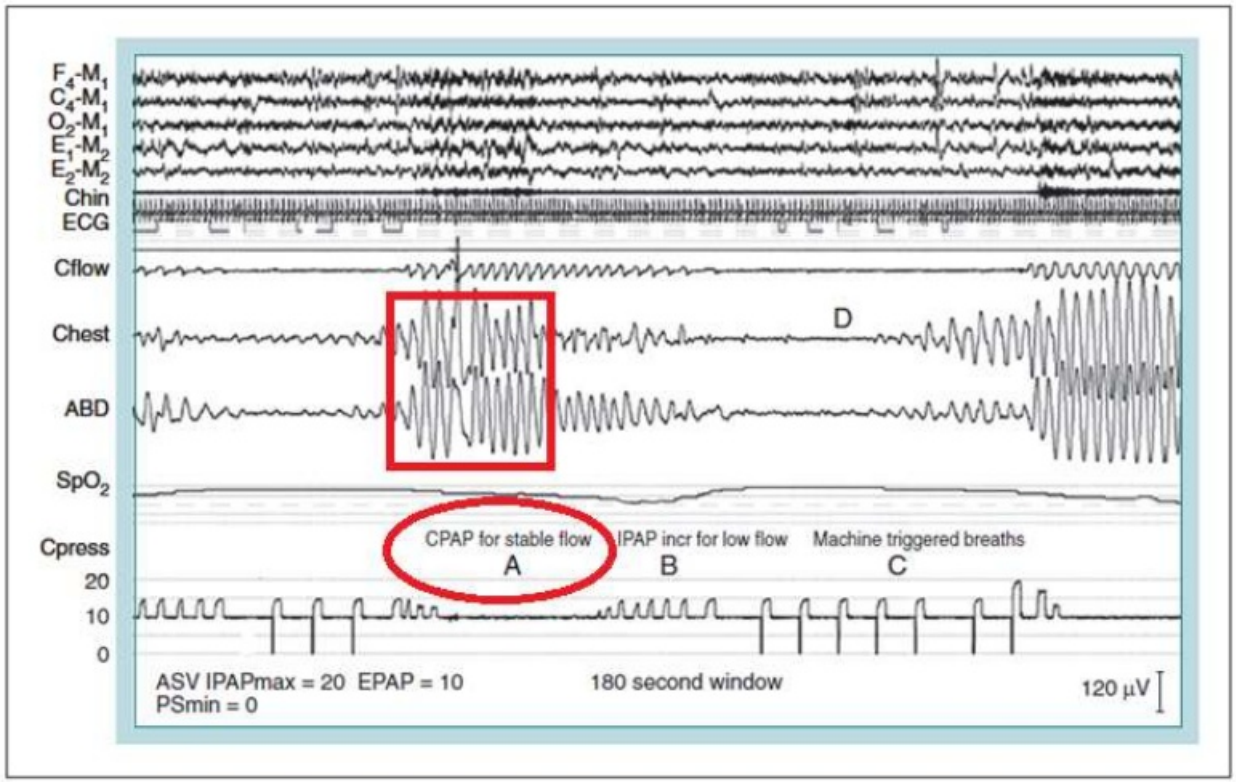


1 = Spontaneously-triggered pressure support breaths.
2 = Time-triggered, pressure-limited, time-cycled breath.

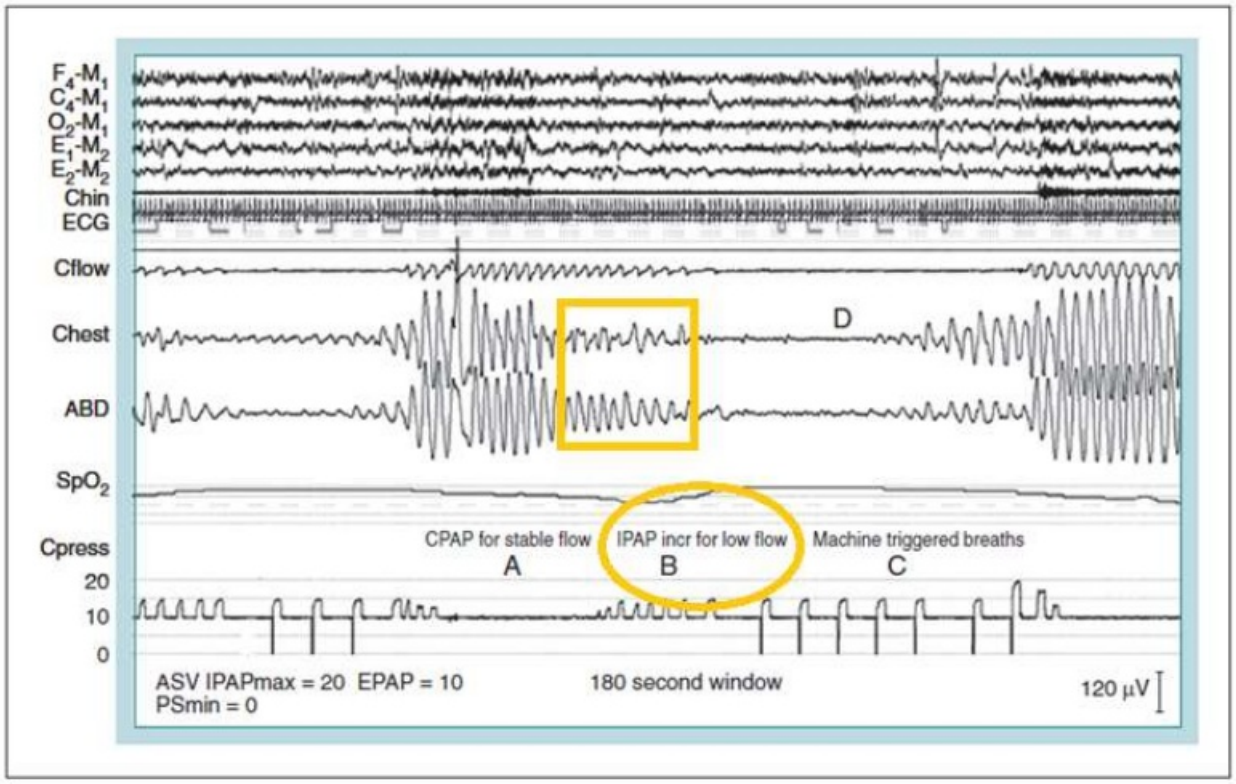
ASV is a variant of BPAP that was developed to treat Cheyne-Stokes central apnea. Both ASV and BPAP devices with a backup rate are approved for use with patients with central apnea and complex sleep apnea



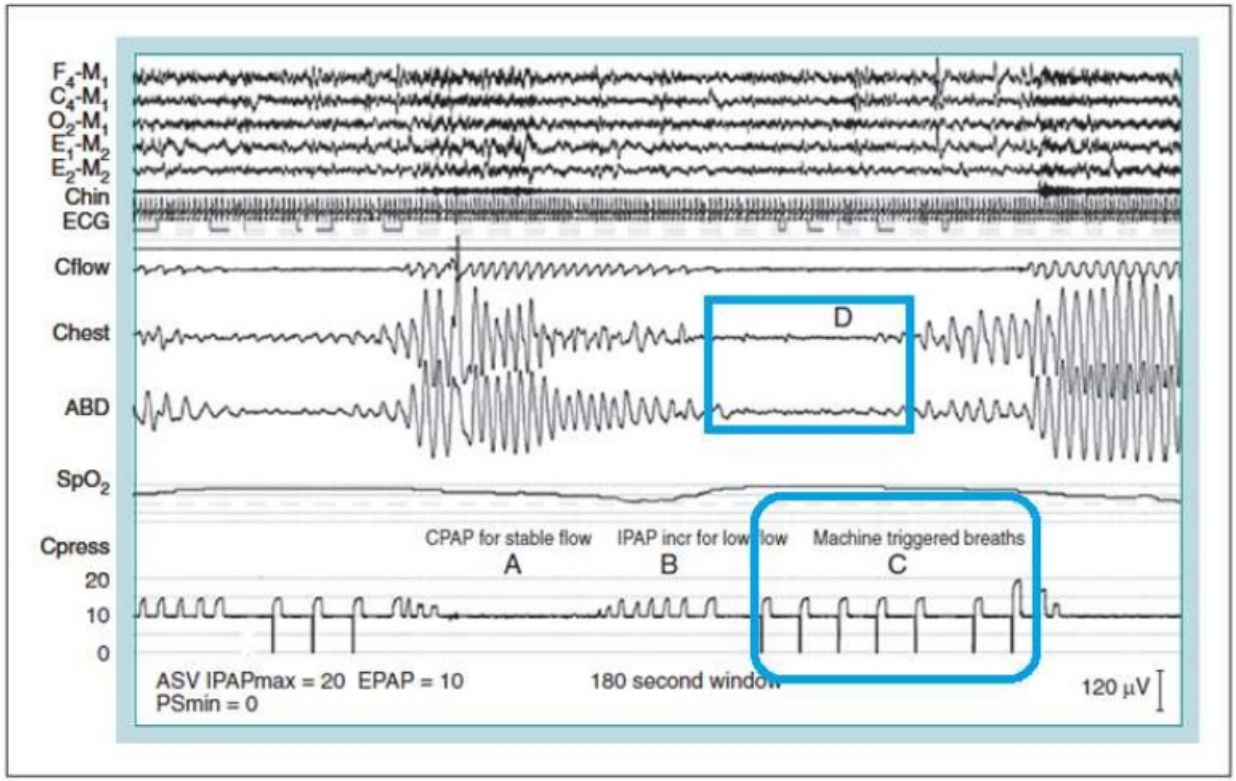
ASV in Cheyne Stokes Breathing



ASV in Cheyne Stokes Breathing



ASV in Cheyne Stokes Breathing

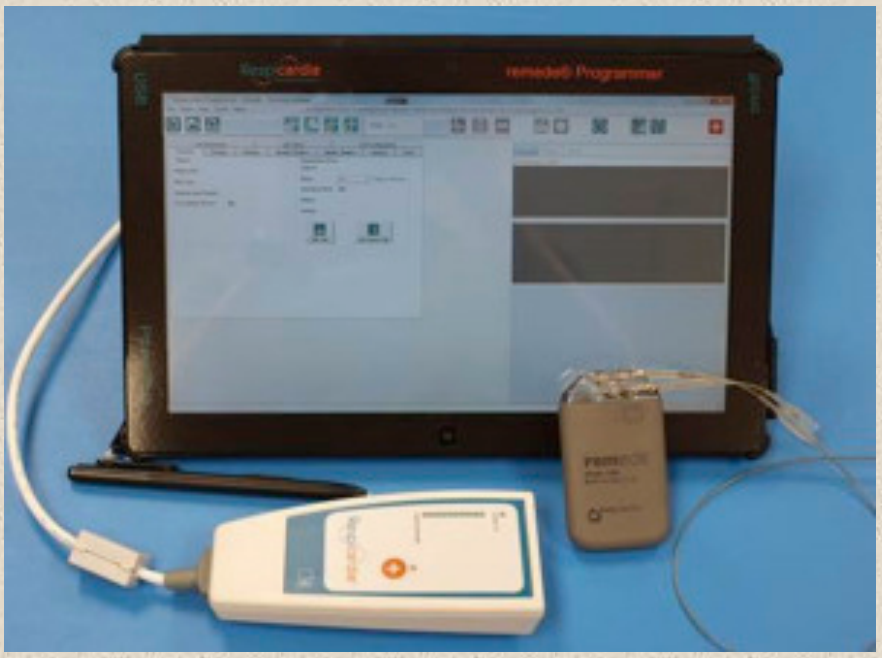


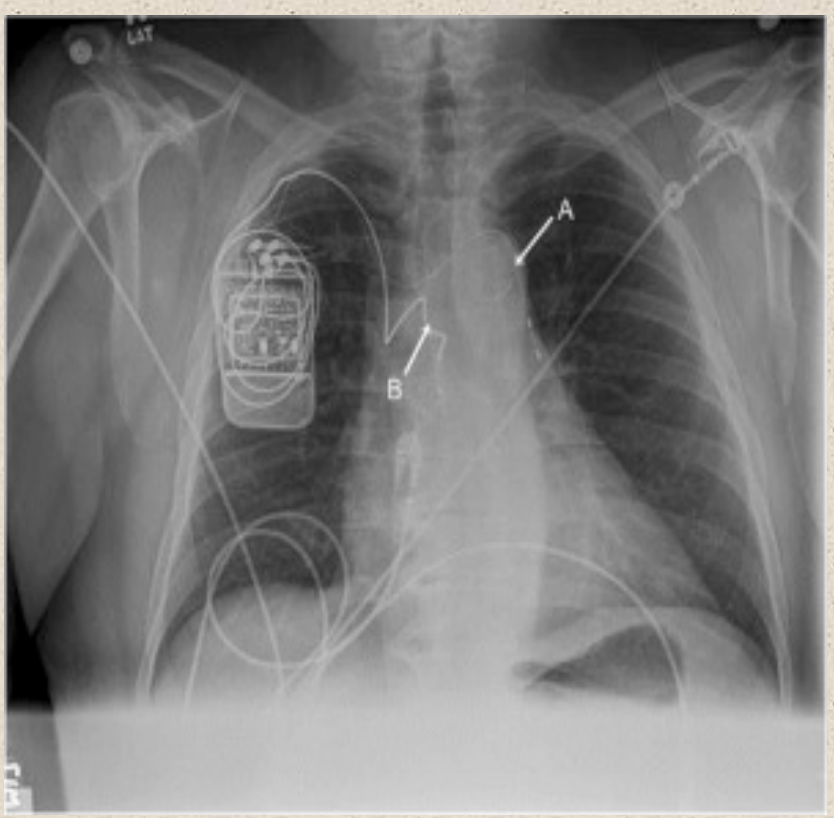
- CSA is defined by cessation of airflow without respiratory effort, in contrast to OSA, where respiratory effort is ongoing.

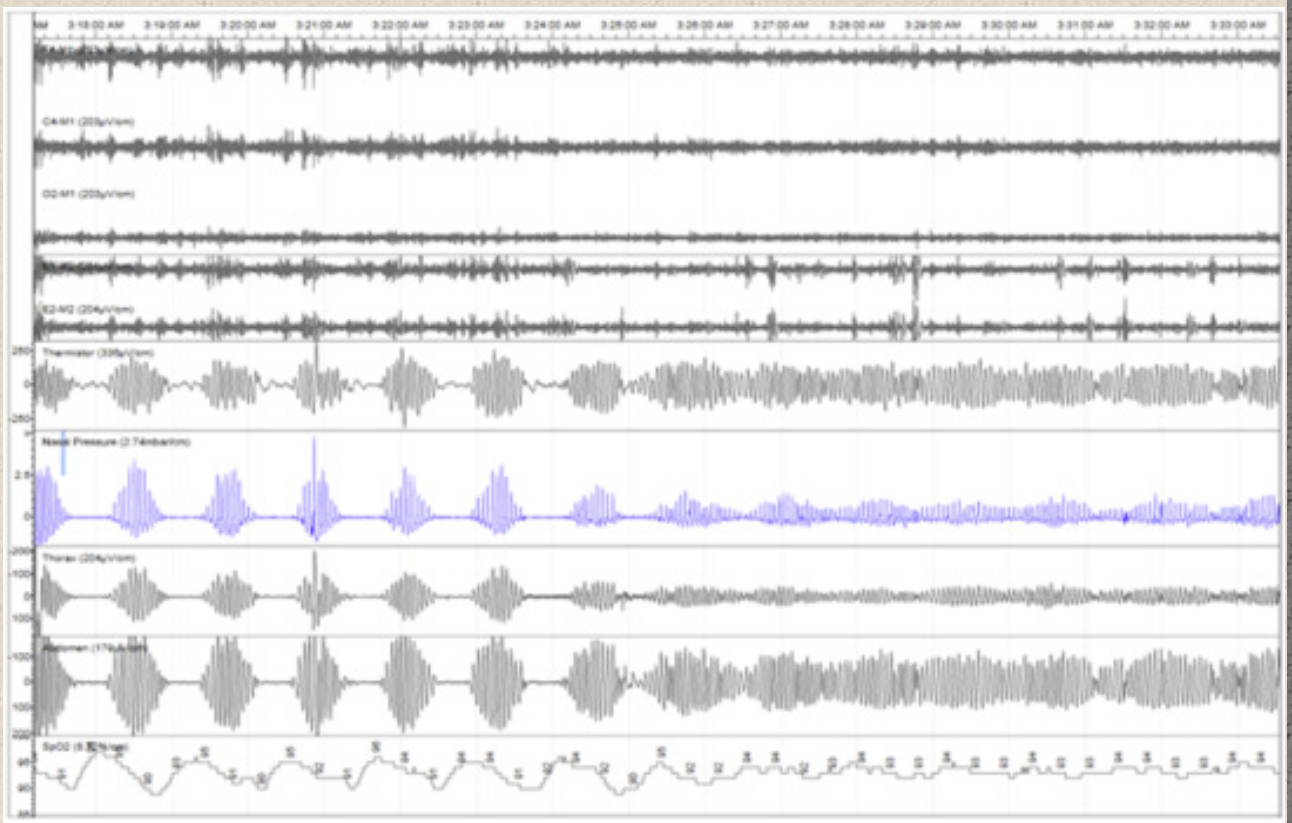
- A minimally invasive process that stimulates the phrenic nerve so that the patient can breathe more naturally. This is the method utilized by the Avery Diaphragm pacemaker.

- All currently available systems involve an external transmitter and an implanted receiver, but fully implantable diaphragmatic pacing systems are being developed.
- The current pacing systems are more affordable and easier to place than the earlier systems. [

- Diaphragmatic pacing should be considered in the following patients:
- Those with apnea from a central cause (the CNS lesion must be higher than C2,C3 because the phrenic nerve originates from the anterior horns of C3-5.
- Patients with amyotrophic lateral sclerosis and polio
- Patients with upper cervical spine injury
- Patients with basilar meningitis or brainstem infarction
- Patients with central sleep apnea

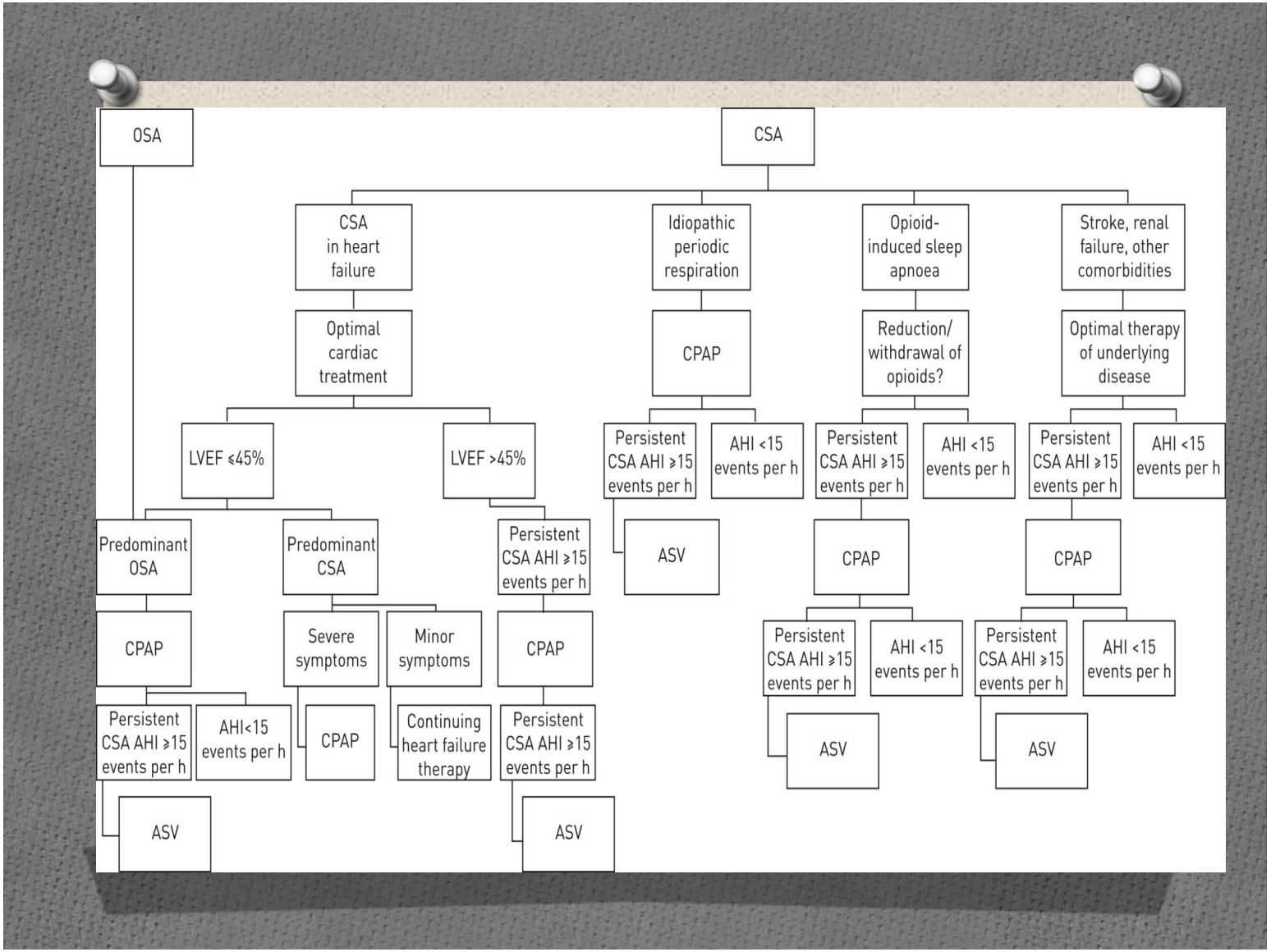

remede® Programmer


A
B



Therapy OFF

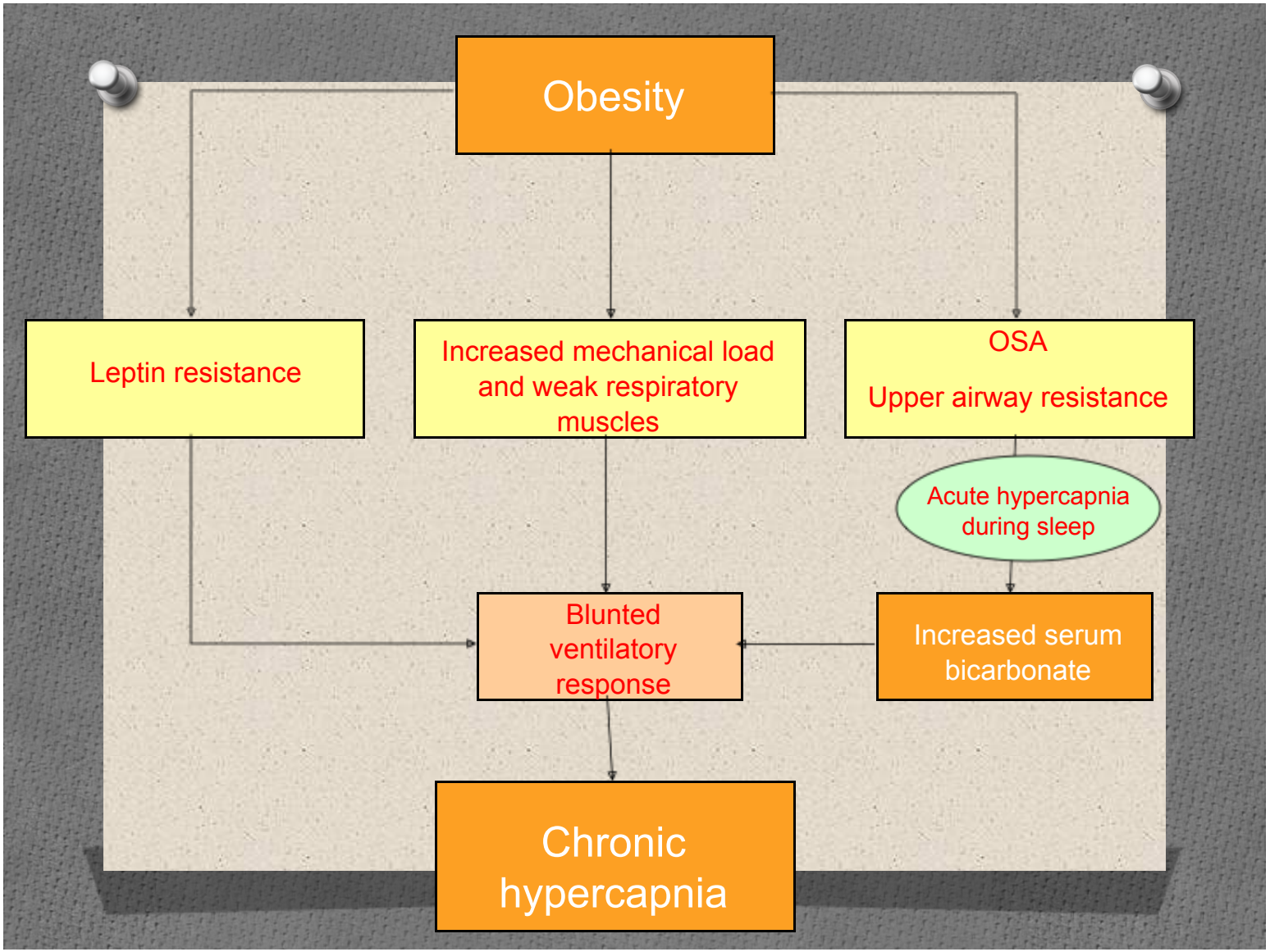
Therapy ON


OSA
CSA
CSA in heart failure
Idiopathic periodic respiration
Opioid-induced sleep apnoea
Stroke, renal failure, other comorbidities
Optimal cardiac treatment
CPAP
Reduction/ withdrawal of opioids?
Optimal therapy of underlying disease
LVEF ≤45%
LVEF >45%
Persistent CSA AHI ≥15 events per h
AHI <15 events per h
Persistent CSA AHI ≥15 events per h
AHI <15 events per h
Persistent CSA AHI ≥15 events per h
AHI <15 events per h
Predominant OSA
Predominant CSA
Persistent CSA AHI ≥15 events per h
ASV
CPAP
CPAP
CPAP
Severe symptoms
Minor symptoms
CPAP
Persistent CSA AHI ≥15 events per h
AHI<15 events per h
CPAP
Continuing heart failure therapy
Persistent CSA AHI ≥15 events per h
Persistent CSA AHI ≥15 events per h
AHI <15 events per h
Persistent CSA AHI ≥15 events per h
AHI <15 events per h
ASV
ASV
ASV
ASV

OHS: Definition

Thomas Nast, The Pickwick Papers

- Obesity (BMI ≥30 kg/m2)
- Hypercapnia (PaCO2 ≥ 45 mmHg)
- Sleep-disordered breathing


Obesity
Leptin resistance
Increased mechanical load and weak respiratory muscles
OSA
Upper airway resistance
Acute hypercapnia during sleep
Blunted ventilatory response
Increased serum bicarbonate
Chronic hypercapnia


با سپاس