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ORIGINAL CONTRIBUTIONS

Impact of Bariatric Surgery on Women Aerobic Exercise Capacity

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Abstract

Rationale Bariatric surgery has a considerable positive effect on weight loss and on metabolic and cardiovascular risks. It has therefore been extensively used this last decade to overcome obesity. However, the impact of this surgery on exercise capacity remains unclear. The aim of this study is to clarify the impact of a surgically induced weight loss on aerobic exercise capacity (VO_2max) in a specific middle-aged female population.

Methods Forty-two women with a body mass index > 40 kg/m^2 (age, 42 ± 13 years; weight, 117 ± 15 kg) underwent blood analyses and a cardiopulmonary exercise test (CPET) before and 1 year after bariatric surgery. CPET was performed on a cycloergometer. The first ventilatory threshold (VT1) was measured according to the V-slope method.

Results Absolute VO_2max was reduced by 10% after surgery (2.0 ± 0.4 vs 1.8 ± 0.4 l/min, $p < 0.01$) or increased when corrected for body weight (18 ± 4 vs 23 ± 4 l/min/kg, $p < 0.001$) or unchanged when expressed as percentage of predicted values (111 ± 21 vs 105 ± 22, $p = 0.06$). Weight loss did not affect ventilatory or chronotropic response but increased maximal respiratory exchange ratio (RER) ($p < 0.001$), decreased maximal O_2pulse ($p < 0.05$) and VT1 in milliliters per minute ($p < 0.01$). By multivariable analysis, decreased absolute VO_2max after weight loss was associated with increased maximal RER and reduced maximal O_2pulse ($p < 0.05$, $p < 0.01$ respectively), possibly related to a muscular mass limitation.

Conclusions Weight loss induced by bariatric surgery may reduce aerobic capacity in women in relation to muscle mass loss.

Keywords Obesity · Weight loss · VO_2max · Sleeve · Gastric bypass

Introduction

Obesity is a life-threatening condition associated with higher rates of cardiovascular, metabolic and oncologic morbidities and mortalities [1–3]. Bariatric surgery is an invasive but effective therapy to reduce obesity-associated comorbidities and mortality [4, 5] and is therefore overspreading worldwide [6]. Contrastingly, whilst surgically induced weight loss is generally believed to be associated with a better quality of life [7, 8] and an increased daily physical activity level [5, 8], some patients still complain about dyspnea and asthenia.

Previous studies evaluating the aerobic exercise capacity (VO_2max) with a cardiopulmonary exercise test (CPET) before and after bariatric surgery showed controversial results with either an increased [9, 10], unchanged [11–15] or decreased VO_2max [16–20]. This discrepancy results emerged from different population characteristics (body mass index (BMI), age, sex, comorbidities, etc.), surgical conditions (type of surgery, complications, etc.), post-surgery testing time, amount of weight loss, exercise testing protocol (weight-carrying vs unweight carrying ergometer, direct or indirect measurements of VO_2max, etc.) and expression of results (absolute VO_2max or corrected for body dimensions). As VO_2max is the gold standard to assess exercise capacity but also an important independent predictor of cardiovascular risks and general mortality, a clear description of the impact of bariatric surgery on this parameter is crucial [21].

In order to clarify the situation, we investigated the effect of a massive weight loss 1 year after bariatric surgery on aerobic exercise capacity measured during an unweight carrying CPET on a cycloergometer. A homogeneous population of middle-aged women with type III obesity (BMI > 40 kg/m^2)

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has been selected as it reflects the predominantly population undergoing this type of surgery [6]. As aerobic exercise capacity is dependent on the integrity of the O_2 transport system from the lungs to the muscular mitochondrion, it is of great interest to investigate on limiting factors to better advise specific rehabilitation of the aerobic exercise capacity during the post-surgery period.

Methods

Forty-two women (42 ± 13 years old; height,164 ± 6 cm; weight, 117 ± 15 kg) with a BMI of 44 ± 4 kg/m^2 were recruited from the Wallonie Picarde Hospital and Erasme Hospital, Belgium. The study was approved by the Ethical Committee of the Erasme University Hospital (reference P2019/179). Patients suffering from cardiovascular or pulmonary disease and taking beta-blockers were excluded. All subjects met the legal criterion for bariatric surgery (BMI > 40 kg/m^2) and underwent either a sleeve gastrectomy (N = 18) or Roux-en-Y gastric bypass (N = 24). After surgery, patients had a dietary follow-up at least every 3 months and were advised to exercise and to increase their daily life activities. However, none of them participated to a rehabilitation programme. All declared themselves to be active but not performing more than 2 h/week of moderate intensity activities.

Experimental Protocol

All subjects underwent a clinical assessment, blood sampling and a CPET maximum of 3 months before bariatric surgery. Tests were repeated strictly 1 year after the intervention. This post-surgery period is characterized by a smoothening of an intense post-surgery weight loss.

Blood Sampling and Anthropometry

Fasting blood tests were analysed for metabolic syndrome and for hematologic assessment by the same hospital laboratory (Wallonie Picarde Hospital, Belgium). Weight was measured to the nearest 0.1 kg on a standing weighting scale (CAE, Bande, Belgium) wearing no shoes and light clothing. Stature was measured to the nearest 0.5 cm with a wall-mounted stadiometer. Weight and height were used to calculate BMI as weight (kg)/height (m^2).

Cardiopulmonary Exercise Test

The CPET was performed with breath by breath measurements of oxygen consumption (VO_2), carbon dioxide production (VCO_2) and ventilation (VE) on a stationary and graded cycle ergometer (Schiller CH640/BAR, Ergoline, Switzerland) at progressively increased workload (W). Exercise started with an unloaded 2 min warm-up period at 60 rpm followed by an increased workload of 20 W/min in a ramp protocol until exhaustion. Gas exchange was measured through a facial mask, collected and analysed every 5 s using a metabolic recording system (MasterScreen CPX, Jeager, Carefusion, Germany) previously calibrated with room air and standardized gas (SBx/CPX, Jeager, Germany). Ear lobe arterial oxygen saturation (SpO_2) and heart rate (HR) were measured continuously. Blood pressure (BP) was recorded every 2 min. VO_2max was considered to be achieved when two of the following criteria were met: an increase in VO_2 of less than 100 ml/min with a further increase in workload, a respiratory exchange ratio (RER) greater than 1.1 or age-predicted maximal HR. The Riddle et al. [22] prediction equation has been used to express the VO_2max in percentage of predicted values as this equation has recently been suggested to be the best option to determine whether the aerobic exercise capacity is normal in women with obesity [23, 24]:

$$\text{Predicted } VO_2\text{max (ml/min)} = (48 - 0.37 \times \text{age}) \times \text{predicted weight (kg)}$$

$$\text{Predicted weight} = (3.55 \times \text{height (cm)}/2.54 - 106)/2.2$$

The VT1 was determined by the V-slope when the angular coefficient of the VCO_2 vs VO_2 relationship increases. With this method, VT1 is considered to be a body mass–independent parameter as VCO_2 and VO_2 are both corrected for body weight [25].

Limiting factors of exercise capacity have been considered. Pulmonary limitation was considered if maximal VE reached 85% of the maximal voluntary ventilation, calculated by 35 × 1-s forced expiratory volume (FEV1). Breathing efficiency was defined as VE (l/min) divided by VO_2 (l/min). Chemosensibility was assessed by the VE/VCO_2 ratio measured at the VT1 and the VE/VCO_2 slope up to the respiratory compensation point. Cardiovascular limitation was assessed if theoretical maximal HR was reached, maximal O_2pulse decreased or reached a plateau, increased HR/VO_2 slope or decreased VO_2/Workload slope. Metabolic limit was reached when RER > 1.1 or VT1 decreased.

Statistical Analyses

Continuous variables were presented with mean and standard deviations after checking for normality of distributions. Student's t tests were used to test the effect of the type of surgery on metabolic and exercise changes 1 year after surgery. Pre- and post-intervention were compared with a paired Student t test. A univariate linear regression was performed to determine the significant variables associated with weight loss or the changes in VO_2max (ml/min) after surgery, followed by a multivariable linear regression to find the independent variables associated with the changes in VO_2max after surgery.

Table 1 Anthropometric measurements and blood sampling before and 1 year after bariatric surgery ($n = 42$)

Variables	Pre-surgery	Post-surgery
Age (years)	42 ± 13	43 ± 13
Height (cm)	164 ± 6	164 ± 6
Weight (kg)	117 ± 15	81 ± 14***
BMI (kg/m^2)	44 ± 4	30 ± 4***
SBP rest (mmHg)	131 ± 15	123 ± 16 **
DBP rest (mmHg)	81 ± 14	73 ± 11**
Glucose (mg/dl)	94 ± 15	83 ± 9***
HDL (mg/dl)	53 ± 12	63 ± 14**
LDL (mg/dl)	108 ± 31	81 ± 24***
TG (mg/dl)	102 ± 31	84 ± 30**
CRP (mg/l)	11 ± 9	6 ± 6**
Albumin (g/l)	39 ± 5	39 ± 4
Haemoglobin (g/dl)	14.0 ± 0.9	13.2 ± 0.9***
Iron (μg/dl)	76 ± 38	85 ± 24
Ferritin (ηg/ml)	92 ± 71	70 ± 61**
Iron saturation coefficient (%)	21 ± 8	27 ± 10
B12 vitamin (pg/ml)	339 ± 105	309 ± 110*
B9 vitamin (ng/ml)	6.1 ± 3.0	8.3 ± 4.6
Creatinine (mg/dl)	0.69 ± 0.13	0.68 ± 0.09

BMI, body mass index; *SBP*, systolic blood pressure; *DBP*, diastolic blood pressure; *HDL*, high-density lipoproteins; *LDL*, low-density lipoproteins; *TG*, triglycerides; *CRP*, C-reactive protein

*$p < 0.05$; **$p < 0.01$; ***$p < 0.001$

The results are presented as mean ± SD and the significance level was set at $p < 0.05$ for all analyses.

Results

Clinical Examination and Blood Sampling

As shown in Table 1, body weight was reduced by more than 30% 1 year after bariatric surgery bringing BMI to the lower limit of the obesity definition. Metabolic syndrome factors were improved after surgery with a decrease in resting systemic BP, triglyceride, low-density lipoprotein (LDL) and fasting blood glucose and an increase in high-density lipoprotein (HDL). Surgery also reduced C-reactive protein (CRP), haemoglobin (Hb), red blood cell count ($p < 0.001$, not shown), ferritin levels and vitamin B_{12} (only in Roux-en-Y gastric bypass surgery (RYGB)) whilst the mean corpuscular volume increased ($p < 0.01$, not shown). Mean blood albumin level, creatinine, folic acid, iron concentration and the iron saturation coefficient did not significantly change with weight loss.

Except for a vitamin B_{12} reduction after RYGB, no difference in anthropometric measurements, blood sampling and aerobic exercise capacity was observed before and after surgery between the RYGB ($N = 24$) vs sleeve gastrectomy ($N = 18$) (data not shown).

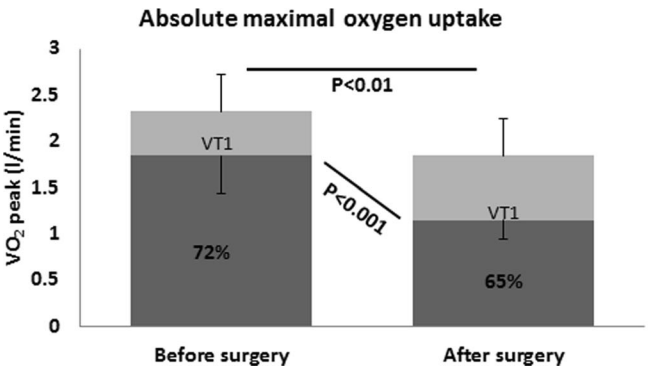
Cardiopulmonary Exercise Testing

Impact of bariatric surgery on aerobic exercise capacity is illustrated in Fig. 1. Bariatric surgery reduced maximal workload, VO_2max (ml/min), end-exercise O_2pulse and the VT1 with increasing end exercise RER. VO_2max expressed in millilitres per minute per kilogramme was increased with body weight loss and remained in the normal range of predicted values (Table 2).

Maximal HR, VE, SpO_2 and VO_2/W, VE/VCO_2, HR/VO_2 slopes remained unaffected after weight loss (Table 2).

Correlations

The magnitude of the surgically induced weight loss was correlated to the increase in HDL ($R = -0.47$, $p < 0.05$) and decrease in LDL ($R = 0.46$, $p < 0.05$) and Hb concentration ($R = 0.39$, $p < 0.05$) but also with changes in VO_2max (ml/min) ($R = 0.31$, $p < 0.05$) and the VE/VCO_2 slope ($R = 0.37$, $p < 0.05$).



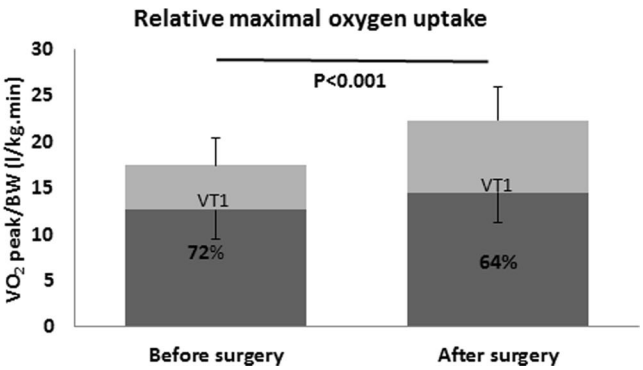


Fig. 1 Comparison between the absolute and relative oxygen uptake below (dark grey) and above (light grey) the ventilatory threshold (VT1) before and 1 year after surgically induced weight loss. The number in the columns indicate the percentage of oxygen uptake below the ventilatory threshold. This figure illustrates the impaired VO_2max and VT1 after surgery

Table 2 Cardiopulmonary exercise testing before and after bariatric surgery

Variables	Pre-surgery	Post-surgery
W (watts)	139 ± 26	133 ± 19*
VO_2max (ml/min)	1995 ± 416	1851 ± 367**
VO_2max (ml/kg/min)	18 ± 4	23 ± 4***
VO_2max (% predicted)	111 ± 21	105 ± 22
HRmax (bpm)	158 ± 13	155 ± 21
VEmax (l/min)	76 ± 16	81 ± 16
SpO_2max (%)	98 ± 2	97 ± 4
RER	1.15 ± 0.09	1.26 ± 0.08***
O_2Pulse (ml/bpm)	12.7 ± 2.6	12.0 ± 2.4***
SBPmax (mmHg)	179 ± 23	169 ± 16*
DBPmax (mmHg)	87 ± 11	82 ± 9*
Ventilatory threshold		
Workload (W)	98 ± 28	79 ± 14***
VO_2 (ml/kg/min)	13 ± 3	14 ± 3*
VE/VCO_2	28 ± 2	29 ± 3
Slopes		
VE/VCO_2	30.3 ± 4.6	30.4 ± 2.8
VO_2/W (ml/kg/min/W)	9.2 ± 1.6	10.6 ± 0.7
HR/VO_2 (b/ml/kg)	5.0 ± 1.9	5.2 ± 1.3

W, workload; *VO_2*, oxygen uptake; *HR*, heart rate; *VE*, minute ventilation; *SpO_2*, pulse oximetry oxygen saturation; *RER*, respiratory exchange ratio; *VCO_2*, carbon dioxide output

*$p < 0.05$; **$p < 0.01$; ***$p < 0.001$

The post-surgery changes in Hb concentrations were positively correlated to the changes in vitamin B_9 and B_{12} (both $R = 0.41$, $p < 0.01$).

Serum creatinine changes after surgery were correlated to the changes in VT1 ($R = 0.30$, $p < 0.05$), O_2pulse ($R = 0.42$, $p < 0.01$) and VO_2max ($R = 0.42$, $p < 0.01$).

The magnitude of the changes in VO_2max after surgery was positively correlated to the changes in Hb ($R = 0.34$, $p < 0.05$) and iron concentration ($R = 0.50$, $p < 0.05$), coefficient of iron saturation ($R = 0.35$, $p < 0.05$), creatinine concentration ($R = 0.42$, $p < 0.01$), maximal W ($R = 0.59$, $p < 0.01$), VT1 ($R = 0.48$, $p < 0.01$), maximal O_2pulse ($R = 0.63$, $p < 0.001$) and VO_2/W slope ($R = 0.57$, $p < 0.01$), and inversely correlated to the changes in the end exercise RER ($R = -0.49$, $p < 0.01$).

The multivariable analysis showed that only surgically induced changes in end exercise RER and maximal O_2pulse were independently associated with the changes in VO_2max (ml/min) (adjusted R^2 of the mode 0.404).

Discussion

The present results confirm that bariatric surgery reduces cardiovascular and metabolic risk factors proportional to weight loss. In a middle-aged women population with obesity, surgically induced weight loss increased relative maximal aerobic capacity but decreased absolute VO_2max with a lower ventilatory or dyspnea threshold. This could be explained by a combination of a reduction in haemoglobin concentration and a muscular limitation.

Aerobic Exercise Capacity

VO_2 is commonly expressed relative to body mass allowing for body dimensions correction. This penalizes heavier individuals and women with an unfavourable body composition [26, 27]. Tanner discussed the fallacy of this ratio method 70 years ago by demonstrating that expressing the VO_2 relative to mass is valid in an approximate normal adult population, but an insidious drift will be introduced in any deviating situation [28]. It also introduces a confounding factor when VO_2max is compared in specific situations where body mass changes occur with growth, training, sickness, diet, etc. The increased VO_2max corrected for body weight observed after bariatric surgery in the present and previous studies is highly dependent on body mass loss after the intervention. This information may be relevant in terms of exercise capacity in body weight–bearing efforts, frequent in daily life activities (walking, running) although a recent study demonstrated that the metabolic cost for walking or climbing stairs is identical in obesity and normo-weighted subjects [23]. However, treadmill exercise testing studies before and after bariatric surgery showed indeed reduced metabolic costs of body weight–bearing activities with lower ventilation cost and a higher dyspnea threshold level [8–10, 17, 19, 29]. However, treadmill studies showed unchanged or reduced absolute VO_2max after surgery [10, 12, 17, 19, 20].

In the present study, CPET performed on a cycloergometer revealed a 7% average reduction of absolute VO_2max. This is a greater loss than the 1% decrease observed per year with ageing [30]. Lund et al. described similar absolute VO_2max decrease 4 and 18 months after RYGB surgery and concluded that patients do not adopt new exercise habits following surgery [18]. However, it is to mention that this analysis refers to mean values. Intersubject variability confirms that weight loss does not universally reduce exercise capacity but that an individual post-surgery follow-up should be encouraged focusing on the improvement of the exercise limiting factors of aerobic capacity.

Considering the inconsistent consensus on expressing VO_2max in obesity, it remains difficult to quantify individual survival prognosis. It has recently been recommended to quantify VO_2max in percentage of the prediction equations from Riddle et al. [22–24]. Accordingly, the present subjects had an above normal cardiorespiratory fitness before surgery only mildly reduced after surgery. It is however not clear if this reduction could account for worst prognostic as predicted

VO_2max remains in the normal range and as, paradoxically, blood cardiovascular risks predictors are improved after weight loss. It also remains unclear if similar results would appear in a male population.

Table 3 lists the main studies with available data on the impact of bariatric surgery on cardiorespiratory fitness changes measured directly with CPET. Analysis of these changes allows for a better understanding of limiting factors of oxygen transport from the lungs to the mitochondria.

Respiratory Limitation

Hypoventilation and a poor compensatory hyperventilation at high-intensity exercise is common in obese people with increased mechanical load on the thorax [15, 31]. Zavorsky et al. compared pulmonary function and arterial blood-gas at maximal exercise before and 10 weeks after bariatric surgery, showing that overall fat mass loss allowed adequate compensatory hyperventilation resulting in improved pulmonary gas exchange at peak exercise [15]. In the present study, bariatric surgery only slightly increased maximal ventilation (+ 7%, $p = 0.052$), but achieved at a lower maximal workload confirming a mechanical facilitation to fill the lung [10, 31]. Moreover, the VE/VCO_2 slope was inversely correlated to the amount of weight loss. The VE/VCO_2 slope is known to be an independent predictor of mortality in heart failure and other chronic diseases, probably related to increased chemosensibility [32]. This observation confirms the weight loss–induced reduction of ventilatory cost at a given metabolic level. However, those trivial ventilatory changes did not influence SpO_2 or VO_2max positively.

Convective Oxygen Transport Limitation

It has been previously mentioned, although not directly measured, that maximal oxygen transport represented by maximal cardiac output and end exercise arterial oxygen content is not impaired after bariatric surgery [10, 17, 26]. In the present study arterial oxygen content is reduced by a slight post-surgery decrease in haemoglobin concentration independent of the type of surgery (bypass or sleeve). In respiratory-limited patients, a slight decrease in haemoglobin affects aerobic exercise capacity, functional capacity and quality of life by triggering an early anaerobic metabolism with consequent stimulus to ventilation [33]. However, according to previous studies in healthy subjects, the 0.88 g/dl decrease of haemoglobin observed in the present study would only account for 1–2% reduction in VO_2max [34] and can therefore not be considered as the main aerobic exercise limitation factor. The reason for this post-surgery haemoglobin reduction remains unclear, but a relation with a malabsorption or insufficient supplementation of vitamins B_{12} and B_9 could be suspected as the post-surgery changes in Hb levels are associated with changes in those vitamins, essential for erythropoiesis.

Chronotropic response remained unchanged but changes in VO_2max were associated to changes in maximal O_2pulse, a composite index including stroke volume and arterio-venous oxygen difference. Previous studies demonstrated that weight reduction in patients with type III obesity usually improves cardiac function by decreasing ventricular wall thickness, systemic arterial pressure and blood volume and improving left ventricular diastolic function [17, 35]. This latest observation has been shown to be correlated to exercise capacity 6 months after vertical-banded gastroplasty. Accordingly, it seems reasonable to assume that the reduced O_2pulse observed in the present study is more likely related to muscular rather than cardiac limitation. In a similar study, Seres et al. offer an additional argument by showing that post-surgery reduction in O_2pulse disappeared after normalization for fat free mass (FFM) reflecting minimal influence of a cardiac limitation [10].

Muscular and Metabolic Limitation

In the present study, post-surgery VO_2max changes were correlated to changes in maximal RER, O_2pulse and the ventilatory threshold level. With an additional post-surgery maximal workload reduction, all arguments are in favour of a muscular limitation. Unfortunately, a limitation of this study is that it does not provide body composition measurements, which preclude any direct conclusion on muscular mass changes. However, numerous studies have previously indicated that lean body mass loss occurs with post-surgery fat mass loss [10, 12, 13, 16, 18, 36]. Lower muscle mass and oxidative capacity would indeed lead to a deconditioned profile with a low absolute VO_2max and VT1. In the present study, a positive correlation exists between the post-surgical changes in creatinine level, highly dependent on muscle mass, and changes in VO_2max and VT1.

Previous studies confirm early VT1 after surgery [13, 14, 17] but this parameter remains understudied and inconstantly reported although it reflects the activity level triggering dyspnea and is therefore related to quality of life. VT1 is indeed reduced in deconditioning situations when the purely aerobic performance of the muscle is reduced due to oxygen transport, muscular deconditioning or muscle mass loss.

However, only changes in maximal RER and in maximal O_2pulse were independent predictors of the changes in VO_2max. A high RER could be suggestive of high motivation but a higher RER at a lower maximal workload most likely reflect a higher anaerobic metabolism rate on less muscular fibres.

A mitochondrial affection after surgery could be excluded as it would decrease VO_2max relative to FFM

Table 3 Comparisons of studies on the influence of bariatric surgery on cardiopulmonary exercise testing

Study (year)	*N*	Months post-surgery	BMI (kg/m^2) pre-post	Ergometer	VO_2max (ml/min)	VO_2max (ml/min/kg)	VO_2max (ml/min/FFM)	Wmax (W)	RER	VEmax (L/min)	HRmax (bpm)	O_2puls (ml/min/bpm)	SpO_2 (%)	Respiratory equivalent	VT	Exercise economy
Wilms et al. 2013	18 (11f)	28	46–33	Bicycle	=	↑	NA	=	↑	=	=	=	NA	=	↓	=
Zavorsky et al. 2008	15 (11f)	2.5	47–40	Bicycle	=	↑	=	=	=	=	=	=	↑	=	NA	↑
Stegen et al. 2011	15 (11f)	4	40–32	Bicycle	=	↑	=	=	=	NA	=	NA	NA	NA	=	=
Dereppe et al. 2019	42 (42f)	12	44–30	Bicycle	↓	↑	NA	↓	↑	=	=	↓	=	=	↓	=
Neunhaeuserer et al. 2017	26 (18f)	6	45–33	Treadmill	↓	↑	NA	↑	=	↓	=	↓	↑	NA	NA	↑
Seres et al. 2006	31	12	51–33	Treadmill	=	↑	=	↑	↑	↑	↑	↓	NA	=	NA	NA
Kanoupakis et al. 2011	16 (10f)	6	49–34	Treadmill	↓	↑	NA	↑	=	↓	=	=	NA	=	↓	NA
Browning et al. 2017	9 (9f)	3	43–34	Treadmill	=	↑	=	↑	=	=	=	=	NA	NA	NA	NA

f female subjects, *BMI* body mass index during pre- and post-testing, *VO_2max* maximal oxygen uptake, *FFM* fat free mass, *Wmax* maximal workload, *RER* respiratory exchange ratio, *VEmax* maximal ventilation, *HRmax* maximal heart rate; *SpO_2* pulse oximetry oxygen saturation, *VT* ventilatory threshold, *NA* not available

which has consistently been shown to be preserved [10, 12, 13, 18]. Moreover, there is some evidence of mitochondrial remodelling after bariatric surgery associated with increased phosphorylation capacity in the absence of electron transport system capacity or mitochondrial abundance changes [16].

Another indirect argument of a muscle mass limitation is that exercise training has a positive effect on FFM and aerobic capacity [16, 37, 38]. This is of great interest and should be widely promoted. Indeed, a recent meta-analysis showed that in the absence of peri-surgical exercise promotion, moderate-to-vigorous physical activities are reduced within the first 6 months after bariatric surgery despite a greater active time which would lead to muscular deconditioning [39]. This finding might be explained by a reduction in VT1 after surgery, reducing the spontaneous exercise threshold level under dyspnea symptoms. Therefore, this study supports the crucial current idea that promoting adequate physical activity at a sufficiently high intensity is necessary to overcome the muscle mass loss and reverse the sedentary viscous circle. This should be recommended in parallel with adequate dietary (protein intake, etc.) and haematological (Hb, etc.) follow-up. It is, however, to mention that a longer follow-up is necessary to confirm a long-term muscular adverse effect of bariatric surgery.

In conclusion, although bariatric surgery has a multitude of health benefits, a slight haemoglobin decrease and muscle mass reduction with post-surgery weight loss may negatively affect the aerobic exercise capacity in women. This could be avoided by an individual exercise activity promotion and by an adequate hematologic follow-up before and after bariatric surgery.

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Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

Statement of Informed Consent Informed consent was obtained from all individual participants included in the study.

Statement of Human and Animal Rights/Ethical Approval The study was approved by the local Ethical Committee (Erasme University Hospital, reference P2019/179).

References

1. Flegal KM, Graubard BI, Williamson DF, et al. Cause-specific excess deaths associated with underweight, overweight and obesity. JAMA. 2007;298:2028–37.
2. Maru S, van der Schouw YT, Gimbrere CH, et al. Body mass index and short-term weight change in relation to mortality in Dutch women after age 50 y. Am J Clin Nutr. 2004;80:231–6.
3. Stevens J, Cai J, Everson KR, et al. Fitness and fatness as predictors of mortality from all causes and from cardiovascular disease in men and women in the lipid research clinics study. Am J Epidemiol. 2002;156:832–41.
4. Adams TD, Gress RE, Smith SC, et al. Long-term mortality after gastric bypass surgery. N Engl J Med. 2007;357:753–61.
5. Sjöström L, Lindroos A-K, Peltonen M, et al. Lifestyle, diabetes and cardiovascular risk factors 10 years after bariatric surgery. N Engl J Med. 2004;351:2683–93.
6. Buchwald H, Oien DM. Metabolic/bariatric surgery worldwide 2008. Obes Surg. 2009;19:1605–11.
7. Horchner R, Tuinebreijer W. Improved physical functioning of morbidly obese patients who have undergone a lap-band operation: one-year study. Obes Surg. 1999;9:399–402.
8. Karason K, Lindroos AK, Stenlöf K, et al. Relief of cardiorespiratory symptoms and increased physical activity after surgically induced weight loss. Arch Intern Med. 2000;160:1797–802.
9. de Souza SA, Faintuch J, Sant'anna AF. Effect of weight loss on aerobic capacity in patients with severe obesity before and after bariatric surgery. Obes Surg. 2010;20(7):871–5.
10. Serés L, Lopez-Ayerbe J, Coll R, et al. Increased exercise capacity after surgically induced weight loss in morbid obesity. Obesity. 2006;14(2):273–9.
11. Brissman M, Ekbom K, Hagman E, et al. Physical fitness and body composition two years after Roux-En-Y gastric bypass in adolescents. Obes Surg. 2017;27(2):330–7.
12. Browning MG, Franco RL, Herrick JE, et al. Assessment of cardiopulmonary responses to treadmill walking following gastric bypass surgery. Obes Surg. 2017;27(1):96–101.
13. Stegen S, Derave W, Calders P, et al. Physical fitness in morbidly obese patients: effect of gastric bypass surgery and exercise training. Obes Surg. 2011;21(1):61–70.
14. Wilms B, Ernst B, Thurnheer M, et al. Differential changes in exercise performance after massive weight loss induced by bariatric surgery. Obes Surg. 2013;23:365–71.
15. Zavorsky GS, Kim DJ, Christou NV. Compensatory exercise hyperventilation is restored in the morbidly obese after bariatric surgery. Obes Surg. 2008;18(5):549–59.
16. Coen PM, Tanner CJ, Helbling NL, et al. Clinical trial demonstrates exercise following bariatric surgery improves insulin sensitivity. J Clin Invest. 2015;125(1):248–57.
17. Kanoupakis E, Michaloudis D, Fraidakis F, et al. Left ventricular function and cardio-pulmonary performance following surgical treatment of morbid obesity. Obes Surg. 2001;11:552–8.
18. Lund MT, Hansen M, Wimmelmann CL, et al. Increased postoperative cardiopulmonary fitness in gastric bypass patients is explained by weight loss. Scand J Med Sci Sports. 2016;26:1428–34.
19. Neunhaeuserer D, Gasperetti A, Savalla F, et al. Functional evaluation in obese patients before and after sleeve gastrectomy. Obes Surg. 2017;27(12):3230–9.
20. Notarius CF, Rhode B, MacLean LD, et al. Exercise capacity and energy expenditure of morbidly obese and previously obese subjects. Clin Invest Med. 1998;21(2):79–87.
21. Ross R, Blair NB, Arena R, et al. Importance of assessing cardiorespiratory fitness in clinical practice: a case for fitness as a clinical vital sign. Circulation. 2016;134:653–99.

22. Riddle W, Younes M, Remmers JE, et al. Graphical analysis of patient performance in the pulmonary function laboratory. Proc Annu Symp Comput Appl Med Care. 1980;1:282–90.
23. Dereppe H, Verbeke M, Debruxelles C, et al. Energy expenditure of household activities and cardiorespiratory fitness in women with obesity. Clin Obes. 2018;8(6):391–7.
24. Lorenzo S, Babb TG. Quantification of cardiorespiratory fitness in healthy nonobese and obese men and women. Chest. 2012;141(4): 1031–9.
25. Reybrouck T, Mertens L, Vinckx J, et al. Assessment of cardiorespiratory exercise function in obese children and adolescents by body mass-independent parameters. Eur J Appl Physiol. 1997;75: 478–83.
26. Loftin M, Sothern M, Trosclair L, et al. Scaling VO2 peak in obese and non-obese girls. Obes Res. 2001;9:290–6.
27. Vanderburgh PA, Katch F. Ration scaling of VO2max penalizes women with larger percent body fat, not lean body mass. Med Sci Sports Exerc. 1996;28:1204–8.
28. Tanner JM. Fallacy of per-weight and per-surface area standards, and their relation to spurious correlation. J Appl Physiol. 1949;2(1): 1–15.
29. Öhrström M, Hedenbro J, Ekelund M. Energy expenditure during treadmill walking before and after banded gastroplasty: a one-year follow-up study in 11 obese women. Eur J Surg. 2001;167:845–50.
30. Hawkins S, Wiswell R. Rate and mechanism of maximal oxygen consumption decline with aging: implications for exercise training. Sports Med. 2003;33:877–88.
31. Refsum H, Holter P, Loving T, et al. Pulmonary function and energy expenditure after marked weight loss in obese women: observations before and one year after gastric banding. Int J Obes. 1990;14:175–83.
32. Paolillo S, Veglia F, Salvioni E, et al. Heart failure prognosis over time: how the prognostic role of oxygen consumption and ventilatory efficiency during exercise has changed in the last 20 years. Eur J Heart Fail. 2019;21(2):208–17.
33. Ferrari M, Manea L, Anton K, et al. Anemia and hemoglobin serum levels are associated with exercise capacity and quality of life in chronic obstructive pulmonary disease. BMC Pulm Med. 2015;15:58.
34. Philo SU, Garvican-Lewis L, Schmidt WF, et al. Relationship between changes in haemoglobin mass and maximal oxygen uptake after hypoxic exposure. Br J Sports Med. 2013;47:i26–30.
35. Scaglione R, Dichiara MA, Indovina R, et al. Left ventricular diastolic and systolic function in normotensive obese subjects: influence of degree and duration of obesity. Eur Heart J. 1992;13:138–42.
36. Carnero EA, Dubis GS, Hames KC, et al. Randomized trial reveals that physical activity and energy expenditure are associated with weight and body composition after RYGB. Obesity. 2017;25(7): 1206–16.
37. Hassannejad A, Khalaj A, Mansournia MA, et al. The effect of aerobic or aerobic-strength exercise on body composition and functional capacity in patients with BMI>35 after bariatric surgery: a randomized control trial. Obes Surg. 2017;27(11):2792–801.
38. Shah M, Snell PG, Rao S, et al. High-volume exercise program in obese bariatric surgery patients: a randomized, controlled trial. Obesity (Silver Spring). 2011;19(9):1826–34.
39. Herring LY, Stevinson C, Davies MJ, et al. Changes in physical activity behaviour and physical function after bariatric surgery: a systematic review and meta-analysis. Obes Rev. 2016;17(3):250–61.