Consequences of Sleep Disordered Breathing

from the SHHS, WSCS, NHHS

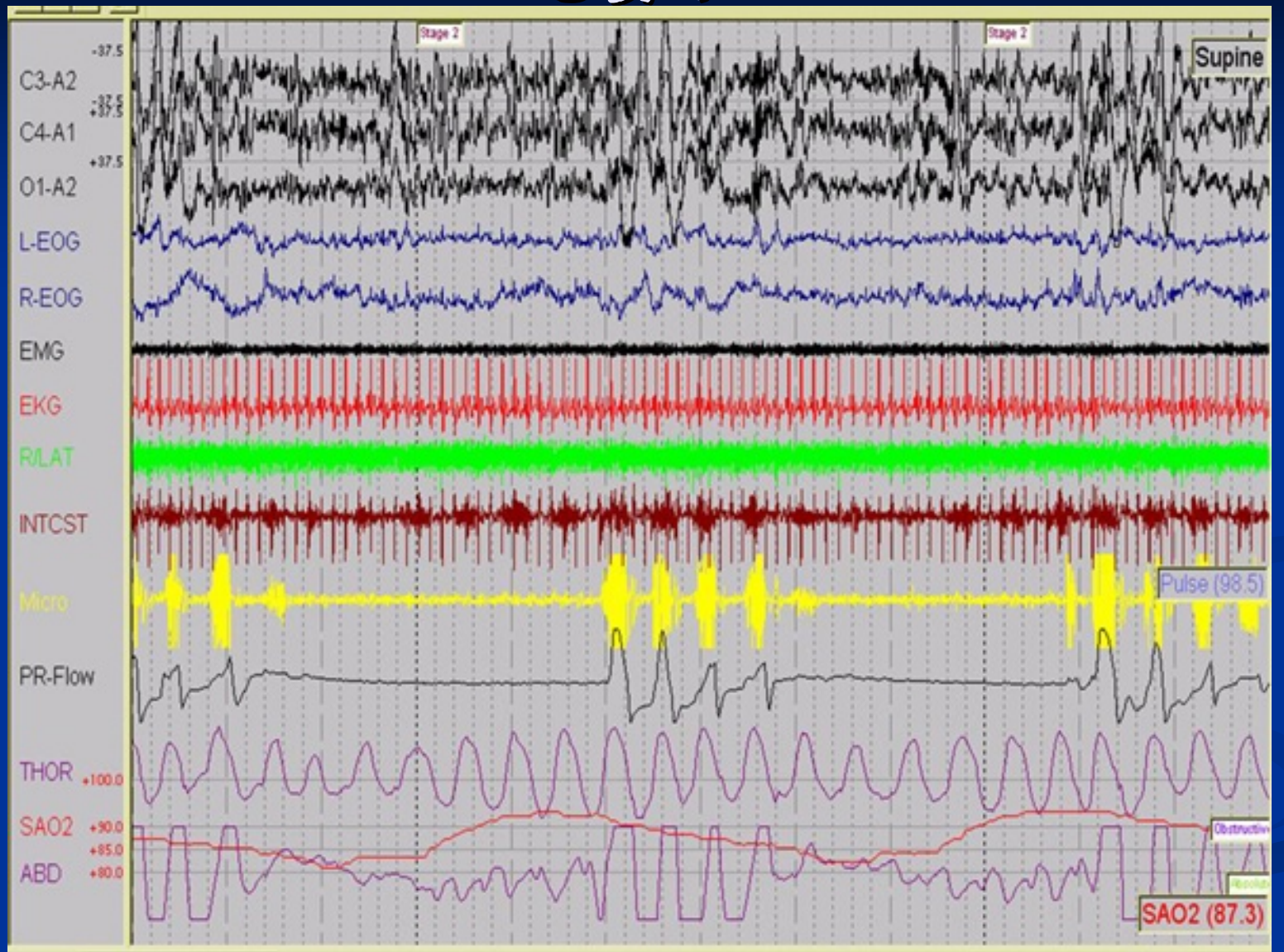
Andrew G Veale

New Medical Knowledge

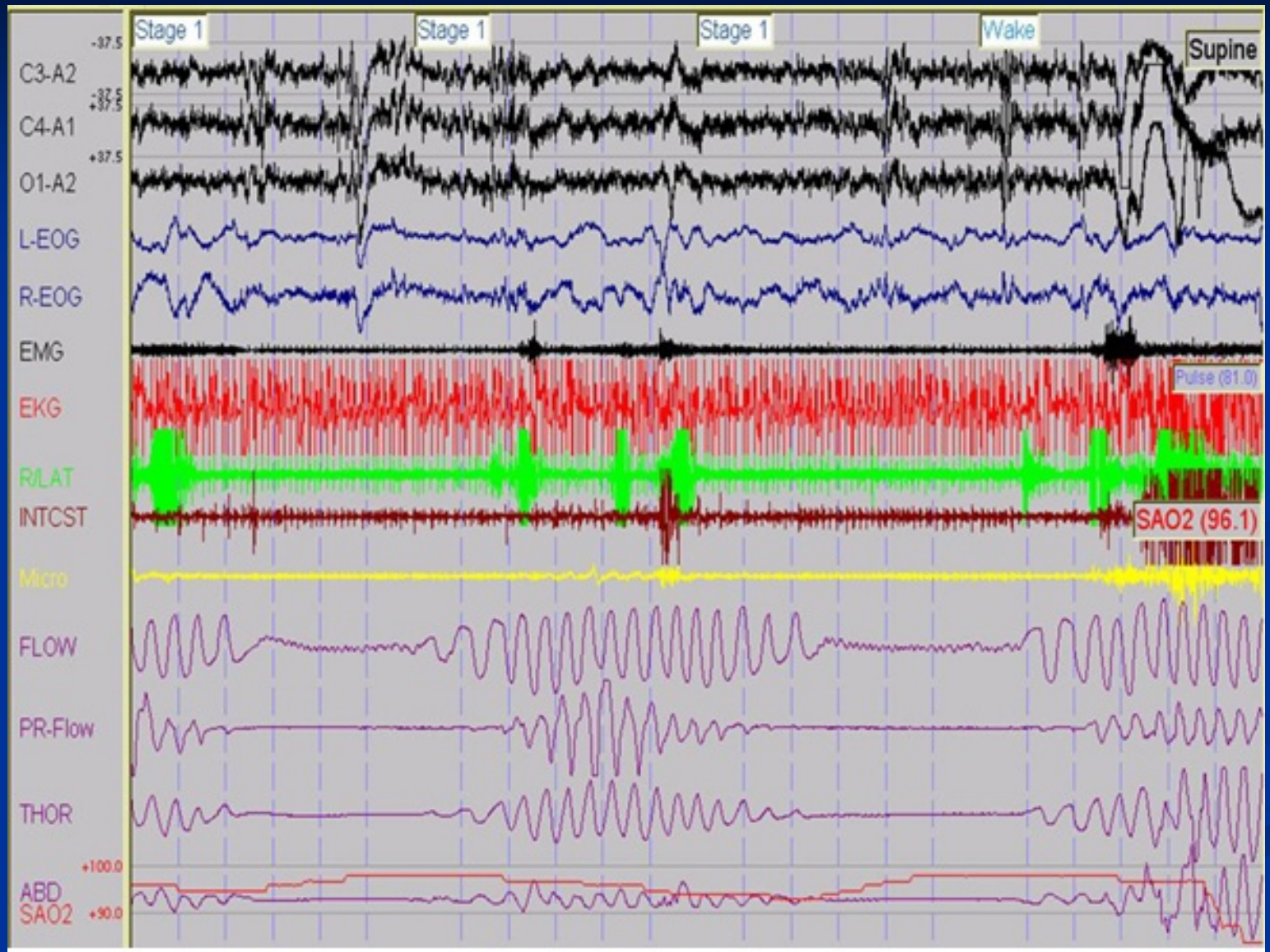
- Observe a problem
- Describe the problem
- Look for associations
- Find a test
- Work out a treatment
- Treat more patients (on research money)
- Ask for more money
- Management notice
- Epidemiology - size of the problem
- Show the treatment works
- Determine why the treatment works
- Randomize to prove the treatment works
- Show that it saves money

Lofty

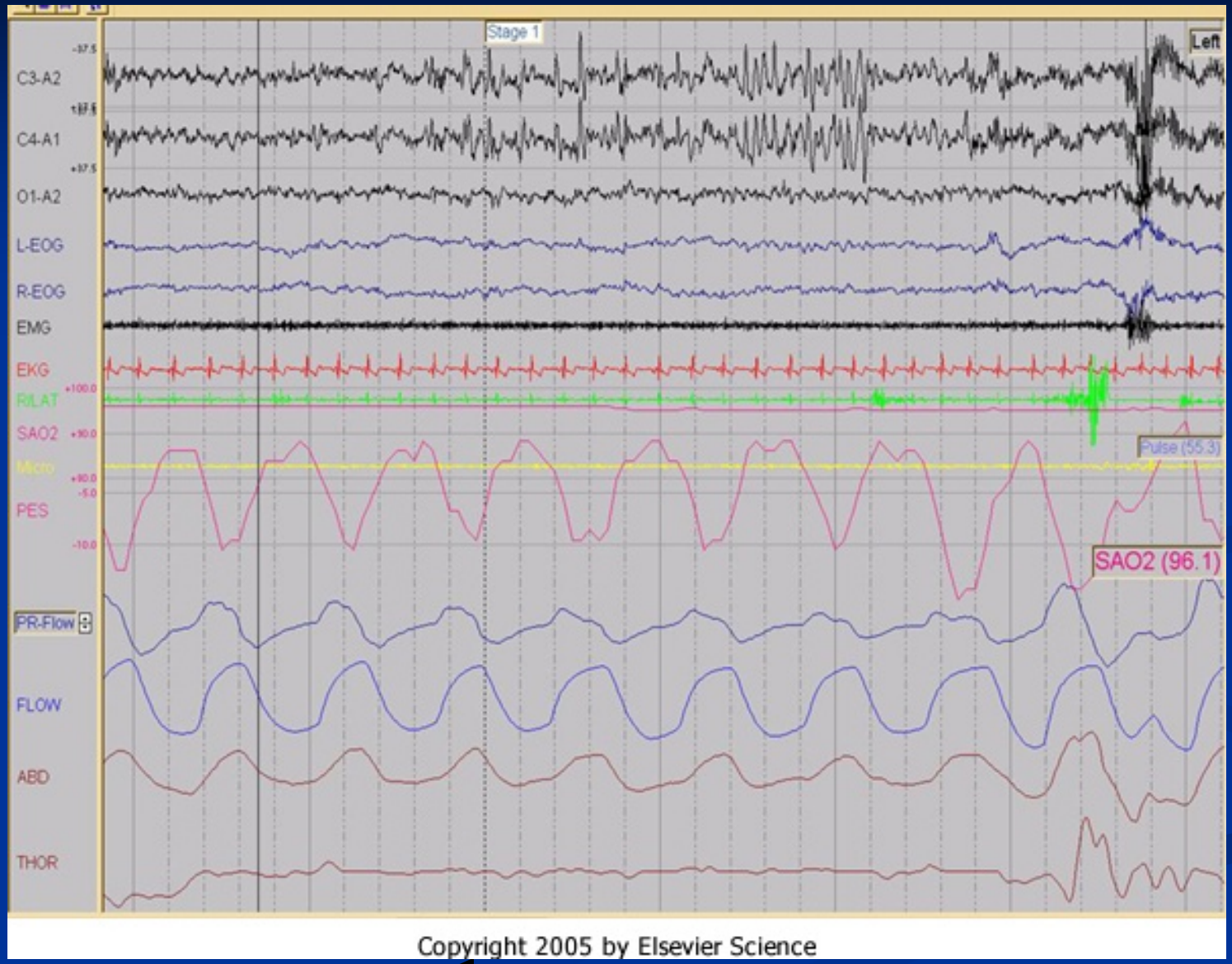
OSA



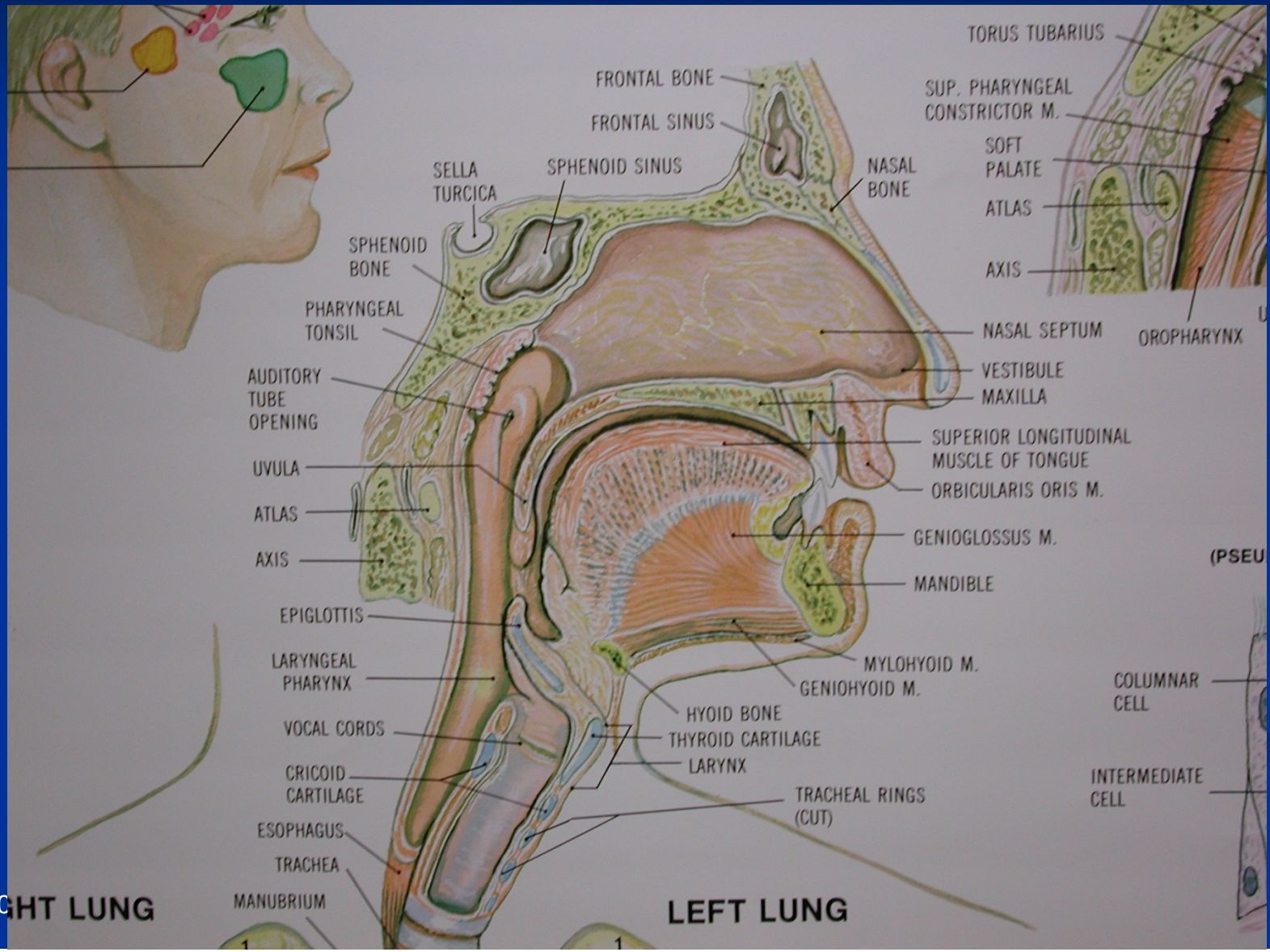
CSR



UARS



Anatomy


TORUS TUBARIUS
FRONTAL BONE
SUP. PHARYNGEAL CONSTRICTOR M.
FRONTAL SINUS
SOFT PALATE
SELLA TURCICA
SPHENOID SINUS
NASAL BONE
ATLAS
SPHENOID BONE
AXIS
PHARYNGEAL TONSIL
NASAL SEPTUM
OROPHARYNX
VESTIBULE
AUDITORY TUBE OPENING
MAXILLA
SUPERIOR LONGITUDINAL MUSCLE OF TONGUE
UVULA
ORBICULARIS ORIS M.
ATLAS
GENIOGLOSSUS M.
AXIS
MANDIBLE
EPIGLOTTIS
MYLOHYOID M.
LARYNGEAL PHARYNX
GENIOHYOID M.
COLUMNAR CELL
HYOID BONE
VOCAL CORDS
THYROID CARTILAGE
LARYNX
CRICOID CARTILAGE
INTERMEDIATE CELL
TRACHEAL RINGS (CUT)
ESOPHAGUS
TRACHEA
MANUBRIUM
LEFT LUNG

Pathophysiology

- Abnormally narrowed airway
- Increased collapsibility
- Airway collapse – multi-level problem
  - Palate, base of tongue, pharynx, supraglottis or all levels
- Increased effort
- Sympathetic outpouring
- Desaturation
- Arousal

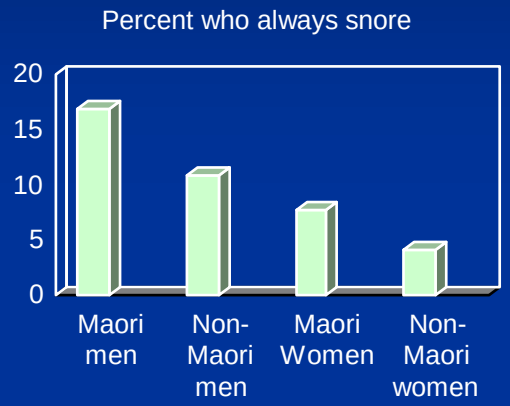
Presentation

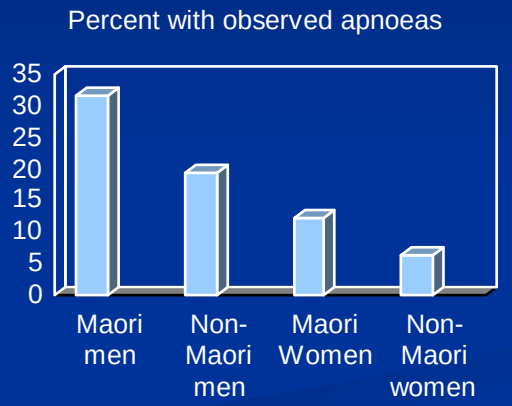
- Patient complains of:
  - Poor sleep quality
  - Always tired
  - Impotence
  - Headaches
  - Enuresis
  - GE reflux
- Wife complains of:
  - Snoring
  - Apnoeas
  - Mood changes
  - Impotence
- Doctor should notice:
  - Asleep in waiting room
  - Difficult Hypertension
  - Difficult diabetes
  - Congestive cardiac failure
  - Obesity
  - Known associations (Hypothyroidism, Acromegally, Abnormal facies, Ehlers Danlos Syndr. Etc)

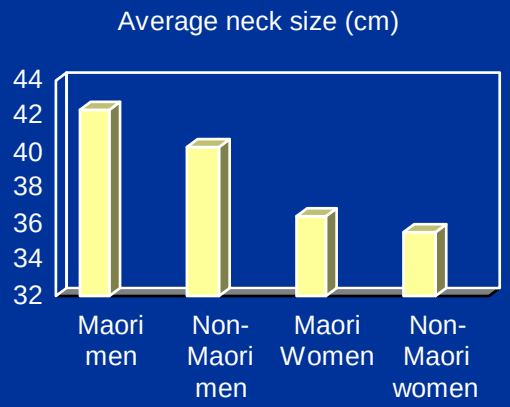
Epidemiology - Obstructive Sleep Apnea Syndrome

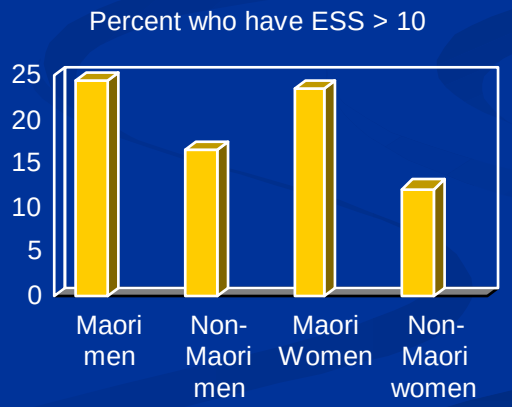
Reference,	Country	Methods	Subjects, N	Age (yr)	Criteria	Prevalence, %
Lavie (1983),163	Israel	Questionnaire. PSG	1262 (m)	18-67	AI >10, symptomatic	1.0-5.9
Peter et al. (1985),164	Germany	Questionnaire. PSG	354 (m)	25-55	AI >10, symptomatic	2.3
Telakivi et al. (1987),42	Finland	Questionnaire. PSG	1939 (m)	30-69	Snoring, EDS, and RDI >10	0.4-1.4
Gislason et al. (1988),41	Sweden	Questionnaire. PSG	3201 (m)	30-69	Snoring, EDS, and AHI >10	0.7-1.9
Cirignotta et al. (1989),156	Italy	Questionnaire. PSG	1170 (m)	30-39	AI >10, symptomatic	0.2 – 1.0
				40-59	AI >10, symptomatic	3.4 – 5.0
				60-69	AI >10, symptomatic	0.5 – 1.1
Stradling & Crosby (1991),165	Great Britain	Ambulatory oximetry recordings	893 (m)	35-65	ODI4 >20, symptomatic	0.3
					ODI4 >10	1.0
					ODI4 >5	4.6
Haraldsson et al. (1992),166	Sweden	Questionnaire. PSG	846 (m)	30-69	History and PSG	2.8-5.5
Young et al. (1993),32	Wisconsin, USA	PSG	352 (m)	30-60	RDI>5 Sleepy	4.0
			250 (f)	30-60	RDI>5 Sleepy	2.0
Gislason et al.(1993),167	Iceland	Questionnaire. PSG	2016 (f)	40-59	EDS, PSG	>2.5
Olson et al. (1995),168	Australia	Questionnaire home SS	1233 (m)	35-69	AHI ≥15	4-18
			969 (f)	35-69	AHI ≥10	7-35
					AHI ≥5	14-69
Bearpark et al. (1995),169	Australia	MESAM IV	294 (m)	40-65	RD ≥10	10.0
					Subjective EDS and RDI ≥5	≥3.0
Gislason (1995),49	Iceland	Questionnaire. PSG	555 children	6 mo to 6 yr	Snoring or apnea & ODI4 > 3	>2.9
Esnaola et al. (1995),170	Spain	Questionnaire. PSG	1077 (m)	30-70	AHI ≥5	15.3
					AHI ≥10	13.4
					AHI ≥5 and EDS	6.5-9.1
hayon et al. (1997),171	Great Britain	Telephone (Sleep-EVAL)	2078 (m)	35-64	NA	2.4-4.6
			2894 (f)	35-64	NA	0.8-2.2
Kripke et al. (1997),172	San Diego, US	Telephone, oximeter, snoring	165 (m)	40-64	ODI4 >20	5.4-13.2
			190 (f)	40-64	ODI4 > 20	2.1-8.3
Bixler et al. (1998),38	United States	Telephone PSG (sample)	4364 (m)	20-100	AHI > 10 and clinical criteria	All: 3.3

Symptoms, Risk Factors





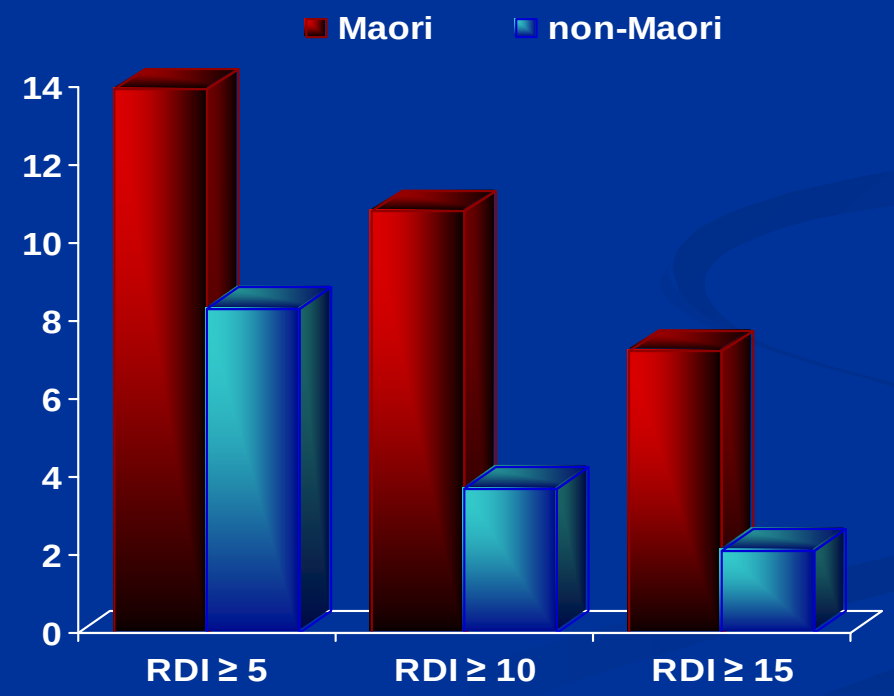




Source: Random sample of 10,000 NZ adults aged 30-60 yrs, 71% response rate
Data courtesy of Dr Ricci Harris

4% O_2 Desaturations / Hour

- Random sample from electoral roll, 30-60 years
- 169 Maori, 195 non-Maori
- Overnight MESAM4 monitoring at home



Data courtesy of Kara Mihaere

Hypertension

STUDY	Age	N	RDI <1	RDI 1-4.9	RDI 5-14.9	RDI > 15	RDI >30
WSCS	30-65	709	1.0	1.2	2.0	2.9	
SHHS	40-97	6132	1.0	1.1	1.2	1.3	1.4
S Penn	20-100	1741			2.3	6.9	
Vitoria	30-70	552	1.0	2.5	1.3	2.3	

Hypertension treatment

Prospective study 420,000 over 10 years

Reduction of BP	Reduction in Stroke	Reduction in CAD
5mHg	34%	21%
7.5mmHg	46%	29%
10mmHg	56%	37%

CPAP reduction 5 – 10 mmHg

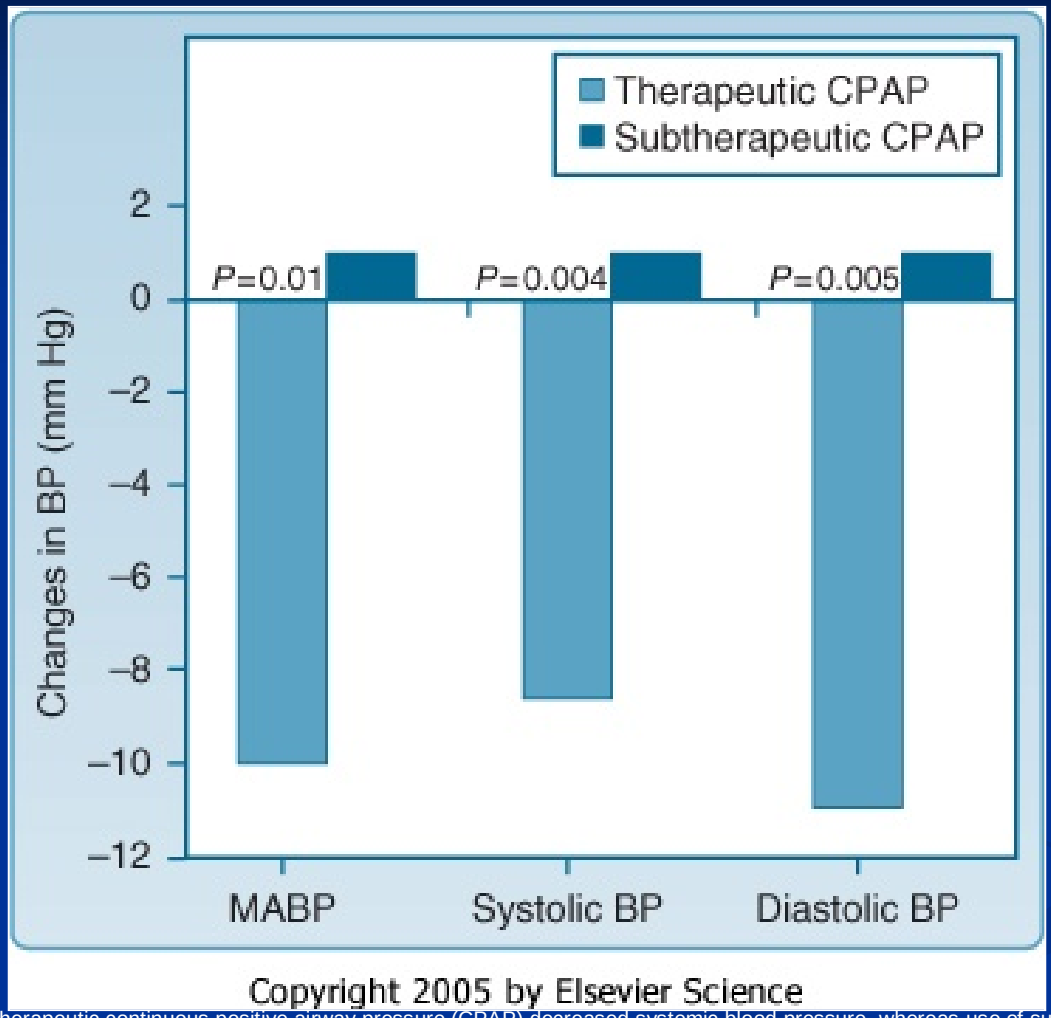


Figure 100-2 Treatment with therapeutic continuous positive airway pressure (CPAP) decreased systemic blood pressure, whereas use of subtherapeutic CPAP failed to decrease blood pressure. (From Becker HF, Jerrentrup A, Ploch T, et al: Effect of nasal continuous positive airway pressure treatment on blood pressure in patients with obstructive sleep apnea. Circulation 2003;107:68-73.)

Recommendation – When to test

- All new hypertensives
- All hypertensive patients not controlled on 1 drug
- All patients with severe HT
- All patients with loss of nocturnal dip in BP
- All patients with diastolic dysfunction

Pulmonary Hypertension

Study	N	Criteria	Prevalence
Chaouat	220	AHI > 20	17% PAP > 20
Laks	100	AHI > 20	42%PAP > 20 (20-52)
Sanner	92	AHI > 10 (10-100)	20% PAP > 20
			8 had increased PCWP
			all were hypertensive

Pre-capillary factors	Hypoxia, hypercapnia, Intrathoracic pressure changes, Endothelial damage.
Capillary factors	Reduction of capillary bed from co-morbidities.
Post-capillary factors	Increased LVEDP

Atrial Fibrillation

- Cardioverted patients with OSA
  - Untreated – 82% recurrence @ 12 mths
  - CPAP treated – 42% recurrence @ 12 mths
  - Non OSA Pts – 53%

Sudden death

- 46% OSA patients die between 12mn and 8am
- 21% in people without OSA

- RDI >40 have a 40% greater risk of nocturnal death than RDI 5-39

- 5 to 7% risk in non snorers
- 12%risk in habitual snorers
- 20% risk in OSA OR 3.6

- OR 1.4 in SHHS

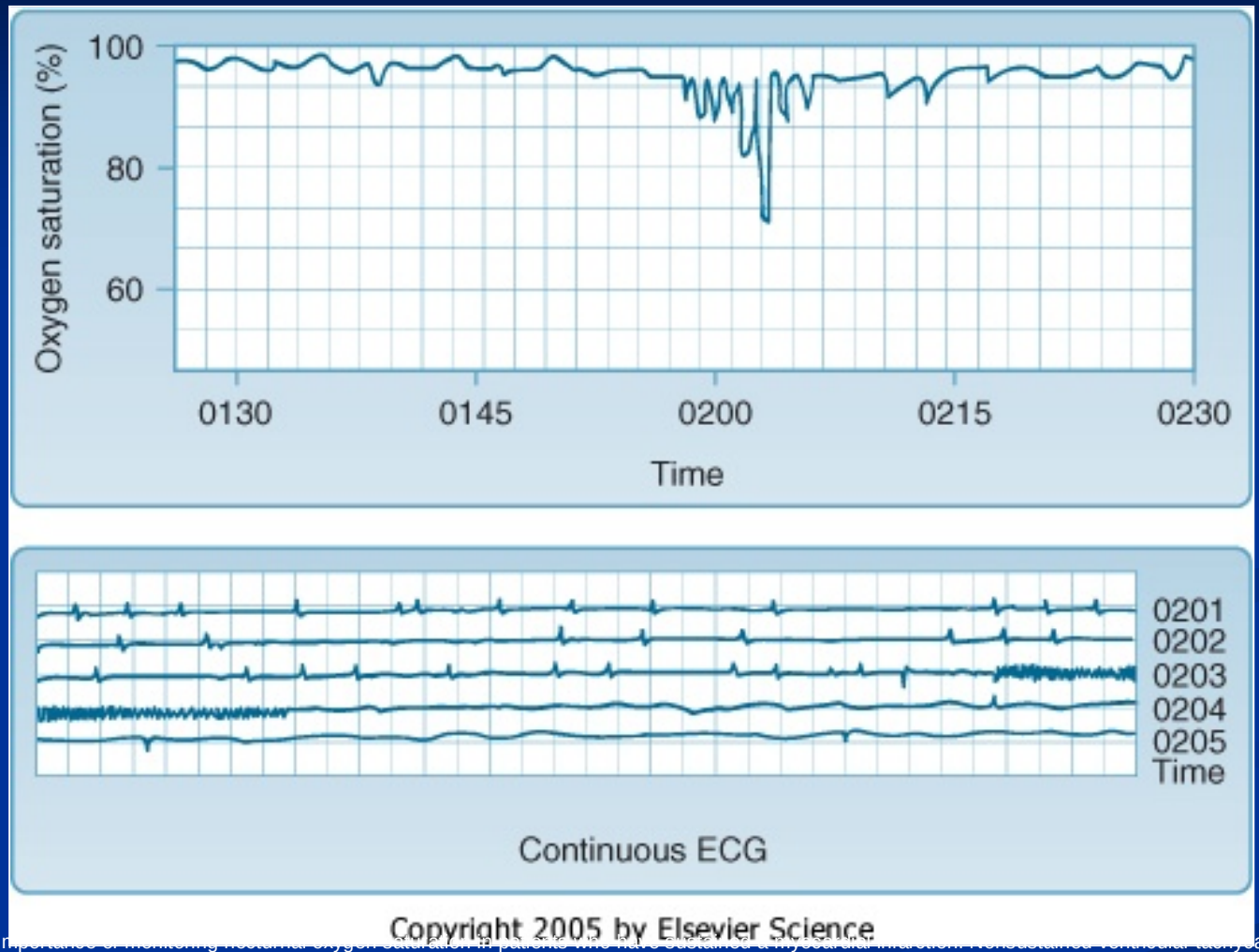


Figure 97-4 Importance of monitoring nocturnal oxygen saturation in patients who have suffered myocardial infarction. Nonsustained ventricular tachycardia (lower panel) and hypoxemia measured by pulse oximetry (upper panel) occurred simultaneously in a patient on the third night after infarction. The patient died on the following day of cardiogenic shock. ECG, electrocardiogram. (From Galatius-Jensen S, Hansen J, Rasmussen V, et al: Nocturnal hypoxemia after myocardial infarction: Association with nocturnal myocardial ischaemia and arrhythmias. Br Heart J 1994; 72:23-30.)

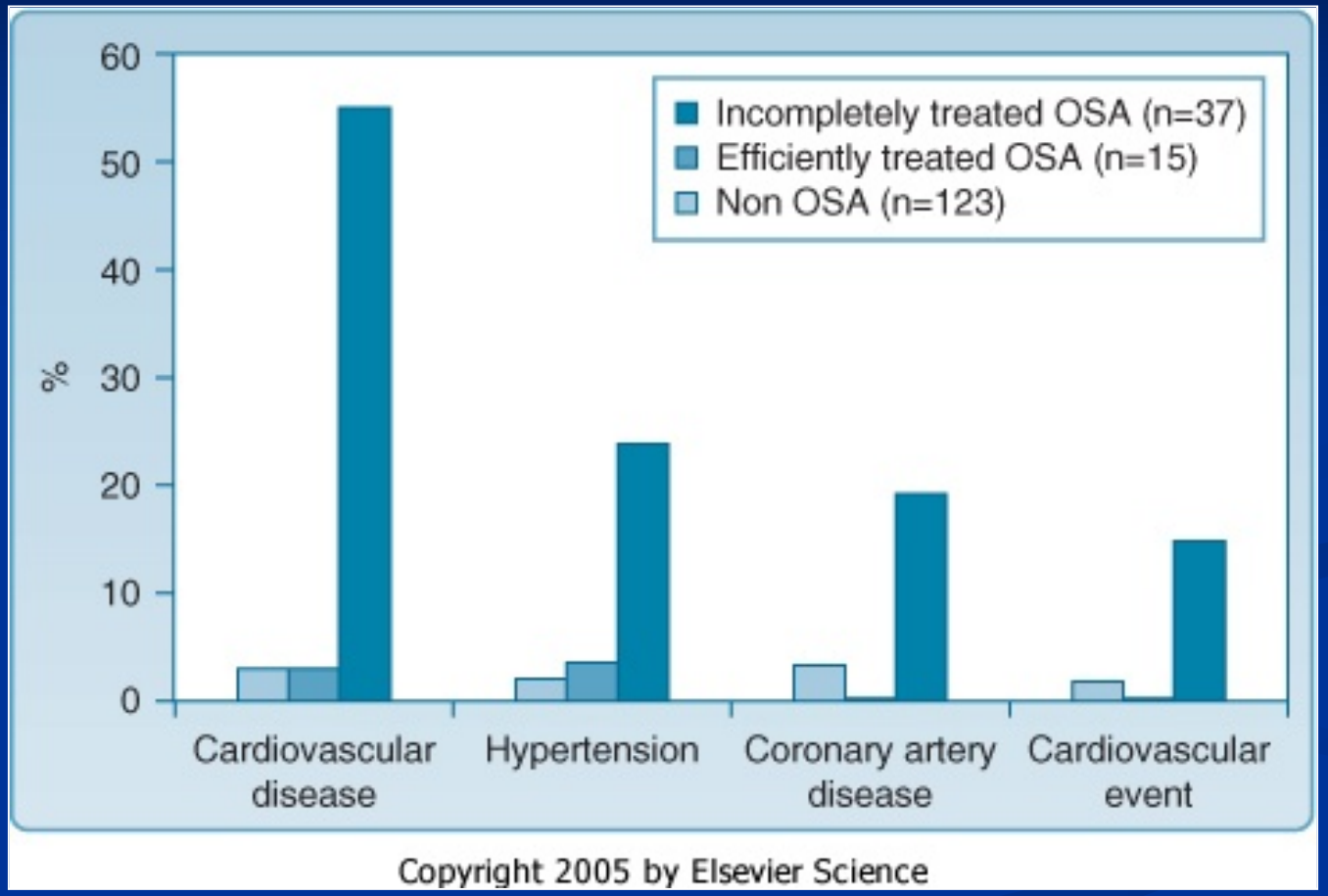


Figure 101-1 Incidence of cardiovascular disease during a 7-year follow-up in middle-aged men otherwise healthy at baseline. Fraction of individuals with incidence of cardiovascular disease, hypertension, coronary artery disease (CAD), and cardiovascular event (stroke, myocardial infarction [MI], or cardiovascular death). Depicted are data from patients without OSA (non OSA) as well as from those incompletely or efficiently treated for their sleep and breathing disorder. (Reprinted from Peker Y, Hedner J, Norum J, et al: Increased incidence of cardiovascular disease in middle-aged men with obstructive sleep apnea: A seven-year follow-up. Am J Respir Crit Care 2002;166:159-165.)

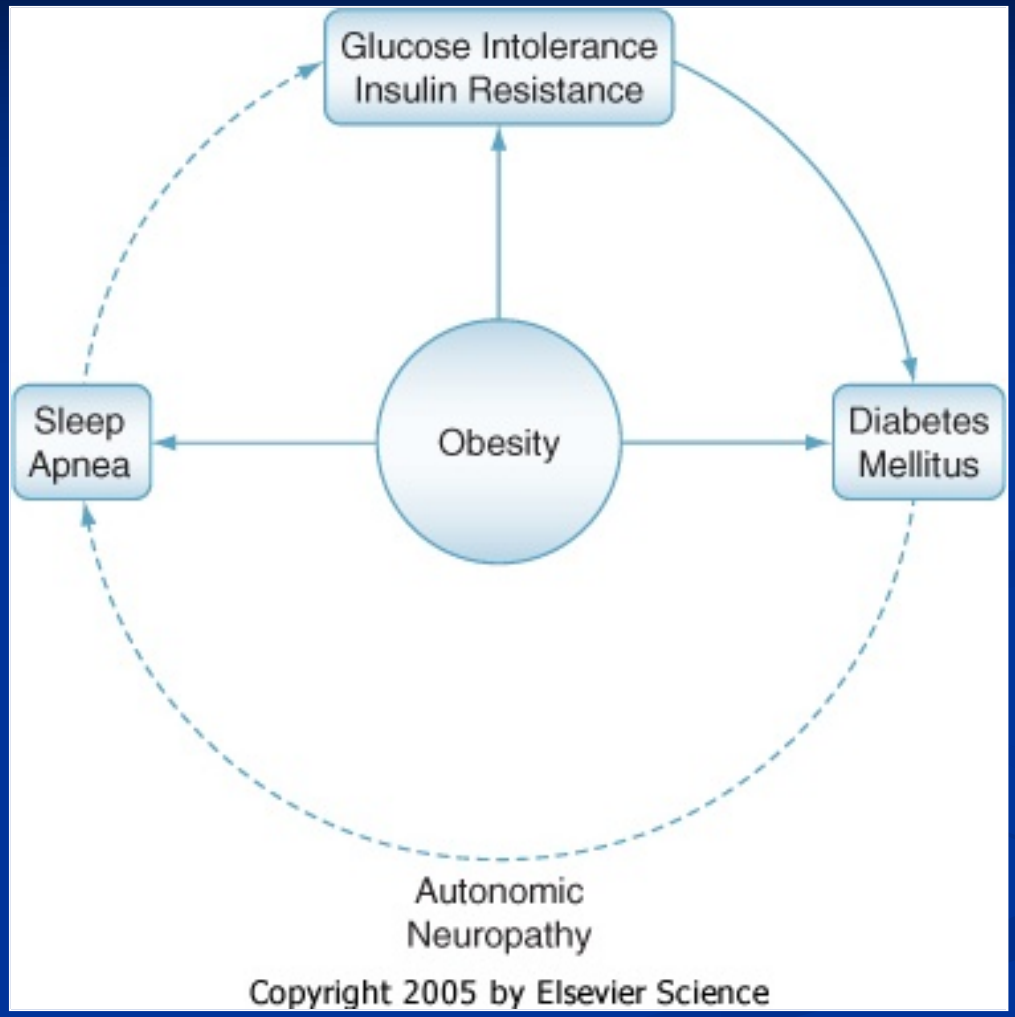


Figure 86-1 Sleep apnea and diabetes mellitus.

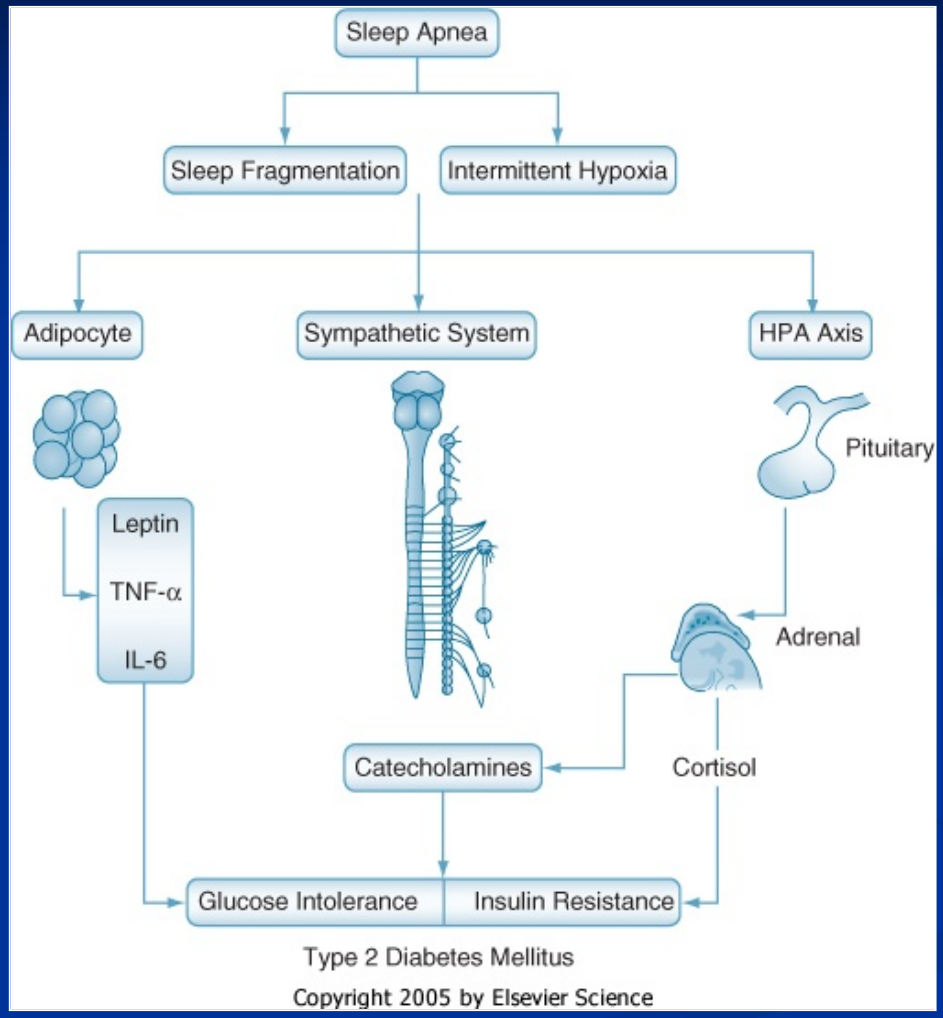


Figure 86-2 Intermediate pathways linking sleep apnea, glucose intolerance, and insulin resistance.

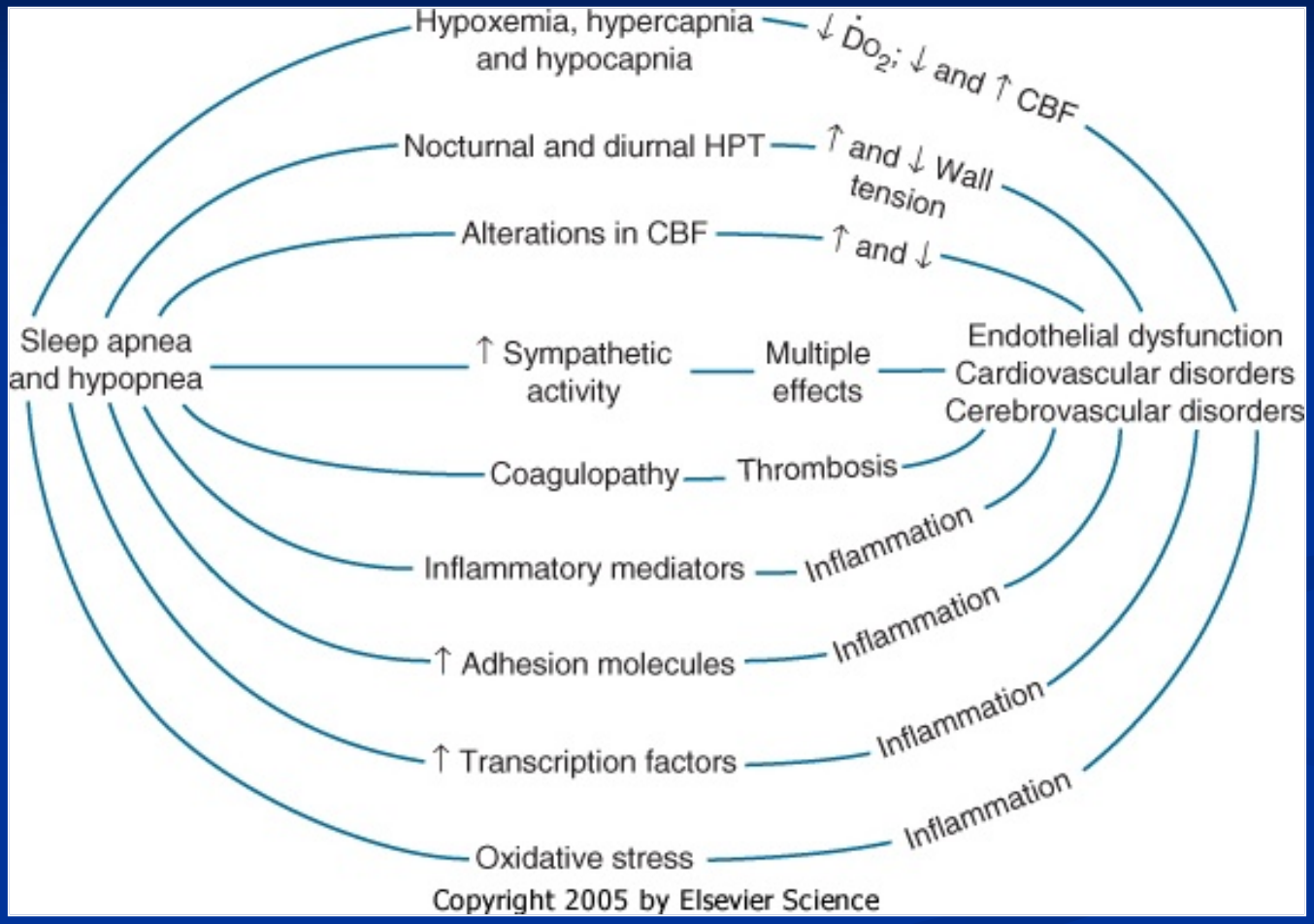


Figure 96-2 The mechanisms by which sleep apnea may result in endothelial dysfunction and cerebrovascular and cardiovascular disorders. CBF, coronary/cerebral blood flow; Do2, oxygen delivery; HPT, hypertension; ↑, increase; ↓, decrease. (Adapted from Javaheri S: Heart failure and sleep apnea: Emphasis on practical therapeutic options. Clin Chest Med 2003;24:207-222.)

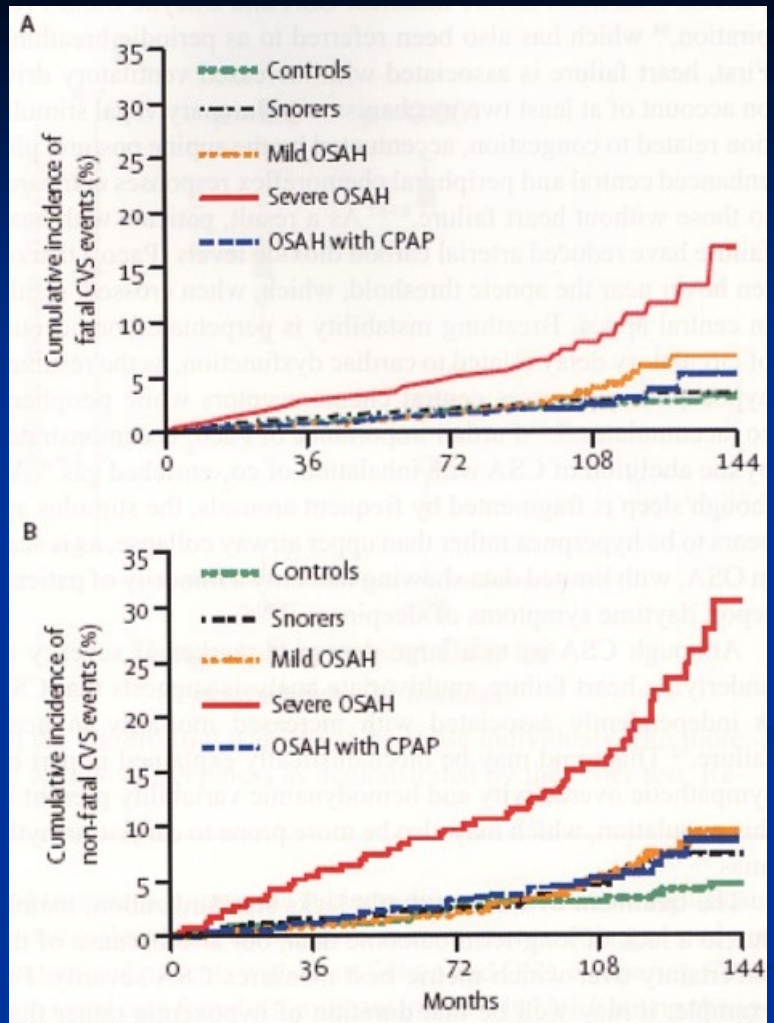


Figure 6—In a cohort of more than 1600 men, a higher incidence of fatal (top) and non-fatal (bottom) cardiovascular events was observed in the 36% with severe OSA who were noncompliant with CPAP treatment. Reproduced with permission from Reference 140.

Quality of Life

- 80% of OSA patients have EDS & cognitive impairment
- 50% of OSA patients report personality changes
- 4% of a random community sample 18 – 84 – Habitual sleepy drivers
- 34% of OSA patients have had a crash in previous 5 years (24% of controls)

Driving

- OSA in Driving Simulators
  - Increase off road incidents
  - Slower brake reaction time
  - Increased lateral position deviations

Driving & the Law

- Falling asleep whilst driving = recklessness
  - Maggie's Law
- Inform the patient that they should not drive and document this in the record. Inform the patient that this advice will be given to the GP. If the patient continues to drive, despite this advice, then the Licensing Authority MUST be notified.
  - But not all patients with OSA will have a crash
  - If they do crash Low speed bunt or high speed fatal
  - Nod off driving, nod of driving daily, run off road or out of lane, previous crash, risk to others, size of vehicle.

Quality of Life

- Quality of life
- Health related quality of life
- Functional status

- Generic (SF36)
- Disease specific
  - Functional outcomes of sleep questionnaire (FOSQ)
  - Calgary sleep apnoea quality of life instrument (SAQLI)
  - OSA Patient oriented severity index (OSAPOSI)
- Threshold effect between RDI 1 and 15