Central European Journal of Sport Sciences and Medicine | Vol. 53, No. 1/2026: 17–25
ISSN (print): 2300-9705 | ISSN (online): 2353-2807 | DOI: 10.18276/cej.2026.1-02

MECHANISMS OF EXERCISE LIMITATION DURING INCREMENTAL TESTING IN POSTMENOPAUSAL OBESITY: A CARDIOPULMONARY CASE SERIES

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Abstract **Background:** Obese postmenopausal women are at high risk for cardiometabolic disease, yet the specific physiological patterns of their exercise intolerance remain poorly described. Emerging evidence suggests that this intolerance reflects the combined effects of reduced cardiovascular reserve, impaired metabolic flexibility, and autonomic dysregulation, which may be detectable using cardiopulmonary exercise testing (CPET). This case series aimed to characterize the cardiorespiratory and metabolic responses to graded exercise in this population.

Case summary: We present nine sedentary, obese postmenopausal women (mean age 56.78±9.02 years; BMI 33.87±3.8 kg/m²) who underwent maximal cardiopulmonary exercise testing using graded cycle ergometry. Key parameters (VO_2, VCO_2, respiratory exchange ratio (RER), heart rate (HR), and O_2 pulse) were recorded at rest (Rest.3), the anaerobic threshold (AT), maximum workload (Wmax), and at 1 (Rec.1) and 3 (Rec.3) minutes into recovery. Results revealed severely impaired functional capacity, with peak oxygen uptake (VO_2max) ranging from (4±1) to (16±3) ml/kg/min. Key findings included profound metabolic inflexibility (high resting and recovery RER) and delayed heart rate recovery, indicative of autonomic dysfunction.

Conclusion: This case series highlights a triad of physiological impairments including reduced cardiovascular reserve, metabolic inflexibility, and autonomic dysfunction that contribute to severe exercise intolerance in obese postmenopausal women. These findings support the clinical value of CPET for identifying individual-specific physiological limitations and guiding personalized exercise interventions in this high-risk population.

Key words: obesity, postmenopausal women, incremental exercise testing, cardiopulmonary exercise testing, metabolic response, respiratory exchange ratio

Introduction

Obesity prevalence has increased dramatically worldwide (World Health Organization Obesity and Overweight, 2025) and is recognized as a chronic relapsing disease (Bray et al., 2017). Excess adiposity is a major independent

risk factor for type 2 diabetes, cardiovascular disease, and metabolic syndrome (Calle & Kaaks, 2004; Haslam & James, 2005; Lavie et al., 2009). These effects arise from complex interactions of genetics, diet, and lifestyle (Bray et al., 2017; Hill et al., 2012) and are perpetuated by low-grade inflammation and altered gut microbiota in obesity (Hotamisligil, 2006; Turnbaugh et al., 2009). Effective management requires a multifactorial approach: dietary energy balance (Hall et al., 2011), regular physical activity (Donnelly et al., 2009; Swift et al., 2014), and weight-loss maintenance (Jakicic et al., 2018) are all essential. Indeed, current guidelines emphasize integrated lifestyle interventions for weight control (Hruby & Hu, 2015). Because postmenopause brings a decline in estrogen, increased visceral fat, and worsening insulin resistance, these factors synergistically impair cardiometabolic function in middle-aged and postmenopausal women with obesity (Després, 2012; Lavie et al., 2009). In addition to these metabolic changes, postmenopausal women with obesity commonly exhibit reduced cardiorespiratory fitness, impaired substrate utilization during exercise, and altered autonomic regulation, which together contribute to marked exercise intolerance and elevated cardiovascular risk (Monferrer-Marín et al., 2024; Vasconcelos et al., 2025).

Incremental cardiopulmonary exercise testing (CPET) can unmask early maladaptations in this group, such as a disproportionate rise in ventilatory drive or early anaerobic shift. Importantly, CPET provides an integrated assessment of cardiovascular reserve, metabolic flexibility, and autonomic recovery responses, enabling identification of the dominant physiological limitations to exercise in individual patients (Woodward et al., 2025; Sánchez-Delgado et al., 2023).

In this case series, we evaluated the cardiorespiratory and metabolic responses during graded cycle ergometry in nine obese postmenopausal women, to characterize the magnitude and nature of their exercise intolerance. While cohort studies have established low mean fitness in this population, the specific patterns of physiological limitation and the degree of inter-individual variability remain poorly characterized. Therefore, the purpose of this case series was to provide a detailed characterization of the cardiorespiratory and metabolic responses during incremental exercise in nine obese postmenopausal women, with particular emphasis on the interaction between cardiovascular reserve, metabolic flexibility, and autonomic function as determinants of exercise intolerance.

Methods

Participants

A total of nine postmenopausal women (mean age 56.78±9.02 years) clinically diagnosed with obesity (BMI≥30 kg/m²) were enrolled in this study (World Health Organization Obesity and Overweight, 2025). Inclusion criteria required participants to be sedentary (no regular exercise for at least six months) and within an age range of 44 to 69 years. Participants were free of overt cardiovascular or pulmonary disease as determined by a clinical evaluation and medical history. Standard exclusion criteria were applied, including orthopedic or neuromuscular conditions that would contraindicate exercise and the use of any medications known to alter energy metabolism. Before testing, all participants underwent a physical examination and provided written informed consent. The study was conducted in accordance with institutional ethical standards and received approval from the institutional ethics committee. Anthropometric measurements, including age, body weight, height, and BMI were recorded. Body surface area (BSA) was calculated using the Du Bois & Du Bois formula (Du Bois & Du Bois, 1989). Baseline vital signs were recorded after 3 minutes of seated rest. This case series was prepared and reported in accordance with the CARE guidelines (Gagnier et al., 2013).

Exercise protocol and measurements

Participants completed an incremental exercise test to exhaustion on a programmable cycle ergometer (Marquette, Ergocomp EL 1200 – USA). The protocol began with a seated rest period, followed by a workload that increased continuously by 20 watts per minute. The test was terminated when a participant could no longer maintain a steady pedaling rate of at least 50 rpm; this point was recorded as the maximum workload (W_{max}), a functional indicator of the participant's exercise capacity.

Throughout the test, respiratory gases were analyzed breath-by-breath using a mass spectrometer (II-CPX Express, Medgraphics, USA) to determine oxygen consumption (VO_2) and carbon dioxide production (VCO_2), fundamental parameters for assessing metabolic efficiency and ventilatory response to exercise.

Beat-by-beat heart rate (HR) was continuously monitored using a chest strap monitor (Polar, Italy), synchronized with the metabolic analysis system for the assessment of cardiovascular response during exercise.

From these direct measurements, the following variables were calculated:

- **Respiratory exchange ratio (RER):** This was calculated as the ratio of VCO_2/VO_2. RER is an indicator of the type of energy substrate used and the predominance of aerobic or anaerobic metabolism.
- **Anaerobic threshold (AT):** This was identified at the workload where the RER value first reached 1.00 (Liguori & Medicine, 2022), which corresponds to increased lactate production and activation of anaerobic metabolism, with a consequent increase in perceived effort.
- **Oxygen pulse (O2 Pulse):** This was calculated as the ratio of VO_2/HR, serving as an indirect index of cardiac efficiency and stroke volume.

The sequence of study procedures, from participant recruitment to data analysis, is summarized in Figure 1.

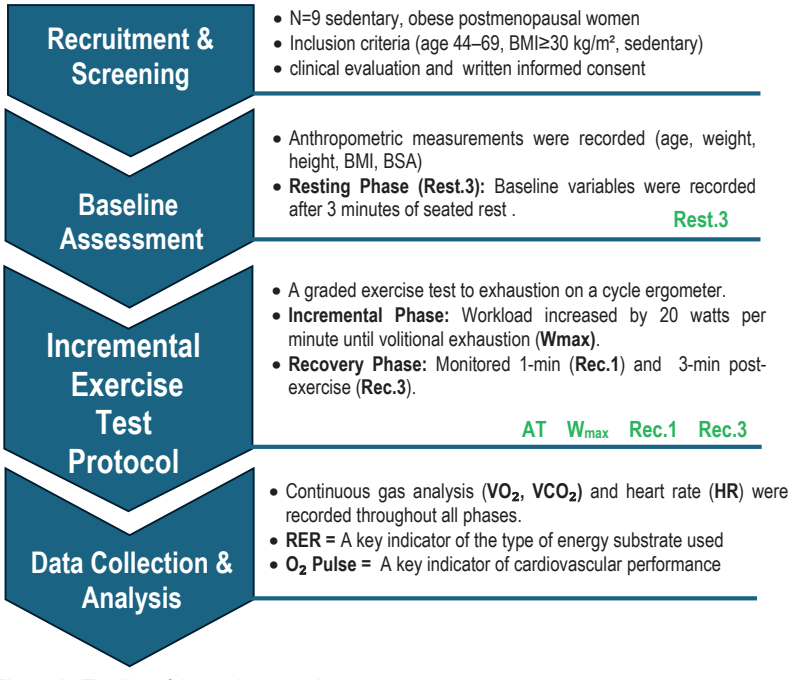


Figure 1. Timeline of the study protocol.

Statistical analysis

Data were analyzed to yield mean, standard deviation (SD), and range to describe the central tendency and dispersion of responses across the group. The primary analysis is focused on the descriptive presentation of individual and group physiological trajectories. All results are presented as mean±SD unless otherwise specified.

Case presentation

Participant characteristics

The characteristics of the nine participants are detailed in Table 1. The group had a mean age of 56.78±9.02 years and a mean BMI of 33.87±3.80 kg/m², classifying them as obese. Maximal exercise capacity was severely limited across the group, with a mean VO_{2max} of 16±3 ml/kg/min and a mean W_{max} of 111±24 W.

Table 1. Participant characteristics and peak exercise performance

Subject	Age (years)	Weight (kg)	Height (cm)	BMI (kg/m²)	BSA (m²)	AT (W)	Wmax (W)
1	66	82	161	36,4	1,77	60	100
2	59	81	150	34,6	1,78	60	100
3	61	71	153	29,6	1,70	80	100
4	69	84	155	34,8	1,83	40	80
5	52	83	155	32,8	1,85	80	140
6	44	70	159	29,1	1,69	60	120
7	64	78	155	29,7	1,83	40	80
8	47	106	162	37,6	2,14	40	140
9	49	104	168	37,3	2,11	80	140
Mean	56.8	84.3	157.6	33,55	1,86	60.0	111.1
SD	9.0	12.7	5.5	3,39	0,16	17.3	24.7

Age, body weight, and anthropometric indexes are shown with means±SD. AT: Anaerobic threshold, W_{max}: Maximum workload, BSA: Body surface area, BMI: Body mass index.

Metabolic and gas exchange responses

The physiological responses to the incremental exercise test are summarized in Table 2 and Figure 2. Significant changes were observed across the five experimental phases. From Rest.3 to peak exercise, there were significant increases in HR, VO_2, VCO_2, and O_2 Pulse (all $p<0.001$). During recovery, HR and VCO_2 remained significantly elevated above baseline at Rec.3 ($p<0.0001$), while VO_2 also remained slightly elevated ($p<0.002$). The mean power output achieved at the AT was 60±17 W, which corresponded to 54% of the mean maximal workload (W_{max}) of 111±24 W (Table 2).

The mean heart rate (HR) increased from 69±17 bpm at Rest.3 to 143±16 bpm at Wmax. At Rec.1 and Rec.3, HR was 125±22 bpm and 92±18 bpm, respectively. There was a significant difference between HR at Rest.3 and Rec.3 ($p<0.0001$) (Table 2). VO_{2max} increased from 4±1 at Rest.3 to 12±2 at AT and 16±3 at W_{max}. Although workload increased ~50% from AT to Wmax, VO_2 increased only modestly beyond that point. The decline of VO_2 in recovery was slow, remained ~27% above baseline at Rec.3 ($p<0.002$).

Carbon dioxide production (VCO_2) increased from a mean of 263±11 ml/min at Rest.3 to 1558±347 ml/min at Wmax. At Rec.3, the mean VCO_2 was 516±97 ml/min, representing a 96% increase above resting levels ($p<0.0001$; Table 2). The mean RER was 0.93±0.05 at Rest.3, 1.15±0.06 at Wmax, and 1.38±0.15 at Rec.3 (Table 2).

The oxygen pulse (VO_2/HR) increased from 4.4±1.0 ml/beat at Rest.3 to a peak of 9.5±2.1 ml/beat at W_{max}, before returning to 4.2±1.0 ml/beat after Rec.3, with no statistically significant changes ($p>0.05$; Table 2).

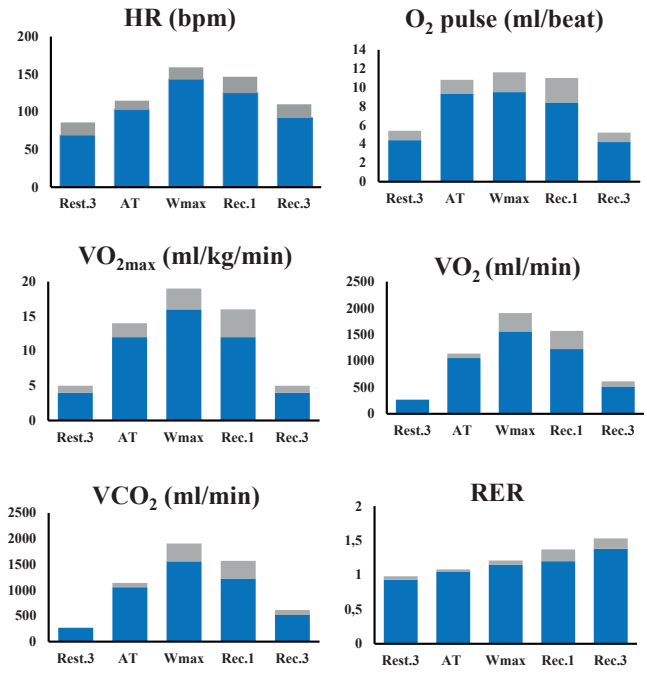
Table 2. Cardiorespiratory and metabolic responses at five stages of the exercise test

Phase	REST.3	AT	Wmax	REC.1	REC.3
HR (bpm)	69±17	103±11.9	143±16.1	125±21.6	92±18.1
VO_{2max} (ml/kg/min)	4±1	12±2	16±3	12±4	4±1
VO_2 (ml/min)	295±25	1004±75	1343±234	1018±266	374±56
VCO_2 (ml/min)	263±11	1055±8	1558±347	1224±344	516±97
RER	0.93±0.05	1.05±0.03	1.15±0.06	1.20±0.17	1.38±0.15
O_2 Pulse (ml/beat)	4.4±1.0	9.3±1.5	9.5±2.1	8.4±2.6	4.2±1.0

Rest.3: rest, AT: Anaerobic threshold, W_{max}: Maximum workload, Rec.1: 1-min recovery, Rec.3: 3-min recovery

Summary of group responses

As shown in Figure 2, while all participants demonstrated severe deconditioning, considerable heterogeneity was observed in their metabolic and autonomic responses. For instance, RER during the recovery period ranged from 1.25 to 1.50. Similarly, the rate of heart rate decline in recovery varied substantially among individuals (Figure 2).



Rest.3: rest, AT: Anaerobic threshold, Wmax: Maximum workload, Rec.1: 1-min recovery, Rec.3: 3-min recovery

Figure 2. Cardiorespiratory and metabolic responses to incremental exercise.

Discussion

This case series demonstrates that obese postmenopausal women have profoundly impaired exercise physiology. All key parameters (VO_2max, workload, chronotropic reserve) were well below reference values for sedentary peers, reflecting the synergistic negative effects of obesity, aging, and menopause (Després, 2012; Lavie et al., 2009).

The findings can be explained by a triad of factors:

(1) **Reduced cardiovascular reserve:** Lower maximal stroke volume and chronotropic response (peak HR ~143 bpm vs. 220–age) limit cardiac output.

(2) **Impaired peripheral oxygen utilization:** Likely due to diminished mitochondrial density/function in muscle (Larsen et al., 2011) coupled with a predominance of glycolytic (type II) fibers (Andersen & Henriksson, 1977), causing rapid lactate accumulation.

(3) **Chronic sympathetic overactivity:** Excess adiposity and menopause-related hormonal shifts sustain a higher resting metabolic rate (evidenced by high resting RER) and delay recovery.

Together, these produce the marked deconditioning observed.

The mean VO_2max of 16±3 ml/kg/min is alarmingly low – far under the ~22 ml/kg/min threshold considered necessary for basic functional independence in older women (Liguori & Medicine, 2022) and is a strong independent predictor of cardiovascular morbidity and all-cause mortality.

Peak workload (111±24 W) was ~30–40% lower than normative values for sedentary women, underlining a severe loss of integrated heart–lung–muscle efficiency (Larsen et al., 2011). Post-exercise VO_2 fell slowly, indicating prolonged oxygen debt and inefficient aerobic recovery, a pattern linked to reduced mitochondrial density and impaired lactate clearance (Larsen et al., 2011).

The early anaerobic shift is evidenced by RER behavior. Resting RER was ~0.93, suggesting high baseline glycolysis. In recovery, it soared to 1.38, indicating heavy metabolic acidosis driving compensatory hyperventilation to expel excess CO_2. This pattern reveals impaired metabolic flexibility and implies that even modest exercise provokes major acid-base disturbances (Brooks, 1986).

Importantly, the O_2 Pulse was maintained at expected levels (Crisafulli et al., 2007). This is a critical clinical finding, as it suggests that the heart's intrinsic contractile function was preserved and helps to rule out primary cardiac disease as the cause of exercise intolerance. The limitation is therefore due to central and peripheral deconditioning (low stroke volume and poor tissue O_2 extraction) (Crisafulli et al., 2007; Larsen et al., 2011). The blunted HR response and delayed HR recovery further highlight autonomic nervous system dysfunction, a known prognostic marker for increased cardiovascular risk (Cole et al., 1999; Haddock et al., 1998).

Our observations are in line with prior reports. For instance, Hulens et al. (2001) found that obese subjects use a greater fraction of their VO_2max at AT, significantly reducing their aerobic reserve (Hulens et al., 2001). Despite not including a control group, the relative intensities here (AT at 54% Wmax) mirror those findings, showing that these individuals operate closer to their physiological limits even during moderate effort, leading to early fatigue.

Clinically, these data underscore the need for tailored interventions. Our findings emphasize that a simple measure of peak VO_2 alone is insufficient. The abnormal RER kinetics observed in several of our cases point to an underlying metabolic inflexibility missed by standard exercise tests. We therefore recommend that comprehensive CPET with full gas exchange analysis be considered for risk stratification in this population. Beyond weight management, the goal is to address the compounding physiological deficits of aging and menopause, such as

the decline in mitochondrial function exacerbated by estrogen loss. In line with the guidelines of the ACSM and the American Heart Association (Lavie et al., 2015), progressive aerobic training (starting ~40–50% of VO_2max) and resistance training should be prescribed as a form of physiological therapy (Westcott, 2012) to increase mitochondrial biogenesis (Larsen et al., 2011), shift muscle fiber phenotype toward more oxidative types (Andersen & Henriksson, 1977), and raise the lactate threshold. Nutritional strategies (caloric deficit, anti-inflammatory diet) are equally important to reduce visceral fat and improve metabolic profile (Hruby & Hu, 2015; Lavie et al., 2009). Together, these central strategies can increase stroke volume and O_2 extraction, breaking the vicious cycle of sedentary behavior, obesity, and functional decline.

Furthermore, only through comprehensive, multidisciplinary management can long-term adherence be encouraged, patient empowerment be promoted, and sustainable clinical outcomes be achieved. These results strongly support prescribing structured aerobic and resistance training as physiological therapy to restore metabolic flexibility, improve mitochondrial function, and enhance quality of life (Lavie et al., 2015; Liguori & Medicine, 2022). Early recognition of this cardiometabolic drift can prompt interventions to prevent further decline and reduce long-term cardiovascular risk.

Limitations

This study is limited by its small sample (n=9), which restricts statistical power and generalizability. No lean control group or mechanistic biomarkers (e.g. lactate levels, inflammatory markers) were measured. Furthermore, we did not collect detailed data on comorbidities or medications that could have influenced the results. However, the detailed gas-exchange and HR data provide a strong pathophysiological basis. Future studies should involve larger cohorts, include biochemical and inflammatory markers, and track longitudinal responses to interventions to define clear operational guidelines for this population.

Conclusion

In conclusion, this case series provides a detailed physiological portrait of severe deconditioning in obese postmenopausal women, characterized by a triad of reduced cardiovascular reserve, metabolic inflexibility, and autonomic dysfunction. Peak VO_2, W_{max}, and chronotropic response were all markedly reduced, while RER and VCO_2 remained abnormally high into recovery. The significant inter-individual variability highlights the value of comprehensive CPET in unmasking the specific pathophysiological drivers of exercise intolerance to guide personalized interventions. Therefore, these findings emphasize that comprehensive CPET, including assessment of aerobic capacity and autonomic recovery, should be an integral part of clinical monitoring to identify at-risk individuals and implement timely preventive strategies.

Acknowledgments The authors would like to acknowledge all patients for their participation in this study.

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Cite this article as: Pusceddu, M., Balaghi Inaloo, F. (2026). Mechanisms of Exercise Limitation during Incremental Testing in Postmenopausal Obesity: A Cardiopulmonary Case Series. *Central European Journal of Sport Sciences and Medicine*, *1*(53), 17–25. https://doi.org/10.18276/cej.2026.1-02