

Case Report

Effect of a very low-energy diet on moderate and severe obstructive sleep apnoea: case reports

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Summary

In this article, we report two cases from our study in which we investigated the effect of a very low-energy diet (VLED) for 9 weeks followed by a weight maintenance program for a total duration of 1 year in obese men with moderate to severe obstructive sleep apnea. The first case was a 43-year-old man with a body weight of 126 kg and a body mass index (BMI) of 38.8 kg/m² at baseline. Over the 1-year treatment program he lost 26% of his initial weight and his apnea-hypopnea index (AHI) was improved by 60%. The second case was at baseline a 54-year-old man with a weight of 87 kg and BMI of 32.1 kg/m². Over the 1-year treatment program he lost 10% of his initial weight and his AHI improved by 66%. In summary, treatment with a VLED followed by a weight maintenance program significantly improved obstructive sleep apnea in both cases.

Keywords: Obesity, obstructive sleep apnea, very low-energy diet, weight loss.

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Introduction

The majority of patients with obstructive sleep apnea (60–70%) are either overweight or obese (1,2). Given the close association between obstructive sleep apnea and obesity, weight loss has been advocated as a primary treatment option in obese patients, but randomized controlled trials supporting this concept has until recently been lacking. During 2009, three controlled trials (3–5) in various patient categories were published investigating the effect of weight loss, with different intensity and duration, on obstructive sleep apnea. In this article, we report two cases from our study in which we investigated the effect of a very low-energy diet (VLED) for 9 weeks (5) followed by a weight maintenance programme (Johansson unpublished data) for a total duration of 1 year in obese men with moderate to severe obstructive sleep apnea.

Case 1

The first case was a 43-year-old man with a body weight of 126 kg and a body mass index (BMI) of 38.8 kg m⁻² (Table 1). Weight gain gradually started after he stopped playing soccer at the age of 30 years. At baseline, the patient had severe sleep apnea (apnea-hypopnea index [AHI] > 30) and was treated with continuous positive airway pressure (CPAP). He also had hypertension, dyslipidemia and metabolic syndrome. Prior to the study he had a sedentary lifestyle.

During the VLED phase the patient lost 30 kg, BMI was reduced to 29.5 kg m⁻² and AHI was reduced from 37 to 12 events h⁻¹. During the weight maintenance programme he lost additional weight but had a slight increase in AHI. Over the 1-year treatment programme he lost 26% of his initial weight, AHI was improved by 60% and he went from severe (AHI > 30) to mild (AHI 5–15) sleep apnea

Table 1 Changes in anthropometric, sleep and metabolic variables for case 1 and 2 during the 1-year weight-loss programme

	Case 1			Case 2		
	Baseline	After VLED change 0–9	After 1 year change 0–52	Baseline	After VLED change 0–9	After 1 year change 0–52
Anthropometrics						
Weight (kg)	125.8	95.7	92.9	86.9	72.8	78.3
Change		–30.1	–32.9		–14.1	–8.7
BMI (kg m ⁻²)	38.8	29.5	28.7	32.1	26.9	28.9
Change		–9.3	–10.1		–5.2	–3.2
Waist circumference (cm)	121	96	92	111	94	98
Change		–25	–29		–17	–13
Neck (cm)	45	40	40	44	40	41
Change		–5	–5		–4	–3
Percentage body fat (%)	34.5	25.9	23	31.7	22.3	28.0
Change		–8.6	–11.5		–9.4	–3.7
Sleep data						
AHI (events h ⁻¹)	37	12	15	56	21	19
Change		–25	–22		–35	–37
Snoring time > 50 dB (%)	36	19	1	63	28	28
Change		–17	–35		–35	–35
Nadir oxygen saturation (%)	83	82	81	80	84	82
Change		–1	–2		+4	+2
Treatment	CPAP	No	No	CPAP	CPAP	CPAP
Change						
Metabolic variables						
Systolic (mmHg)	155	120	120	110	112	120
Change		–35	–35		–2	+10
Diastolic (mmHg)	90	70	80	78	80	90
Change		–20	–10		–2	+12
Hypertension	Yes	No	No	No	No	Borderline
Metabolic syndrome	Yes	No	No	No	No	No
Triglycerides (mmol L ⁻¹)	2.0	0.8	0.5	1.4	1.1	1.1
Change		–1.2	–1.5		–0.3	–0.3
Cholesterol (mmol L ⁻¹)						
Total	6.5	4.6	5.2	4.6	3.6	4.7
Change		–1.9	–1.3		–1.0	+0.1
LDL	4.0	2.8	2.7	2.7	2.1	2.5
Change		–1.2	–1.3		–0.6	–0.2
HDL	1.6	1.5	2.3	1.2	1.1	1.7
Change		–0.1	+0.7		–0.1	+0.5
Dyslipidemia	Yes	No	Borderline	No	No	No
Fasting glucose (mmol L ⁻¹)	5.2	4.8	4.4	5.6	4.7	6.1
Change		–0.4	–0.8		–0.9	+0.5
Fasting insulin (mU L ⁻¹)	10.9	5.8	5.4	9.9	6.0	9.8
Change		–5.1	–5.5		–3.9	–0.1
HOMA-IR	2.5	1.2	1.1	2.5	1.3	2.7
Change		–1.3	–1.4		–1.2	+0.2

Hypertension was defined as systolic/diastolic blood pressure of 140/90 mmHg or greater and/or anti-hypertensive drug treatment. **Metabolic syndrome** was defined according to the *International Diabetes Federation and the American Heart Association/National Heart, Lung, and Blood Institute, joined by the World Heart Federation, International Atherosclerosis Society, and International Association for the Study of Obesity* (6).

Dyslipidemia was defined as total cholesterol > 5.0 mmol L⁻¹ and/or LDL > 3.0 mmol L⁻¹ and/or HDL < 1.0 mmol L⁻¹ and/or triglycerides > 1.7 mmol L⁻¹ in patients without cardiovascular disease and as total cholesterol > 4.5 mmol L⁻¹ and LDL > 2.5 mmol L⁻¹ in patients with cardiovascular disease and/or drug treatment. **Insulin resistance** was calculated by use of the homoeostasis model assessment HOMA-IR, which is defined as fasting plasma insulin (mU L⁻¹) times fasting plasma glucose (mmol L⁻¹) divided by 22.5 (7).

AHI, apnea-hypopnea index; BMI, body mass index; CPAP, continuous positive airway pressure; HDL, high-density lipoprotein; HOMA-IR, homoeostasis model assessment of insulin resistance; LDL, low-density lipoprotein; VLED, very low-energy diet.

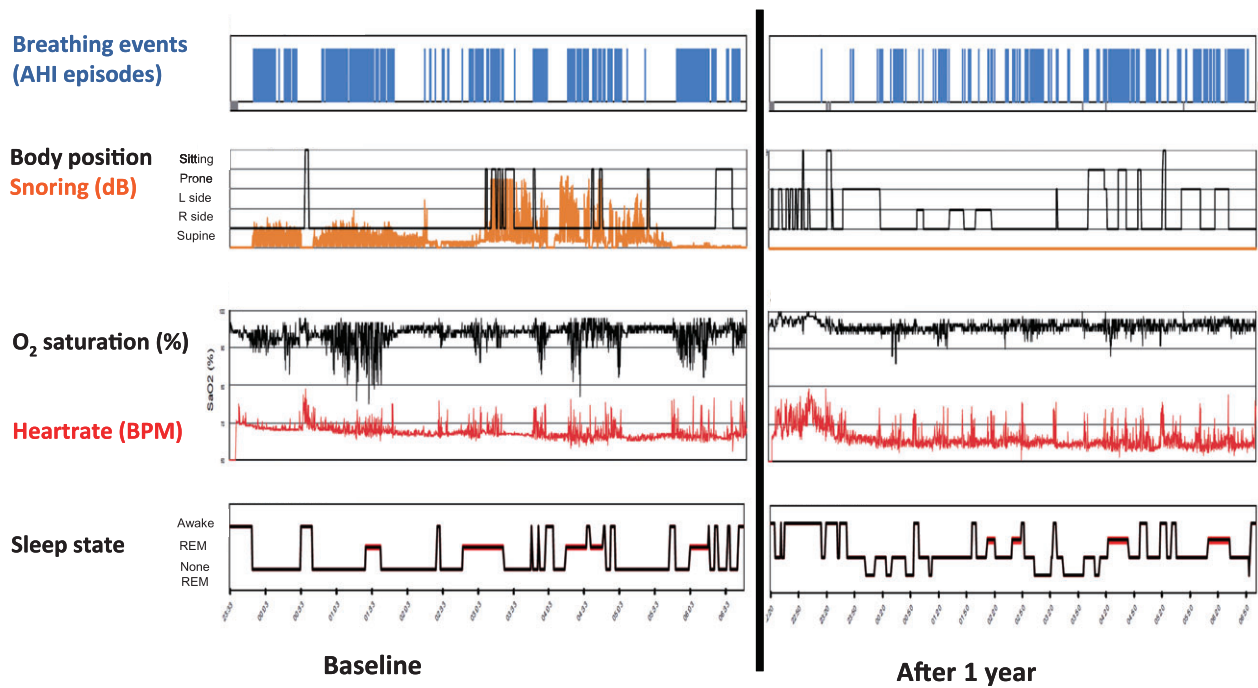


Figure 1 Sleep study before and after 1 year for case 1. AHI, apnea-hypopnea index; BPM, beats per minute; REM, rapid eye movement.

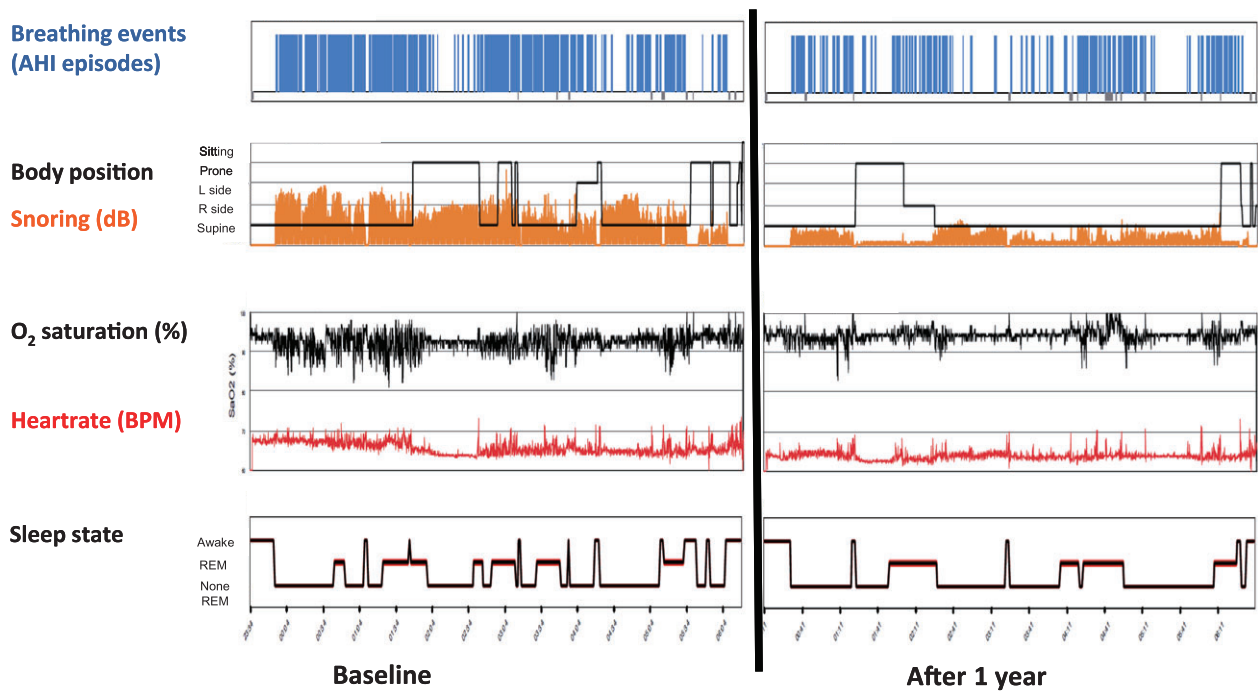


Figure 2 Sleep study before and after 1 year for case 2. AHI, apnea-hypopnea index; BPM, beats per minute; REM, rapid eye movement.

and no longer needed treatment with CPAP (Table 1 and Fig. 1).

In addition, all his comorbidities were eliminated and he suffered no adverse events to the diet. During and after the weight-loss period he started to exercise again and 1 year after the weight-loss period he completed a half marathon.

Case 2

The second case was a 54-year-old man with a weight of 87 kg and BMI of 32.1 kg m⁻² (Table 1). His weight gain had started after he stopped smoking at the age of 43 years. At baseline, he had severe sleep apnea and was treated with CPAP, but had no other comorbidities. Prior to the study he had a sedentary lifestyle.

During the VLED phase the patient lost 14 kg, BMI was reduced to 26.9 kg m⁻² and AHI was reduced from 56 to 21 events h⁻¹. During the weight maintenance programme the patient increased some in weight but his AHI decreased further. Over the 1-year treatment programme he lost 10% of his initial weight. His AHI improved by 66% and he went from severe (AHI > 30) to moderate (AHI 15–30) sleep apnea. Despite this he still needed CPAP treatment (Table 1 and Fig. 2). In contrast to the previous case the current patient had two adverse events during the 1-year programme. After the VLED period he had a temporary increase in alanine aminotransferase (a liver enzyme) and he also contracted a gallstone, which he was operated for, 16 weeks after the VLED phase. Despite the adverse events the patient was positive to the weight-loss programme since he experienced large improvements in quality of life.

Conflict of Interest Statement

No conflict of interest was declared. Kari Johansson has received a travel grant from Cambridge Weight Plan to attend a scientific meeting.

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