# EPIDEMIOLOGY AND PREVENTION

# Cardiopulmonary Function and Exercise Capacity in Patients With Morbid Obesity

Luis Serés,<sup>a</sup> Jordi López-Ayerbe,<sup>a</sup> Ramón Coll,<sup>b</sup> Oriol Rodríguez,<sup>a</sup> José M. Manresa,<sup>e</sup> Jaume Marrugat,<sup>e</sup> Antonio Alastrue,<sup>c</sup> Xavier Formiguera<sup>d</sup> and Vicente Valle<sup>a</sup>

<sup>a</sup>Servicios de Cardiología, <sup>b</sup>Rehabilitación y <sup>c</sup>Cirugía General. <sup>d</sup>Unidad de Trastornos de la Alimentación, Hospital Universitario Germans Trias I Pujol, Badalona, Barcelona. Spain. <sup>e</sup>Instituto Municipal de Investigación Médica, Barcelona, Spain.

**Introduction and objectives.** The effect of obesity on cardiac function is still under discussion. The objective of this study was to assess cardiopulmonary capacity in morbidly obese patients.

**Patients and method.** A symptom-limited cardiopulmonary exercise stress test was carried out in 31 morbidly obese patients (BMI 50  $\pm$  9 kg/m<sup>2</sup>) and 30 normal controls (BMI 24  $\pm$  2 kg/m<sup>2</sup>). Cardiovascular function was evaluated using the oxygen pulse (oxygen uptake/heart rate).

**Results.** There were no differences in age, sex and height between both groups. During the effort the obese subjects presented greater oxygen uptake, heart rate, systolic arterial pressure and minute ventilation and shorter test duration than control group ( $14 \pm 3 \text{ vs } 27 \pm 4 \text{ min}$ ; p < 0.001). Oxygen pulse values were higher in obese patients. However, after oxygen uptake indexation by fat free mass, these differences disappeared, suggesting a similar cardiovascular function. At the end of the exercise, the control group reached 96% of their age-predicted maximal heart rate and their respiratory exchange ratio was  $1 \pm 0.2$ . Obese patients only reached 86% and  $0.87 \pm 0.2$ , respectively.

**Conclusions.** Due to their need of more energy output to move total body mass morbidly obese patients have a reduced exercise capacity. They finish the test having done a submaximal exercise. However, during this effort they show a normal cardiopulmonar capacity.

Key words: Obesity. Exercise. Cardiopulmonary capacity.

Full English text available at: www.revespcardiol.org

Este estudio forma parte de un proyecto de investigación aprobado por el FIS (expediente 99/1021), con el título: Alteraciones de la anatomía y función cardíaca en pacientes con obesidad mórbida. Modificaciones tras la pérdida ponderal secundaria a cirugía bariátrica.

Correspondence: Dr. L. Serés García.

Servici<sup>o</sup> de Cardiología. Hospital Universitario Germans Trias i Pujol. Carretera de Canyet, s/n. 08916 Badalona. Barcelona. España. E-mail: seres@hugtip.scs.es

Received 19 June 2002. Accepted for publication 29 January 2003.

# Función cardiopulmonar y capacidad de ejercicio en pacientescon obesidad mórbida

**Introducción y objetivos.** La repercusión de la obesidad sobre la función cardíaca es motivo de controversia. El propósito del presente estudio ha sido determinar la capacidad cardiopulmonar en pacientes con obesidad mórbida.

**Pacientes y método.** Hemos realizado una ergoespirometría limitada por síntomas a 31 pacientes con obesidad mórbida (IMC 50  $\pm$  9 kg/m<sup>2</sup>) y a 30 individuos como grupo control (IMC 24  $\pm$  2 kg/m<sup>2</sup>). La función cardiovascular ha sido valorada mediante el pulso de oxígeno (consumo de oxígeno/frecuencia cardíaca).

Resultados. No existían diferencias en edad, sexo y talla entre ambos grupos. Durante el esfuerzo, los sujetos obesos presentaron un consumo de oxígeno, frecuencia cardíaca, presión arterial sistólica y ventilación por minuto significativamente más elevados que el grupo control, con menor duración de la prueba (14 ± 3 frente a 27 ± 4 min; p < 0,001). Los valores de pulso de oxígeno fueron más altos en los pacientes obesos. Sin embargo, tras corregir el consumo de oxígeno por la masa magra, las diferencias en el pulso de O2 desaparecieron, demostrando una función cardiovascular similar. Al final del ejercicio, el grupo control alcanzó el 96% de su frecuencia cardíaca máxima teórica y su cociente respiratorio fue de 1 ± 0,2. Los pacientes obesos sólo alcanzaron el 86% de la frecuencia cardíaca máxima teórica y su cociente respiratorio fue de 0,87 ± 0,2.

**Conclusiones.** Los pacientes con obesidad mórbida tienen una capacidad de trabajo reducida debido al gran consumo energético que realizan al mover su masa corporal. Finalizan la prueba habiendo realizado un esfuerzo submáximo. No obstante, durante este esfuerzo demuestran una capacidad cardiopulmonar normal.

**Palabras clave:** Obesidad. Ejercicio. Capacidad cardiopulmonar.

# INTRODUCTION

Obesity is a metabolic problem. Its prevalence continues to increase in the developed world, and is reaching almost epidemic proportions.<sup>1,2</sup> Chronic obesity is associated with an increased left ventricular mass<sup>3</sup>

# ABBREVIATIONS

BMI: body mass index.
VO<sub>2</sub>: oxygen consumption.
VCO<sub>2</sub>: production of carbon dioxide.
VE: ventilation per minute.
MTCF: maximum theoretical cardiac frequency.
ME: metabolic equivalent.
RQ: respiratory quotient.
MO: morbid obesity.

and with high cardiovascular morbidity and mortality.<sup>4,5</sup> Its effects on cardiac function, however, are still controversial. While some authors describe alterations of systolic<sup>6,7</sup> or diastolic<sup>8,9</sup> function, others indicate cardiac function to be normal.<sup>10,11</sup>

The cardiopulmonary exercise test offers objective measurements of functional capacity and cardiac reserve. Several studies have evaluated the exercise capacity of obese patients, but results have been contradictory. Some authors believe obese people to have a cardiopulmonary response within normal limits, but that their exercise capacity is compromised by the large body mass they have to carry.<sup>12,13</sup> Others indicate that they have reduced aerobic capacity compared to people of normal weight, their fat mass interfering with cardiac and pulmonary function and limiting their aerobic response to excessive.<sup>14-18</sup> Some of the discrepancies in the results of these studies might be due to the different methodologies employed, and because they examined different populations with different ages and degrees of obesity.

Using treadmill exercise test and gas analysis, the present cross-sectional study prospectively analyzed cardiopulmonary functional capacity in a group of patients with morbid obesity (MO), and in a group of healthy, volunteer controls of normal body weight.

# PATIENTS AND METHODS

# Study group

The study subjects were 55 patients of both sexes, all of whom suffered OM. All were receiving treatment at the Nutritional Disorder Unit of our hospital and were included in a bariatric surgery program. MO was defined as a having a body mass index (BMI) equal to or greater than 40 kg/m<sup>2</sup>. All patients had suffered obesity for more than 15 years. In nine patients, exercise tests could not be performed because of the physical difficulty experienced in walking. The BMI of these latter patients was significantly greater than those who were able to undergo exercise tests (BMI 57.7  $\pm$  10 compared to 50  $\pm$  10 kg/m<sup>2</sup>; *P*<.001). Of those who did undergo this testing, 15 were excluded because of high blood pressure (their results were, however, reserved for future studies). This was to try to control for the potential negative influence that this variable might have on cardiac function. Thirty one MO patients with normal blood pressure therefore made up the final sample (56% of the initial population).

The control group was made up of 30 healthy, normotensive volunteers of normal body weight (BMI<27 kg/m<sup>2</sup>). These were recruited from the patients' families and from among healthcare personnel via advertisement of the study. Pairing with the OM group was performed on the basis of age ( $\pm$  5 years) and height ( $\pm$  5 cm). Before starting, all participants underwent physical examination and a normal 12 lead electrocardiogram. None of the participants practiced sport regularly nor did they take any type of medication that might interfere with the exercise test results.

The study protocol was assessed and accepted by the Clinical Trials and Research Committee of our hospital. All participants received detailed information about the aim of the study and the methods to be used. They all provided written consent to participate.

# Cardiopulmonary exercise test

A symptom-limited cardiopulmonary exercise test (Enraf Nonius Holland ergometer) with analysis of respiratory gases was performed at least 3 h after breakfast. After trying several different tests with a patient weighing 244 kg, an appropriate experimental protocol (a modification of Balkes protocol<sup>19</sup>) was designed. The belt speed and the gradient settings were: stage 1-2.5 km/h, 0%; stage 2-2.5 km/h, 2%; stage 3-2.5 km/h, 4%; stage 4-2.5 km/h, 6%; stage 5-2.5 km/h, 8%; stage 6-3 km/h, 10%; stage 7-3 km/h, 12%; stage 8-3 km/h, 14%; stage 9-3 km/h, 16%; stage 10-3 km/h, 18%; stage 11-3.5 km/h, 20%; stage 12-3.5 km/h, 22%; stage 13-3.5 km/h, 24%; stage 14-3.5 km/h, 25%. From this point on, both belt speed and gradient were held constant. Each stage lasted 2 min. The patients were asked to keep going until they could continue no longer.

Cardiac frequency (CF) was monitored by continuous electrocardiographic recording. Blood pressure was monitored at the beginning of the test and every 2 mi-nutes thereafter, during both exercise and recovery, using a sphygmomanometer attached to the arm. An ergospirometer (Mintjarth 4, Holland) with a Hans Rudolf one-way mask was used for the analysis of gases expired during rest and exercise. Tidal volume (TV, in mL), breathing frequency (BF, in breaths per min), ventilation per minute(VE, in L/min), oxygen consumption  $O_2$  (VO<sub>2</sub>, in mL/min), carbon dioxide production (VCO<sub>2</sub>, in mL/min) and respiratory quotient (RQ = VCO<sub>2</sub>/VO<sub>2</sub>) were measured every 30 s. Before each session, the system was calibrated using standard gases with known O<sub>2</sub> and CO<sub>2</sub> concentrations. The metabolic equivalent (ME) is a unit of oxygen consumption at rest with the subject sitting<sup>20</sup> (3.5 mL of O<sub>2</sub> per kg of body weight per min [mL/kg/min]).

The efficiency of the cardiovascular system during exercise was evaluated by the  $O_2$  pulse (the amount of  $O_2$  consumed during a complete cardiac cycle; calculated by dividing  $O_2$  consumption by the cardiac frequency[VO<sub>2</sub>/CF]). If an individual's VO<sub>2</sub> is expressed according to the principle of Fick:<sup>21</sup>

 $VO_2$ =cardiac usage × arterio-venous difference in  $O_2$ 

If cardiac usage is equal to the stroke volume multiplied by the CF, then  $O_2$  pulse equals the stroke volume multiplied by the arterio-venous difference in  $O_2$ . Given that during exercise this difference has a physiological limit<sup>20</sup> of 15-17 vol/%, if a large physical effort is made then the  $O_2$  pulse allows the behavior of the stroke volume to be evaluated.

The maximum theoretical cardiac frequency (MTCF) is calculated using the algorithm

MTCF and RQ were used to determine the effort made.  $^{\rm 22}$ 

#### Body densitometry

Total body mass, fat mass and lean mass were measured by dual densitometry with an x-ray source using a Lunar Prodigy densitometer (Lunar Corp., Madison, WI, USA). Precision controls were performed daily using an external calibrator. The margin of error for total body mass was 1%.

#### Statistical analysis

Categorical variables were expressed as percentages. Quantitative values were expressed as mean  $\pm$  SD. The Student *t* test was used to compare means, and Persons  $\chi^2$  test to assess gender proportions. The influence of obesity was studied by repeated measures analysis of variance (RM ANOVA). To compare the O<sub>2</sub> pulse between the groups, the 25th, 50th and 75th percentiles were calculated for each subject, as well as baseline and maximum values. RM ANOVA was used to test the hypothesis that VO<sub>2</sub>, VE, CF and systolic blood pressure (SBP) vary differently throughout the exercise test in patients and in controls. Significance was set at *P*<.05. All analyses were performed using SPSS statistical software (version 10.0.6) for Windows.

#### RESULTS

#### **Baseline characteristics**

No differences were seen between the groups with respect to age, sex or height. Patients with MO had significantly greater weight, BMI, and lean and fat masses (Table 1). Table 2 shows the results for the parameters recorded at rest and during maximum effort. When baseline and maximum values are taken into account, ventilation patterns (BF, TV and VE) were no different between groups. Although under baseline conditions the SBP was significantly higher in the patients, the maximum SBP reached by both groups was similar.

The duration of exercise endured by the patients was shorter than that endured by the controls  $(14 \pm 3 \text{ com-}$ pared to  $27 \pm 4 \text{ min}$ ; *P*<.001). The distance patients traveled was therefore much shorter (661 ± 175 m compared to  $1.363 \pm 290$  m; *P*<.001) (Table 2).

#### Variables during exercise

Important differences were seen in the behavior of the CF, SBP, VO<sub>2</sub> and VE curves (Figure 1). From the beginning, and throughout exercise, the patients showed marked increases in the values of these variables. Compared to the control group, this determined an upward shift of their curves steeper slopes. This reflects the patients' greater energy consumption. After 4 min of exercise walking at 2.5 km/h on a 2% gradient (Table 3), the patients reached 75% of their maximum CF, 86% of their maximum blood pressure and 58% of their peak VO<sub>2</sub> whereas control subjects had only reached 57, 75 and 34% respectively. After 14 min of exercise, when the patients had all ended the test through exhaustion, they were consuming 2.17 L/min of O<sub>2</sub>-almost double that seen in the controls (1.12 L/min) (Table 3). Since the abscissa represents time, the graphs for all these variables were shorter in patients, corresponding to the shorter duration of their tests (Figure 1).

Baseline and final  $VO_2$  were higher in the patients (Figure 1 and Table 2). In both situations, if  $VO_2$  is corrected for body weight, the relationship inverts and

TABLE 1.	Characteristics of	participants
----------	--------------------	--------------

	Obese (n=31)	Control (n=30)	Ρ
Age, years	40 ± 6	39 ± 7	NS
Women	22 (71%)	21 (70%)	NS
Height, cm	$164 \pm 11$	164 ± 9	NS
Weight, kg	137 ± 34	64 ± 10	<.001
BMI, kg/m <sup>2</sup>	$50 \pm 9$	$24 \pm 2$	<.001
Lean mass, kg	58 ± 11	43 ± 9	<.001
Fat mass, kg	65 ± 17	19 ± 5	<.001

BMI indicates body mass index; NS: nonsignificant.

	Obese (n=31)	Control (n=30)	Р
	(11=31)	(11=50)	r
Resting			
BR, breaths/min	15 ± 3	14 ± 2	NS
TV, L/min	$0.74 \pm 0.2$	0.71 ± 0.2	NS
V, L/min	11 ± 3	10 ± 2	NS
SBP, mm Hg	131 ± 15	118 ± 15	<.002
DBP, mm Hg	84 ± 10	78 ± 9	<.03
CF, beats/min	90 ± 12	85 ± 13	NS
VO <sub>2</sub> , L/min	0.40 ± 0.1	$0.29 \pm 0.2$	<.001
VO <sub>2</sub> , mL/min/kg	$2.9 \pm 0.9$	4.5 ± 1.2	<.001
VO <sub>2</sub> , mL/min/kg lean mass	6.8 ± 2	6.7 ± 2	NS
O <sub>2</sub> pulse, mL/beat	4.3 ± 1.3	3.4 ± .9	<.02
$O_2$ pulse, mL/kg lean	$7.4 \pm 2.3$	8.2 ± 2	NS
mass/beat			
RQ	0.71 ± 0.2	0.81 ± 0.2	<.001
Maximum effort			
BF, breaths/min	32 ± 4	32 ± 5	NS
TV, L/min	1.9 ± .5	$1.9 \pm 0.4$	NS
V, L/min	59 ± 17	$60 \pm 14$	NS
SBP, mm Hg	184 ± 30	180 ± 17	NS
DBP, mmHg	95 ± 14	88 ± 9	<.02
CF, beats/min	156 ± 16	173 ± 15	<.001
VO <sub>2</sub> , L/min	2.37 ± .5	$2 \pm 0.5$	<.05
VO2, mL/min/kg	$17 \pm 3.4$	32 ± 4	<.001
VO <sub>2</sub> , mL/min/kg lean mass	42 ± 8	47 ± 6	<.02
O <sub>2</sub> pulse, mL/beat	15 ± 3	12 ± 2	<.001
O <sub>2</sub> pulse, mL/kg lean	0.28 ± 0.05	0.27 ± 0.04	NS
mass/beat			
RQ	0.87 ± 0.2	1 ± 0.2	<.001
Duration, min	14 ± 3	27 ± 4	<.001
Distance, m	661 ± 175	1.363 ± 290	<.001

TABLE 2. Resting and maximum effort of patients and controls

RQ indicates respiratory quotient; CF, cardiac frequency; BF, breathing frequency; DBP, diastolic blood pressure; SBP, systolic blood pressure; TV, tidal volume; VE, ventilation per minute; VO<sub>2</sub>, oxygen consumption; O<sub>2</sub> pulse (mL/kg mm/beat), O<sub>2</sub> pulse having corrected VO2 for kg lean body mass; NS: nonsignificant.

 $VO_2$  becomes much greater for the control group (Table 2). However,  $VO_2$  per kg of lean mass was the same in baseline conditions in both groups and, although the maximum was slightly lower in the patients, no significant differences were seen between the two groups during exercise.

The baseline, maximum (Table 2) and in-exercise  $O_2$  pulse values of patients were significantly greater than those of the controls. This variable was always higher in the patients whether comparisons were made for the 25th, 50th or 75th percentiles, at rest, or at the point of maximum effort (*P*<.001) (Figure 2). However, when  $O_2$  pulse was calculated after correcting for VO<sub>2</sub> for lean body mass, the differences between the groups disappeared (Figure 2).

When exercise was finished, the controls had reached 95% of their MTCF, and their RQ was 1 (Table 2); therefore the effort made by these subjects was

TABLE 3. Comparison of CF, blood pressure and VO <sub>2</sub>
between the two groups at 4 and 14 min of exercise

	Obese	Control
CF, beats/min		
4 min	117 (75%)	99 (57%)
14 min	149 (95%)	121 (70%)
Maximum value	156	173
SBP, mm Hg		
4 min	160 (86%)	136 (75%)
14 min	184 (100%)	152 (84%)
Maximum value	184	180
VO <sub>2</sub> (L/min)		
4 min	1.37 (58%)	0.68 (34%)
14 min	2.17 (91%)	1.12 (56%)
Maximum value	2.37	2

CF indicates cardiac frequency; SBP, systolic blood pressure;  $VO_2$ , oxygen consumption. Values in parentheses are percentages of the maximum value for the variable.

practically their maximum. When the patients reached the end of exercise, however, they had only reached 86% of their MTCF and their RQ was 0.87 (Table 2); therefore, they had not reached the limit of their cardiopulmonary capacity and their effort was sub-maximum. The gradients of the patients' VO<sub>2</sub> and CVO<sub>2</sub> curves during exercise were almost parallel (Figure 3); the expected increase in CO<sub>2</sub> production with increased O<sub>2</sub> consumption—seen in the control group—did not occur (Figure 3).

# DISCUSSION

The patients endured the exercise test for much shorter times than the controls-the former therefore covered only half the distance achieved by the latter. As soon as effort began, the patients had higher CF, SBP, VO<sub>2</sub> and VE levels (Figure 1), showing them to consume more energy from the beginning of exercise. This might be needed to move their much heavier bodies.<sup>23</sup> When walking at 2.5 km/h and with only a very slight gradient, the patients had already reached 58% of their maximum VO<sub>2</sub>. In contrast, the controls had only reached 34%. These results agree with those reported by other authors.<sup>18,23,24</sup> For these patients, a simple walk therefore exacted a metabolic output much greater than that required of the normal weight controls. After 14 minutes of exercise, when the patients could no longer continue the test (and their effort ended), the controls had consumed only 5 ME, i.e., the  $VO_2$  needed to perform the basic activities of daily life.20

Although the majority of authors agree on the limitation of effort by obese people, controversy remains with respect to their cardiopulmonary capacity. Some authors consider it to be normal<sup>12,13</sup>

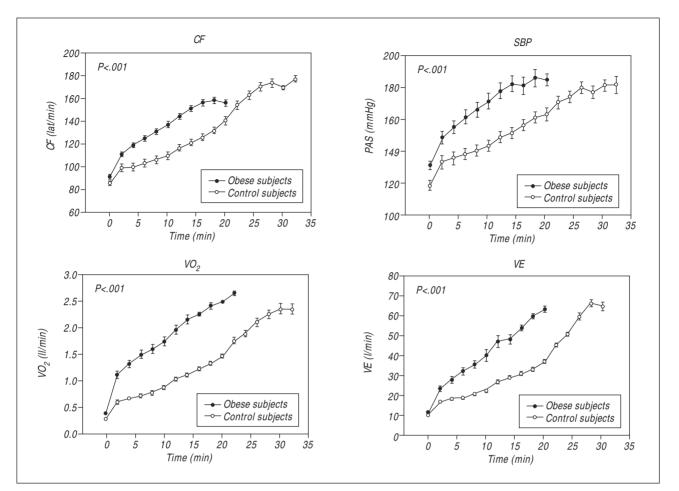
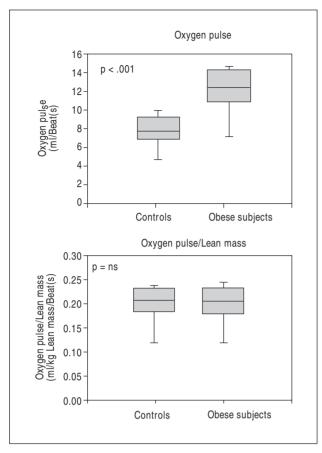


Fig. 1. Behavior of the different variables during exercise. From the outset, the patients showed higher values in general than those of controls. CF indicates cardiac frequency; SBP, systolic blood pressure; VE, ventilation per minute; VO<sub>2</sub>, oxygen consumption; beats/min, beats per minute. Points are means, bars are standard errors.

while others believe it to be affected.<sup>14-18</sup> The patients in the present study showed higher O<sub>2</sub> pulse rates during exercise. Bearing in mind that  $O_2$  pulse depends on stroke volume and the arterio-venous difference in O<sub>2</sub><sup>25</sup> and given that during maximum exercise the latter is similar in obese and normal weight people,<sup>26</sup> the higher O<sub>2</sub> pulse values of the patients must correspond to a greater stroke volume.<sup>27</sup> This res ponse has also been described in people who practice top level sport.<sup>28</sup> For this reason, it is indicated by some that obese people are physically more able because of the training that carrying their excess weight provides.<sup>29</sup> On the contrary, when the stroke volume is incapable of increasing in response to exercise, the  $O_2$  pulse is low.<sup>30</sup>

The controversy surrounding cardiopulmonary response to exercise in obese people stems from the lack of agreement on how to compare populations with different body sizes. When the absolute  $VO_2$  of different populations with different weights is compared, their is wide consensus that the hea-

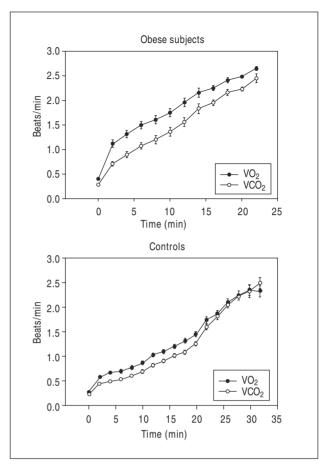
viest individuals will have the greatest O<sub>2</sub> consumption. But if VO<sub>2</sub> is corrected for body weight, those who are obese show much lower values. This criterion has been used to argue that their cardiopulmonary functional capacity is deficient.14,18 Howe-ver, the normalization of variables by weight for obese people has been criticized by several authors for not taking into account the different metabolic needs of the various body tissues.<sup>31,32</sup> Recently, it has been suggested that lean body mass might be a better variable to use since it is metabolically very active and correlates strongly with  $VO_2^{12,33}$  In the present study, when the  $O_2$ pulse of the two groups is compared after correcting  $VO_2$  for lean mass (Figure 2), the differences between the groups disappear. This supports the idea that cardiopulmonary capacity is similar in both groups, and, therefore, normal. The small capacity the patients showed for exercise is due to the high metabolic cost of their daily life activities. Their greater O<sub>2</sub> consumption is insufficient to compensate for the overload of their fat mass, as



**Fig. 2.** The upper figure compares the  $O_2$  pulse values (VO<sub>2</sub>/CF) of the two groups using a box chart. Values for the patient group are much higher (*P*<.001). The lower figure shows how these differences disappear when  $O_2$  pulse is calculated after having corrected VO<sup>2</sup> for lean body mass (VO<sub>2</sub>/kg lean body mass/CF)(NS). CF indicates cardiac frequency; VO<sub>2</sub>, oxygen consumption.

shown by their low  $VO_2$  per kg body weight figures (Table 2).

RQ is equivalent to the carbon dioxide produced divided by the oxygen consumed. At high levels of exercise, the production of  $CO_2$  is greater than  $VO_2$  and, therefore, the RQ is greater than 1. This is one of the parameters used to determine the level of effort.<sup>20</sup> Reaching the MTCF is another indicator of having reached the limit of cardiovascular capacity. In the patients, the production of CO<sub>2</sub> throughout the test was always lower than O<sub>2</sub> intake, and their RQ at the end of exercise was below 0.9 (Figure 3). Further, only 86% of MTCF was reached. Therefore, the patients finished their effort without having reached the maximum limit of their cardiopulmonary capacity. The present study does not allow us to determine whether this is due to a subjective sensation of poor tolerance to effort,<sup>34</sup> the incapacity to perform functions in anaerobiosis,<sup>35</sup> or an alteration in pulmonary fucntion<sup>36,37</sup>. Hulens<sup>18</sup> obtained the same results—in that particular



**Fig. 3.** The upper figure shows that throughout exercise, the VCO<sub>2</sub> of patients is lower than their VO<sub>2</sub>. This suggests that their effort was not maximum. The lower figure shows how, in the control group at the end of effort, VCO<sub>2</sub> was greater than VO<sub>2</sub>. Points represent the mean; bars are standard error. L/min indicates liters per minute; VCO<sub>2</sub>, production of carbon dioxide; VO<sub>2</sub>, oxygen consumption.

study, only 18% of patients ended their effort due to skeletomuscular discomfort.

## Limitations of the study

Since only 56% of the original obese population was studied, it could be argued that the present results are biased since the least affected subjects were those chosen. However, those who were analyzed were a wide selection and showed an acute degree of obesity.

Since the patients did not reach their maximum cardiopulmonary capacity and only managed a sub-maximum effort, this study does compare two groups with different effort levels. In any event, the patients showed normal cardiopulmonary capacity for the effort they made.

## CONCLUSIONS

The patients finished the test only having made a sub-

maximum effort. Despite this, they showed cardiopulmonary capacity within the normal limits for the effort made. After correcting VO<sub>2</sub> for lean body mass, the O<sub>2</sub> pulse of the patients was no different from that of the normal weight controls. However, as soon as exercise began, the patients showed high energy consumption–necessary to move their large mass. This metabolic cost determines the reduced exercise capacity they suffer, as reflected in the short duration of their tests.

#### REFERENCES

- 1. Wilding J. Science, medicine, and the future: obesity treatment. BMJ 1997;315:997-1000.
- Flegal KM, Carroll MD, Kuczmarski RJ, Johnson CL. Overweight and obesity in the United States: prevalence and trends, 1960-1994. Int J Obes 1998;22:39-47.
- Vasan RS, Larson MG, Levy D, Evans JC, Benjamin EJ. Distribution and categorization of echocardiographic measurements in relation to reference limits. The Framingham Heart Study: formulation of a height and sex specific classification and its prospective validation. Circulation 1997;96:1863-73.
- Levy D, Garrison RJ, Savage DD, Kannel WB, Castelli WP. Prognostic implications of echocardiographically determined left ventricular mass in the Framingham heart study. N Engl J Med 1990; 322:1561-6.
- De Simone G, Devereux RB, Daniels SR, Koren MJ, Meyer RA, Laragh JH. Effect of growth on variability of left ventricular mass: assessment of allometric signals in adults and children and their capacity to predict cardiovascular risk. J Am Coll Cardiol 1995;25:1056-62.
- Alpert MA, Lambert CR, Terry BE, Cohen MV, Mukerii V, Massev CV, et al. Interrelationship of left ventricular mass, systolic function and diastolic filling in normotensive morbidly obese patients. Inter J Obes 1995;19:550-7.
- Scaglione R, Dichiara MA, Indovina R, Lipari R, Ganguzza A, Parrinello G, et al. Left ventricular diastolic and systolic function in normotensive obese subjects: influence of degree and duration ob obesity. Eur Heart J 1992;13:138-42.
- Chakko S, Mayer M, Allison MD, Kessler KM, Materson BJ, Myerburg RJ. Abnormal left ventricular diastolic filling in eccentric left ventricular hypertrophy of obesity. Am J Cardiol 1991; 68:95-8.
- Grossman E, Oren S, Messerli FH. Left ventricular filling in the systemic hypertension of obesity. Am J Cardiol 1991;68:57-60.
- Stoddart MF, Tseuda K, Thomas M, Dillon S, Kupersmith J. The influence of obesity on left ventricular filling and systolic function. Am Heart J 1992;124:694-9.
- Crisostomo LL, Batista Araujo LM, Câmara E, Carvalho C, Silva FA, Vieira M, et al. Comparison of left ventricular mass and function in obese versus nonobese women < 40 years of age. Am J Cardiol 1999;84:1127-9.
- Maffeis C, Schena F, Zaffanello M, Zoccante L, Schutz Y, Pinelli L. Maximal aerobic power during running and cycling in obese and non-obese children. Acta Paediatr 1994;83:113-6.
- Rowland TW. Effects of obesity on aerobic fitness in adolescent females. Am J Dis Child 1991;145:764-8.
- Salvadori A, Fanari P, Fontana M, Buontempi L, Saezza A, Baudo S, et al. Oxygen uptake and cardiac performance in obese and normal subjects during exercise. Respiration 1999;66: 25-33.

- Reybrouck T, Mertens L, Schepers D, Vinckx J, Gewillig M. Assessment of cardiorespiratory exercise function in obese children and adolescents by body mass-independent parameters. Eur J Appl Physiol Occup Physiol 1997;75:478-83.
- Alpert MA, Singh A, Terry BE, Kelly DL, Villarreal D, Mukerji V. Effect of exercise on left ventricular systolic function and reserve in morbid obesity. Am J Cardiol 1989;63:1478-82.
- Davies CT, Godfrey S, Light M, Sargeant AJ, Zeidifard E. Cardiopulmonary responses to exercise in obese girls and young women. J Appl Physiol 1975;38:373-6.
- Hulens M, Vansant G, Lysens R, Claessens AL, Muls E. Exercise capacity in lean versus obese women. M Scand J Med Sci Sports 2001;11:305-9.
- Balke B, Ware RW. An experimental study of physical fitness of Air Force personnel. US Armed Force Med J 1959;10:675-9.
- Fletcher GF, Balady G, Froelicher VF, Hartley LH, Haskell WL, Pollok ML. Exercise Standards. A statement for healthcare professionals from the American Heart Association. Circulation 1995;91:580-615.
- Fargard R, Conway J. Measurement of cardiac output: Fick principle using catheterization. Eur Heart J 1990;11(Suppl I):1-5.
- Misquita NA, Davis DC, Dobrovolny CL, Ryan AS, Dennis KE, Nicklas BJ. Applicability of maximal oxygen consumption criteria in obese, postmenopausal women. J Women Health Gend Based Med 2001;10:879-85.
- Salvadori A, Fanari P, Mazza P, Agosti R, Longhini E. Work capacity and cardiopulmonary adaptation of the obese subject during exercise testing. Chest 1992;101:674-9.
- 24. Mattsson E, Evers Larsson U, Rossner S. Is walking for exercise too exhausting for obese women? Int J Obes 1997;21:380-6.
- López J, Fernández A. Fisiología del ejercicio. 2.ª ed. Madrid: Editorial Médica Panamericana, 1998; p. 274.
- DeDivitiis O, Fazio S, Petitto M, Maddalena G, Conta F, Mancini M. Obesity and cardiac function. Circulation 1981;64:477-82.
- Wasserman K, Hansen JE, Sue DY, Casaburi R, Whipp BJ. Measurements during integrative cardiopulmonary exercise testing. En: Weinberg R, editor. Principles of exercise testing and interpretation. Baltimore: Lippincott Williams & Wilkins, 1999; p. 62-94.
- Ekblom B, Astrand P-O, Saltin B, Stenberg J, Walstron J. Effect of training on circulatory response to exercise. J Appl Physiol 1968; 24:518-28.
- Farebrother MJB. Respiratory function and cardiorespiratory response to exercise in obesity. Br J Dis Chest 1979;73:211-25.
- Nery LE, Wasserman K, French W, Oren A, Davis JA. Contrasting cardiovascular and respiratory responses to exercise in mitral valve and chronic obstructive pulmonary diseases. Chest 1983; 83:446-53.
- De Simone G, Devereux RB, Daniels SR, Mureddu G, Roman MJ, Kimball TR, et al. Stroke volume and cardiac output in normotensive children an adults. Assessment of relations with body size and impact of overweight. Circulation 1997;95:1837-43.
- Lauer MS, Larson MG, Levy D. Gender-specific reference Mmode values in adults: population-derived values with consideration of the impact of height. J Am Coll Cardiol 1995;26:1039-46.
- Goran M, Fields DA, Hunter GR, Herd SL, Weinsier RL. Total body fat does not influence maximal aerobic capacity. Int J Obes 2000;24: 841-8.
- Jones NL, Killian KJ. Exercise limitation in health and disease. N Engl J Med 2000;343:632-41.
- 35. Ardévol A, Adán C, Franco L, García-Lorda P, Rubio F, Remesar X, et al. During intense exercise, obese women rely more than lean women on aerobic energy. Eur J Physiol 1998;435: 495-502.
- Ray CS, Sue DY, Bray G, Hansen JE, Wasserman K. Effect of obesity on respiratory function. Am Rev Respir Dis 1983;128: 501-6.
- Whipp BJ, Davis JA. The ventilatory stress of exercise in obesity. Am Rev Respir Dis 1984;129(Suppl):S90-2.