

# Surgical vs Conventional Therapy for Weight Loss Treatment of Obstructive Sleep Apnea

## A Randomized Controlled Trial

John B. Dixon, MBBS, PhD, FRACGP  
 Linda M. Schachter, MBBS, PhD  
 Paul E. O'Brien, MD, FRACS  
 Kay Jones, MT&D, PhD  
 Mariee Grima, BSc, MDiet  
 Gavin Lambert, PhD  
 Wendy Brown, MBBS, PhD, FRACS  
 Michael Bailey, PhD, MSc  
 Matthew T. Naughton, MD, FRACP

**O**BESITY IS A MAJOR RISK FACTOR for obstructive sleep apnea (OSA). The estimated prevalence of OSA in obese adults varies from 42% to 48% in men and 8% to 38% in women.<sup>1</sup> Obstructive sleep apnea is strongly related to obesity and associated conditions such as type 2 diabetes and hypertension.<sup>2</sup> Individuals with OSA are at greater risk of stroke, cardiac disease, psychosocial morbidity, cognitive dysfunction, and impaired quality of life.<sup>3-5</sup>

Therapy for symptomatic OSA focuses on methods to reduce upper airway resistance and prevent obstruction, such as continuous positive airway pressure (CPAP),<sup>6</sup> oral appliances,<sup>7</sup> and upper airway surgery. However, in severely obese patients, none of these options treats the accompanying obesity. Observational studies of major weight loss following bariatric surgery suggest substantial remission of OSA symptoms in up to 60% to 80% of patients.<sup>8</sup> However, in the studies where repeat polysomnography was avail-

For editorial comment see p 1160.

**Context** Obstructive sleep apnea (OSA) is strongly related to obesity. Weight loss is recommended as part of the overall management plan for obese patients diagnosed with OSA.

**Objective** To determine whether surgically induced weight loss is more effective than conventional weight loss therapy in the management of OSA.

**Design, Setting, and Patients** A randomized controlled trial of 60 obese patients (body mass index:  $\geq 35$  and  $\leq 55$ ) with recently diagnosed ( $\leq 6$  months) OSA and an apnea-hypopnea index (AHI) of 20 events/hour or more. These patients had been prescribed continuous positive airway pressure (CPAP) therapy to manage OSA and were identified via accredited community sleep clinics. The trial was conducted between September 2006 and March 2009 by university- and teaching hospital-based clinical researchers in Melbourne, Australia. Patients with obesity hypoventilation syndrome, previous bariatric surgery, contraindications to bariatric surgery, or significant cardiopulmonary, neurological, vascular, gastrointestinal, or neoplastic disease were excluded.

**Interventions** Patients were randomized to a conventional weight loss program that included regular consultations with a dietitian and physician, and the use of very low-calorie diets as necessary (n=30) or to bariatric surgery (laparoscopic adjustable gastric banding; n=30).

**Main Outcome Measures** The primary outcome was baseline to 2-year change in AHI on diagnostic polysomnography scored by staff blinded to randomization. Secondary outcomes were changes in weight, CPAP adherence, and functional status.

**Results** Patients lost a mean of 5.1 kg (95% CI, 0.8 to 9.3 kg) in the conventional weight loss program compared with 27.8 kg (95% CI, 20.9 to 34.7 kg) in the bariatric surgery group (P<.001). The AHI decreased by 14.0 events/hour (95% CI, 3.3 to 24.6 events/hour) in the conventional weight loss group and by 25.5 events/hour (95% CI, 14.2 to 36.7 events/hour) in the bariatric surgery group. The between-group difference was -11.5 events/hour (95% CI, -28.3 to 5.3 events/hour; P=.18). CPAP adherence did not differ between the groups. The bariatric surgery group had greater improvement in the Short Form 36 physical component summary score (mean, 9.3 [95% CI, 0.5 to 18.0]; P=.04).

**Conclusion** Among a group of obese patients with OSA, the use of bariatric surgery compared with conventional weight loss therapy did not result in a statistically greater reduction in AHI despite major differences in weight loss.

**Trial Registration** anzctr.org Identifier: 12605000161628

JAMA. 2012;308(11):1142-1149

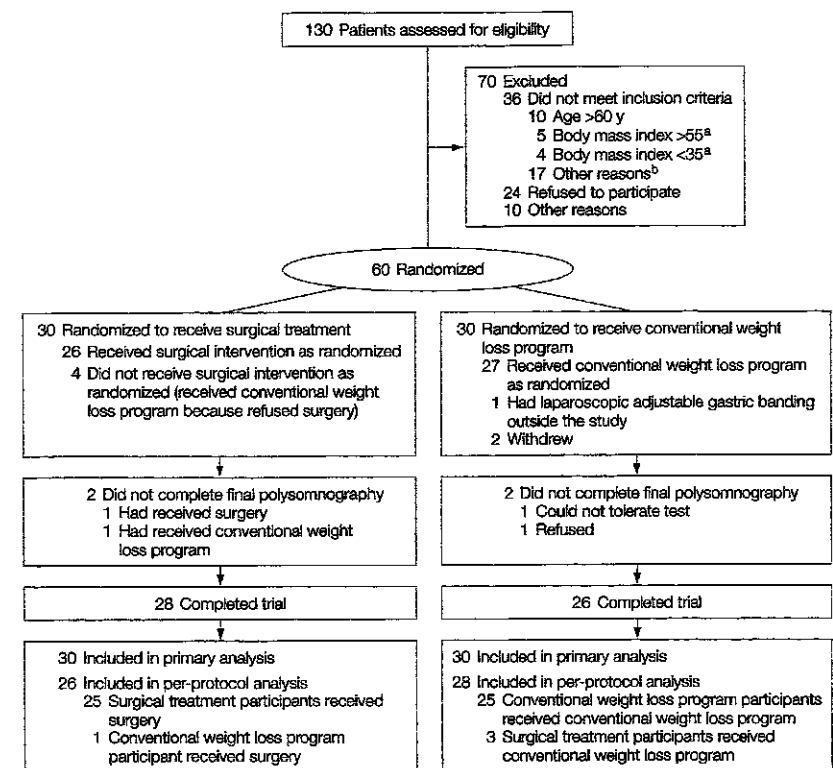
www.jama.com

**Author Affiliations:** Obesity Research Unit, Department of General Practice (Drs Dixon and Jones), Centre for Obesity Research and Education (Drs Schachter, O'Brien, and Brown), and Departments of Medicine (Drs Schachter, Lambert, and Naughton), Surgery (Dr Brown), Epidemiology and Preventive Medicine (Dr Bailey), Monash University, Melbourne, Victoria, Australia; Department of Allergy, Immunology, and

Respiratory Medicine, Alfred Hospital, Melbourne, Victoria, Australia (Dr Naughton); and Baker IDI Heart and Diabetes Institute, Melbourne, Victoria, Australia (Drs Dixon and Lambert and Ms Grima).

**Corresponding Author:** John B. Dixon, MBBS, PhD, FRACGP, Baker IDI Heart and Diabetes Institute, 75 Commercial Rd, Melbourne 3004, Australia (john.dixon@bakeridi.edu.au).

Figure 1. Participant Recruitment, Exclusions, and Flow Throughout the Study



<sup>a</sup> Calculated as weight in kilograms divided by height in meters squared.

<sup>b</sup> Included obesity hypoventilation syndrome, previous bariatric surgery, contraindications to bariatric surgery, or significant cardiopulmonary, neurological, vascular, gastrointestinal, or neoplastic disease.

Table 2. Longitudinal Analysis With Multiple Imputation for Missing Data for Polysomnography Variables and Weight at 2 Years

	Mean (95% CI)			No. (%) Missing at 2 y	P Value
	Surgical Treatment (n = 30)	Conventional Weight Loss Program (n = 30)	Between-Group Difference		
Weight, kg	107 (99 to 116)	121 (113 to 129)			
Change in weight, kg	-27.8 (-34.7 to -20.9) <sup>a</sup>	-5.1 (-9.3 to -0.8) <sup>a</sup>	-22.7 (-31.1 to -14.3)	2 (3)	<.001
Apnea-hypopnea index, events/h	39.5 (28.4 to 50.5) <sup>a</sup>	43.2 (34.9 to 51.9) <sup>a</sup>			
Change in apnea-hypopnea index, events/h	-25.5 (-36.7 to -14.2)	-14.0 (-24.6 to -3.3)	-11.5 (-28.3 to 5.3)	6 (10)	.18
Total sleep time, min	373 (348 to 399)	333 (297 to 370)			
Change in total sleep time, min	22.0 (-5.5 to 46.5)	10.5 (-25.5 to 46.4)	11.5 (-39.0 to 62.1)	6 (10)	.65
Sleep latency, min	18.6 (11.3 to 26)	24.5 (15.0 to 34.1)			
Change in sleep latency, min	-6.3 (-13.7 to 1.0)	4.2 (-4.4 to 12.8)	-10.6 (-24.4 to 2.3)	6 (10)	.11
Sleep efficiency, %	79.8 (75.8 to 83.8)	72.4 (65.3 to 79.6)			
Change in sleep efficiency, %	1.6 (-3.4 to 6.6)	-3.04 (-9.70 to 3.70)	4.7 (-4.6 to 13.9)	6 (10)	.32
Supine sleep, %	47.1 (35.5 to 58.6)	37.0 (25.1 to 48.8)			
Change in supine sleep, %	2.9 (-7.6 to 13.4)	-1.7 (11.5 to 8.0)	4.6 (-11.3 to 20.5)	6 (10)	.57
Slow wave sleep, %	15.3 (10.5 to 20.1)	21.1 (13.1 to 29.1)			
Change in slow wave sleep, %	0.7 (-2.7 to 4.1)	2.6 (-3.5 to 8.7)	-1.9 (-10.0 to 6.3)	7 (12)	.65
Rapid eye movement sleep, %	15.5 (12.8 to 18.2)	11.3 (8.4 to 14.2)			
Change in rapid eye movement sleep, %	2.6 (-1.1 to 6.3)	-1.6 (-4.7 to 1.5)	4.2 (-1.1 to 9.5)	7 (12)	.12
Apnea-hypopnea index rapid eye movement sleep, events/h	34.0 (21.8 to 46.2)	54.5 (43.6 to 65.4)			
Change in apnea-hypopnea index rapid eye movement sleep, events/h	-32.2 (-49.7 to -14.7) <sup>a</sup>	-13.5 (-26.7 to -0.3) <sup>a</sup>	-18.7 (-44.8 to 7.5)	10 (18)	.16
Arousal index, events/h	29.9 (23.2 to 36.7)	33.6 (27.8 to 39.4)			
Change in arousal index, events/h	-14.9 (-28.0 to -1.8) <sup>a</sup>	-24.9 (-62.5 to 12.7)	10.0 (-32.0 to 52.5)	15 (27)	.64
Available hemoglobin saturated with oxygen					
During sleep, %	95.5 (94.9 to 96.1)	95.2 (94.9 to 95.9) <sup>a</sup>			
Change during sleep, %	1.2 (0.3 to 2.0) <sup>a</sup>	0.2 (-1.1 to 1.4)	1.0 (-0.6 to 2.6)	9 (17)	.22
Minimum, %	79.0 (73.9 to 84.0)	76.2 (68.0 to 84.3)			
Change in minimum, %	7.2 (1.9 to 12.5)	3.7 (-2.8 to 10.2)	3.4 (-6.5 to 13.4)	9 (17)	.50
Period when level <90%, min	23.4 (10.3 to 36.5)	28.9 (9.1 to 48.7)			
Change in period when level <90%, min	-59.4 (-95.6 to -23.2) <sup>a</sup>	-28.1 (-50.8 to -5.3)	-31.3 (-77.7 to 15.1)	13 (22)	.19

<sup>a</sup> Indicates significant within-group improvement (P<.05).

outcome