Work Capacity and Cardiopulmonary Adaptation of the Obese Subject during Exercise Testing*

Alberto Salvadori, M.D.; Paolo Fanari, M.D.; Paola Mazza, M.D.; Roberto Agosti, M.D.; and Erminio Longhini, M.D.

Twelve obese patients (six male subjects) aged 17 to 42 years with a mean body mass index (BMI) of 40 kg/m² and 12 normal subjects (six male subjects) aged 19 to 39 years with a mean BMI of 22 kg/m² underwent a cycloergometric test until exhaustion to assess work capacity and cardiopulmonary adaptations of the two groups. Minute ventilation, expiratory gas concentrations, and heart rate, together with anaerobic threshold (AT) were determined in each subject during the test. The same subjects were then submitted to normocapnic hyperpnea at rest to assess the oxygen cost of breathing. We found that in the obese patients, the maximal sustainable work rate was not different from that of controls (120 vs 136 W) while AT was significantly lower (78 vs 110 W). Nevertheless, there was no difference in maximum \( \dot{V}O_2 \) and in \( \dot{V}O_2 \) at AT levels (expressed in milliliters per minute) indicating that cardiac, pulmonary, vascular, and muscle performance did not differ from obese to normal subjects. Greater muscular effort was needed by obese patients when moving their heavier legs and less when doing external work with a decreased gross mechanical efficiency and an identical net mechanical efficiency between the two groups. (Chest 1992; 101:674-79)

Obese patients usually exhibit lung function abnormalities related to the increase in body weight. These include a decrease in the functional residual capacity (FRC) due mainly to a decrease in the expiratory reserve volume (ERV)\(^1\) and a decrease in compliance of the respiratory system.\(^2\) These functional abnormalities cause an increase in the energy cost of breathing.\(^3\) In addition, according to Whipp and Davis,\(^4\) the increased body mass is associated with greater metabolic energy requirements during muscular exercise which results in further ventilation stress.

To our knowledge, there have been no previous studies dealing with cardiopulmonary adaptation in obese patients during incremental exercise testing.

In a group of obese patients and in a matched group of normal individuals, the aim of this study was to investigate the maximal sustainable work capacity and the anaerobic threshold, which is generally regarded as a useful index of fitness in both patients with cardiorespiratory diseases\(^5\)\(^,\)\(^6\) and normal subjects, including athletes.\(^9\)\(^,\)\(^14\)

Moreover, we assessed the oxygen consumption (\( \dot{V}O_2 \)) minute ventilation (\( V_e \)), and heart rate during both exercise and the subsequent recovery. We also investigated the influence of obesity on \( \dot{V}O_2 \) during normocapnic hyperpnea at rest to assess the oxygen cost of breathing.

**METHODS**

**Subjects**

We studied 12 obese patients and 12 normal subjects matched for age and sex, all employed as administrative or teaching staff, both groups being untrained and without cardiorespiratory disorders.

In the obese group there were six male and six female subjects whose mean age (± SE) was 25.9 ± 2.3 years (range, 17 to 42 years). The control group consisted of six male and six female subjects whose mean age was 27.7 ± 1.8 years (range, 19 to 39 years).

The body mass index (BMI, kg/m²) was taken as a measure of obesity.\(^13\)\(^,\)\(^16\) with a BMI <25 for normal subjects. In our obese patients the mean BMI was 39.9 ± 1.1 kg/m² and in the normal subjects it was 22.2 ± 0.8 kg/m² (p<0.001) (Table 1). All the obese patients were hospitalized in the Division of Endocrinology in our Center, and were tested within three days of hospital admission. None was receiving medical therapy and all were receiving the same diet (1,500 kcal/day).

**Pulmonary Function**

Total lung capacity and its subdivisions were measured using the helium closed circuit dilution method,\(^9\) together with the flow-volume curve. The pulmonary diffusing capacity for CO was measured using the single breath method.\(^16\)

**ergospirometry**

The ergospirometric test was performed on a cycloergometer (Gould) at least 3 h after lunch, by subsequent 20-W increases every 4 min until exhaustion. This was followed by a 20-min prolonged recovery period in a sitting position (modified protocol of Sjöstrand).\(^8\)

The base values were recorded at a preset pedaling rate (60

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Table 1—Anthropometric and Lung Function Data*

<table>
<thead>
<tr>
<th></th>
<th>Normal Subjects</th>
<th>Obese Subjects</th>
<th>p Value†</th>
</tr>
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<tbody>
<tr>
<td>No. of subjects</td>
<td>12</td>
<td>12</td>
<td>NS</td>
</tr>
<tr>
<td>Sex, M/F</td>
<td>6/6</td>
<td>6/6</td>
<td>NS</td>
</tr>
<tr>
<td>Age, yrs</td>
<td>27.8±1.8 (19-39)</td>
<td>25.9±2.3 (17-42)</td>
<td>NS</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>64.9±3.4 (49-85)</td>
<td>110.7±8.9 (91-144)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Height, cm</td>
<td>170±3 (154-194)</td>
<td>165.3±3.1 (150-190)</td>
<td>NS</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>22.2±0.8 (19-25)</td>
<td>39.9±1.1 (35-46)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>VC, L</td>
<td>5.8±0.4 (120%)</td>
<td>4.6±0.3 (107%)</td>
<td>NS</td>
</tr>
<tr>
<td>ERV, L, BTPS</td>
<td>1.8±0.3 (95%)</td>
<td>0.9±0.1 (104%)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>FRC, L, BTPS</td>
<td>3.9±0.4 (114%)</td>
<td>2.1±0.2 (124%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>RV, L, BTPS</td>
<td>2.1±0.3 (138%)</td>
<td>1.2±0.1 (154%)</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>TLC, L, BTPS</td>
<td>7.9±0.8 (122%)</td>
<td>5.8±0.4 (107%)</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>FEV₁, L, BTPS</td>
<td>4.6±0.4 (115%)</td>
<td>3.7±0.3 (101%)</td>
<td>NS</td>
</tr>
<tr>
<td>FEV₁/VC, %</td>
<td>79.3±2.6</td>
<td>80.4±2.3</td>
<td>NS</td>
</tr>
<tr>
<td>Dco, ML/MM Hg STPD</td>
<td>44.8±9.6 (142%)</td>
<td>33.8±2.7 (121%)</td>
<td>NS</td>
</tr>
<tr>
<td>HbO₂ Sat, %</td>
<td>97.6±3.3</td>
<td>96.3±3.1</td>
<td>NS</td>
</tr>
</tbody>
</table>

*Values are mean±SE. In parentheses, the range values of anthropometric data and the values percent predicted of lung function data are given. BMI = body mass index; VC = vital capacity; ERV = expiratory reserve volume; FRC = functional residual capacity; RV = residual volume; TLC = total lung capacity; FEV₁ = forced expiratory volume in 1 s; Dco = diffusing capacity of lung for CO; HbO₂ Sat = O₂ saturation of hemoglobin in arterial blood.
†By two-tailed analysis of variance.

cycles/min) without any load. An ergospirometer (MMC Horizon System 4400 TC; Sensor Medics) was used. The gas was sampled at the mouth by means of a 210-cm-long heated tube (100°C). A personal computer (HP series 300) was connected to the equipment to analyze data.

Calibrations were performed prior to each test. We took into consideration the mean data obtained during the last minute of each work rate, which should represent steady-state values. The HbO₂ saturation was determined at 20-s intervals during the test by means of a percutaneous oximeter (Radiometer). The heart rate was monitored (by a Kontron 504 Heartstation). At the end of each work rate, systolic and diastolic arterial blood pressure were measured.

![Figure 1. Mean V̇O₂ during exercise testing for obese patients compared with normal subjects is plotted; only the SE is graphically representable. AT = anaerobic threshold; *= p<0.05; **= p<0.01; p values by two-tailed analysis of variance and Dunnett method. At the bottom of the figure are the reported mean values of V̇O₂, the corresponding SE, and the number of subjects who have reached each work rate.](http://journal.publications.chestnet.org/)
pressure were determined.

The anaerobic threshold was determined as previously described17,42 by using the following criteria: (1) inflection point on the minute ventilation ($V_{E}$) vs $V_{O_{2}}$ diagram; (2) point of increase in end-tidal $P_{CO_{2}}$ (PetCO$_{2}$); and (3) point of increase in the ventilatory equivalent of $O_{2}$ ($V_{E}/V_{O_{2}}$) without a concomitant reduction of end-tidal $P_{CO_{2}}$ (PetCO$_{2}$).

Voluntary Hyperpnea Test

The subjects were asked to voluntarily increase $V_{E}$ at rest while breathing in an open circuit. CO$_{2}$-enriched air was added to keep the PetCO$_{2}$ constant during the test. In all subjects the magnitude of $V_{E}$ during voluntary hyperpnea corresponded to ventilations achieved during muscular exercise at 40 and 80 W. These ventilations were the more easily reproducible ventilatory frequencies considering our aim.

The voluntary hyperpnea tests lasted 2 min, when the steady state was reached, and, as in the ergospirometric test, the mean data obtained during the last minute of each test are reported.

The investigation was approved by the institutional ethics committee and informed consent was obtained from each volunteer.

Statistical Analysis

The ergospirometric data and the results obtained in the other tests were compared across groups by analysis of variance, and the Dunnett method was used to determine which of the obese group values differed from those of the controls at a given step of the exercise. A $p$ value less than 0.05 was considered statistically significant. Values are expressed as mean ± SE.

RESULTS

As shown in Table 1, the ERV and the FRC were significantly decreased in the obese patients. There was no significant difference in all the other lung function variables measured. The oxyhemoglobin saturation was within normal limits in the obese and normal subjects both during rest and muscular exercise.

<table>
<thead>
<tr>
<th>Table 2—Mean $V_{E}$ During Exercise Testing*</th>
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<tr>
<td></td>
</tr>
<tr>
<td>Normal subjects</td>
</tr>
<tr>
<td>SE</td>
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<tr>
<td>Obese patients</td>
</tr>
<tr>
<td>SE</td>
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</tbody>
</table>

*p Abbreviations and the number of subjects who reached each work rate is same of Fig 1. AT = anaerobic threshold.

†By two-tailed analysis of variance and Dunnet method.

**Figure 2.** Mean heart rate during exercise testing and initial recovery phase; for other details see legend to Fig 1.
The peak work rate achieved did not differ significantly from normal to obese subjects (136 ± 11.6 W in controls, 120 ± 9 W in the obese patients). The anaerobic threshold, expressed as work rate was, however, significantly lower (p<0.05) in the obese patients (78 ± 9 W vs 110 ± 11 W).

The oxygen consumption at the different exercise levels was significantly higher in the obese patients except at the anaerobic threshold level and at peak exercise work rate (Fig 1). During recovery, the average values of \( \dot{V}O_2 \) were consistently higher in the obese patients but a significant difference relative to controls was found only at 2, 3, 4, and 15 min of recovery.

Minute ventilation was significantly higher in the obese patients at rest, while pedaling at zero resistance and at 20 W (Table 2). As work rates increased, the average values of \( \dot{V}E \) were consistently higher than those of normal subjects but the difference was not significant. On the contrary, both at anaerobic threshold and peak work rate levels, \( \dot{V}E \) was higher in normal subjects, although not significantly. During recovery, \( \dot{V}E \) did not differ significantly from one group to the next.

The heart rate was significantly higher in the obese patients at rest, pedaling at zero resistance up to a work rate of 40 W (Fig 2). At peak activity, however, it was significantly lower than in controls. During recovery, it continued its low tendency in obese patients, the difference being significant for the first minute.

The \( \dot{F}ETO_2 \) at rest and during exercise did not differ significantly between the two groups (Fig 3) and decreased from rest to free pedaling in both groups, significantly in obese patients; at peak exercise and throughout recovery, however, it was significantly higher in obese patients.

During normocapnic voluntary hyperpnea, \( \dot{V}O_2 \) increased progressively with \( \dot{V}E \), there being no significant difference between obese and nonobese subjects (Fig 4). Since during muscular exercise up to 120 W \( \dot{V}O_2 \) was significantly higher in the obese patients (Fig 1), this difference cannot be attributed to the increased \( O_2 \) cost of breathing associated with the higher \( \dot{V}E \) of the obese patients.

**DISCUSSION**

Our results do not show a significant difference in the maximal sustainable work capacity between normal subjects and markedly obese patients (BMI: 39.9 ± 1.1 kg/m²). The anaerobic threshold, however, was reached at a significantly lower work rate in the obese patients (p<0.05). According to Wasserman,21 this implies that the endurance time at maximal effort is less in obese patients than in normal subjects as a result of the increase in arterial lactate with subse-
quent metabolic acidosis. Thus, our obese patients appear to have a decreased working capacity compared with normal subjects.

They use a greater amount of O2 to accomplish the same work rate. In line with previous reports,1,24 at rest they have a higher VO2 than normal subjects, reflecting the increased body weight. During the free wheeling there is a further increase in the VO2 difference between obese and normal subjects that most likely reflects the fact that the obese patients undergo greater postural stress when sitting on the cycloergometer.

The results obtained during normocapnic hyperpnea indicate that only a small proportion of the increase in VO2 of the obese patients can be attributed to the increased O2 cost of breathing (Fig 4). A preferential utilization of the lipid substrate, which involves a greater O2 consumption, may explain the results in our obese patients,25 but are probably less important mechanisms.

Maximum VO2 and VO2 at anaerobic threshold levels did not differ from obese to normal subjects when expressed as milliliters per minute. This indicates that global cardiac, pulmonary, vascular, and muscle performance, which contributes to the delivery of oxygen, shows no difference between the two groups.

As indicated by the increase in VO2, more muscular effort was needed by obese patients when moving the heavier legs and less when doing external work, as expressed by the lower work rate performed at a given VO2 when compared with normal subjects (Fig 1).

The term "mechanical efficiency" defines the energy used to do a given amount of work26-28 and is conceptually compared with the "muscular contraction efficiency," which is based on thermodynamic considerations during modest steady-state exercise. Total energy utilization and total work performed are necessary to obtain the measurement of muscular efficiency in the condition of anaerobiosis.29

During the muscular exercise performed by our subjects, the increase in VO2 is the same in both groups, as reflected by the same slope of the relationship between VO2 and watts. This indicates that during the aerobiosis period of the test, the gross mechanical efficiency (watts/VO2 exercise) is less in the obese patients than in the normal subjects, and the net mechanical efficiency (watts/VO2 - VO2 free wheeling) is virtually identical in obese and normal subjects.29-31

The relationship between heart rate and cardiac output (Q) has been well studied in normal subjects. During exercise, the increase of stroke volume is limited to 30 percent of the initial value, and as a result, there is a close correlation between heart rate and Q.32 To our knowledge, this relationship has not been explored yet in obese patients.

In our obese patients, the heart rate was significantly higher than that of normal subjects at rest and during

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**Figure 4.** Mean VO2 at rest, at 40 and 80 W, and mean VO2 during voluntary normocapnic hyperpnea; for other details see legend to Fig 1.
exercise up to the anaerobic threshold, while it was significantly lower at peak activity when metabolic acidosis was present.\\textsuperscript{21}

This may reflect the greater increase in $Q$ by the obese patients during the initial phases of the incremental exercise and a defective adjustment proceeding in anaerobiosis.

Our obese patients have a higher body mass that probably depends on an increase in fat as well as in lean body muscular mass.

In line with what Wasserman and Whipp\\textsuperscript{33} say, the abrupt fall of PET\textsubscript{O2} at the beginning of the exercise (Fig 3), which was more pronounced in obese patients ($p<0.001$), could be related to a reduced mixed venous O\textsubscript{2} content that should be heavier in the obese patients.

To be more precise, obesity at rest results in a reduced O\textsubscript{2} extraction and in an increased $Q$ when compared with controls, while at the beginning of exercise, it grossly increases O\textsubscript{2} extraction, probably as a consequence of a preferential redistribution of Q to the increased mass of exercising muscles.

In agreement with the same authors, the subsequent slow increase of PET\textsubscript{O2} toward the initial values both in controls and in obese patients could be put down to a diffusion limitation of O\textsubscript{2} transport during exercise due to a lack of time to reach an O\textsubscript{2} equilibrium between alveolar gas and end capillary blood.

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