

Obesity and Obstructive Sleep Apnoea

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Outline of talk

- How might OSAS and obesity be linked?
- What is the evidence that weight loss influences OSAS?
- Why might patients with OSAS be at increased peri-operative risk?
- How should this group of patients be managed peri-operatively?

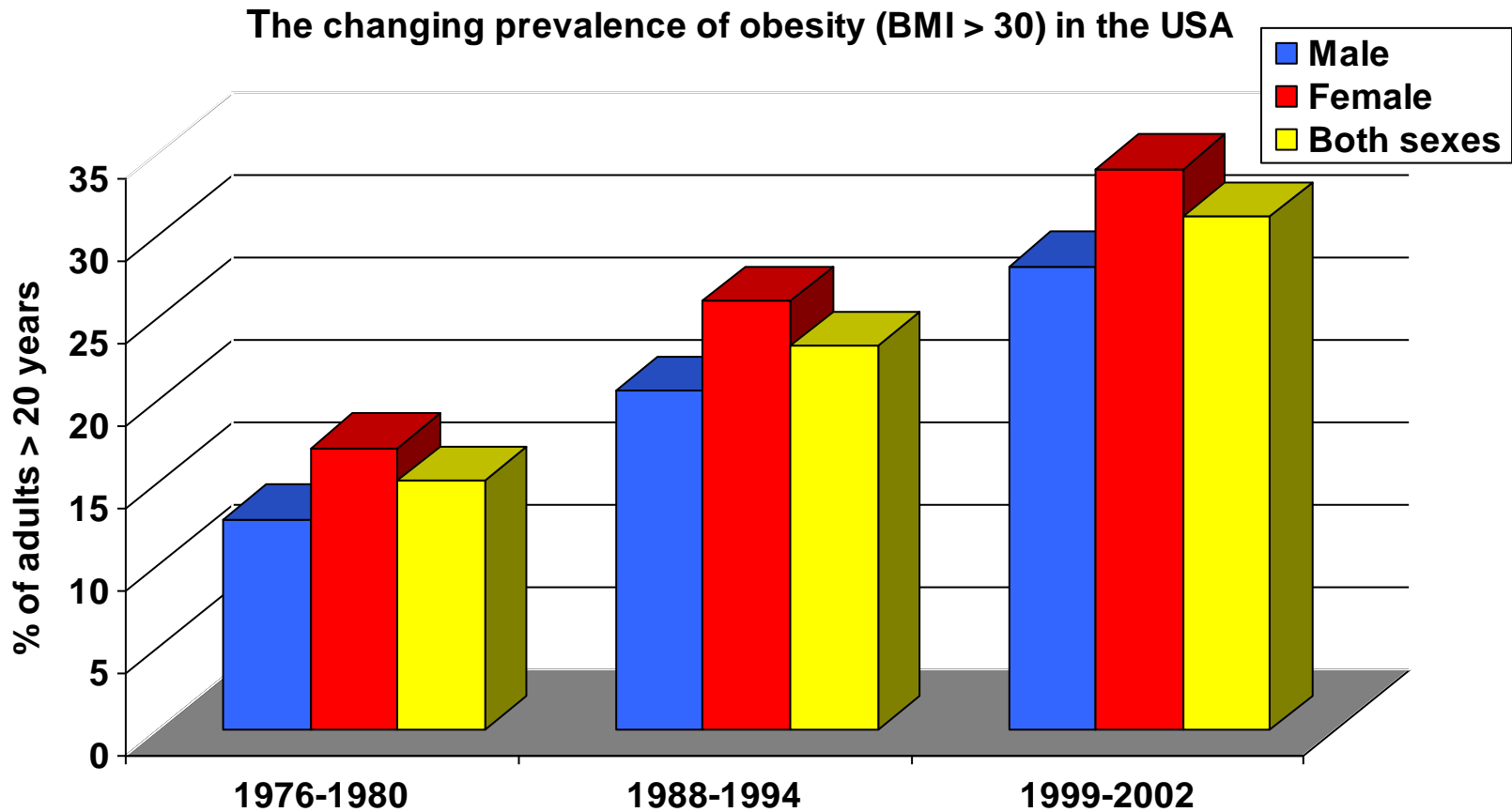
BMI is the most commonly used measurement of obesity

Body mass index (kg/m²)	Classification in adults
<18.5	Underweight
18.5–24.9	Healthy
25–29.9	Overweight
30–34.9	Obesity class I
35–39.9	Obesity class II
≥40	Obesity class III (morbid obesity)

Definition of Obstructive Sleep Apnoea Syndrome

- Repetitive upper airway collapse (either total or partial)
- Occurring > 5 times/hour of sleep
- Associated with daytime sleepiness

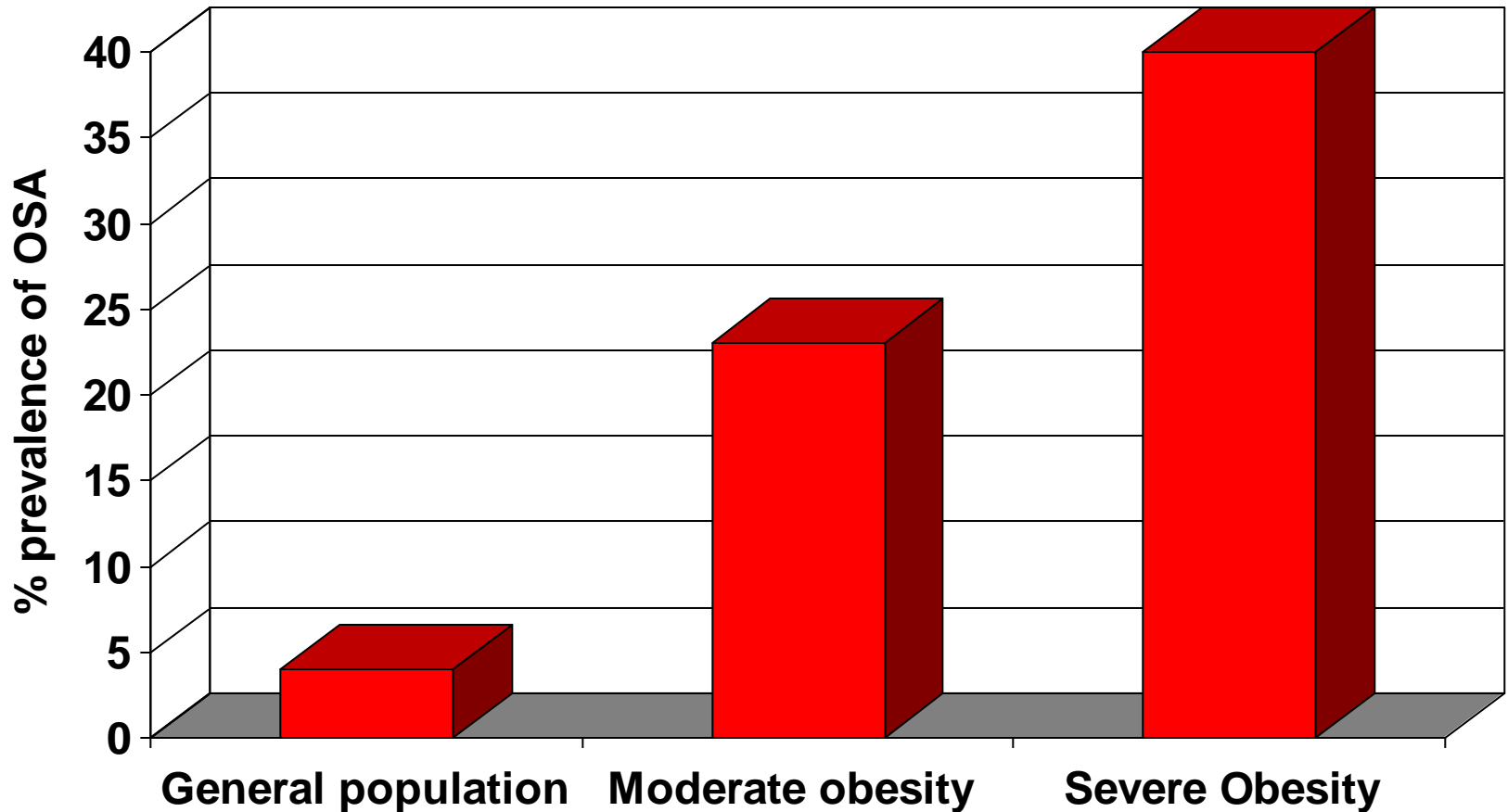
Prevalence of Obesity



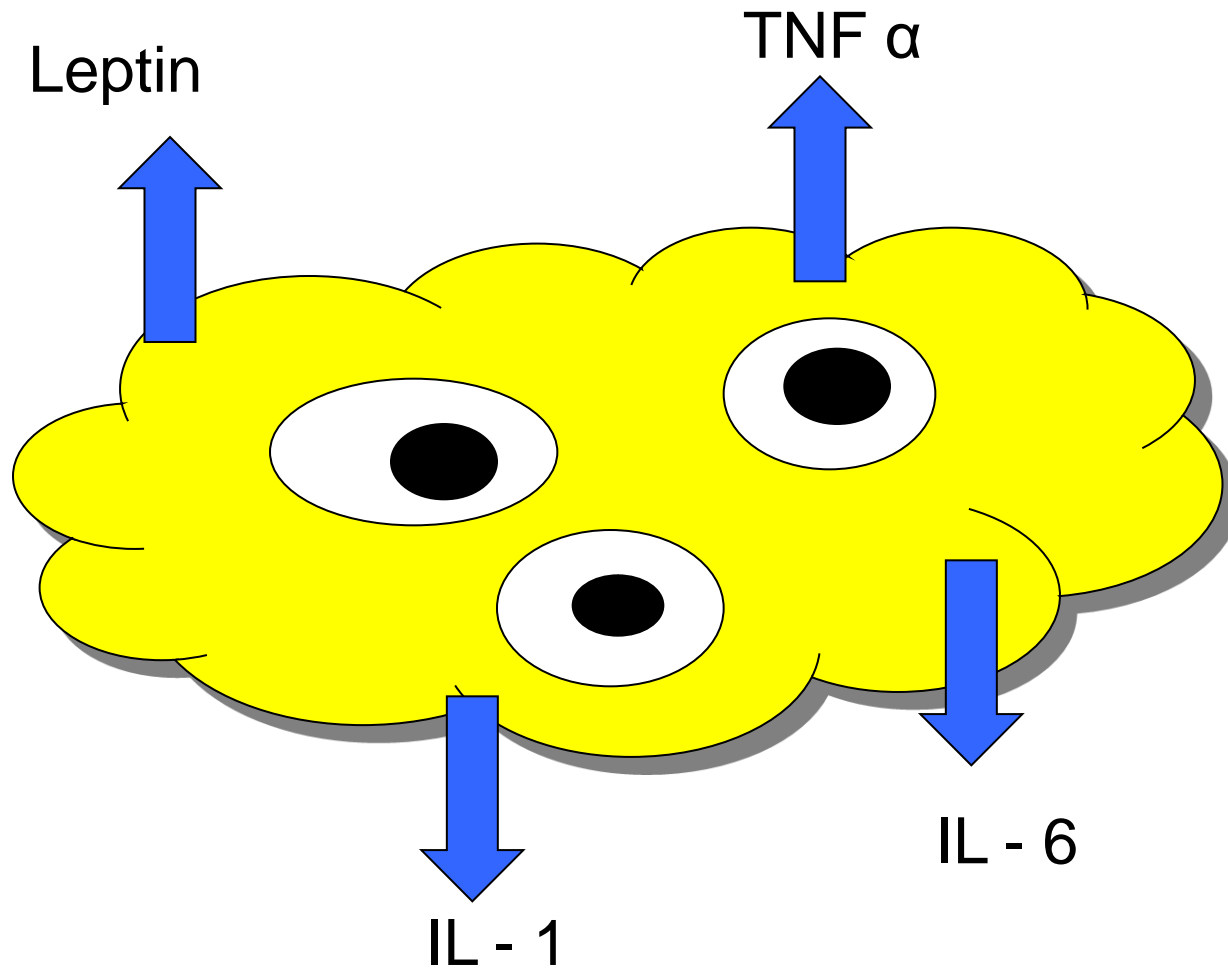
National Centre for health statistics
2004

60,000,000 people in USA classified as obese

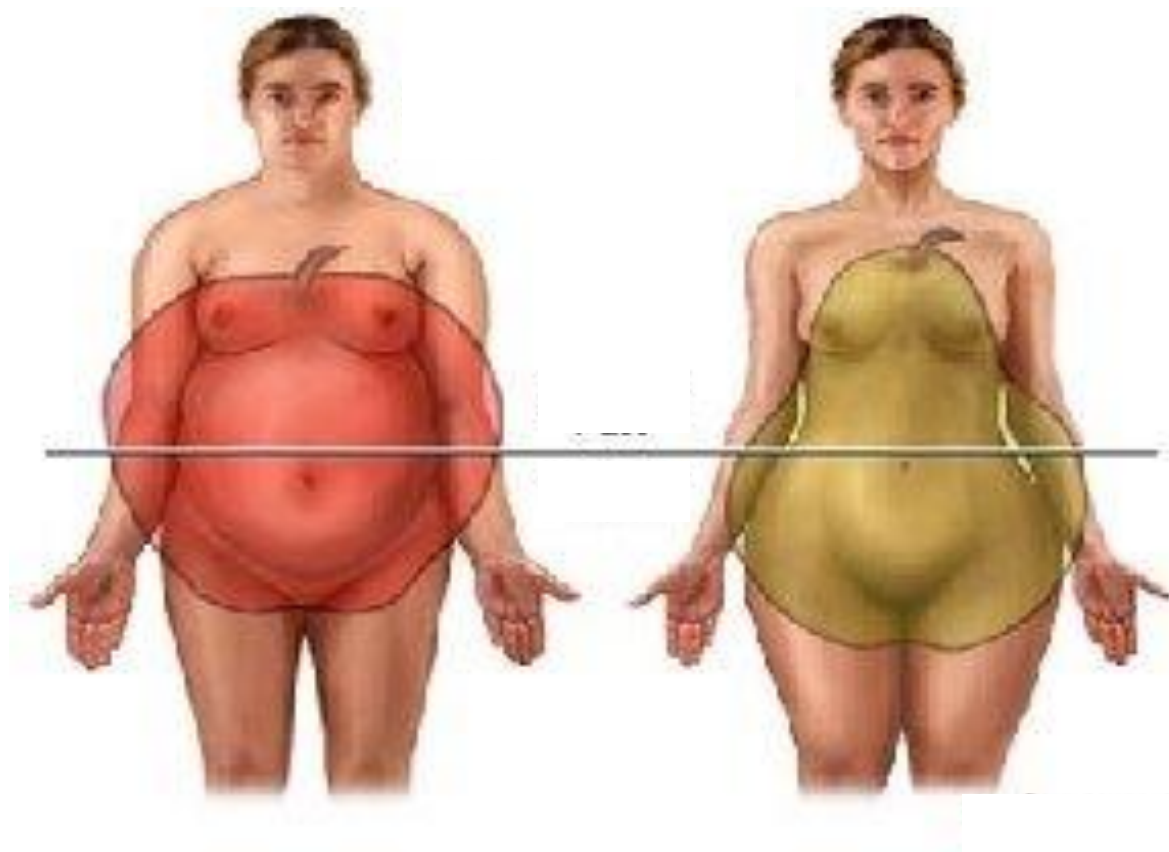
Obese population has an increased prevalence of OSA



Fat – a metabolically active tissue



Android vs. gynaecoid fat distribution



Summary

- BMI is the most commonly used measure of obesity
- Obesity levels continue to rise at alarming levels
- Fat is a metabolically active tissue
- Fat distribution varies between the sexes

Weight gain more detrimental in men

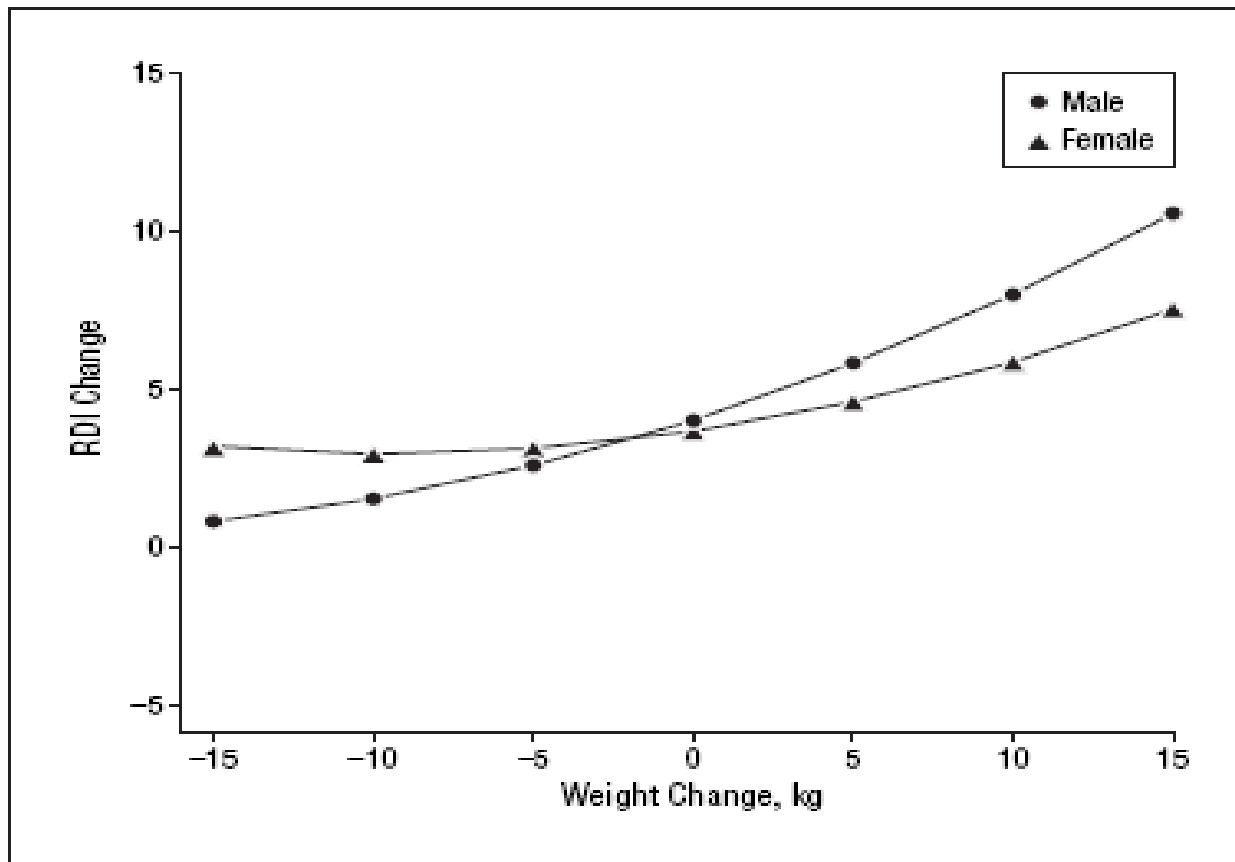
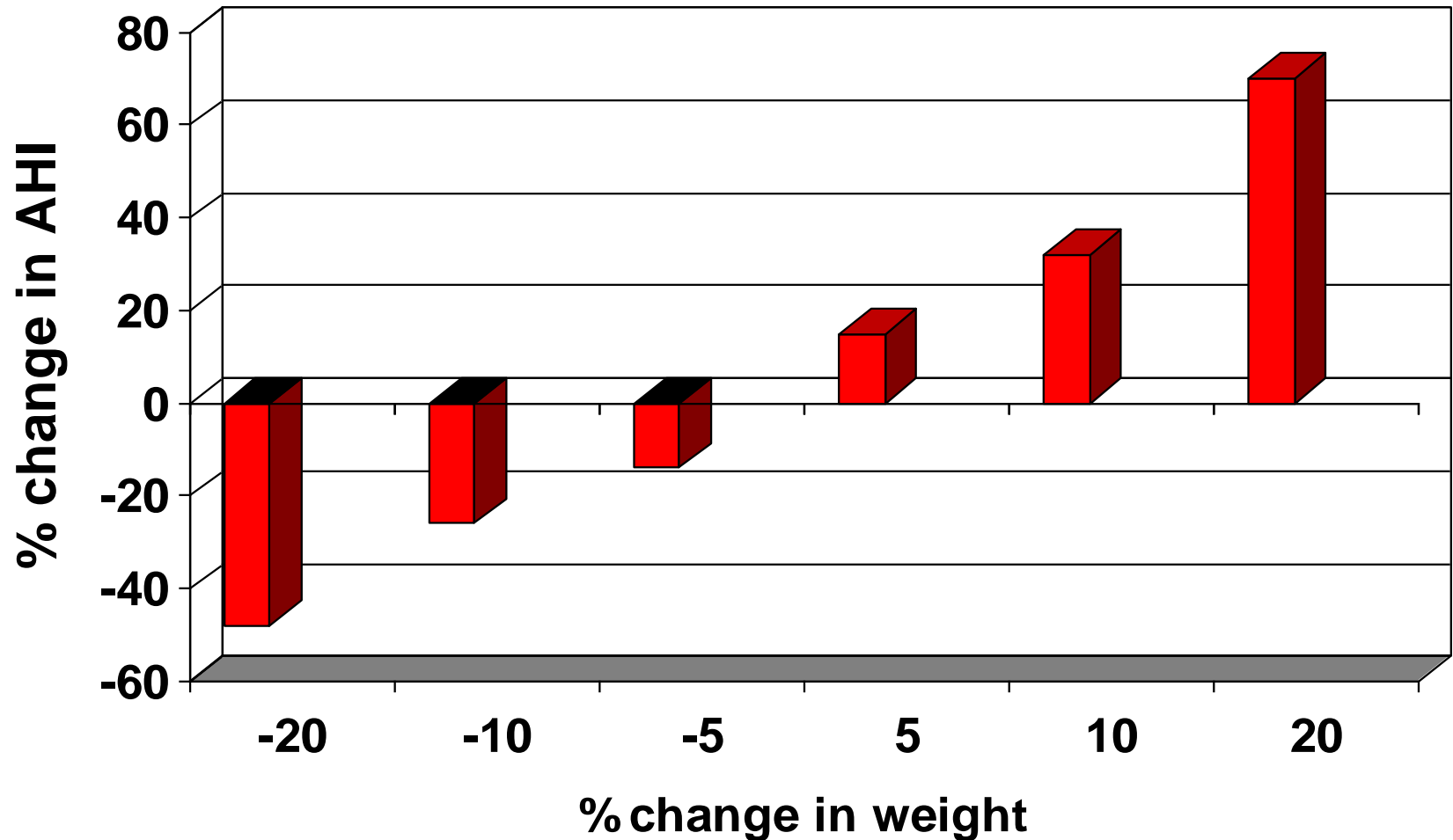


Figure 2. Change in Respiratory Disturbance Index (RDI) by change in weight.

The link between SDB and obesity



Peppard et al, JAMA 2000; 284: 3015-3021

The pathogenesis of OSA

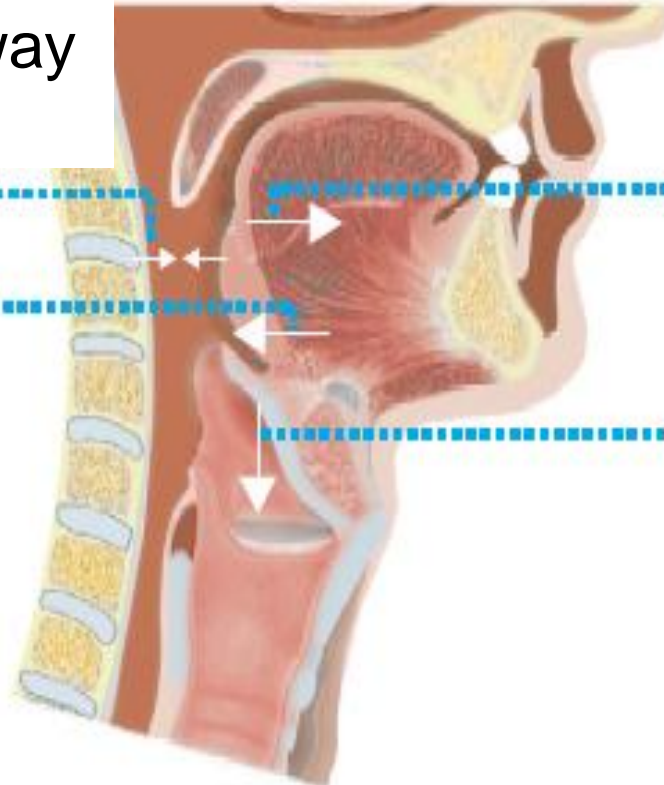
Neuromuscular control

Promotion of airway collapse

Negative pressure on inspiration

Extraluminal positive pressure

Fat deposition
Small mandible



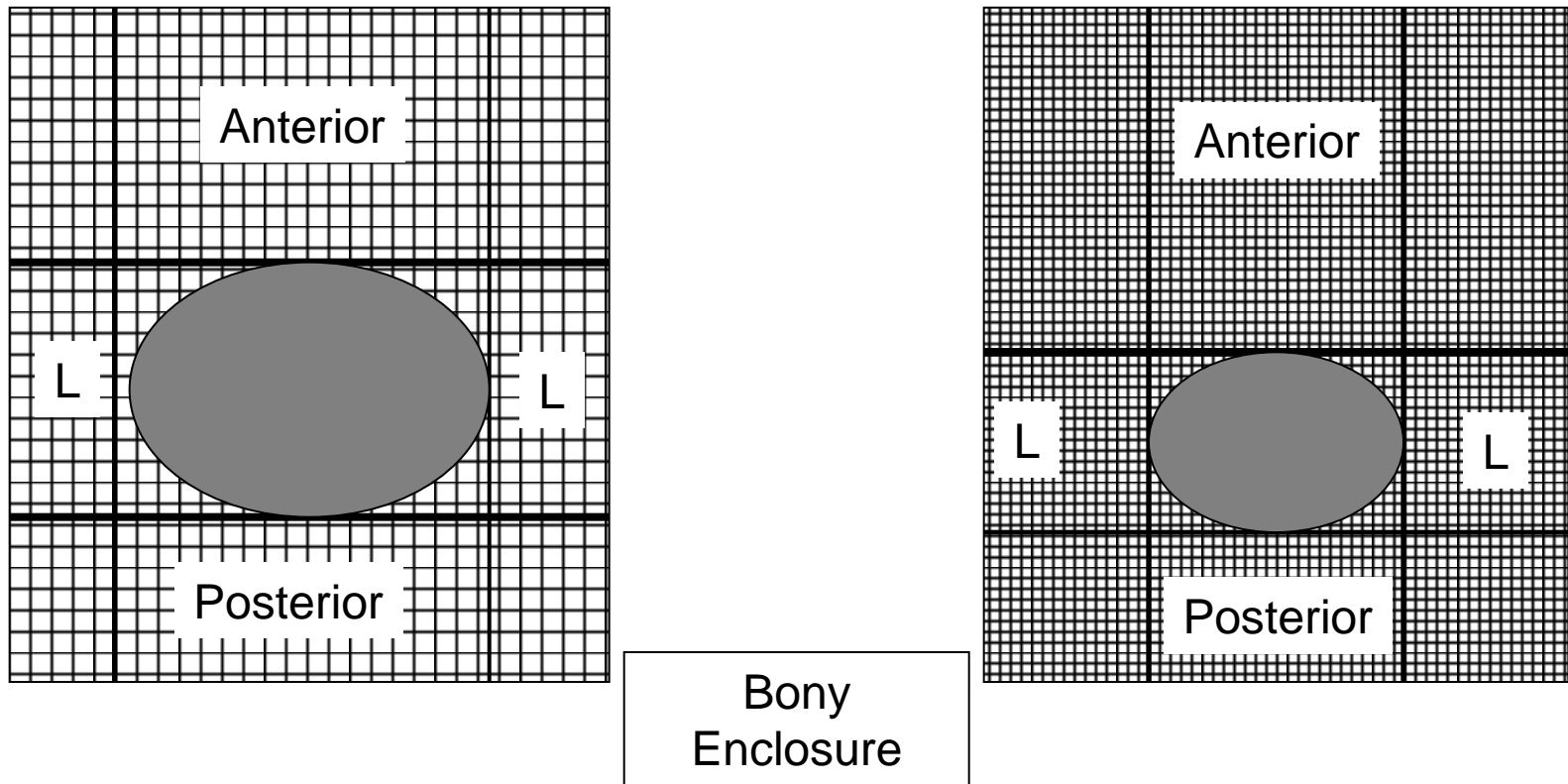
Promotion of airway patency

Pharyngeal dilator muscle contraction (genioglossus)

Lung volume (longitudinal traction)

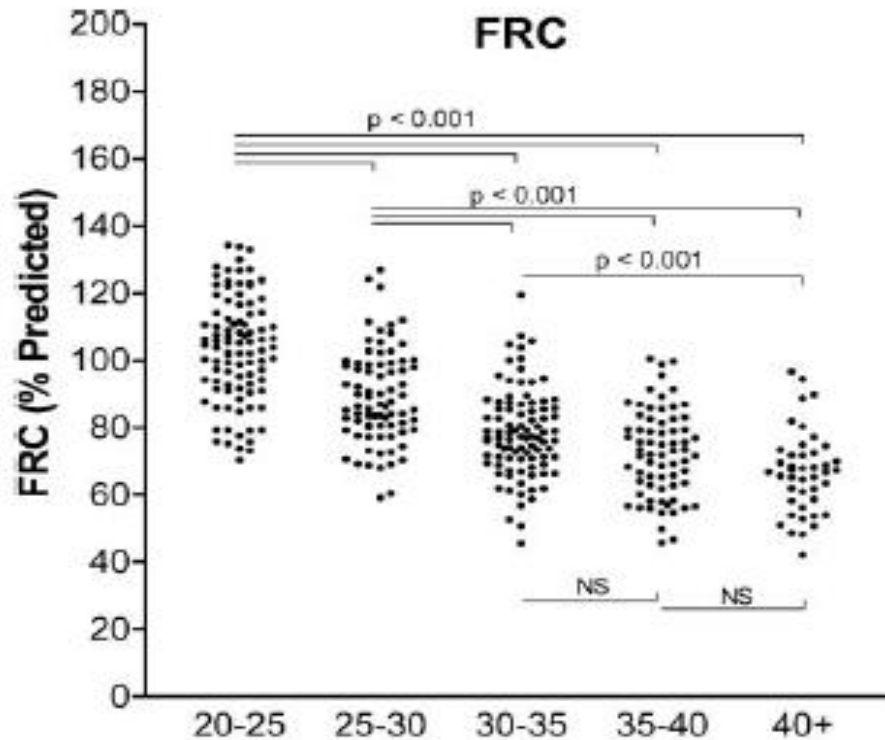
? Effects of adipokines

Fat deposition increases tissue pressure and airway collapsibility



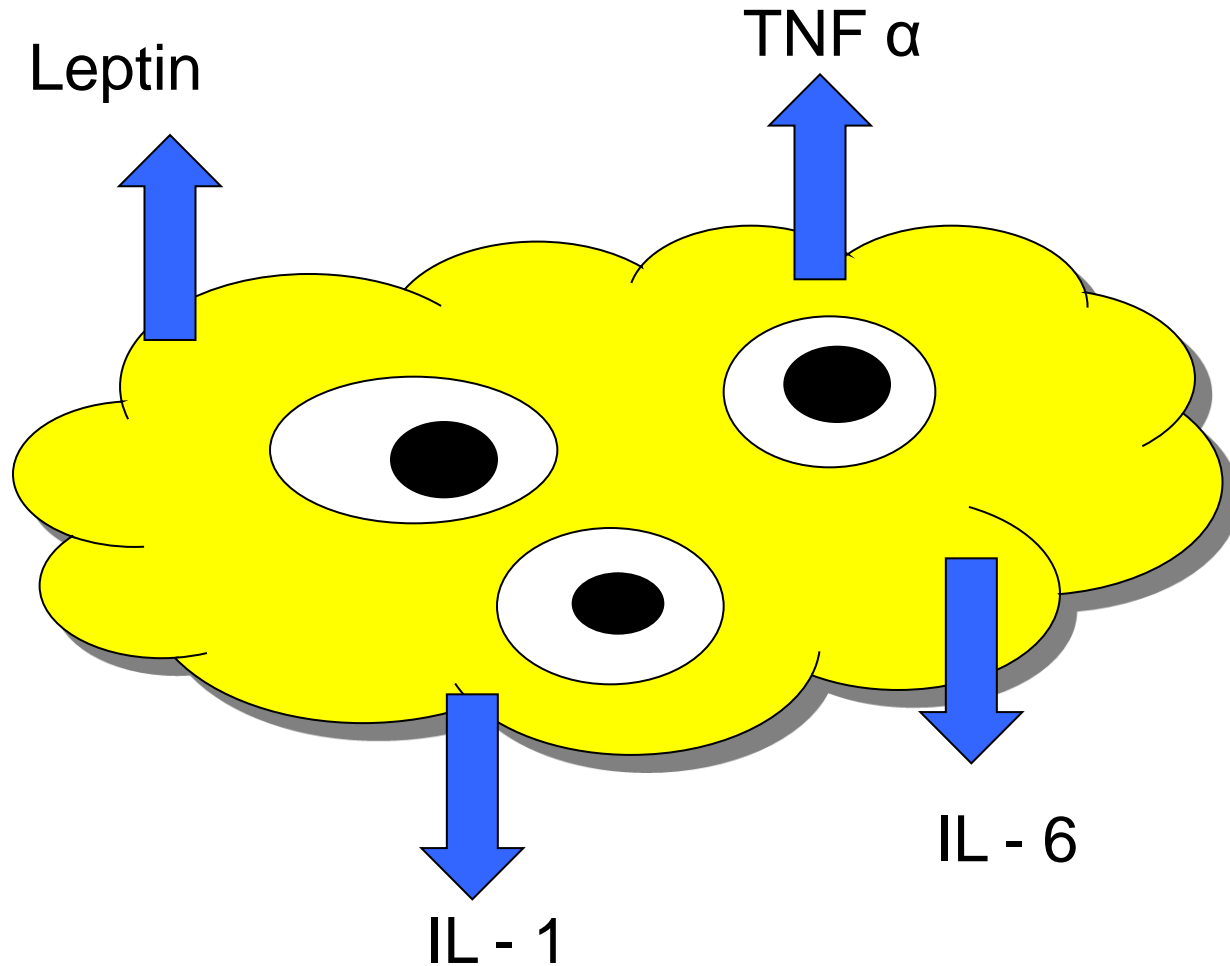
Adapted from Watanabe et al, AJRCCM 2002; 165:260-265

FRC declines with obesity



Jones et al, Chest 2006: 130; 827 - 833

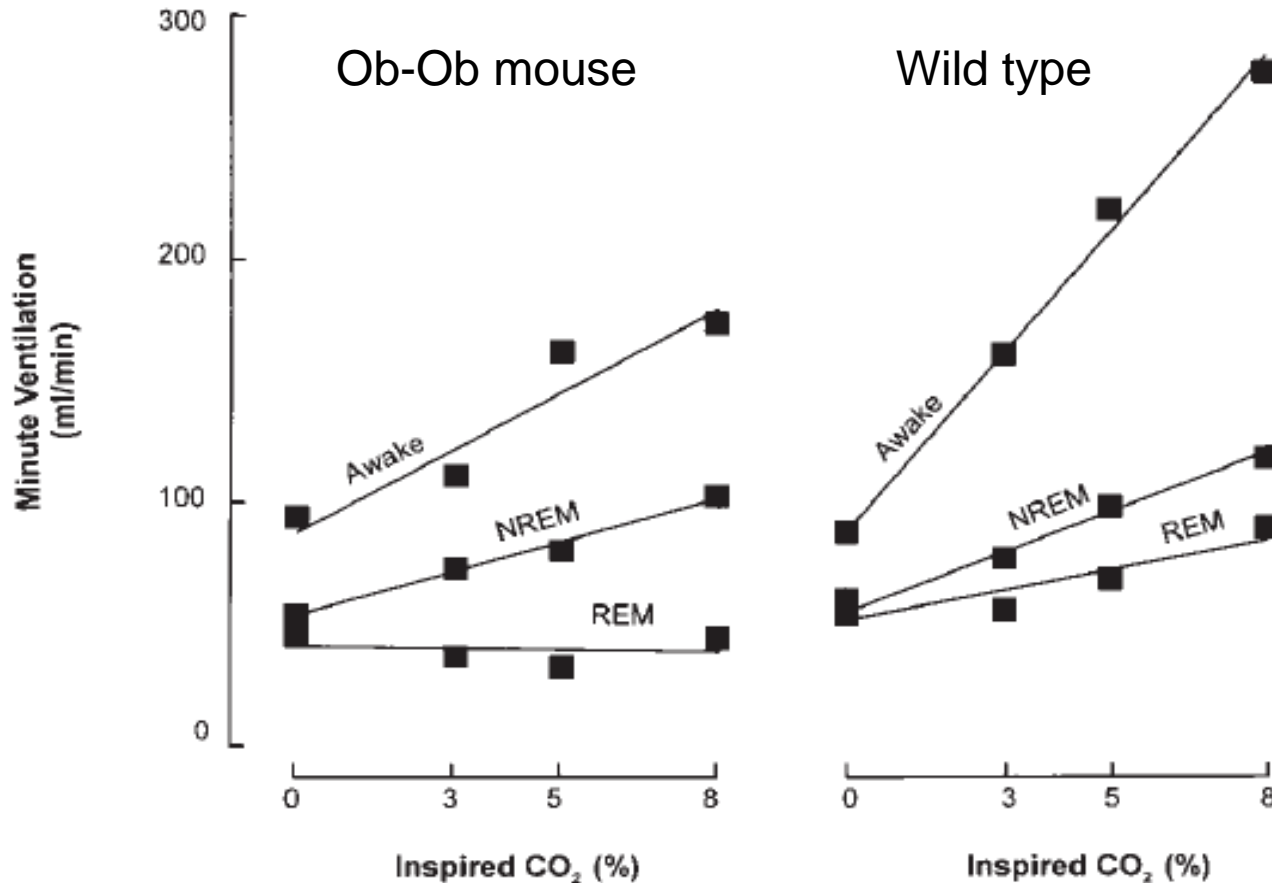
Can adipokines affect ventilation?



Leptin

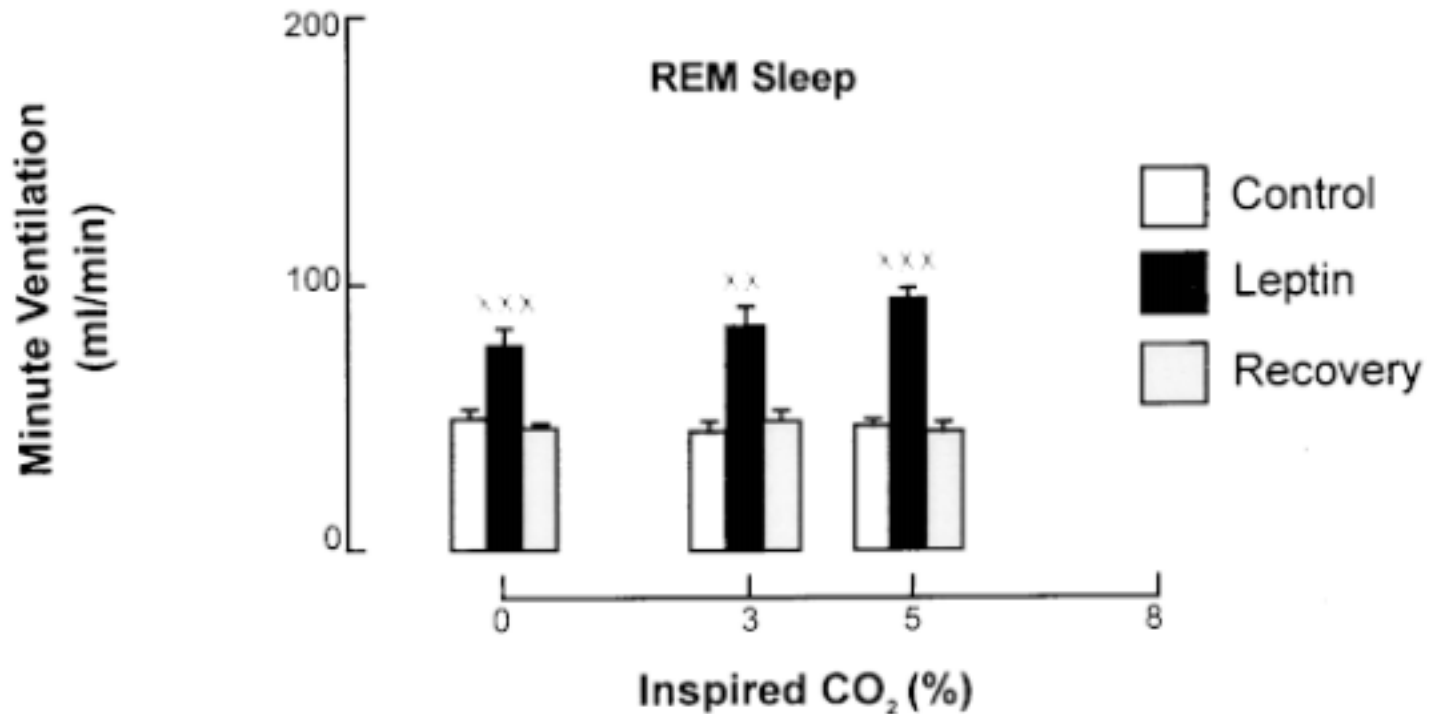
- Adipocyte derived hormone
- Has central hypothalamic effects to signal satiety
- Appears to have a role in ventilatory control in animal models

Leptin absence blunts ventilation in mouse model



O'Donnell et al, AJRCCM 1999; 159: 1477-1484

Leptin replacement stimulates ventilation



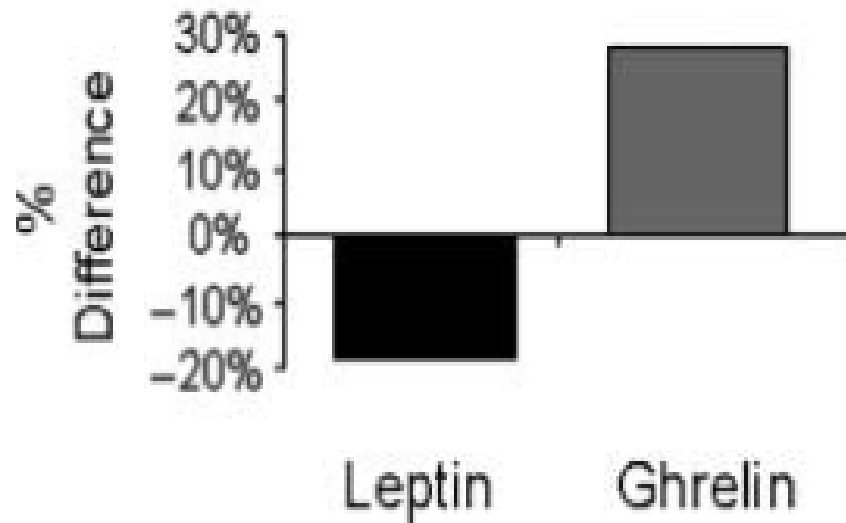
Can adipokines affect ventilation in humans?

- Seems that human obesity associated with high leptin levels – indicative of a degree of resistance
- High levels may attenuate response to hypercapnic stimulus and impair arousal response in OSA¹
- TNF α and IL 6 known to be somnogenic and depress CNS activity ²

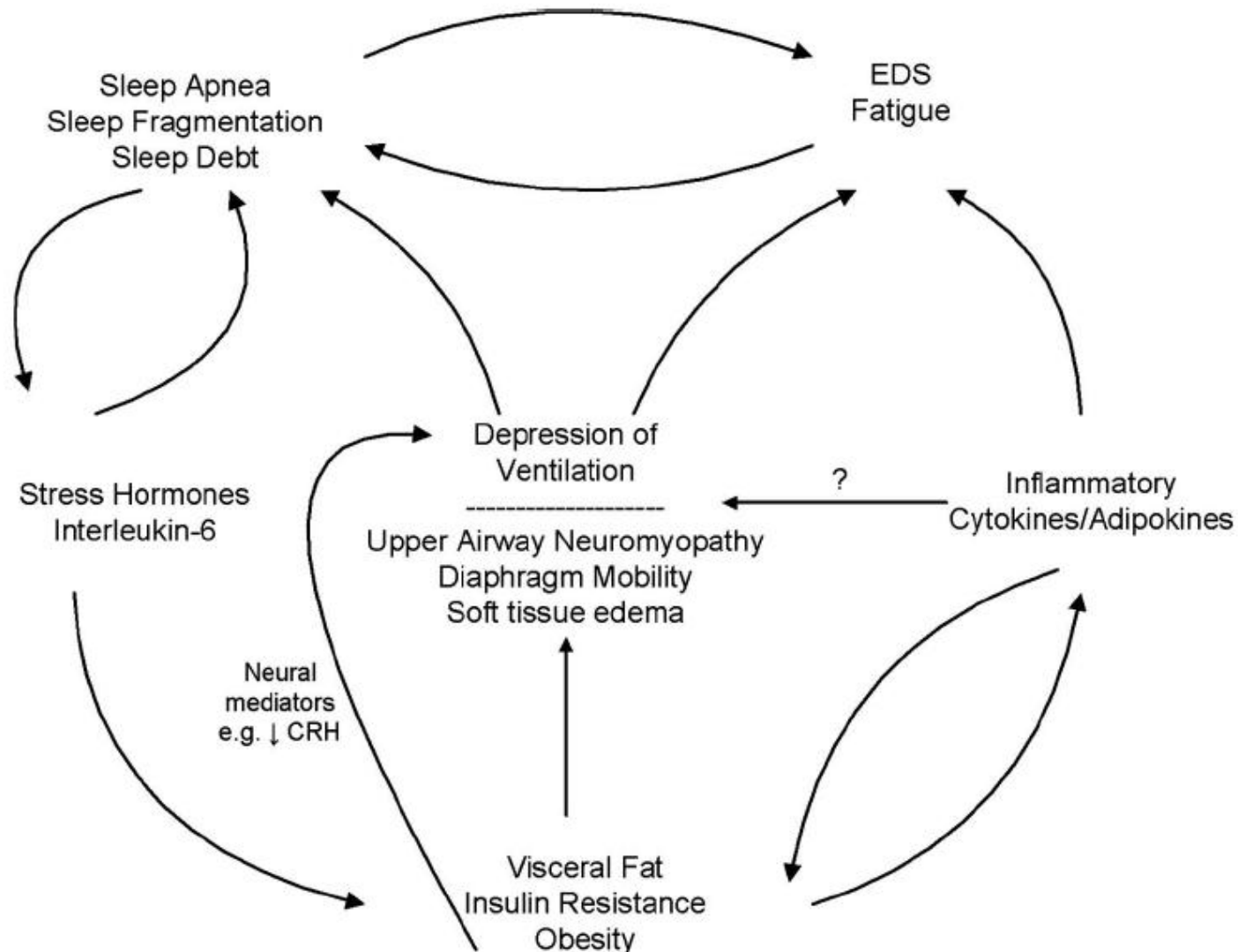
1 Campo et al, ERJ 2007; 30:223-231

2 Opp, Sleep Med Rev 2005; 9:355-364

Can sleep restriction cause obesity?



Cytokines - the link between obesity and OSA?



Meta-analysis of effects of surgical weight loss

	Resolved	Resolved/Improved	↓ in AHI
Patients evaluated	1195	726	92
No (%) with improvement in characteristic	1051 (87.9)	585 (80.6)	
No of treatment groups	38	24	4
Mean (95% CI)	85.7 (79.2-95.4) %	83.6 (71.8-95.4) %	-33.9 (-52.8- -16.0)

Buchwald et al, JAMA 2004; 292:1724-37

VLCD and in the treatment of mild SDB

	Control group	Intervention group
Number of patients	37	35
BMI (kg/m ²)	31.4 (2.7) *	33.4 (2.8) *
Waist circumference (cm)	105.3 (8.3) *	112.5 (8.7) *
ESS	9.9 (4.8)	10.1 (5.0)
AHI	9.3 (3.0)	10.0 (3.0)
Supine AHI	21.3 (17.3)	20.1 (14.1)
% Time with SpO ₂ < 90%	1.7 (3.6)	2.8 (5.2)

* p < 0.01

Lifestyle intervention is effective up to one year

	Control group	Intervention group
Change in BMI (kg/m ²)	-0.8 (2.0) *	-3.5 (2.1)*
Change in waist circumference (cm)	-3.0 (6.0) *	-11.6 (6.6) *
Change in ESS	-2.1 (2.9)	-3.1 (4.0)
Change in AHI	0.3 (8.0) *	-4.0(5.6) *
Change in supine AHI	-5.9 (23.9)	-6.5 (13.0)
Change in % Time with SpO ₂ < 90%	1.8 (6.3)	-1.7 (4.1)

* P ≤ 0.01

Weight loss is associated with improvements in severe SDB

	Intervention (n=30)	Control (n=33)
BMI (kg/m ²)	34.4 (2.9)	34.8 (2.9)
AHI	37 (17)	37 (14)
Change in BMI after 9 weeks	-25 (17)*	-2 (11) *
Change in AHI after 9 weeks	-5.7 (1.1) *	0.3 (0.6) *

Summary

- The predominance of OSA in middle aged men and post-menopausal women may reflect differences in fat distribution
- Cross-sectional data shows that weight gain worsens OSA
- Biologically plausible that obesity contributes OSA
- Possible that metabolic consequences of obesity may contribute to OSA
- Obesity is the only modifiable risk factor in OSA

Relevance of OSAS and obesity to the anaesthetist

- Why might these patients be vulnerable in the peri-operative period?
- What evidence is there that they are at peri-operative risk?
- How should this group of patients be managed?

Potential peri-operative risks in obesity

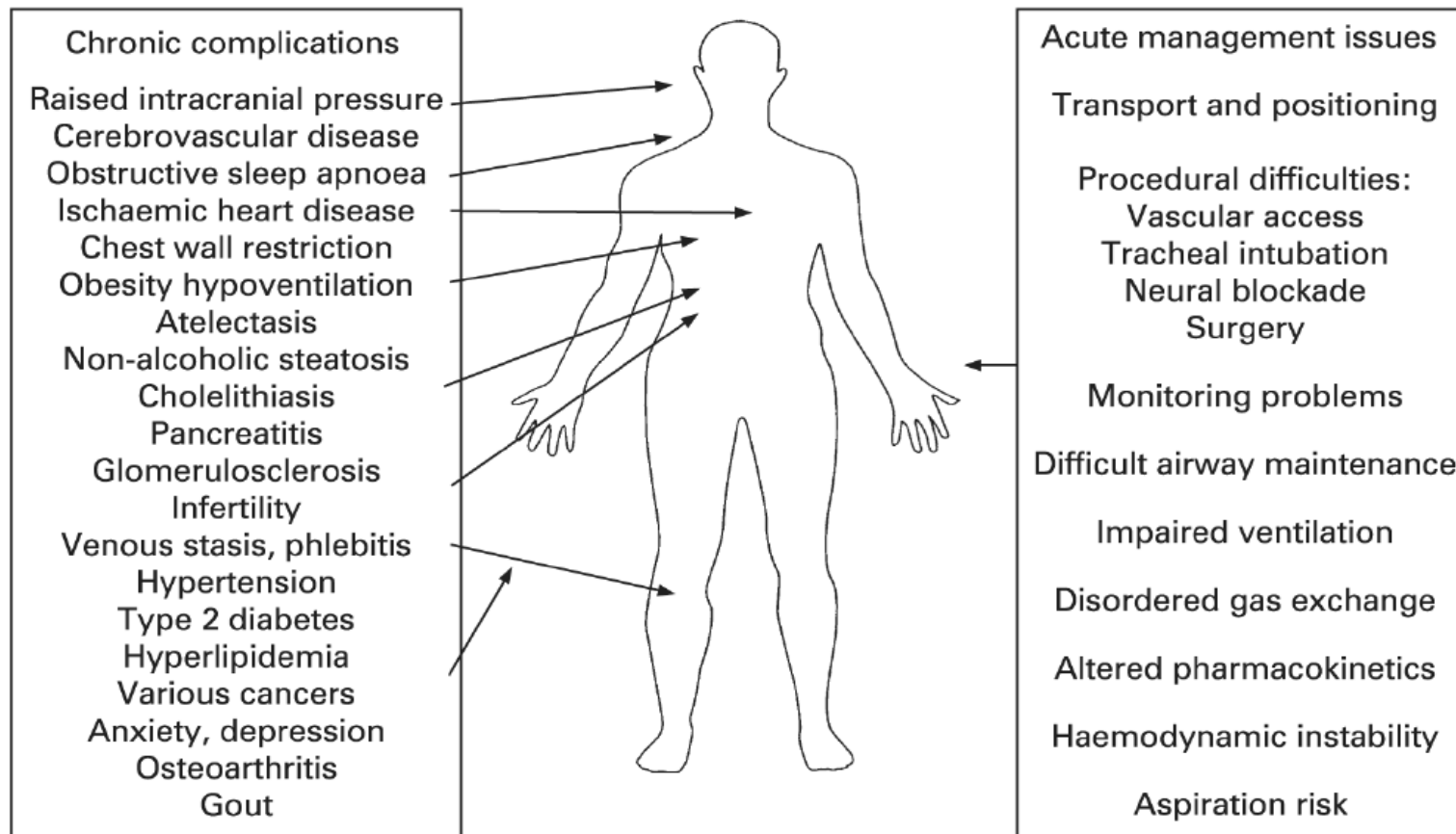
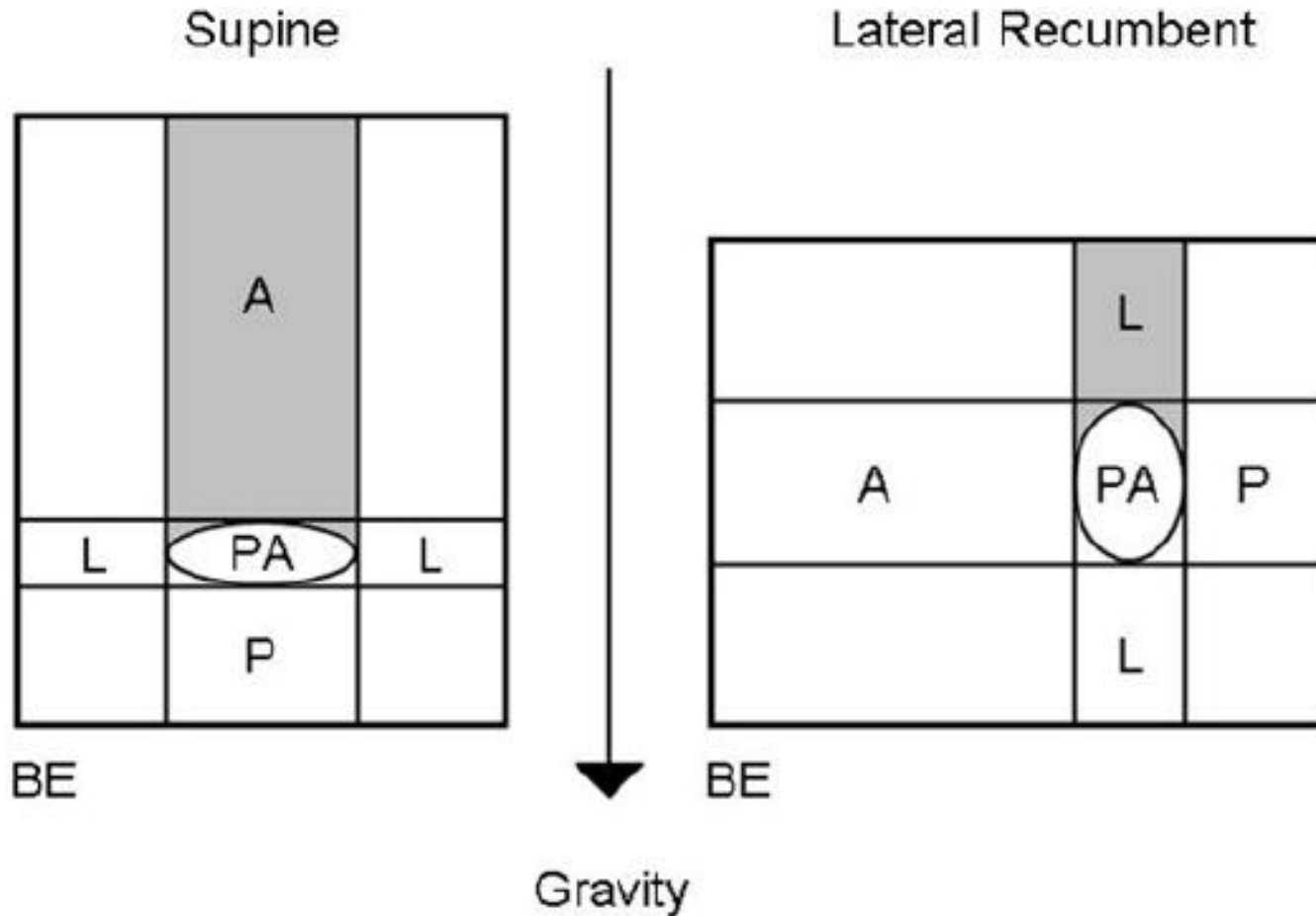


Figure 1.
Obesity-related complications.

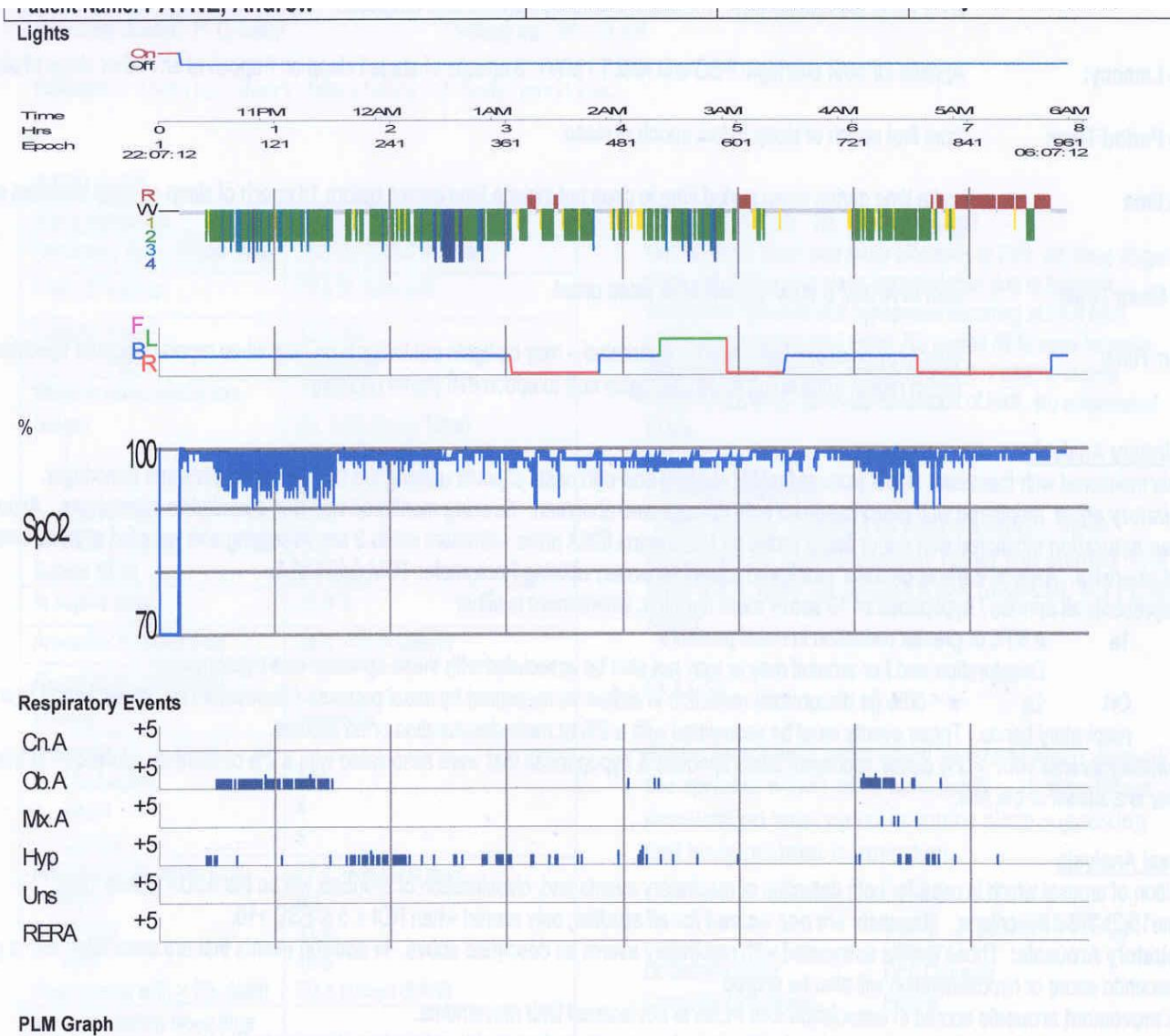
OSA and peri-operative risk

- Effects of sedatives and anaesthetic agents on a vulnerable airway
- Post-operative patients may be positioned in the supine position

Changes in gravity affect pharyngeal size



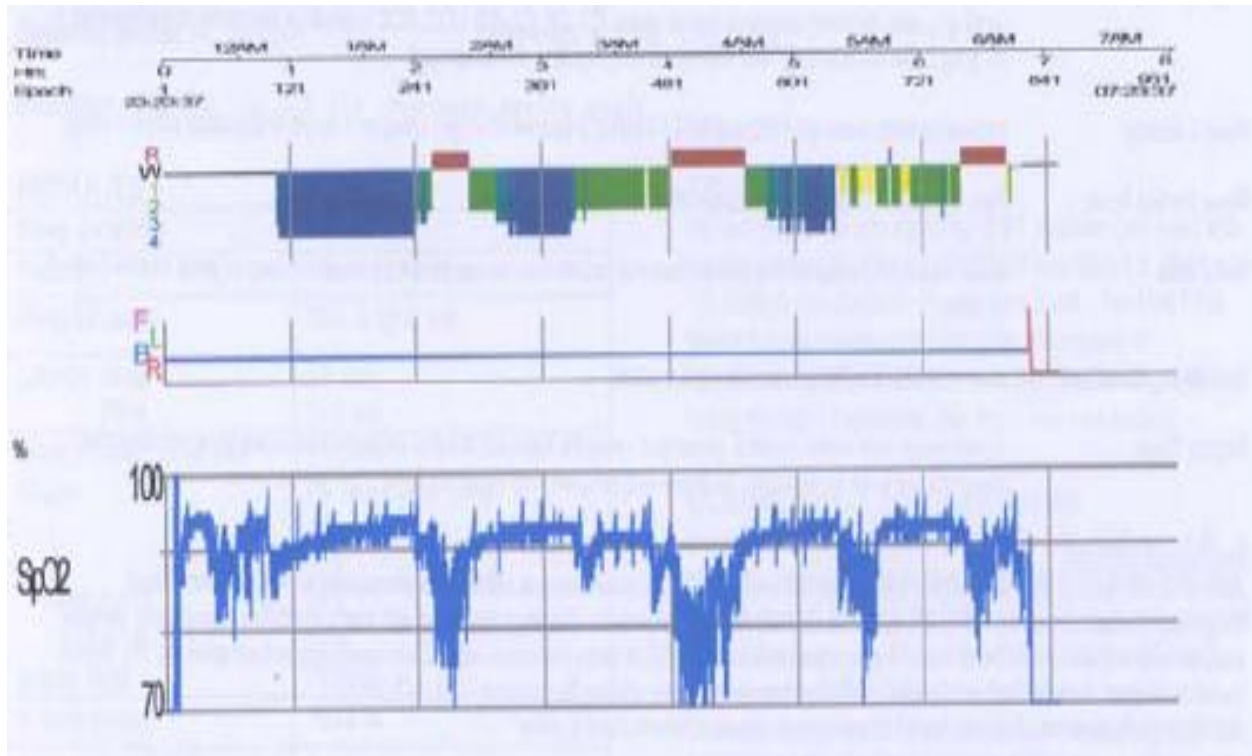
Positionality in OSA



OSA and peri-operative risk

- Effects of sedatives and anaesthetic agents on a vulnerable airway
- Post-operative patients may be positioned in the supine position
- Post-operative REM rebound

OSA worsens in REM sleep



OSA and peri-operative risk

- Effects of sedatives and anaesthetic agents on a vulnerable airway
- Post-operative patients may be positioned in the supine position
- Post-operative REM rebound
- Association between OSA and cardiac arrhythmias

Association between OSA and arrhythmias

Table 2—Daytime and Nocturnal Rhythm Disturbances and ST-Segment Depression Episodes in Patients With OSAHS, Snoring Subjects, and Control Subjects*

Variables	OSAHS Patients	Snoring Subjects	Control Subjects
Daytime sinus tachycardia	3.92 ± 3.041	0.54 ± 1.35‡	0.13 ± 0.18§
<u>Nocturnal sinus tachycardia</u>	8.99 ± 4.52	2.61 ± 3.45‡	0.19 ± 0.17‡
Daytime SB	2.00 ± 2.99	0.20 ± 0.27§	0.09 ± 0.13‡
<u>Nocturnal SB</u>	14.90 ± 14.06	2.62 ± 5.93‡	0.37 ± 0.46‡
Daytime pauses	0.24 ± 0.43	0.03 ± 0.06	0.05 ± 0.06
Nocturnal pauses	3.03 ± 5.38	0 ± 0	0.10 ± 0.12
Daytime PSVB	0.36 ± 0.42	0.25 ± 0.50	0.05 ± 0.05‡
<u>Nocturnal PSVB</u>	3.24 ± 4.66	0.65 ± 1.30	0.06 ± 0.08‡
Daytime SVT	0.03 ± 0.05	0 ± 0‡	0 ± 0‡
Nocturnal SVT	0.63 ± 1.08	0.10 ± 0.33	0.02 ± 0.04‡
Daytime couplets	0.55 ± 0.73	0.14 ± 0.26	0.07 ± 0.10‡
Nocturnal couplets	2.46 ± 3.64	0.35 ± 0.58‡	0.17 ± 0.18‡
Daytime bigeminy	0.13 ± 0.27	0.05 ± 0.18	0.00 ± 0.02
Nocturnal bigeminy	0.51 ± 1.02	0.19 ± 0.61	0.02 ± 0.05
Daytime ST-segment depression	0.079 ± 0.097	0.006 ± 0.021‡	0.004 ± 0.0152‡
<u>Nocturnal ST-segment depression</u>	0.565 ± 0.826	0.096 ± 0.140‡	0 ± 0‡

*Data are presented as mean ± SD episodes per hour.

Alonso - Fernandez et al, Chest 2005; 127:15-22

OSA and peri-operative risk

- Effects of sedatives and anaesthetic agents on a vulnerable airway
- Post-operative patients may be positioned in the supine position
- Post-operative REM rebound
- Association between OSA and cardiac arrhythmias
- Sleep deprivation worsens OSA – anxiety, pain, noise levels in hospital ¹

¹ Stoohs et al, NEJM 1993;328:1279

OSA may be associated with difficult intubation

	Laryngoscopy grade > 3 (n=152)(%)	Laryngoscopy grade < 3 (n=1320)
Age (yr)	50.6 ±17.5 *	43.5 ± 15.2 *
BMI (kg/m ²)	29.6 ± 7.6	29.0 ± 7.3
Female	54(35.5) *	670(50.8)
OSA	20 (77) *	6 (23) *
Impaired TMJ	12 (7.9) *	8 (0.6) *
TMD < 6cm	46 (30.3) *	184 (13.9) *

* p< 0.05

What evidence is there that OSA carries peri-operative risk?

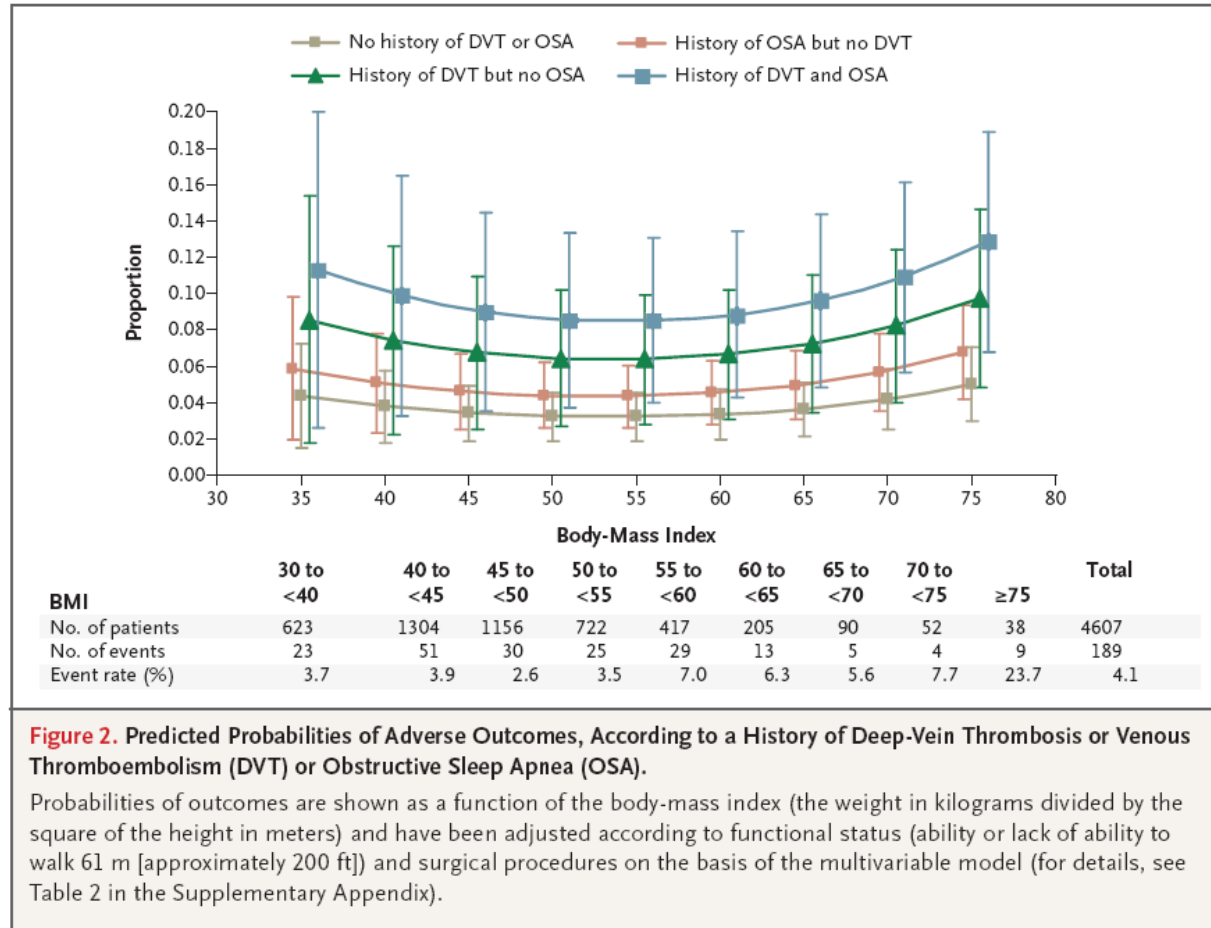
Study design	Number of patients	Definitive OSA Δ	Outcome
Case report	3	yes	Post -op heart block
Retrospective	101 with matched controls	variable	\uparrow LOS, \uparrow complications, \uparrow ICU transfer
Case report	8	yes	Arrest after opioids
Retrospective	234 with matched controls	yes	No differences in admission or adverse effects
Retrospective	37	yes	\uparrow infection rate, \uparrow LOS ICU

Peri-operative risk in OSAS

Table 3. Postoperative Outcomes*

	Group 1 (OSAS)				Group 2 (control) (n=101)	<i>P</i> value†
	1B (n=65)			Total (n=101)		
	1A (n=36)	No home CPAP (n=32)	Home CPAP (n=33)			
Any complication	18 (50)	12 (37.5)	9 (27.3)	39 (39)	18 (18)	.001
Serious complication‡	12 (33.3)	9 (28.1)	3 (9.1)	24 (24)	9 (9)	.004
Total ICU	14 (38.9)	8 (25.0)	1 (3.0)	23 (23)	8 (8)	.003
Unplanned ICU	12 (33.3)	7 (21.9)	1 (3.0)	20 (20)	6 (6)	.003
Hospital stay (d)	7.4±2.9	6.9±3.3	6.0±2.1	6.8±2.8	5.1±4.1	.007

Does OSA carry peri-operative risk in bariatric surgery?



Physiological effects of CPAP

- Pneumatic splinting of upper airway
- Recruitment of atelectatic alveoli and improvement in FRC
- Reduction of upper airway oedema
- ? Residual effects on neuromuscular function

What evidence is there for the use of CPAP in post-op OSA patients

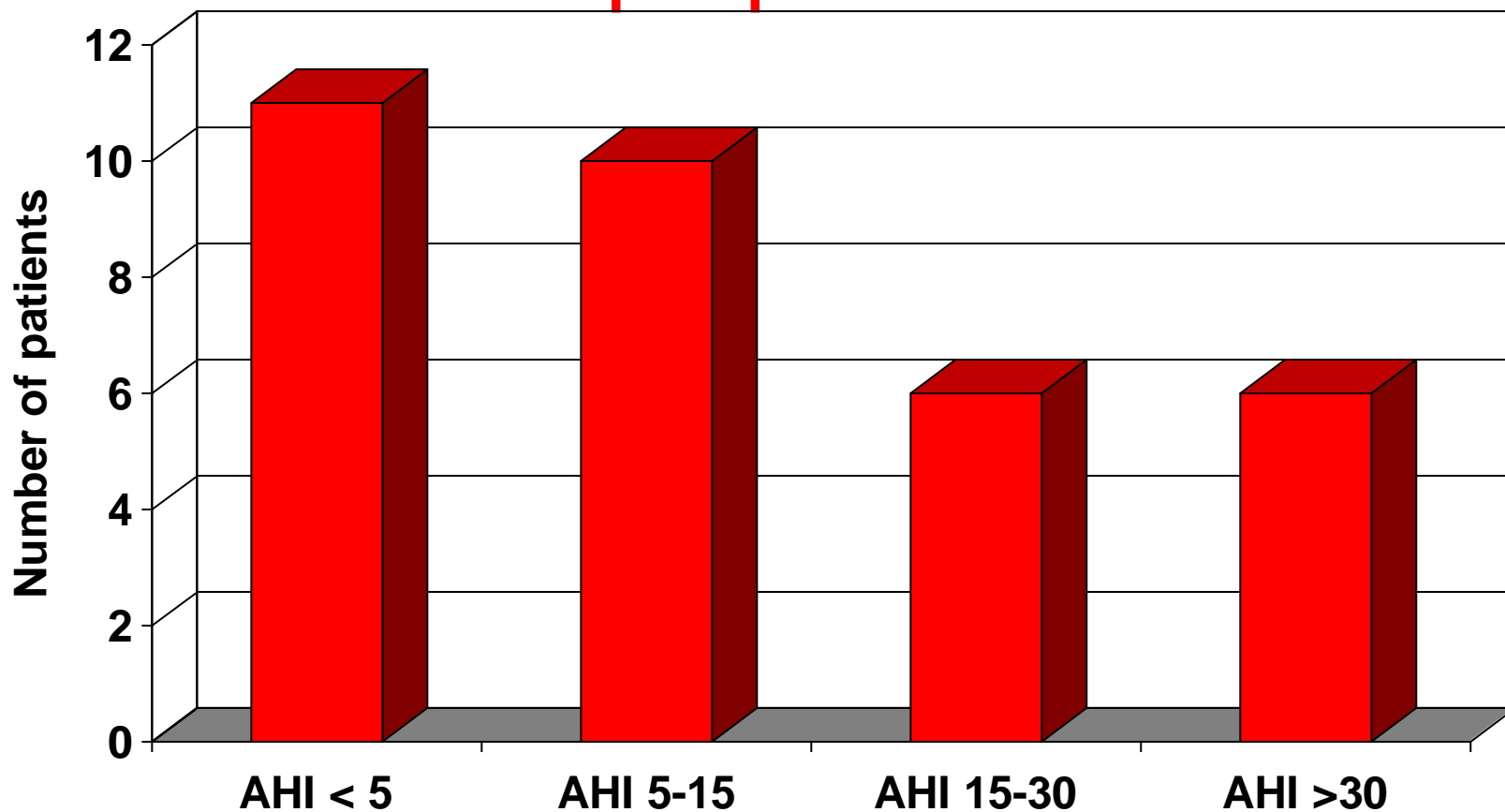
	No home CPAP (n=68)	Home CPAP (n=33)	<i>P</i> value§
Any complication	30 (44.1)	9 (27.3)	.10
Serious complication‡	21 (30.9)	3 (9.1)	.02
Total ICU	22 (32.3)	1 (3)	.001
Unplanned ICU	19 (27.9)	1 (3)	.003
Hospital stay (d)	7.2±3.1	6.0±2.1	.03

Gupta et al, Mayo Clin Proc 2001; 76(9): 897-905

How should these patients be managed?

- Attempt to identify OSA pre-operatively
- Consider use of CPAP in peri-operative period
- Adaptation of anaesthesia and analgesia
- Avoidance of supine position when possible
- Extended close observation
- Largely expert opinion

Should patients deemed to be a difficult intubation be assessed for sleep apnoea?



OSA patients had more post-op respiratory compromise

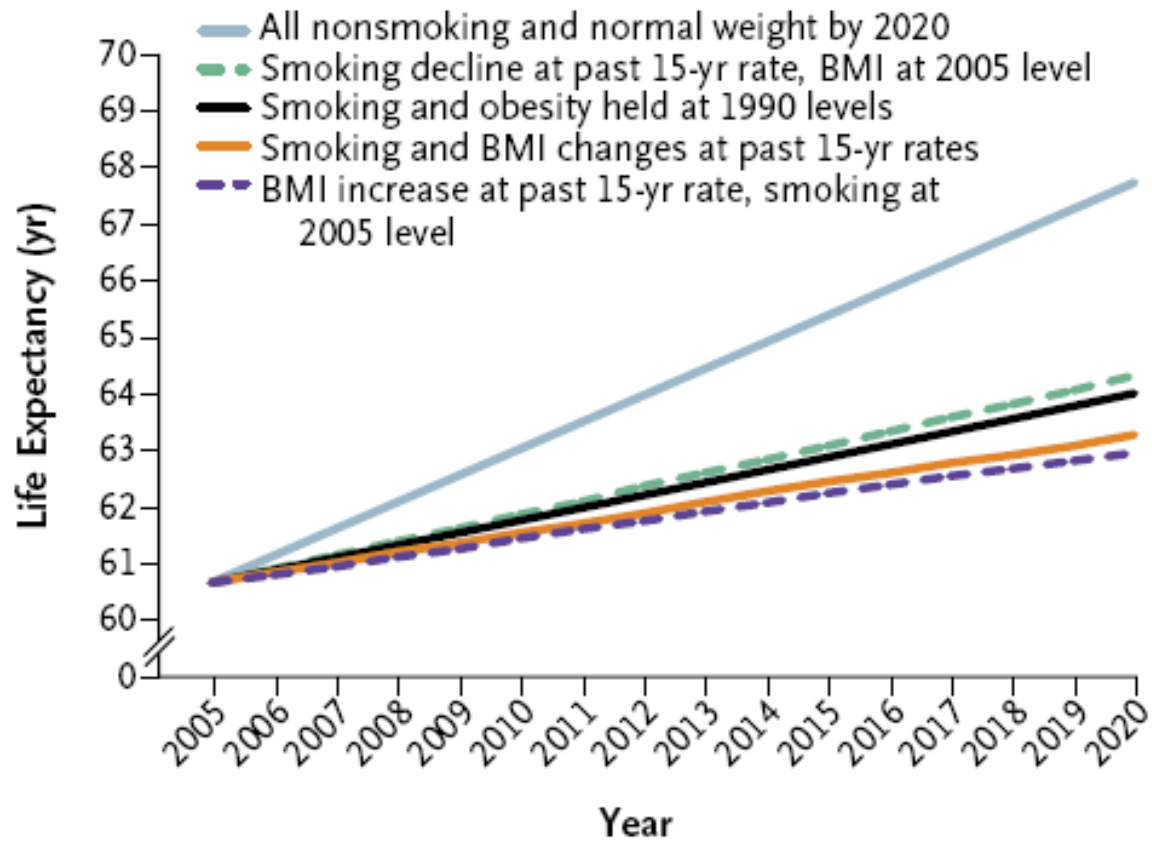
	AHI >5 (n=22)	AHI < 5 (n=11)
Mild desaturation (SpO ₂ 90-95%)	1	1
Severe desaturation (SpO ₂ <90%)	4	0
Required oxygen on ward	7	0
Cardiac complications	0	0

Summary

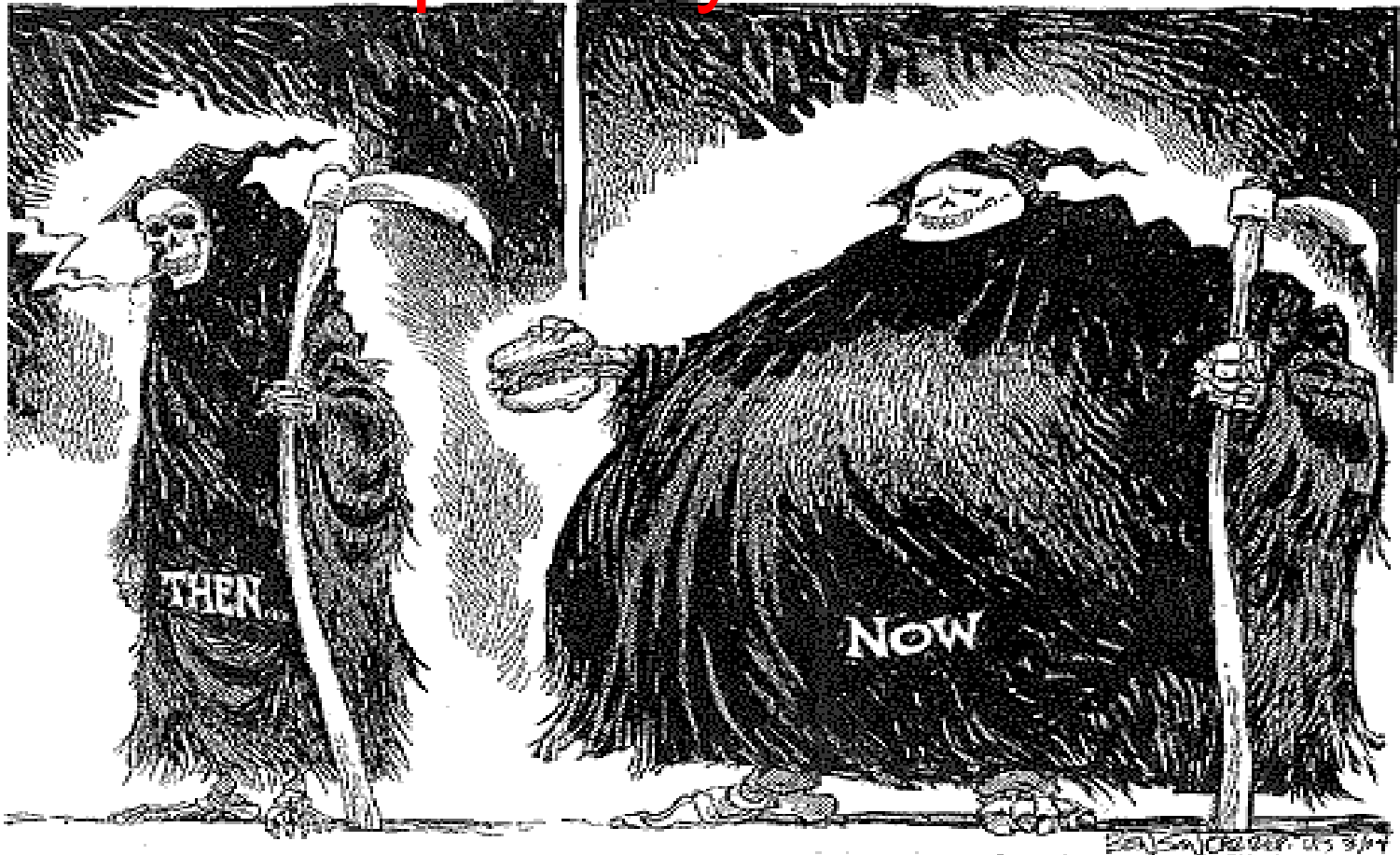
- OSA patients may be vulnerable in the peri-operative period
- Difficult to quantify the degree of risk based on current available evidence
- Guidelines for management are largely based on current opinion
- Symptoms of OSA should be sought in patients deemed difficult to intubate

Obesity – a risk to life?

A Life Expectancy at 18 Years of Age



Obesity – the next scourge of respiratory health?



BIG TOBACCO AND BIG BURGER VIE FOR THE TITLE AS THE NATION'S No. 1 KILLER.