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Original Research

Aerobic exercise capacity is normal in obesity with or without metabolic syndrome

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ABSTRACT

Background: Obesity might be a cause of limited aerobic exercise capacity. It is often associated with metabolic syndrome (MS) that includes cardiovascular comorbidities as arterial hypertension. Cardiopulmonary exercise testing (CPET) is the gold-standard to assess aerobic capacity and discriminate causes of dyspnea.
Aim: To evaluate aerobic capacity in obesity and if MS or hypertensive treatment impacts on the CPET profile.
Methods: CPET of 146 obese patients, whom 33 and 31 were matched for MS and antihypertensive medication, were analyzed. VO_{2peak} (mL/min/Kg) was reported in percentage of predicted value, or, divided by body weight, fat free mass (FFM) or body weight expected for a body mass index of 24 (BMI24).
Results: VO_{2peak} (20,8 ± 4,4 mL/min/Kg) was normal when expressed in percentage predicted for obesity (111 ± 22%pred) or divided by FFM and $weight_{BMI24}$ (33,6 ± 5,6 and 30,6 ± 6,2 respectively). The latter correlated better with maximal work rate ($r = 0,7168$, $p < 0,001$). Obese patients showed normal ventilatory efficiency (ventilation to carbon dioxide production slope: 28 ± 4), VO_2 to work rate (10,2 ± 1,6 mLO_2/Watt) and, slightly elevated heart rate to VO_2 slope (4,0 ± 1,1 bpm/mL/min/Kg). Compared to normotensives, hypertensive medicated patients had higher blood pressure at anaerobic threshold (142 ± 23 vs 158 ± 26 mmHg, $p = 0,001$) but not at maximal exercise (189 ± 31 vs 201 ± 23 mmHg, p = NS), and, had lower actual maximal heart rate (155 ± 23 vs 143 ± 25 bpm, $p = 0,03$). There was no difference between obese patients with or without MS.
Conclusion: Obese people with or without MS present with similar and normal aerobic profile related to the excessive body weight. VO_{2peak} divided by $weight_{BMI24}$ is an easy and clinical meaningful index for obese patients.

1. Introduction

Obesity is a chronic condition of excessive body fat accumulation leading to too high body weight. Worldwide prevalence of obesity is increasing and it is now recognised as a global epidemic [1]. Obese patients are prone to develop metabolic dysfunction with an increased risk of hypertension, diabetes, high serum triglyceride and low high-density cholesterol (HDL). Those dysfunctions, together with high waist circumference, define the metabolic syndrome that increases the risk of cardiovascular events, heart failure and mortality [2–4]. Therefore, obese patients are often highly medicated, even with beta-adrenergic receptors antagonists that aim to reduce cardiovascular activation [5].

Dyspnea is an usual symptom in obese patients. Its mechanisms are not completely known, however excessive weight, movement impairment, deconditioning, impaired cardiovascular function [6–9] and low oxidative muscle capacity [10,11] are suggested among more common causes. Therefore, the evaluation of aerobic capacity of obese patients is mandatory to define the cardio-muscular-respiratory integrity.

The cardiopulmonary exercise testing (CPET) is the gold-standard to assess functional disorders [12]. Moreover, the assumption of aerobic capacity relies on measurement of oxygen consumption (VO_2) and its expected value. Studies have shown normal absolute maximal oxygen consumption (in mL/min) in obesity but lower to normal when

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expressed relative to body mass or fat free mass (FFM) [13,14] This assessment may however be confusing as there is no clear predicted value of maximal oxygen consumption (VO_{2peak}) for obese patients, and, dividing VO_2 by body weight is obviously biased by excessive fat mass. This is of particular matter when comparing exercise capacity before and after bariatric surgery [15].

In the present study, we aimed to evaluate which assumption of aerobic capacity in obesity would be more relevant. We therefore considered different ways of reporting VO_{2peak} (1. in absolute value (mL/min), 2. divide by body weight (mL/min.Kg), 3. divided by fat free mass (mL/min.Kg_{FFM}) 4. Divided by the weight expected for BMI of 24 (mL/Min.Kg_{BMI24}) and 5. in percent of predicted value (%pred)) and their relation to workload. Furthermore, we wondered if metabolic syndrome and antihypertensive treatment may affect the aerobic capacity.

2. Methods

2.1. Protocol - Population

We retrospectively included obese patients (defined by a BMI higher than 30 kg m^{-2}) taking part to a multidisciplinary weight loss program. The program consisted of nutritional and psychological follow up and every patient was invited to a personalized trianing program after a careful medical assessment for contraindications. About 60–70% of the patients enrolled themselves to the exercise program. CPET were performed after 2–4 exercise training sessions for optimization of training prescription. Patients unable to cycle or weighting more than 150 Kg (limit afforded by the bicycle) did not perform CPET. Fourthy one male and 105 women were recruited. Height, weight, BMI, waist circumference, FFM, blood pressure at rest and fasting blood analysis were obtained at the beginning of the weight loss program (Table 1). The study protocol was approved by the institutional review board at University Hospital Erasme of ULB. Considering the risk/benefit ratio of the study, informed consent was waived.

2.2. Cardiopulmonary exercise testing

A standard cycle ergometer incremental cardiopulmonary exercise test was applied until the symptom-limited maximum [16].

After a warm-up pedalling at 20 W during 3 min, a load incremental protocol of 10–25 W/min was applied aimed to obtain an exercise duration of 8–12 min. Ventilation and gas analysis were performed using a Vmax (Sensormedics, Yorba Linda, CA). Gas analysers and pneumotachograph were calibrated prior to each test. Heart rate and blood pressure were obtained via automatic standard ECG and sphygmomanometer.

VO_{2peak} was defined as the highest VO_2 measured during a period of 20 s at the end of the CPET. The ventilatory equivalents for carbon dioxide (CO_2) was determined by dividing VE (L/min) by VCO_2 (L/min) and the oxygen pulse (O_2pulse) was obtained by dividing VO_2 (ml/min) by HR (bpm). Anaerobic threshold was determined using the V-slope method and the corresponding accordance with the evolution of VE/VO_2 [16]. The slope between ventilation and CO_2 production (VE/VCO_2 slope) was determined as well as VO_2/WR and HR/VO_2 slopes on the linear part of the relationship.

2.3. Fat Free Mass

Before performing the CPET, all subjects underwent electrical bioimpedance assessment (Seca, Hamburg, Germany), in order to measure the total weight and the fat free mass (FFM).

2.4. Statistics and analysis

Results are expressed as mean ± SE. Paired t-tests were used to compare the clinical and exercise variables between paired subjects. Pearson coefficient correlations were calculated by linear regression analysis.

Aerobic exercise capacity was expressed as VO_{2peak} in absolute value (VO_{2peak}, mL/min), or, divided by actual weight ($VO_{2peakKg}$, mL/min•Kg), fat free mass ($VO_{2peakFFM}$, mL/min.Kg_{FFM}) and ideal body weight calculate on a basis of a BMI of 24 ($VO_{2peakBMI24,}$ mL/min.Kg_{BMI24}). Predicted VO_{2peak} was calculated by Karlman Wassermann published formulas for obese people [16].

Patients with and without metabolic syndrome (MS) [2,4] or with and without antihypertensive treatment (AHT) were matched according to the following criteria: sex (mandatory), age (± 3 years) and height (± 10 cm) and weight (± 25 Kg). MS was defined by the presence of a waist circumference of more than 80 and 94 cm for women and men respectively and the presence of more than one criterion as an HDL >50 or 40 mg/dL for women and men respectively, systolic and/or diastolic blood pressure (sBP/dBP) above 130/85 mmHg and/or the taking of an AHT, and a fasting glycemia >100 mg/dL. AHT was categorized according to the presence of at least one of the following drugs: beta-adrenergic receptors blockers, angiotensin conversing enzyme inhibitors, angiotensin-receptor blockers, calcium channel antagonists, diuretics and/or mineral-receptor corticoid antagonists.

Table 1
Descriptive characteristics of the participants (n = 146).

VARIABLE	Obese	Normal value
Gender, m (%)	41–(28%)	/
Age, y	49 ± 12	/
Height, cm	167 ± 10	/
Weight, Kg	104 ± 20	/
BMI, kg/m^2	37 ± 6	18–24
Waist circumference (m/f), cm	130 ± 20/114 ± 26	<94/<80
Fat free mass (male/female), Kg	75 ± 7/54 ± 8	64 ± 6/48 ± 4
Fat free mass (male/female), %	67 ± 3/55 ± 4	80-85/70-75
sBP, mmHg	129 ± 17	<140
dBP, mmHg	82 ± 12	<90
mBP, mmHg	96 ± 17	<105
Glycemia, mg/L	104 ± 26	70–100
HbA1c, %	5,9 ± 0,9	4–6
Total cholesterol, mg/L	190 ± 36	<190
HDL, mg/L	51 ± 16	>40
TG, mg/L	130 ± 63	<150

BMI: body mass index, sBP: systolic blood pressure, sBP: diastolic blood pressure, HbA1c: *glycated haemoglobin, HDL: high density lipoproteins, TG: triglycerid*

3. Results

Hundred fourthy six subjects were included in the analysis. High prevalence of female (72% of total) with mean mid-age was found. Averaged normal body blood pressure at rest was measured. Glycemic and lipidic profile showed mean values at upper limit of normal. General characteristics of overall population are given in Table 1.

Obese patients presented with normal response to exercise (Table 2) reaching 111 ± 22% of obese predicted value of VO_{2peak} and normal ventilatory response (VE/VCO_2@AT = 29 ± 3 and VEVCO_2 slope = 28 ± 4) but slightly elevated HR/VO_2 slope (4,0 ± 1,1 bpm/mL/min/Kg).

As shown in Table 3, $VO_{2peakBMI24}$ was 10% lower than $VO_{2peakFFM}$. VO_{2peak} (in mL/min) correlated better with FFM (n = 62, r = 0,7128, p < 0,001) or weight at BMI24 (r = 0.6153, p < 0,001), than actual body weight (r = 0,5427, p < 0,001) (Fig. 1, abc).

Maximal work rate was respectively better correlated with VO_{2peak} in mL/min (r = 0,8750, p < 0,001), than with $VO_{2peakBMI24}$ (r = 0,7168, p < 0,001), than with $VO_{2peakKg}$ (r = 0,6264, p < 0,001) or $VO_{2peakFFM}$ (n = 62, r = 0,4451, p < 0,001). Fig. 2, abcd)

Heighty-height patients among the 146 (60%) were treated, with at least one medication; for hypertension (n 66/146), or abnormal lipidic

Table 2
Exercise characteristics of the 146 obese subjects.

n = 146	
Anaerobic threshold	
Work, Watt	80 ± 36
VO_2, mL/min•kg	13,4 ± 3,4
Pet CO_2, mmHg	39 ± 4
VE/VCO_2	29 ± 3
HR, bpm	119 ± 20
sBP, mmHg	153 ± 30
dBP, mmHg	85 ± 12
O2 pulse, mL/beat	11,3 ± 3,3
VO_{2PEAK}	
Work, Watt	136 ± 44
VO_2, mL/min	2067 ± 548
VO_2, mL/min•kg	20,8 ± 4,4
VO_2 pred, %	111 ± 22
VE, L/min	60,8 ± 17,5
RQ	1,14 ± 0,08
HR, bpm	151 ± 25
HR pred, %	88 ± 12
sBP, mmHg	199 ± 29
dBP, mmHg	92 ± 16
O2 pulse, mL/beat	13,9 ± 3,8
SLOPES	
VE/VCO_2sl	28 ± 4
VO_2/WRsl, mL/Watt	10,2 ± 1,6
HR/VO_2sl, bpm/mL/min/Kg	4,0 ± 1,1

VO_2: oxygen consumption, VO_2pred: percentage predicted in peak oxygen consumtion, VE: ventilation, RQ: respiratory quotient, PetCO_2: CO_2 partial pressure at end tidal, VE/VCO_2: ventilatory equivalent for carbone dioxyde, HR: Heart rate, HRpred: percentage of maximal predicted heart rate, sBP: systolic blood pressure, dBP diastolic blood pressure, O_2pulse: oxygen pulse, VEVCO_2sl: slope of ventilation slope to dioxyde production insncrease, VO_2/WRsl: oxygen uptake slope to work rate increase, HR/VO_2sl: Heart rate slope to oxygen consumption increase.

Table 3
Five different ways of reporting aerobic capacity for obese patients.

VO_{2PEAK}	
VO_2, mL/min	2067 ± 548
VO_2, mL/min.kg	20,8 ± 4,4
VO_2 pred, %	111 ± 22
VO_2, ml/min.Kg_{FFM} [a]	33,6 ± 5,6
VO_2, ml/min.Kg_{BMI24}	30,6 ± 6,2

VO_2: oxygen consumption, VO_2pred: peak percentage predicted oxygen consumtion, FFM: fat free mas, BMI24: weight for a BMI of 24.
[a] n = 62.

profile (n = 37/146), or high glycaemic level (n 37/146). Those 88 patients were older and heavier (respectively, 53 ± 11 vs 43 ± 11 yo, p < 0,001; 38 ± 6 vs 36 ± 4 kg/m^2, p < 0,02) and showed more impaired clinical assessment with greater sBP (132 ± 17 vs 125 ± 16 mmHg (p = 0,03), altered fasting glycemic level (111 ± 30 vs 93 ± 12 mg/L, p < 0,001) and HbA1c (6,2 ± 1,0 vs 5,6 ± 0,3%,p < 0,001), higher serum TG level (145 ± 65 vs 107 ± 52 mg/L, p < 0,001) and lower HDL (48 ± 13 vs 55 ± 20 mg/L,p < 0,01).

We paired 33 patients with and without MS and 31 with or without AHT (Fig. 3). Optimal sample size calculation estimated from data from another study of us [15] and from the presents results indicated that groups of that magnitude would be large enough to detect a 15–20% difference in aerobic exercise capacity with a type I error of 0,05 and type II error of 0,9.

The 33 patients with MS, matched with patients without MS, showed higher resting sBP (131 ± 18 vs 123 ± 15 mmHg, p = 0,04), glycaemic level (110 ± 25 vs 90 ± 7 g/dL, p < 0,001), HbA1c (6,0 ± 0,8 vs 5,5 ± 0,3%, p < 0,01 and higher serum TG level (157 ± 84 vs 104 ± 30 mg/L, p < 0,01). No difference was shown during CPET.

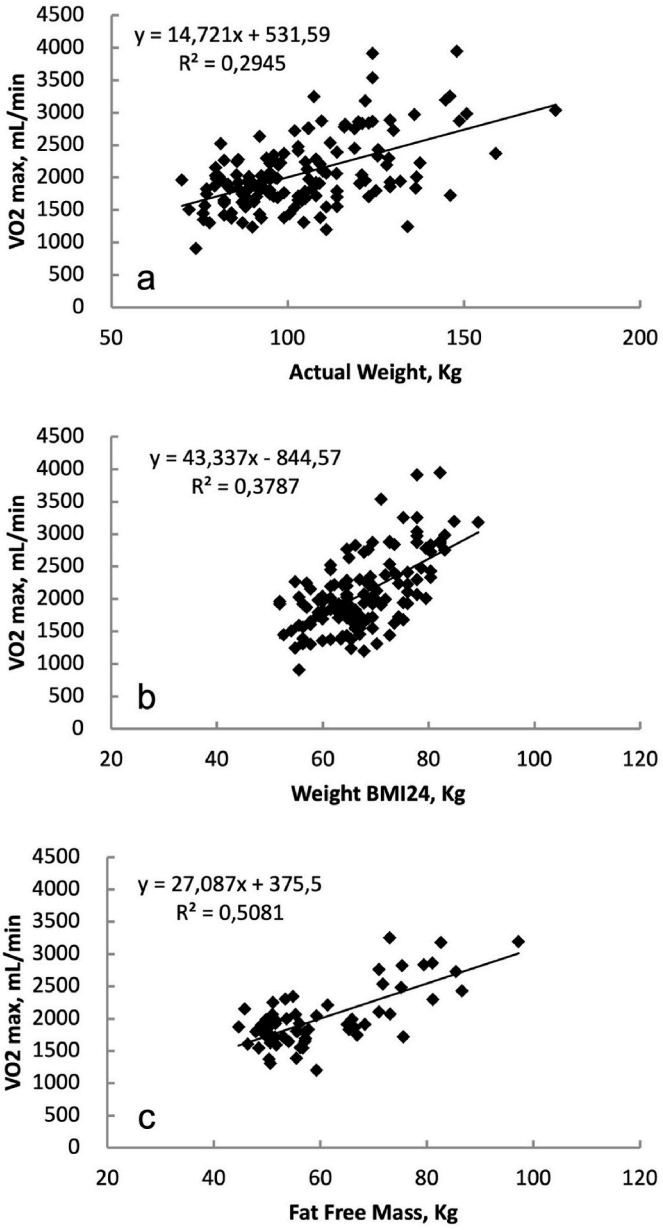

Fig. 1. Relation between oxygen consumption (mL/min) and body weight, body weight expected for a BMI of 24 and Fat Free Mass. (graph 1c: n = 62).

The 31 subjects with AHT, matched with patients without AHT, did not show any difference for BP and metabolic profile at rest. At CPET, AHT patients showed greater sBP at AT (158 ± 20 vs 142 ± 23 mmHg, p = 0,001), and lower absolute and predicted HR at peak exercise (143 ± 25 vs 155 ± 23 bpm, p = 0,03; 86 ± 13 vs 92 ± 12%, p = 0,05). No other difference was shown.

4. Discussion

Our results showed normal or slightly higher than expected exercise capacity in obese people engaging in a weight loss program. Normalizing exercise capacity by considering the expected bodyweight for a BMI of 24 was shown to be promising and clinically meaningful.

Obese people have been suggested to perform submaximal exercise testing [17–19] due to earlier fatigue sensation and truncated perception of exercise induced discomfort [20]. However, our patients were able to perform high intensity exercise with maximal RER (1,14 ± 0,08) and HR (88 ± 12%predicted) what suggests that our patients were

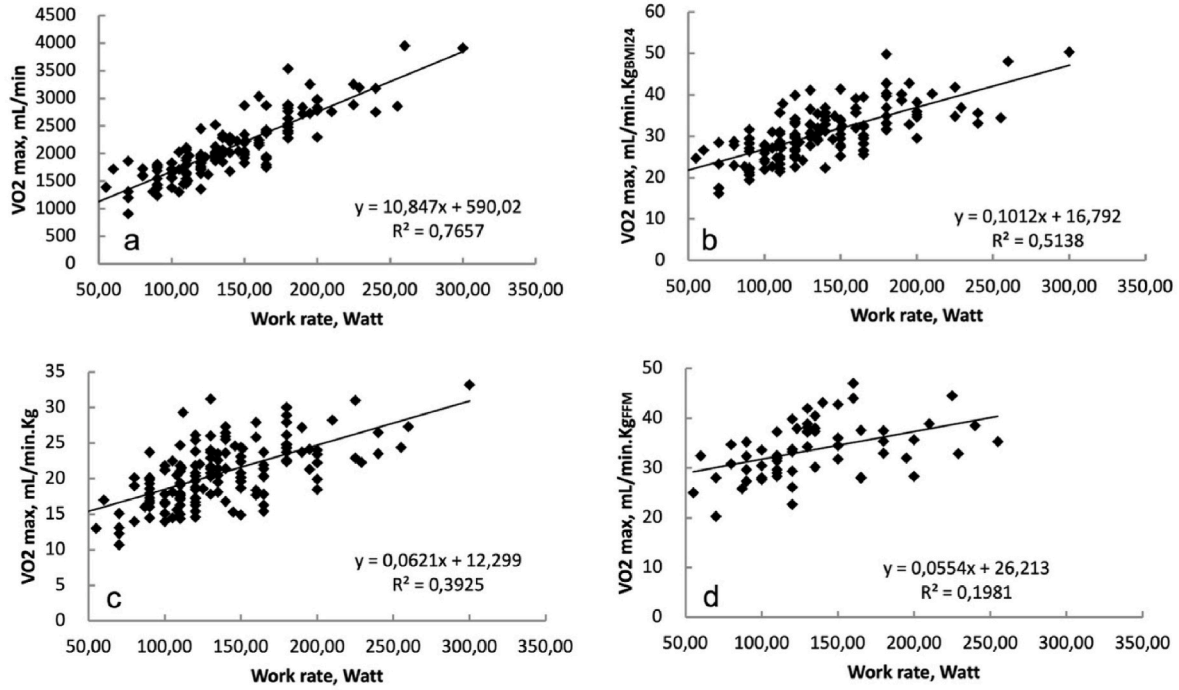

Fig. 2. Relations between aerobic capacity and work rate (graph 2d: n = 62).

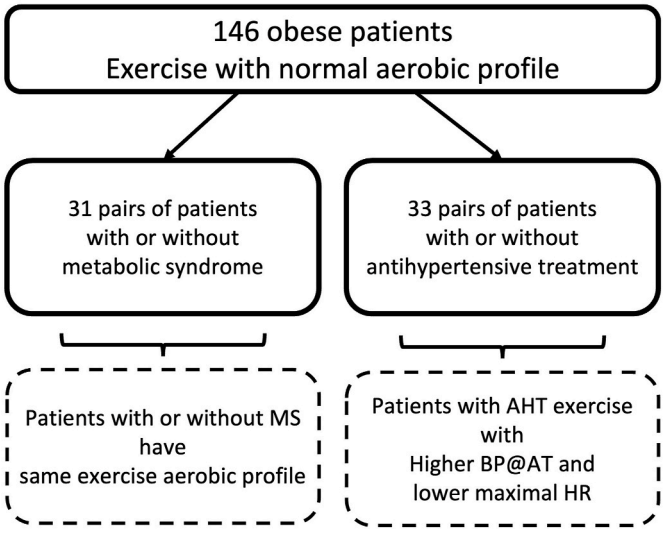

Fig. 3. Pairing patients resulted in 31 pairs of patients with or without metabolic syndrome (MS) and 33 pairs of patients with or without anithypertensive treatment.

highly motivated to perform good exercise testing. FEV1 was not measured in our population, but our patients reported no respiratory discomfort during medical examination prior to exercise rehabilitation. It seems therefore unlikely that ventilatory limitation have occurred in those patients like has been reported earlier [21].

Meanwhile, our patients reached a mean VO_{2peak} of 20,8 mL/min.Kg what is demonstrating poor aerobic capacity, and, a cardiovascular/muscular deconditioning should be considered [16]. Our data tend to show that this is not the case as, when dividing VO_{2peak} by the expected weight at a BMI of 24 (BMI24), mean aerobic capacity was about 30,6 mL/min.Kg. Moreover, exercise capacity was estimated at 111% of obese predicted values [16] with an anaerobic threshold occurring at 65% of VO_{2peak} and normal CPET cardiovascular adaptation. This profile is in line with other reports showing no good aerobic exercise capacity but still no evidence of deconditioning in obese patient [22].

We could however consider that the VO_2 standard reference formulas from Wassermann are not adapted anymore, or, that cultural, social and/or demographic differences explain this underestimation. Determining expected exercise capacity of a specific population is a challenging task, and particularly in obesity. However, it might be considered that wearing excessive body mass in every activity of their life is a kind of exercise training, especially in active people volunteering for exercise weight loss program.

Using different expression of aerobic capacity for obese people might be sought to add to the understanding of functional limitation. Indeed, even if absolute VO_{2peak} is well correlated to maximal workload, it does not appropriately transcribe the functional handicap of wearing excessive bodyweight. Dividing VO_2 by the weight is therefore a good appreciation of functional capacity in normal weighted subjects. It is the case in cardiac or pulmonary disease as well as in normal population where VO_{2peak} mL/min.Kg correlates to workload and refers to prognosis [23–25]. In people with abnormal fat mass proportion, this ratio stays relevant as to refer to handicap even if, in obesity, VO_2 in mL/min.Kg is largely lowered by excessive weight.

Of consequence, oxygen consumption in mL/min was better correlated to FFM or $weight_{BMI24}$ than actual weight (Fig. 1) what must underline the leading role of the muscle tissue in consuming oxygen during exercise. Accordingly, VO_2 in mL/min was highly correlated to work rate. However, $VO_{2peakBMI24}$ was interestingly better correlated to work rate than $VO_{2peakKg}$ or $VO_{2peakFFM}$ (Fig. 2). Indeed, because oxygen is used by fat free mass, we would have expected a better correlation with $VO_{2peakFFM}$. It is however of clinical interest that BMI_{24} is an easier index to use than FFM and that it will not be influenced by the modification of body composition that occurs with diet. Therefore, $VO_{2peakBMI24}$ could be a better clinical meaningful index for the evaluation of aerobic capacity of obese people whereas it is unknown if it is related to quality of life or survival.

Because of the linear relation between cardiac output and oxygen consumption during exercise, evaluating cardiodynamic adaptation with CPET is possible by the analysis of VO_2/WR slope, given the muscular oxygen utilization is normal [16]. Normal oxygen use efficacy

has been shown in obese people [7] and our patients showed normal VO_2/WRslope (Table 2). It is therefore reasonable to assume that, in obese people, cardiac output increase during exercise is normal. However, unloaded cycling during warm up for obese people, generates a high oxygen demand because of moving heavy legs. This phenomenon leads the patient to higher VO_2 at maximum exercise than what could be expected for normal weighted people at same maximal WR. This was adequately taken into account by Wasserman predicted formulas for obese people [16].

Adaptation of stroke volume during exercise is usually appreciated by HR/VO_2 slope that relates to the needed increase in heart rate to adapt the cardiac output. This is thereby expected to be low in well trained athletes and high in deconditioning or in pulmonary hypertension and heart failure patients [16]. However, because the normal HR/VO_2 slope is generally calculated in beats/mL/min.Kg, this consideration might be biased in overweight people. Indeed, it might be calculated that, this slope will be increased by 1,5 points just by modifying the BMI of a subject (from 24 to 35). However, in our population the HR/VO_2 slope is only slightly above the limit of normal (i.e. 3-4 beats/mL/min•Kg-1) what contrasts with previous results of Dereppe et al. [15] showing that HR/VO_2 slope equal to 5 beats/ml•Kg before bariatric-surgery. To note, the higher BMI in this population in comparison to ours (44 ± 4 vs 37 ± 6 kg/m^2). We can also speculate that patients enrolling in a weight loss program might be naturally more active and therefore fitter than heavier patients consulting for a surgery. Moreover, end exercise oxygen pulse was normal in patients with or without any treatment, suggesting no alteration of stroke volume in our patients as either suggested by Dereppe et al. However, several patients had antihypertensive medication, what could also be part of the explanation of a low HR to VO_2 slope because of hemodynamic depressive effect [5]. Among our 146 obese patients, indeed, the 58 patients that were without any treatment were younger, lighter and fitter (22,7 ± 4,3 vs 19,15 ± 3,9 mL/min/Kg, p:<0,001) but still presented with higher slope than treated ones. (4,4 ± 1,1 vs 3,8 ± 1,0 beat/mL/min/Kg, p = 0, 0003). However, HR/VO_2slope was not different between our matched obese patients for anti-HT treatment, even though only slightly elevated in treated group (i.e., 4,2 ± 1,1 beat/mL/min.Kg).

Our population was not characterized for the presence of heart failure (HF), despite it is conceivable that those with multiple cardiovascular risk factor may have developed a form of HF with preserved ejection fraction. The ratio between minute ventilation and carbon dioxide production (VE/VCO_2) is a measure of cardiorespiratory efficiency and leads prognosis in cardiovascular disorders [23,26–28]. VE/VCO_2 slope is independent of subject effort and BMI [29] and has been explored in different studies on lean and obese patients [15,29–32]. Accordingly, we reported normal VE/VCO_2 slope. However, values of VE/VCO_2 slope and optimal threshold predicting survival have been shown to be lower in overweight and obese HF patients compared to normal weighted [26]. The MECKI study showed moreover an inverse correlation between BMI and VE/VCO_2slope in HF patients with obesity [33]. Some authors declared a relative hypoventilation due to mechanical constraints and low pulmonary compliance caused by abdominal fat mass [30,31]. Those accordingly showed an increased VE/VCO_2 slope after bariatric surgery [31,34], while, others did not [15].

The BMI paradox, known as the protective effect of BMI on survival has been shown to be attenuated by a good aerobic capacity in obese HF patients [33]. It could therefore be of interest, also in HF patients, to consider dividing VO_{2peak} by weight at BMI24. This could be a way to overcome the BMI paradox by decreasing the bias caused by excessive weight when assessing aerobic capacity in HF.

It is known that comorbidities are progressively causing systemic structural modification associated with complications leading to symptoms, cardiovascular risks and a loss of function [2,35,36]. This loss of function may impair quality of life, shorten lifespan and lead to psycho-social problems through influencing physical and exercise capacity that is associated with prognosis. Despite metabolic syndrome associates comorbidities like dyslipidemia, diabetes and hypertension to obesity [2,3], in our population it was not related with impaired aerobic capacity nor cardiovascular efficiency [37]. Indeed, in our groups of patients, metabolic syndrome was not associated with worse exercise capacity, and it was not possible to highlight a specific CPET profile for those patients.

In conclusion, we showed that obese people with or without comorbidities present with a normal aerobic exercise profile. Abnormal exercise profile should be considered as pathological and not related to obesity. Exercise related dyspnoea should therefore improve with a progressive and regular loss of weight [15]. Moreover VO_{2peak} divided by predicted weight for a BMI of 24 might be more clinically meaningful when classifying aerobic exercise capacity of obese patients. We suggest this index might be useful for the clinical follow-up of obese patients and that further research to be done with it.

CRediT authorship contribution statement

G. Deboeck: Conceptualization, Formal analysis, Writing (original and review), Supervision, Writing – original draft, Writing – review & editing. **M. Vicenzi:** Formal analysis, Writing (original and review), Writing – original draft, Writing – review & editing. **V. Faoro:** Conceptualization, Writing (original). **M. Lamotte:** Writing (original and review), Supervision.

Declaration of competing interest

The authors have no conflicts of interests to declare.

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The authors report that this study was not funded and that no conflict of interest exists. The results of the study are presented clearly, honestly, and without fabrication, falsification, or inappropriate data manipulation. All data might be available by contacting first author. Adrien Bocquet helped in the preparation of the study.

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