

# HEMODYNAMIC AND STRUCTURAL ALTERATIONS OF THE CARDIOVASCULAR SYSTEM IN PATIENTS WITH MORBID OBESITY: A LITERATURE REVIEW

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## ABSTRACT

**Introduction:** Obesity, defined as a Body Mass Index (BMI) equal to or greater than 30, is a critical factor for cardiovascular health, manifesting itself in three distinct degrees. **Objective:** The objective of this review was to explore the cardiovascular changes associated with obesity and their implications for health. **Method:** Using databases such as PubMed and the Virtual Health Library (VSL), articles published between 2019 and 2024 were selected, focusing on "cardiovascular disease," "obesity," and "hemodynamics." The method involved filtering relevant studies, resulting in 11 articles addressing relevant issues regarding the cardiovascular effects of obesity. **Results:** The results showed that obesity induces significant hemodynamic changes, such as left ventricular remodeling and right ventricular dysfunction, and contributes to chronic inflammation and cardiac fibrosis. It was also observed that, paradoxically, individuals with moderate obesity may have a better cardiovascular prognosis compared to normal-weight individuals with heart failure. **Conclusion:** In conclusion, a multifaceted approach to the treatment of obesity, including lifestyle changes, pharmacological therapies, and bariatric surgery, may potentially reverse or mitigate the adverse cardiovascular changes associated with this condition.

**Keywords:** obesity; cardiovascular disease; hemodynamics.

## INTRODUCTION

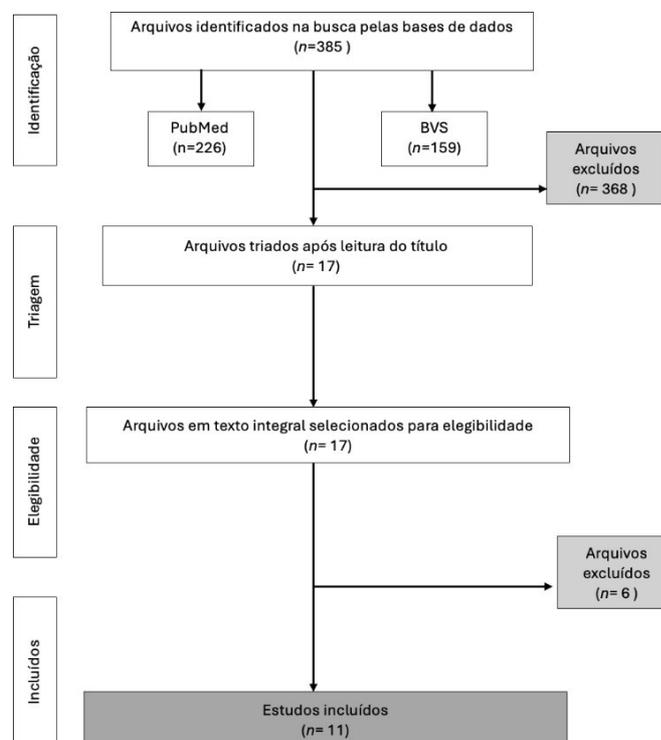
Obesity is characterized by a Body Mass Index (BMI) equal to or above 30, with excessive accumulation of body fat<sup>1</sup>. Furthermore, it can be classified into three degrees: degree I (moderate excess weight) when the BMI is between 30 and 34.9; grade II (mild or moderate obesity) with a BMI between 35 and 39.9; and grade III (morbid obesity) when the BMI exceeds 40<sup>2</sup>. Undeniably, excess adiposity causes damage, especially to body homeostasis, significantly impairing the cardiovascular system in both structural and functional terms<sup>3</sup>.

## METHODOLOGY

In this expanded review, we used the PubMed and Virtual Health Library (VHL) electronic databases with the DECS (Health Sciences Descriptors) in English, “cardiovascular disease,” “obesity,” and “hemodynamic,” with the Boolean “AND” between the descriptors. In addition, the following inclusion filters used were articles in English, Portuguese, and Spanish, available free of charge online and published in the last 5 years (2019-2024). Articles that did not address the topic under investigation were also excluded.

Figure 1 shows the methodological flowchart. First, articles that did not adhere to the topics related to the review in terms of their title and/or abstract were excluded. Next, those that did not present alternatives to the guiding question were excluded. After applying the before mentioned filters, a total of 11 articles in English were found that answered the guiding question, including original articles, cohort studies, case-control studies, and systematic reviews.

**Figure 1.** Methodological flowchart



Source: Own production, 2024.

Note: Virtual Health Library (VHL)

## RESULTS

Obesity is measured by established anthropometric values, such as abdominal circumference, waist circumference, and BMI<sup>4</sup>. Unfortunately, high adipose tissue content interferes with the interpretation of possible cardiovascular changes in tests, such as echocardiographic parameters and natriuretic peptide levels. Non-invasive assessment methods include Doppler and echocardiogram<sup>5</sup>.

In simple terms, three main changes can be observed in obese individuals: neurohormonal effects (increased activation of the sympathetic nervous system with sodium retention), inflammatory effects (increased inflammatory cytokines produced by adipose tissue), and plasma volume expansion, which causes a decrease in arterial resistance<sup>6</sup>. Some hemodynamic changes are observed in obese individuals, such as concentric remodeling of the left ventricle, increased metabolic demand and cardiac effort, and dilation of the heart chambers<sup>7</sup>.

At an immunological level, obesity is considered a chronic inflammation due to the high production of cytokines by adipocytes. With the increase in inflammatory mediators, cardiomyocyte hypertrophy occurs with the proliferation of myofibroblasts, leading to cardiac fibrosis, which leads to heart failure with preserved ejection fraction, atrial and ventricular tachyarrhythmias<sup>8</sup>. It is important to note that central organs are the main targets of blood pressure, such as the heart and brain<sup>3</sup>.

Obesity is associated with right ventricular dysfunction, as proven by invasive measures that investigate hemodynamic changes<sup>9</sup>. Some factors that drive these changes are hyperactivation of the sympathetic system, remodeling of arterial compliance, increased microcirculatory resistance, and increased arterial effort<sup>10</sup>. In addition, natriuretic peptides, protective factors that cause natriuresis and cardiac vasodilation, are decreased in obesity<sup>11</sup>.

Obesity has a multifactorial impact on the body, affecting sleep quality and leading to the development of sleep apnea syndrome. This syndrome affects the cardiovascular system by promoting nocturnal hypertension, stress on the heart walls during apnea, and increased inflammatory cytokines, triggering myocardial fibrosis<sup>12</sup>.

The discovery of the obesity paradox came as a surprise to the scientific community, which had previously correlated obesity only with harmful effects. In other

words, patients with moderate obesity did not experience a worsening of cardiorespiratory function, but rather an improvement. Overweight and grade I obese patients with heart failure had a better prognosis compared to normal weight or underweight patients with the same disease, as they had increased muscle mass and improved cardiorespiratory capacity<sup>13</sup>.

## **CONCLUSION**

In summary, it is clear that cardiovascular changes in individuals with severe obesity can be reversed through physical activity, nutrition, emotional health, pharmacological therapies, or bariatric surgery.

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## **REFERENCES**

1. Wanderley EN, Ferreira VA. Obesity: a plural perspective. Collective Health Science, 2010.
2. Ministry of Health [<https://bvsms.saude.gov.br/11-10-dia-nacional-de-prevencao-daobesidade/#:~:text=The%20severity%20of%20obesity%20is,in%20which%20BMI%20exceeds%2040>]. 11/10 – National Obesity Prevention Day [accessed on 11 Sep 2024]. Available at: <https://bvsms.saude.gov.br>.

3. Bittencourt JC, Scheinbein GHA, Junior WCO, Bassi RL, Moura LB, Correa ALD, *et al.* Arterial stiffness indices, pulse wave velocity and central systolic blood pressure, are able to discriminate between obese and non-obese children. *European Journal of Pediatrics*, 2023.
4. Talib A, Roebroek YGM, Paulus GF, Loo KV, Winkes B, Bouvy ND, *et al.* Left Ventricular Geometrical Changes in Severely Obese Adolescents: Prevalence, Determinants, and Clinical Implications. *Pediatric Cardiology*, 2021.
5. Gutiérrez-Cuevas J, Sandoval-Rodríguez A, Meza-Ríos A, Monroy-Ramírez HC, Galicia-Moreno M, García-Bañuelos J, *et al.* Molecular Mechanisms of Obesity-Linked Cardiac Dysfunction: An Up-Date on Current Knowledge. *Cells*, 2021.
6. Harada T, Obokata M. Obesity-Related Heart Failure with Preserved Ejection Fraction Pathophysiology, Diagnosis, and Potential Therapies. *Heart Failure Clin*, 2020.
7. Aryee EK, Ozkan B, Ndumele CE. Heart Failure and Obesity: The Latest Pandemic. *Progress in Cardiovascular Diseases*, 2023.
8. Preda A, Carbone F, Tirandi A, Montecucco F, Liberale L. Obesity phenotypes and cardiovascular risk: From pathophysiology to clinical management. *Reviews in Endocrine and Metabolic Disorders*, 2023.
9. Ma JI, Zern EK, Parekh JK, Owunna N, Jiang N, Wang D, *et al.* Obesity Modifies Clinical Outcomes of Right Ventricular Dysfunction. *Circ Heart Fail*, 2023.
10. Mandry D, Girerd N, Lamiral Z, Huttin O, Filippetti L, Micard E, *et al.* Arterial and Cardiac Remodeling Associated With Extra Weight Gain in an Isolated Abdominal Obesity Cohort. *Frontiers in Cardiovascular Medicine*, 2021.
11. Ozkan B, Ndumele CE. Exploring the Mechanistic Link Between Obesity and Heart Failure. *Current Diabetes Reports*, 2023.
12. Chen HHL, Bhat A, Gan GCH, Khanna S, Ahlenstiel G, Negishi K, *et al.* The impact of body mass index on cardiac structure and function in a cohort of obese patients without traditional cardiovascular risk factors. *International Journal of Cardiology Cardiovascular Risk and Prevention*, 2023.
13. Carbone S, Canada JM, Billingsley HE, Siddiqui MS, Elagizi A, Lavie CJ. Obesity paradox in cardiovascular disease: where do we stand? *Vascular Health and Risk Management*, 2019.