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Italian Association of Hospital Cardiologists position paper—obesity in adults: a clinical primer

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Obesity is a chronic and relapsing disease characterized by the interaction between individual predispositions and an obesogenic environment. Recent advances in understanding the mechanisms of energetic homoeostasis paved the way to more effective therapeutic approaches compared with traditional treatments. Since obesity is a complex disease, it necessitates a multi-disciplinary approach whose implementation remains challenging. Nonetheless, emerging pharmacological interventions appear promising. Currently, therapeutic success is discreet in the short term but often fails to maintain long-term weight loss due to a high likelihood of weight

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regain. Cardiologists play a key role in managing patients with obesity, yet often lack familiarity with its comprehensive management. The aim of this document is to summarize knowledge to consolidate essential knowledge for clinicians to effectively treat patients living with obesity. The paper emphasizes the pivotal role of a strong patient–clinician relationship in navigating successful treatment. We analyse the criteria commonly used to diagnose obesity and point out the strengths and limitations of different criteria. Furthermore, we discuss the role of obesiologists and the contributions of cardiologists. In addition, we detail key components of effective therapeutic strategies, including educational aspects and pharmacological options.

Introduction

Obesity is a chronic, complex, and multi-factorial disease resulting from an excessive accumulation of adipose tissue that causes damage to health.[1]

The disease is spreading in all countries in a pandemic manner.[2]

Currently, around 59% of the European population and one in three children are overweight or affected by obesity.[3]

The prevalence of obesity worldwide has nearly tripled since 1975.[1]

In Italy, 35% of the population is overweight, and more than 9.8% of adults are obese.[4] Obesity represents a significant concern in terms of prevalence and health implications, so much so that the World Health Organization's (WHO) European Work Programme 2020–25—'United Action for Better Health' has prioritized it as a key area for sustainable development.[5]

Enhancing skills to assist patients living with overweight and obesity is imperative for several reasons. Firstly, overweight and obesity are linked with an increased likelihood of illness,[6] higher mortality rates,[7] and reduced life expectancy.[8] Secondly, individuals living with obesity often face stigma and prejudice, even amongst healthcare professionals,[9,10] which adversely affect the quality of care they receive. Furthermore, weight stigma is significantly and negatively correlated with both mental and physical quality of life.[11]

Cardiologists are deeply involved in the treatment of obesity. Often, patients with obesity consult cardiologists due to numerous comorbidities associated with this condition. More than two-thirds of deaths linked to high body mass index (BMI) are due to cardiovascular diseases.[6,7] The cardiovascular risk profile is one of the most crucial factors to be quantified in order to understand the additional risk due to obesity and how much each patient can benefit from losing fat mass.[12] Since most anthropometric measures can assess cardiovascular risk with an accuracy of 60–70%,[13,14] the judgement of an experienced clinician becomes vital in guiding the patient towards effective medical treatment.

The purpose of this paper is to provide a practical guide specifically for cardiologists, presenting key information derived from scientific evidence and outlining the most appropriate behaviours and resources to be used from initial patient contact to therapy prescription.

Patient engagement

The initial engagement between the clinician and his patient is a pivotal step in the treatment continuum for patients living with obesity. This is because all subsequent interactions might be handed over to other healthcare professionals, such as nurses and dieticians, or to colleagues specialized in obesity management (as detailed in the section on the initial obesity consultation). In contrast, the initial patient relationship cannot be delegated; if not well-established, it can potentially lead to harming the patient's trust in healthcare or causing a sense of alienation, reinforcing feelings of failure, prejudice, and negative judgment that the patient has likely already experienced in the past. In clinical practice, every first 'contact' with a patient, regardless of the clinical setting—whether during hospitalization or an outpatient visit—should be seen as an opportunity to initiate a personalized therapeutic journey. The first key principle outlined in the guidelines is that not all patients living with obesity are ready to address this issue with their physician.[15] Therefore, discretion, sensitivity, and tact are required during this initial engagement, offering help in a respectful and open-minded manner. What patients most frequently seek when engaging with a physician is a flexible and non-judgmental support.[16]

Figure 1 illustrates a hypothetical approach that could be implemented at the time of the first patient contact.

Table 1 outlines a series of questions aimed at fostering a motivational approach. Some of them can be used to motivate the patient and reassure him. In this way, the patient, if willing, can begin a treatment journey that is free from stigma and prejudice.[17]

Table 2 indicates some precautions to implement when communicating with the patient.

In most cases, patients interact with cardiologists for reasons unrelated to obesity. It seems appropriate to confine the focus to discerning if the patient wants to discuss and address the issue, and, accordingly, to provide motivational support and conduct screenings regarding the present phenotype. This entails the collection of data such as weight, height, and waist circumference (WC) to calculate the BMI and, potentially, the waist–hip ratio (WHR) and/or waist–height ratio (WHtR).

This data gathering should be as routine as measuring blood pressure and heart rate, without emphasizing any specific interest or implying that obesity and its associated measurements have more significance than the reason for the patient's clinic visit.

The rest of the process should be postponed to a specific setting. This is done with the aim on one hand to conserve the necessary time to address the health issue for which the patient has presented and, on the other hand, to

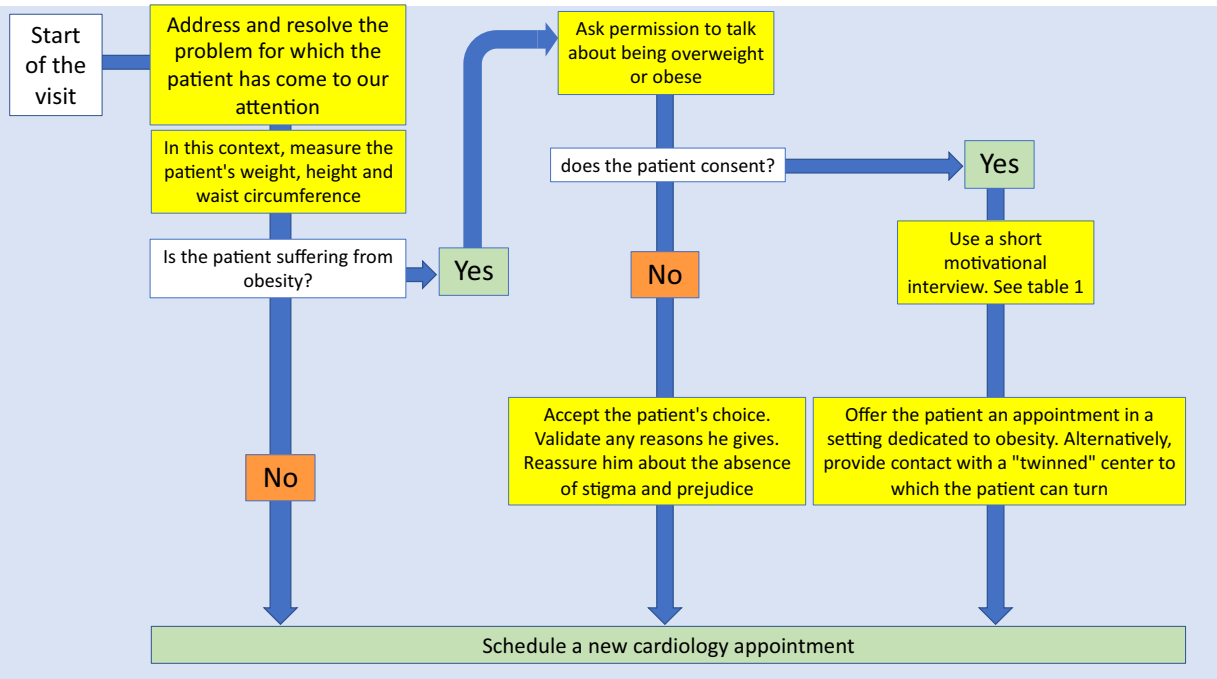

Figure 1 Flowchart for a hypothetical pathway at the first contact with a patient living with obesity. Flowchart for the first contact with the patient suffering from obesity.

Table 1 Useful questions for a motivational interviewing[17]

How do you feel about your weight currently?
How important is it for you to change your behaviour now?
How confident are you that you can change your lifestyle?
Are there now any stressful events in your life that might be an obstacle to change?
If you decide to change your lifestyle, what would you change?
What would be two or three benefits you could get from it?
If you decide to make this change, how would you do it?
How has our discussion helped you move forward?
How do you see the sequel?
What does 'healthy eating' mean to you?
Do you feel sensations of hunger and satiety?
Do you eat when you are bored, stressed, or sad?
What is the best time of day (or evening) to be physically active?
What does physical activity mean to you?
What type of physical activity do you enjoy now or have enjoyed in the past?
What are some reasonable goals you could set regarding your weight?
What kind of changes would you be willing to start with?
What kind of help would you like to receive in order to achieve your goals?

List of questions that can be used to test and strengthen the patient's motivation. Modified from Schutz *et al.*[17]

allocate more time for a specific obesity-related consultation. If a patient presents at the clinic for hypertension or dyslipidaemia, diverting full attention to the issue of obesity, while clinically legitimate, tends to reinforce the well-known health bias that the patient has likely previously experienced, which leads to mistrust of conventional treatment paths. Instead, a professional response should be offered for the primary health issue presented by the patient, and, only afterwards, if the patient desires, a specific appointment with a dedicated clinic or a referral to a collaborating centre should be provided.

Diagnosis

The diagnosis of obesity is typically established using the BMI, calculated as the ratio between weight in kilograms and the square of height in meters.

Table 3 lists the four main categories derived from the BMI according to WHO criteria, along with an additional category pertaining to under-nutrition.

Within the same table, additional anthropometric criteria are reported to enhance the quality of diagnosis and to refine the associated risk, in an effort to overcome the inherent limitations of the BMI.[19-21] This measure, in fact, does not provide any information regarding the tissue contributing to the overall patient weight,[22] and, at least theoretically, significant muscular hypertrophy, as occurs in some high-level athletes, could lead to achieving BMI values classified under overweight or obesity categories. Moreover, the BMI fails to provide information on the distribution of adipose tissue, while it is well established that the abdominal and visceral adipose tissue (VAT) localization is itself a significant cardiovascular risk factor.[23,24]

Therefore, it is possible for a patient to have a normal BMI yet an excess of VAT. Conversely, not all patients with elevated BMI due to increased adipose tissue necessarily

Table 2 Fundamental points in the relationship with the patient[17]

1. **Sensitivity and non-judgment.** Avoid making negative comments or making judgments about the patient's weight.
2. **Active listening.** Physician should listen carefully to the patient's concerns regarding the reasons for the visit. Even if obesity is not the focus of the patient's expressed concerns, physician should be open to discussion and providing information on weight management if appropriate and relevant for the management of the overall clinical status.
3. **Respecting the patient's priorities.** Physician should focus on resolving the medical conditions or concerns expressed by the patient. Weight loss could be a long-term goal.
4. **Inclusion of obesity in assessments.** Focus the visit on the patient's concerns, and collect height, weight, and waist circumference, without focusing exclusively on this aspect.
5. **Empathic communication.** The doctor should adopt empathetic and open communication and encourage the patient to express all his concerns. The patient must feel listened to and understood.
6. **Semantics.** It is important to use the expression a patient 'with obesity' rather than an 'obese' patient; this approach recognizes the individual first rather than its condition, aligning with contemporary standards of respectful and non-stigmatizing communication.
7. **Complexity.** Recognize that obesity has a complex and multi-factorial aetiology, caused by both individual and external factors that are not always under the patient's control.

General criteria to refer to when relating to a patient with obesity.

exhibit clinically significant metabolic dysfunctions. This specific condition, termed metabolically healthy obesity, is associated with good functional capacity and in the absence of risk factors and is generally linked with normal or only moderately increased VAT.[19] The actual existence of this category is debated in the literature, potentially representing a transitional phenotype towards metabolically unhealthy obesity.[25]

The measurement that most significantly complements the informational value of BMI is the WC,[26] an indirect yet directly correlated indicator to VAT. Waist circumference is not a direct measure of VAT as it also includes subcutaneous abdominal tissue; however, there is unanimous consensus in the literature that an increase in WC is associated with a heightened risk of developing various pathologies, including cardiovascular diseases, type 2 diabetes, arterial hypertension, and metabolic syndrome.[17,18,24,26–30] Unfortunately, there is no universal agreement on the precise location for the measurement of WC. It is possible to measure it at different abdominal positions, with these variations being significant predominantly in the realm of clinical research.[26]

The WHO, the International Diabetes Federation, and the World Obesity Federation recommend measuring WC at the midpoint between the lower edge of the last palpable rib and the upper border of the iliac crest.[26] The US National Institutes of Health and the National Heart, Lung, and Blood Institute recommend measurement just above the iliac crests.[26] Some studies and regional guidelines have used alternative measurement sites, such as the level of the navel or the point of maximum circumference, but these are not commonly recommended in major international guidelines.

The cut-off values for WC vary depending on the guidelines and reference populations, as different ethnic groups may have varying levels of risk associated with specific measurements of WC.[18] In the European population, WHO defines an increased cardio-metabolic risk for males when WC is $\geq$94 cm, with a further increased risk at values $\geq$102 cm. For females, the corresponding values for increased risk and very increased risk are $\geq$80 and $\geq$88 cm, respectively.[31]

In the pursuit of cut-off values with the best predictive value for increased cardio-metabolic risk, different WC values have also been proposed for different BMI classes.[20] This approach can undoubtedly be useful in increasing the specificity of the information and also adds more complexity in daily clinical practice.

Table 3 presents normal and pathological values for WC referenced to populations other than the European one. Knowledge of these values is significant, as it is not uncommon to encounter individuals from non-Caucasian populations in clinical practice. *Table 4* describes the correct method for measuring WC.

In conclusion, WC is a valuable measurement and clinically relevant of body fat distribution and gives significant additional information on the risk of obesity-linked diseases, especially in case of lower BMI values.[26] In patients with heart failure and preserved ejection fraction (HFpEF), central adiposity, measured with WC, is associated with a worse prognosis, while there is an inverse correlation between prognosis and BMI.[32] Waist circumference value is so important that it is considered a 'vital' parameter, which must be measured and recorded in each clinical examination, like heart rate and blood pressure.[26]

In a specialist context, other measures can be used for a more sophisticated evaluation of the phenotype in question and to evaluate in follow-up to which type of tissue any changes in weight should be ascribed.

The first obesiologic visit

Who is the specialist who deals with obesity?

A specialist traditionally defined and universally accepted by the scientific community does not exist, and the field is occupied by various professional figures, sometimes even non-medical ones. If we establish that obesity is a disease, the professional reference can only be a physician, which has acquired the necessary skills through an adequate training course. Usually, internists, endocrinologists, and specialists in clinical nutrition are the figures who most frequently dedicate themselves to this field, but any physician is entitled to do so. The general practitioner could be suitable to carry out this role, in cases of clinically non-advanced obesity, due to his knowledge of the patient and the family setting, his

Table 3 Anthropometric measurements necessary to define the patient's phenotype and health risk

			BMI-specific pathological WC values[18,19]	
		BMI (kg/m^2)[18,19]	M (cm)	F (cm)
Severely underweight		<16.5		
Underweight		<18.5		
Normal weight		≥18.5 e < 25	≥90	≥80
Overweight		≥25 e < 30	≥100	≥90
Obesity		>30		
	Class I	≥30 e < 35	≥110	≥105
	Class II	≥35 e < 40	≥125	≥115
Also defined as severe, extreme, massive, or morbid obesity.	Class III	≥40	≥125	≥115

WC limits beyond which an increase in health risk can be documented in different ethnic groups

	M (cm)	F (cm)
European Caucasian/USA/Middle Eastern Mediterranean[a]	≥94	≥80
European Caucasian/USA/Middle Eastern Mediterranean[b]	≥102	≥88
Central and South America[b]	≥94	≥90
Africa sub-Sahariana[b]	≥95	≥99
Sub-Saharan Africa[b]	≥80.5	≥81.5
Koreans[b]	≥90	≥85
Chinese[a]	≥83	≥81
Limits of normality for waist/hip ratio and waist/height ratio[20]		
WHR	0.87	0.78
WHtR	0.56	0.48

Classification was suggested by the World Health Organization of body weight in relation to height for Caucasians. In patients with Asian, Chinese, Middle Eastern, dark-skinned African, or Afro-Caribbean origins, Class II and III obesity is generally identified by reducing the limit value by 2.5 kg/m^2 as these individuals have a greater propensity to central obesity and their cardio-metabolic risk manifests itself at lower BMI values.

In these populations, overweight and obesity are defined, respectively, by BMI values greater than 23 and 27.4 kg/m^2.[8]

BMI, body mass index; F, female; M, male; USA, United States of America; WC, waist circumference; WHR, waist-hip ratio; WHtR, waist-height ratio.

[a]Increased cardiovascular risk

[b]Significantly increased cardiovascular risk.

Table 4 How to measure the waist circumference[20]

Patient should stand with feet close together, arms at their sides.

Utilize a flexible measuring tape, positioning it midway between the lower edge of the last palpable rib and the upper edge of the iliac crest or, alternatively, just above this. The same method should be employed consistently within each centre.

Ensure the tape is parallel to the floor and does not compress the skin.

Measurements should be taken during a normal exhalation.

Although differences in the result are documented depending on the location in which the abdominal circumference is measured, these differences are generally small (greater in women). Regardless of the method, between WC and death from all causes, cardiovascular death, cardiovascular disease, and type II diabetes, there is a correlation.[26] This association is more evident for low BMI values.[26]

ability to observe longitudinally, and the trust and credibility he holds. However, when acting as the attending physician, the family doctor cannot work in isolation; instead, he needs to be included in multi-disciplinary team. This team plays a crucial role in assisting with patient evaluation and the development of therapeutic plans.

Physicians (internists, cardiologists, endocrinologists, diabetologists, clinical nutritionists, pulmonologists, psychiatrists, and other specialists), dieticians, clinical psychologists, physiotherapists, nurses, and social workers must form a team, each providing their own specific skills under the leadership of a recognized team leader.

Table 5 outlines the general criteria that must be adhered to in the treatment of obesity. These criteria need to be explicitly communicated from the onset of patient's care and treatment journey.

Table 6 shows the main causes that cause or perpetuate excess dysfunctional fatty tissue.

The obesiologic examination should pinpoint the body composition. This latter aspect, although already assumed to be performed with the use of BMI and WC, is better defined with the plicometric survey, the instrumental survey of percentage fat mass (%BF), or other derived measures.

Table 7 shows the main measures that can be used in the context of a specialist visit to define in detail the patient's phenotype and, in follow-up, to understand in case of weight change to which tissue this is attributable.

Table 5 General criteria and tools for obesiological medical examinations

Identify and support the patient's motivation for addressing obesity, expanding upon this to strengthen their engagement in the treatment process.
Document the patient's weight at various life stages, from birth to the present.
Clarify patient expectations
Evaluate previous effectiveness and outcomes of any previous obesity treatment.
Evaluate the role of patient's social environment.
Screen for signs of eating disorders, depression, anxiety, stress, and poor sleep quality.
Avoid any form of stigma from healthcare providers.
Employ a multi-disciplinary approach.
Encourage and support shifts towards healthy lifestyles regardless of the immediate result they produce.
Examine internal and external factors that cause or perpetuate obesity (see *Table 6*).
Define the patient's phenotype in more detail (see *Table 7*), possibly carrying out including tests to quantify fat mass.
Monitor weight losses by evaluating the tissue reduced in greater extent but emphasize that this is just one aspect of the treatment goals.
Collect specialized medical history.
Perform a specialized physical examination.
Identify and address comorbidities that have not been already managed elsewhere.

General criteria of behaviour to be observed during a visit aimed at treating a patient living with obesity.

Table 6 Main causes of the development or maintenance of obesity[33-35]

Endogenous factors	Exogenous factors
Genetic predisposition	Social pressure
Specific genetic disease	Nutrition (quality and quantity)
• Associated with syndromes (Bardet-Biedl, Alström, Prader-Willi)	Sedentary lifestyle
• Not associated with syndrome	Sleep duration
Epigenetic causes	Smoking cessation
Endocrinological factors/ comorbidities	Chronic stress
Psychological factors/comorbidities	Medications
Psychiatric factors/comorbidities	Social and family context
Specific eating disorders	

Factors to consider at the time of the obese examination in order to most effectively direct treatment.

Indeed, it is necessary to recognize that beyond the classic categorization into four BMI-based classes, it is possible to identify other forms of adipose accumulation with distinct clinical-prognostic impacts. For instance, there are patients who have a 'normal' weight and BMI, but an increased WC, or more specifically, increased abdominal fat mass. At a glance, these patients might erroneously appear free of increased cardiovascular risk.[37]

Other individuals, despite having a BMI that categorizes them as with obesity or overweight, exhibit normal functional capacity and lack of conditions such as hypertension, diabetes, or metabolic syndrome. These patients can be defined, therefore, as having 'metabolically healthy obesity'. This condition could likely be considered a transitional phase towards metabolically dysfunctional obesity[38,39] and has contributed, at least in part, to the genesis of the 'obesity paradox' phenomenon.

The 'obesity paradox' is an intriguing observation where obesity, typically linked with increased mortality and disease risk, appears to offer some protective benefits in certain conditions. Specifically, patients who are overweight or have first-degree obesity, when afflicted with various diseases, might have a better prognosis compared to their normal-weight counterparts.[22]

This finding, evident across various medical disciplines, has no definitive explanation although it may stem from sampling errors or from the inherent limitations of BMI, which fails to distinguish between phenotypes at higher or lower cardio-metabolic risk.[40] A further important category that needs to acknowledge sarcopenic obesity.[41] Sarcopenic obesity is characterized by reduced in muscle mass and strength or muscle performance, associated with an increase in fat mass.

A critical diagnostic aspect is to identify the loss of lean mass associated with strength deficits that occur along with the increase in fat mass. The concomitant presence of sarcopenia and obesity identifies individuals at increased risk for complications and is a distinct category from both obesity and sarcopenia as isolated entities.[41] The use of instruments to measure the various tissue components of total body mass is essential to make this part of patient the evaluation of the patient living with obesity truly specialized.

In daily clinical practice, the most frequently used tools are as follows:

(1) Bioelectrical impedance analysis. It is based on the principle that different tissues in the body offer different resistance to the passage of an electric current due to their different resistive and capacitive properties.
(2) Air displacement plethysmography. It determines body density by measuring body volume through air displacement inside a sealed chamber and relating the obtained value to weight. By knowing the body density, fat mass can be determined.
(3) Dual-energy X-ray absorptiometry (DXA). Primarily used to measure bone mineral density and assess fracture risk. In addition to bone density, DXA can also be used to determine body composition, measuring fat and lean mass. It employs X-rays at two different energy levels to analyse and distinguish bone, fat, and muscle tissue. As X-rays pass through the body, a varying amount of energy is absorbed depending on the density and type of tissue. Using two different energy levels, the device can accurately calculate tissue composition.

Table 7 Measures to assess adiposity[22,36]

Measurement	International acronym	Measured/calculated
Body mass, kg	BM	Measured (kg)
Body mass index, kg/m^2	BMI	Weight in kg divided by height (h) in square meters (m2)
Fat body mass, kg	FM	Instrumental measurement (BIA, DEXA, MRI, and CT)
Fat body mass index, kg/m^2	FMI	FM/h^2
Fat free mass, kg	FFM	Instrumental measurement (BIA, DEXA, MRI, and CT); calculated: BM-FM
Percentage of fat body mass, %	%BF	FM $*$ 100/BM
Fat free mass index, kg/m^2	FFMI	FFM/h^2
Visceral adipose tissue, L, mL, kg, g, cm^2	VAT	Measured or estimated by anthropometric measurements; reference standard: DXA, MRI, and CT
Waist circumference, cm	WC	Measured
Thigh circumference, cm	TC	Measured
Neck circumference, cm	NC	Measured
Hip circumference, cm	HC	Measured
Total abdominal adipose tissue, cm^2	TAAT	Measured
Subcutaneous adipose tissue, cm^2	SAT	Measured or estimated by anthropometric measurements; reference standard: MRI and CT
Subcutaneous abdominal adipose tissue, cm^2	SAAT	Measured or estimated by anthropometric measurements; reference standard: MRI and CT
Waist–hip ratio	WHR	Calculated (WC/HC)
Waist–height ratio	WHtR	Calculated (WC/h)
Waist–thigh ratio	WTR	Calculated (WC/TC)

Key indicators for more detailed phenotype patients with obesity.
BIA, bioelectrical impedance analysis; CT, computed tomography; DEXA, dual-energy X-ray absorptiometry; MRI, magnetic resonance imaging.

While the reference standard for measuring lean and fat masses and their distribution within the body is nuclear magnetic resonance imaging and computed axial tomography (CT), these advanced techniques are mainly used in scientific research. Further details on body composition assessment tools are described in the work of Borga *et al.*[36]

During obesiologic evaluations, it will be necessary to define whether the patient's health needs extend to psychological or social treatment. Generally, with the patient's consent, an evaluation in these areas appears mandatory for all patients with obesity but especially for all those with a BMI of 35 kg/m^2 or more.

Cardiologist's role in the management of the patient living with obesity

Cardiovascular risk assessment

The relationship between BMI and death risk is observed to follow a J-shaped pattern, with lower and higher BMI categories both associated with increased risk compared with intermediate categories.[42] This kind of relationship has been consistent in all populations studied in primary prevention settings. In secondary prevention settings, populations with overweight or mild obesity may, in some cases, not be disadvantaged or even have a slight prognostic advantage[43] over patients with lower BMI, although there is still insufficient clarity on this aspect.[44] On the other hand, it should be highlighted that the association between higher mortality and lower BMI categories could be ascribed to the pathological conditions causing low BMI and not to the low BMI *per se*. Furthermore, although the excess mortality associated with overweight is relatively small, since many overweight patients tend to gain weight over time in this group of patients, the risk of morbidity and mortality may also increase over time. Usually, the cardiovascular risk estimation is formalized using one of the risk scores recommended by scientific societies. *Table 8* shows the main methods proposed for global cardiovascular risk calculation by the European Society of Cardiology and, as regards Italy, by the Istituto Superiore di Sanità.[45-52]

European guidelines recommend the use of SCORE-2 (for patients aged between 40 and 69 years) or SCORE2-OP (for patients aged ≥70 years) in primary prevention and in the absence of type II diabetes or special risk conditions such as, for example, familial hypercholesterolaemia. It is noteworthy that most of the recommended scores use weight to calculate global cardiovascular risk. Only LIFE-CVD[53] and the DIAL[54] include BMI, with the latter currently under review to correct a systematic enrolment error. The LIFE-CVD score tends probably to underestimate risk. In fact, if we insert into the model a European patient, aged 55, with a total cholesterol of 200 mg/dL, an HDL cholesterol of 55 mg/dL and an LDL of 115 mg/dL, a systolic blood pressure of 135 mmHg, and a BMI of 24 kg/m^2, we obtain a probability of having a myocardial infarction, stroke, or cardiovascular death of 3.6%. If the patient has the same characteristics but a BMI of 41 kg/m^2, we will obtain a probability for the same outcome of 4.4% with an absolute increase of 0.8% that corresponds to an increase in the risk of approximately 22%, while the expected increase should be greater than 40% according to other sources.[55,56]

Although adiposity, particularly visceral, undoubtedly increases the likelihood of disease and death, assessing

Table 8 Main cardiovascular risk scores

Name	Rischio calcolato	Popolazione testata
SMART[45]	Absolute, composite: myocardial infarction, stroke, cardiovascular death.	Primary prevention
ADVANCE[46]	Absolute, composite: non-fatal myocardial infarction, non-fatal stroke, cardiovascular death.	Type 2 diabetes
SCORE[47] (updated in SCORE2[48] and Score2 OP[49])	Absolute: cardiovascular death	Primary prevention without diabetes. European population.
PCEs (ASCVD) Multiple Events Prediction[50]	Absolute, composite: first atherosclerotic cardiovascular event; first myocardial infarction, first stroke, new onset heart failure, al causes mortality.	Primary prevention. US population. Non-Hispanic African Americans and Caucasians aged 40 to 79 years
SCORE2[48]	Absolute, composite: non-fatal myocardial infarction, non-fatal stroke, cardiovascular death.	Primary prevention without diabetes aged 40–69 years
SCORE2-OP[49]	Absolute, composite: non-fatal myocardial infarction, non-fatal stroke, cardiovascular death.	Primary prevention without type 2diabetes. European population aged ≥70 years
SCORE2-DIABETES[51]	Absolute, composite: non-fatal myocardial infarction, non-fatal stroke, cardiovascular death.	Type 2 diabetes. Primary prevention, aged 40–69 years
Progetto Cuore[52]	Absolute, composite: first major cardiovascular event including myocardial infarction established or possible, coronary death, sudden death, stroke, and revascularization procedures.	Primary prevention. Italian population, aged 35–69 years.

PCEs, pooled cohort equations; SCORE, Systematic Coronary Risk Estimation; SMART, Secondary Manifestations of ARTerial disease.

related cardiovascular risk is complex due to confounding elements from associated comorbidities. In other words, it is not clear how much the increased cardiovascular risk is linked to dysfunctional excess adipose tissue, with altered secretion of substances with endocrine effects, altered immune status, and consequent chronic inflammatory processes. It is also unclear how much is a result of the increased prevalence of comorbidities such as hypertension, diabetes, and hypercholesterolaemia that inevitably accompany obesity. On the other hand, concerning HFpEF, there is growing evidence regarding the causal role of obesity that is mediated by its multiple deleterious effects on the structure, function, and metabolism of myocardial cells. This is in addition to its effects on other organs (lungs, muscles, and liver) mediated by altered haemodynamic load, altered autonomic nervous system balance, and the aforementioned inflammatory and endocrine mechanisms.[32,57]

Taking into account what has been said, the assessment of the overall cardiovascular risk of a patient with obesity should consider at least the following aspects:

(1) If the patient has type II diabetes or confirmed atherosclerotic disease or familial hypercholesterolaemia, SCORE-2 and SCORE2-OP are unsuitable.
(2) The ADVANCE score, specific for patients with diabetes, should be used with caution as it not only does not include weight in the risk calculation but is also based on a study conducted more than 12 years ago. The study was designed to provide data on the balance between benefits and risks arising from intensive blood pressure lowering and glycaemic control therapy in high-risk diabetic patients, regardless of initial blood pressure or glucose levels. The follow-up was only 4 years, and the risk calculation was extrapolated to 10 years through a mathematical formula. Additionally, the landscape of cardiovascular drugs available at the time of the study has substantially changed compared with today.
(3) The SCORE2-diabetes does not consider BMI as a factor to establish the risk.
(4) All patients with a BMI above 35 kg/m^2 should be considered as having an increased cardiovascular and metabolic risk.
(5) In patients with a BMI below 35 kg/m^2, the assessment of visceral adiposity becomes crucial in defining cardiovascular risk. Waist circumference, abdominal ultrasound to detect fat deposits in abdominal tissues and particularly in the liver, such as in non-alcoholic fatty liver disease, WHR, and WHtR are simple tools to define this aspect.
(6) The patient's psychological conditions, internalization of stigma, and past medical history should be known and considered to complete the patient's risk assessment. It has been observed that patients with internalized stigma have worse physical conditions with similar metabolic parameters and weight.[11] The use of a clinical staging tool like the Edmonton score[58] appears particularly useful for understanding the extent of obesity's impact on the patient's overall risk. Although the Edmonton score is typically a multi-parametric tool that derives from the collaboration of various professionals, the cardiologists should be familiar with the results of this score as they have a significant impact on risk assessment and, consequently, on defining

therapeutic targets and the temporal sequence in which these should be achieved.

The instrumental cardiological assessment of a patient living with obesity

The clinical and instrumental evaluation of patients living with obesity presents some differences and additional challenges compared with patients with normal weight or only overweight. Normal electrocardiographic criteria, for example, may be different, and electrocardiographic differences can lead to sensitivity and specificity values that are also different.[59] Provocative testing on a cycle ergometer or treadmill may not be satisfactory due to the difficulty of reaching the theoretically predicted maximum age-related heart rate due to chronotropic incompetence,[60] orthopaedic limitations, or pulmonary dysfunction described in these patients,[61] which could result in functional limitations.

Resting or stress echocardiography may prove unsatisfactory due to high acoustic impedance, as well as single-photon emission CT and coronary CT angiography,[59] which have lower sensitivity compared with patients with normal weight. The assessment of coronary calcium may also suffer from the same limitations.[62]

Magnetic resonance imaging with pharmacological stress and positron emission tomography with rubidium, if available, pose fewer problems and are, therefore, when possible, the preferred techniques in patients with obesity.[63] Overall, judging the extent of the patient's global cardiovascular risk with obesity and the benefits achievable with weight loss appears much more difficult and less standardized. It is supported by less evidence and is more subject to individual evaluation rather than extrapolation from results obtained from populations.

The therapy for obesity

Obesity therapy is based on eight fundamental principles:

(1) Therapeutic education.
(2) Behavioural advice
(3) Diet therapy.
(4) Physical activity.
(5) Psychological support, cognitive–behavioural psychotherapy.
(6) Social and family support.
(7) Pharmacotherapy.
   a. Specific for excess fat.
   b. Aimed at cardiovascular risk factors associated with excess fat (hypertension, type II diabetes, hypercholesterolaemia, and hyperuricaemia).
(8) Bariatric surgery.

None of these tools alone can cure obesity. At most, it is possible to manage it and achieve success that is often transient. The integration of different available treatments through relevant professional figures can produce the best results, which currently are satisfying in the short term and unsatisfactory in the medium to long term. It is well known that 50–80% of weight lost after an effective intervention is regained after 2 and 5 years, respectively, regardless of the methodology used to promote loss.[64] Even bariatric surgery is not exempted from this issue.[65]

The purpose of therapeutic treatment is not to modify the BMI, as it is a measure of body size, not health status. Therapeutic management should involve a non-stigmatizing and non-prejudicial approach, requiring a preliminary personalized diagnostic assessment that takes into account social, medical, functional health, and mental aspects. It should guide patients towards a change that allows them to achieve the best possible health conditions.

Therapeutic education

The journey that a chronic patient is forced to take can never be adequately traversed without full awareness of their condition and its consequences. Simple information, the one-way transmission of data from the healthcare provider to the patient, while being a fundamental element of the care process, is not sufficient. To facilitate change, the data sent to the patient must be transformed into information by them and that information takes on a positive emotional value. This educational process can only be achieved through a two-way relationship in which the recipients process the message and present it back to the source to receive it again until they become fully aware of it.[66] This removes resistance and other forms of manipulation that may alter or negate its meaning. Only with this awareness, the patients can manage their behaviour in the countless situations that life will present, consistent with the health goals they have set, and with the serenity of someone who perceives that overcoming difficulties have a positive purpose and goal. This methodology has various execution methods, different types of involved operators, and different meeting settings. For these reasons, a detailed description of achievable processes is beyond our scope. Like all change processes, the patient will go through a series of stages and will be supported by a range of tools aimed at improving their sense of self-efficacy, empowerment, and resilience.

Behavioural advice

After the initiation of the preceding phase, efforts will be made to encourage behavioural changes. A study that pooled the results of 67 studies focusing on behavioural changes overseen by family doctors for weight loss observed a statistically significant difference in weight reduction between the control and treated groups, but the magnitude of the difference was modest (2.39 kg).[67] In the same review, a meta-analysis of 38 studies indicated that patients advised to make behavioural changes were 1.94 times more likely to lose 5% of their weight, with a number needed to treat of 8.[67] At approximately 12 and 18 months after the intervention, both treatment and control group exhibited weight regain, albeit less in the treatment group (average difference of 1.58 kg).[67] Support for lifestyle change is crucial for weight loss and a reduction in WC, but it is unlikely to be effective as the sole tool employed.

Dietary therapy

Evidence-based medical nutritional therapy should form the fundamental pillar of obesity treatment interventions and must be made available to all

Table 9 Available evidence on medical nutritional therapy[27,64,68-70]

Personalized prescription of medical nutrition therapy increases the likelihood of achieving positive changes in adiposity and metabolic factors compared with information provided by brochures.
The total amount of daily calories ingested should be appropriately correlated with the amount consumed daily to achieve a normal body weight.
Caloric restriction exceeding 500 calories per day must be justified for clinical reasons.
Excessive caloric restriction can pave the way for eating behaviour disorders.
The healthiness of a diet and the potential for weight loss are primarily determined by the total caloric intake and the relative balance of macronutrients, regardless of how food is distributed throughout the day.
There are no demonstrable long-term weight loss benefits in reducing specific macronutrients at the same total calorie level.
Clinical evidence supports the health benefits of the DASH, Mediterranean, and vegetarian diets.
Opt for plant-based proteins and lean proteins from marine sources, low in fat, from unprocessed food.
Favour plant-based food and vegetable fat.
Avoid drinks containing added sugars.
Prepare and cook food without adding salt.
Abstain from alcohol or consume it in limited quantities

This is a diet designed to reduce blood pressure but has shown to be a strategy effective to promote lifestyle changes. It was not devised as a weight loss diet, but in practice, it can enable it.

DASH, dietary approaches to stop hypertension.

individuals who live with overweight and obesity.[27] Implementing any intervention for obesity treatment without the simultaneous incorporation of dietary therapy is not advisable. While scientific literature presents diverse dietary approaches, differing in total caloric content, macronutrient percentages, and meal administration methods, the non-scientific, particularly social, sphere offers an abundance of proposals—often unconventional and seldom supported by objective evidence. Discussing all these proposals, which frequently serve primarily commercial purposes, is beyond the scope of this context. For further details, specific guidelines[15,27] are referenced, and key points are outlined in *Table 9*.

If the diet is prescribed for therapeutic purposes, as a treatment for a disease, it must be done under the supervision of a physician specialized or experienced in clinical nutrition. This ensures a proper diagnosis and allows for appropriate re-evaluation over time.[27,68,69] Diet prescription by other professional figures is possible if it is within a framework of collaborative supervision with a medical doctor expert in the field. In general, literature data suggest rather disappointing long-term results of diet therapy. A reasonable result can be achieved in the short term; however, in the long term (2–5 years), most patients regain previously lost weight.[70] The solution for obesity is not isolated dietary therapy.

Physical activity

Increasing physical activity, rediscovering the joy of movement, and setting achievable of a sustainable exercise goal form the three foundational pillars for immediate and long-term health benefits.[71,72] Even the simple transition from ‘no physical activity’ to ‘any level of physical activity’ can be useful to achieve, if possible, a target of 150 min/week of moderate aerobic activity within a reasonable period.[18,27,28] Additional benefits can be achieved by increasing the duration of moderate physical activity or maintaining the same duration but increasing the intensity. The process of changing physical activity takes time, with cycles of at least 3 months to allow the body to adapt and avoid overload damage. Similar to dietary therapy, the personalization of movement is a fundamental point.[71,72] The patient’s age and the presence of cardiovascular or musculoskeletal comorbidities must be taken into account to determine the type of activity that is possible and therefore prescribable. Although physical activity is an essential component for individual well-being and is also associated with an increase in life expectancy, its quantitative contribution to weight reduction is relatively modest.[72] This is because achieving significant calorie expenditure demands considerable physical effort and, secondly, due to compensatory mechanisms activated, such as an increase in the sensation of hunger, which partly nullify the increase in caloric burn. Nevertheless, the role of physical activity in reducing visceral fat and maintaining weight after a period of weight loss is pivotal.[72]

Psychological support and cognitive-behavioural psychotherapy

Cognitive-behavioural therapy (CBT) is the most used psychological intervention for obesity and might be considered a frontline treatment amongst psychological approaches.[73] While this assertion might not be universally agreed upon in the field of clinical psychology, it remains a fact that psychological support appears beneficial for motivating the patient, developing their self-monitoring skills during meals, raising awareness of triggering factors harmful to their goals, and introducing alternative behaviours as needed.

As a methodology delving into the patient’s behaviour, CBT shares some goals and tools with therapeutic education. However, it is a treatment that must be administered by a psychotherapist. Although it can delve deeper into the patient’s behavioural issues, it has the significant limitation of being time-consuming for the operator and is often challenging to obtain in the public domain, especially considering the epidemiological dimension that the obesity problem is assuming. Moreover, as a standalone treatment, although effective in certain eating behaviour disorders such as *binge eating*, it does not necessarily induce a clinically significant weight loss.[74]

The primary purpose of psychological treatment should be to help the patient define realistic goals, sequence them reasonably over time, build confidence in their ability to overcome barriers, and define and sustain personal motivations over time.

Familiar and social support

Living in an obesogenic environment is the most relevant factor for the development and maintenance of obesity. For this reason, meeting familiar members to identify the social barriers to implementing a programme for treating obesity is an obligatory step of the team that takes care of the patient.

Pharmacotherapy

Currently, in Italy, only three drugs have been approved with the indication for the treatment of obesity. However, in terms of obesity therapy, there are promising pharmacologic approaches that could be available in next future.

The knowledge of energetic homoeostasis has allowed the identification of several potential pharmacological targets that, individually or in combination, can facilitate the process of reducing fat mass.

The three drugs approved for the treatment of obesity are orlistat,[75] the bupropion/naltrexone association,[76] and liraglutide.[77] A fourth drug, semaglutide,[78] with a trade name different from that used for diabetes treatment, has already been authorized by the European Medicines Agency (EMA) and by the Agenzia Italiana del Farmaco for the treatment of obesity but is not yet commercially available. Liraglutide is also available with two trade names, one for diabetes and the other for obesity treatment.[77,79] The adoption of two different commercial names for the same drug based on the indication for treatment of obesity or other conditions could probably also characterize the new drugs that will be introduced in the future.

Orlistat. Orlistat is commercially available in 120 mg tablets that must be taken immediately before, during, or within an hour of each main meal.[75]

The drug is an inhibitor of pancreatic and gastric lipase and can induce a reduction in degradation and absorption of around 30% of the eaten fat.[75]

It is indicated if the patient has a BMI of $\geq$30 or $\geq$28 kg/m^2 in the presence of other risk factors, in combination with a moderately hypocaloric diet, and, in any case, containing a fat component. The drug is minimally absorbed and should be discontinued if a weight reduction of 5% of the initial weight is not achieved after 12 weeks.[77]

In randomized controlled studies, it has been demonstrated that orlistat induces a statistically significant but only mild weight reduction compared with placebo (9% of the initial weight vs. 6%, respectively).[80] The major limitation of this drug is associated with its mechanism of action, as it induces steatorrhoea and intestinal disorders. Furthermore, it causes chronic malabsorption, especially of fat-soluble vitamins. Finally, due to the possible pharmacologic interactions, patients taking warfarin should be carefully monitored.

Bupropion/naltrexone. The combination of naltrexone hydrochloride 8 mg and bupropion hydrochloride 90 mg in extended-release tablets has been studied in four randomized and controlled trials.[76] The results of these studies confirm its effectiveness in weight reduction and improvement of metabolic parameters such as blood glucose and lipid profile. However, the medication has several contraindications and requires expertise in its use. One aspect that cardiologists should be aware of is the possibility of unmasking a Brugada-type electrocardiographic pattern.

The drug should be discontinued if a 5% reduction of the initial weight is not achieved after 16 weeks.

Liraglutide. Liraglutide[77,79] is a 31-amino acid polypeptide analogous to human glucagon-like peptide-1 (GLP-1), from which it differs due to the absence of the first six amino acids and the substitution of an amino acid (arginine instead of lysine at position 34). A palmitic acid molecule has also been added to lysine at position 26.

These modifications are necessary to increase its half-life, as the half-life of GLP-1 is less than 2 min due to degradation induced by natural dipeptidyl peptidase-4 (DPP-4). The drug is administered only by subcutaneous injection. It is available in two commercial versions, indicated for two patient groups—one for diabetic patients, for diabetes management, and the other for obese patients for weight reduction. Initially, the same doses of the drug are used in both conditions, injected at the same concentrations. However, in diabetes, the recommended maximum dosage is 1.8 mg/day (0.3 mL of the solution in the pre-filled pen). In contrast, in obesity, the maximum dosage is 3 mg (0.5 mL of the solution in the pre-filled pen).

The therapeutic regimen requires daily subcutaneous administration, starting with 0.6 mg (0.1 mL of the solution in the pre-filled pen) and gradually increasing to the maximum prescribed dosage. Dosage escalation should occur at intervals of no less than 1 week.[77] Clinical indications for drug use include an initial BMI of $\geq$30 kg/m^2 or between 27 and 30 kg/m^2, provided that there is at least one weight-related comorbidity such as pre-diabetes or type 2 diabetes mellitus, hypertension, dyslipidaemia, or obstructive sleep apnoea.[77] The drug should be used in addition to a low-calorie diet and an increase in physical activity for weight management.[77] Treatment with liraglutide should be discontinued if, after 12 weeks at a dose of 3.0 mg/day, patients have not lost at least 5% of their initial body weight.[77]

Scientific evidence demonstrates greater efficacy than placebo, with 63.2% of treated patients losing 5% of body weight vs. 27.1% in the placebo group and 33.1% achieving a 10% weight reduction vs. 10.6% of patients treated with placebo.[81] However, the importance of the drug lies primarily in its innovative role, being the first drug in the incretin class to demonstrate efficacy in obesity. Currently, it is the only incretin available for obesity, although other drugs have already received EMA approval and will soon enter the market.[78]

Semaglutide. Semaglutide is a polypeptide similar to liraglutide but with different fatty acids attached to the amino acid chain and an additional amino acid substitution. Alanine at position 2 (8 of GLP-1) is replaced with a fatty acid, 2-aminobutyric acid, to prevent the proteolytic action of DPP-4. Lysine at position 34 is replaced by arginine, as in liraglutide, to prevent non-specific binding to 18-carbon fatty acids. An 18-carbon fatty acid is attached to lysine at position 26

to increase binding to albumin and circulation time.[82] These molecular modifications allow a much longer half-life and a weekly drug administration. The pharmacodynamics of the drug is analogous to that of liraglutide.

There is a substantial body of literature available for semaglutide. The drug has demonstrated better efficacy than liraglutide in a head-to-head comparison in a randomized controlled study that included a population of patients with obesity and without diabetes.[83] Clinical studies have shown that semaglutide can reduce weight in both diabetic[84] and non-diabetic patients[85] and also improve outcomes in overweight and obese patients without diabetes in secondary prevention.[86] Furthermore, in patients with HFpEF, semaglutide improved symptoms, reduced physical limitations, and inflammation.[87] It is noteworthy that clinical studies on diabetic patients have highlighted that the impact on weight loss is a common effect shared with other GLP-1 receptor agonists.

Other drugs. Setmelanotide should be separately considered because it is a drug reserved for treating obesity and appetite control associated with pro-opiomelanocortin deficiency or a genetic deficiency of both alleles encoding the leptin receptor.[88] This extremely rare genetic condition manifests in childhood, making it unlikely to involve cardiologists.

Other drugs, although not yet approved for obesity treatment but already registered for diabetes mellitus treatment, are emerging on the horizon with more than promising results. The first of these is tirzepatide,[89] the first drug commercially available able to exert an agonistic effect on two distinct receptors involved in the digestive process and glycaemic metabolism: the GLP-1 receptor and the glucose-dependent insulinotropic polypeptide.[90]

Bariatric surgery

The term 'bariatric surgery' refers to all surgical procedures aimed at inducing weight loss through reduced energy absorption introduced with diet.[89,91–93] Amongst these are as follows:

- Sleeve gastrectomy: In this procedure, typically performed endoscopically, about 80% of the gastric volume is removed, leaving a residual stomach in the shape of a 'sleeve', as the name suggests.
- Gastric bypass: In the literature, it is also defined as *Roux-en-Y gastric bypass*. In this intervention, a small part of the stomach, proximal to the oesophagus, is isolated and surgically connected to the ileum. In contrast, the remaining parts of the stomach, duodenum, and proximal ileum are bypassed and isolated from the passage of food.
- Adjustable gastric banding: In this procedure, a silicone device is placed in the upper part of the stomach, restricting the amount of food entering the stomach.
- Biliopancreatic diversion with duodenal switch: In this approach, the stomach is prepared similarly to a sleeve gastrectomy but is then disconnected from the duodenum and connected to a more distal part of the small intestine. With this procedure, duodenum and the first part of the small intestine are bypassed, adding to the sleeve a further mechanism for reducing absorption of food.

For more detailed information on available techniques, as well as their advantages and limitations, further insights can be gained from European guidelines[89] or the American Society of Bariatric Surgery website, which also provides graphical representations.[92]

Bariatric surgery is a well-established and safe surgical technique that allows for significant loss of adipose tissue, resulting in weight loss, improved critical metabolic parameters, and reduced comorbidities associated with obesity.[89] Currently, it remains the most effective method for weight loss and the reduction of obesity-related diseases, including the risk of heart failure onset and mortality.[93,94]

Target to achieve

Obesity treatment aims to induce changes that enhance quality of life, mitigate associated comorbidities, and reduce mortality risk. To achieve all or part of these outcomes, the patient does not need to return to having a BMI between 18.5 and 25 kg/m^2.[95] A loss of 15% of the starting weight is sufficient for most health benefits. Even with a 5% weight loss, measurable improvements can be achieved, such as reduction in systolic blood pressure by 3 mmHg and diastolic blood pressure by 2 mmHg.[95]

Understanding this concept is crucial to provide patients with information containing a more credible hope of success and because the physician must be clear that our goal is not to change body shapes or modify BMI. Rather, the focus should be on guiding patients towards a path to improve their health and prolong their survival by reducing diseases and death risk. Pursuits extending beyond this aim enter the realm of *diet culture*[96] and are placed in a completely different context.

Conclusions

Obesity is a chronic, relapsing disease with a significant impact on health in terms of associated diseases, particularly cardiovascular diseases,[43] including HFpEF,[57] disability, and premature death.[6–8] Its prevalence is reaching epidemic proportions.[1–4]

Cardiologists are in a privileged position to treat obesity as many of the comorbidities associated with cardiovascular diseases fall within their competence.[9]

However, medical treatment for obesity remains unsatisfactory. It requires a multi-disciplinary approach with considerable resources, yielding acceptable short-term results but unsatisfactory already within 2–5 years due to frequent relapse. It is often a source of frustration for the patient and the caregiver.[62,63]

However, the discovery of physiopathological mechanisms involving energy homoeostasis generates many potential solutions that are expected to become valid therapeutic treatments in the near future.[22]

A simple solution based on administering a miraculous drug with an uncomplicated therapeutic regimen to eliminate the problem is unrealistic. Given that it is a problem with deep individual and social motivations, it

is reasonable to believe that without a systemic approach, we cannot change the fate of entire populations, perhaps only that of individual cases. Nevertheless, even this outcome is highly desirable.

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References

1. World Health Organization. Obesity and overweight. 9 June 2021. https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight (26 January 2024).
2. The Lancet Gastroenterology Hepatology. Obesity: another ongoing pandemic. *Lancet Gastroenterol Hepatol* 2021;**6**:411.
3. EpiCentro. Obesità: il rapporto 2022 dell'OMS Europa. https://www.epicentro.iss.it/obesita/report-obesita-oms-2022 (26 January 2024).
4. EpiCentro. Obesità—Dati epidemiologici in Italia. https://www.epicentro.iss.it/obesita/epidemiologia-italia (26 January 2024).
5. World Health Organization. Il programma di lavoro Europeo 2020–2025: un'azione unitaria per una salute migliore in Europa. https://apps.who.int/iris/handle/10665/340793 (26 January 2024).
6. GBD 2015 Obesity Collaborators; Afshin A, Forouzanfar MH, Reitsma MB, Sur P, Estep K *et al*. Health effects of overweight and obesity in 195 countries over 25 years. *N Engl J Med* 2017;**377**:13–27.
7. Abdelaal M, le Roux CW, Docherty NG. Morbidity and mortality associated with obesity. *Ann Transl Med* 2017;**5**:161.
8. Peeters A, Barendregt JJ, Willekens F, Mackenbach JP, Al Mamun A, Bonneux L *et al*. Obesity in adulthood and its consequences for life expectancy: a life-table analysis. *Ann Intern Med* 2003;**138**:24–32.
9. Phelan SM, Burgess DJ, Yeazel MW, Hellerstedt WL, Griffin JM, van Ryn M. Impact of weight bias and stigma on quality of care and outcomes for patients with obesity. *Obes Rev* 2015;**16**:319–326.
10. Westbury S, Oyebode O, van Rens T, Barber TM. Obesity stigma: causes, consequences, and potential solutions. *Curr Obes Rep* 2023;**12**:10–23.
11. Zagaria A, Cerolini S, Mocini E, Lombardo C. The relationship between internalized weight stigma and physical and mental health-related quality of life in a large sample of women: a structural equation modeling analysis. *Eat Weight Disord* 2023;**28**:52.
12. Ryan DH, Deanfield JE, Jacob S. Prioritizing obesity treatment: expanding the role of cardiologists to improve cardiovascular health and outcomes. *Cardiovasc Endocrinol Metab* 2023;**12**:e0279.
13. Macek P, Biskup M, Terek-Derszniak M, Krol H, Smok-Kalwat J, Gozdz S *et al*. Optimal cut-off values for anthropometric measures of obesity in screening for cardiometabolic disorders in adults. *Sci Rep* 2020;**10**:11253.
14. Ashwell M, Gibson S. Waist-to-height ratio as an indicator of "early health risk": simpler and more predictive than using a "matrix" based on BMI and waist circumference. *BMJ Open* 2016;**6**:e010159.
15. Wharton S, Lau DCW, Vallis M, Sharma AM, Biertho L, Campbell-Scherer D *et al*. Obesity in adults: a clinical practice guideline. *CMAJ* 2020;**192**:E875–E891.
16. Crompvoets PI, Cramm JM, van Rossum EFC, Nieboer AP. Views of patients with obesity on person-centred care: a Q-methodology study. *Health Expect* 2022;**25**:3017–3026.
17. Durrer Schutz D, Busetto L, Dicker D, Farpour-Lambert N, Pryke R, Toplak H *et al*. European practical and patient- centred guidelines for adult obesity management in primary care. *Obes Facts* 2019;**12**:40–66.
18. Obesity Canada. Assessment of people with obesity. https://obesitycanada.ca/guidelines/assessment (26 January 2024).
19. Bray GA. Beyond BMI. *Nutrients* 2023;**15**:2254.
20. Weir CB, Jan A. *BMI Classification Percentile and Cut Off Points*. Treasure Island, FL: StatPearls Publishing; 2024.
21. Fang H, Berg E, Cheng X, Shen W. How to best assess abdominal obesity. *Curr Opin Clin Nutr Metab Care* 2018;**21**:360–365.
22. Di Fusco SA, Mocini E, Gulizia MM, Gabrielli D, Grimaldi M, Oliva F *et al*. ANMCO (Italian Association of Hospital Cardiologists) scientific statement: obesity in adults—an approach for cardiologists. *Eat Weight Disord* 2024;**29**:1.
23. Neeland IJ, Ross R, Després JP, Matsuzawa Y, Yamashita S, Shai I *et al*. Visceral and ectopic fat, atherosclerosis, and cardiometabolic disease: a position statement. *Lancet Diabetes Endocrinol* 2019;**7**:715–725.
24. Pischon T, Boeing H, Hoffmann K, Bergmann M, Schulze MB, Overvad K *et al*. General and abdominal adiposity and risk of death in Europe. *N Engl J Med* 2008;**359**:2105–2120.
25. Blüher M. Metabolically healthy obesity. *Endocr Rev* 2020;**41**:bnaa004.
26. Ross R, Neeland IJ, Yamashita S, Shai I, Seidell J, Magni P *et al*. Waist circumference as a vital sign in clinical practice: a consensus statement from the IAS and ICCR Working Group on Visceral Obesity. *Nat Rev Endocrinol* 2020;**16**:177–189.
27. Hassapidou M, Vlassopoulos A, Kalliostra M, Govers E, Mulrooney H, Ells L *et al*. European association for the study of obesity position statement on medical nutrition therapy for the management of overweight and obesity in adults developed in collaboration with the European Federation of the Associations of Dietitians. *Obes Facts* 2023;**16**:11–28.
28. National Institute for Health and Care Excellence. Obesity: identification, assessment and management. Clinical guideline [CG189], 27 November 2014. https://www.nice.org.uk/guidance/cg189 (26 January 2024).
29. Bray GA, Heisel WE, Afshin A, Jensen MD, Dietz WH, Long M *et al*. The science of obesity management: an Endocrine Society scientific statement. *Endocr Rev* 2018;**39**:79–132.
30. Haam JH, Kim BT, Kim EM, Kwon H, Kang JH, Park JH *et al*. Diagnosis of obesity: 2022 update of clinical practice guidelines for obesity by the Korean Society for the Study of Obesity. *J Obes Metab Syndr* 2023;**32**:121–129.
31. World Health Organization. Waist circumference and waist-hip ratio: report of a WHO expert consultation. https://www.who.int/publications-detail-redirect/9789241501491 (26 January 2024).
32. Borlaug BA, Jensen MD, Kitzman DW, Lam CSP, Obokata M, Rider OJ. Obesity and heart failure with preserved ejection fraction: new insights and pathophysiological targets. *Cardiovasc Res* 2023;**118**:3434–3450.
33. van der Valk ES, van den Akker ELT, Savas M, Kleinendorst L, Visser JA, Van Haelst MM *et al*. A comprehensive diagnostic approach to detect underlying causes of obesity in adults. *Obes Rev* 2019;**20**:795–804.
34. National Heart, Lung, and Blood Institute. Overweight and obesity—causes and risk factors. https://www.nhlbi.nih.gov/health/overweight-and-obesity/causes (26 January 2024).

35. Kaspar D, Hastreiter S, Irmler M, Hrabé de Angelis M, Beckers J. Nutrition and its role in epigenetic inheritance of obesity and diabetes across generations. *Mamm Genome* 2020;**31**:119–133.
36. Borga M, West J, Bell JD, Harvey NC, Romu T, Heymsfield SB *et al*. Advanced body composition assessment: from body mass index to body composition profiling. *J Investig Med* 2018;**66**:1–9.
37. Volpe M, Borghi C, Cameli M, Cianflone D, Cittadini A, Maggioni AP *et al*. Otto motivi per i quali i cardiologi non dovrebbero trascurare il problema "obesità". Expert opinion per la pratica clinica. *G Ital Cardiol* 2023;**24**:677–691.
38. Elías-López D, Vargas-Vázquez A, Mehta R, Cruz Bautista I, Del Razo Olvera F, Gómez-Velasco D *et al*. Natural course of metabolically healthy phenotype and risk of developing cardiometabolic diseases: a three years follow-up study. *BMC Endocr Disord* 2021;**21**:85.
39. Eckel N, Li Y, Kuxhaus O, Stefan N, Hu FB, Schulze MB. Transition from metabolic healthy to unhealthy phenotypes and association with cardiovascular disease risk across BMI categories in 90 257 women (the Nurses' Health Study): 30 year follow-up from a prospective cohort study. *Lancet Diabetes Endocrinol* 2018;**6**:714–724.
40. Donini LM, Pinto A, Giusti AM, Lenzi A, Poggiogalle E. Obesity or BMI paradox? Beneath the tip of the iceberg. *Front Nutr* 2020;**7**:53.
41. Donini LM, Busetto L, Bischoff SC, Cederholm T, Ballesteros-Pomar MD, Batsis JA *et al*. Definition and diagnostic criteria for sarcopenic obesity: ESPEN and EASO consensus statement. *Obes Facts* 2022;**15**: 321–335.
42. Angelantonio ED, Bhupathiraju SN, Wormser D, Gao P, Kaptoge S, Berrington de Gonzalez A *et al*. Body-mass index and all cause mortality: individual-participant-data meta-analysis of 239 prospective studies in four continents. *Lancet* 2016;**388**:776–786.
43. Held C, Hadziosmanovic N, Aylward PE, Hagström E, Hochman JS, Stewart RAH *et al*. Body mass index and association with cardiovascular outcomes in patients with stable coronary heart disease—a STABILITY substudy. *J Am Heart Assoc* 2022;**11**:e023667.
44. Tutor AW, Lavie CJ, Kachur S, Milani RV, Ventura HO. Updates on obesity and the obesity paradox in cardiovascular diseases. *Prog Cardiovasc Dis* 2023;**78**:2–10.
45. Dorresteijn JAN, Visseren FLJ, Wassink AMJ, Gondrie MJ, Steyerberg EW, Ridker PM *et al*. Development and validation of a prediction rule for recurrent vascular events based on a cohort study of patients with arterial disease: the SMART risk score. *Heart* 2013;**99**:866–872.
46. Kengne AP, Patel A, Marre M, Travert F, Lievre M, Zoungas S *et al*. Contemporary model for cardiovascular risk prediction in people with type 2 diabetes. *Eur J Cardiovasc Prev* 2011;**18**:393–398.
47. Piepoli MF, Hoes AW, Agewall S, Albus C, Brotons C, Catapano AL *et al*. 2016 European guidelines on cardiovascular disease prevention in clinical practice: the sixth joint task force of the European Society of Cardiology and other societies on cardiovascular disease prevention in clinical practice (constituted by representatives of 10 societies and by invited experts). Developed with the special contribution of the European Association for Cardiovascular Prevention & Rehabilitation (EACPR). *Eur Heart J* 2016;**37**:2315–2381.
48. SCORE2 working group and ESC Cardiovascular risk collaboration. SCORE2 risk prediction algorithms: new models to estimate 10-year risk of cardiovascular disease in Europe. *Eur Heart J* 2021;**42**: 2439–2454.
49. SCORE2-OP working group and ESC Cardiovascular risk collaboration. SCORE2-OP risk prediction algorithms: estimating incident cardiovascular event risk in older persons in four geographical risk regions. *Eur Heart J* 2021;**42**:2455–2467.
50. Sussman JB, Whitney RT, Burke JF, Hayward RA, Galecki A, Sidney S *et al*. Prediction of multiple individual primary cardiovascular events using pooled cohorts. medRxiv 2023:2023.08.01.23293525.
51. SCORE2-Diabetes Working Group and the ESC Cardiovascular Risk Collaboration. SCORE2-Diabetes: 10-year cardiovascular risk estimation in type 2 diabetes in Europe. *Eur Heart J* 2023;**44**: 2544–2556.
52. Il Progetto CUORE. https://www.cuore.iss.it (26 January 2024).
53. Jaspers NEM, Blaha MJ, Matsushita K, van der Schouw YT, Wareham NJ, Khaw KT *et al*. Prediction of individualized lifetime benefit from cholesterol lowering, blood pressure lowering, antithrombotic therapy, and smoking cessation in apparently healthy people. *Eur Heart J* 2020;**41**:1190–1199.
54. Berkelmans GFN, Gudbjörnsdottir S, Visseren FLJ, Wild SH, Franzen S, Chalmers J *et al*. Prediction of individual life-years gained without cardiovascular events from lipid, blood pressure, glucose, and aspirin treatment based on data of more than 500 000 patients with type 2 diabetes mellitus. *Eur Heart J* 2019;**40**:2899–2906.
55. Elmaleh-Sachs A, Schwartz JL, Bramant CT, Nicklas JM, Gudzune KA, Jay M. Obesity management in adults: a review. *JAMA* 2023;**330**: 2000–2015.
56. Khan SS, Ning H, Wilkins JT, Allen N, Carnethon M, Berry JD *et al*. Association of body mass index with lifetime risk of cardiovascular disease and compression of morbidity. *JAMA Cardiol* 2018;**3**:280–287.
57. Lavie CJ, Sharma A, Alpert MA, De Schutter A, Lopez-Jimenez F, Milani RV *et al*. Update on obesity and obesity paradox in heart failure. *Prog Cardiovasc Dis* 2016;**58**:393–400.
58. Sharma AM, Kushner RF. A proposed clinical staging system for obesity. *Int J Obes (Lond)* 2009;**33**:289–295.
59. Powell-Wiley TM, Poirier P, Burke LE, Després JP, Gordon-Larsen P, Lavie CJ *et al*. Obesity and cardiovascular disease: a scientific statement from the American Heart Association. *Circulation* 2021; **143**:e984–e1010.
60. Gondoni LA, Titon AM, Nibbio F, Augello G, Caetani G, Liuzzi A. Heart rate behavior during an exercise stress test in obese patients. *Nutr Metab Cardiovasc Dis* 2009;**19**:170–176.
61. Palma G, Sorice GP, Genchi VA, Giordano F, Caccioppoli C, D'Oria R *et al*. Adipose tissue inflammation and pulmonary dysfunction in obesity. *Int J Mol Sci* 2022;**23**:7349.
62. Boakye E, Grandhi GR, Dardari Z, Adhikari R, Soroosh G, Jha K *et al*. Cardiovascular risk stratification among individuals with obesity: the Coronary Artery Calcium Consortium. *Obesity* 2023;**31**:2240–2248.
63. Chow BJW, Dorbala S, Di Carli MF, Merhige ME, Williams BA, Veledar E *et al*. Prognostic value of PET myocardial perfusion imaging in obese patients. *JACC Cardiovasc Imaging* 2014;**7**:278–287.
64. Hall KD, Kahan S. Maintenance of lost weight and long-term management of obesity. *Med Clin North Am* 2018;**102**:183–197.
65. Cornejo-Pareja I, Molina-Vega M, Gómez-Pérez AM, Damas-Fuentes M, Tinahones FJ. Factors related to weight loss maintenance in the medium-long term after bariatric surgery: a review. *J Clin Med* 2021;**10**:1739.
66. Colivicchi F, Di Fusco SA, Santini M. In: Roncella A, Pristipino C (eds.), *Psycho-educational Interventions and Cardiac Rehabilitation*. Cham: Springer International. Publishing; 2016. p107–120.
67. LeBlanc EL, Patnode CD, Webber EM, Redmond N, Rushkin M, O'Connor EA. Behavioral and pharmacotherapy weight loss interventions to prevent obesity-related morbidity and mortality in adults: updated evidence report and systematic review for the US Preventive Services Task Force. *JAMA* 2018;**320**:1172–1191.
68. Società Italiana dell'Obesità. Linee guida della SICOB Società Italiana di Chirurgia dell'Obesità e delle Malattie Metaboliche. https://sio-obesita.org/linee-guida (26 January 2024).
69. Gardner CD, Vadiveloo MK, Petersen KS, Anderson CAM, Springfield S, Van Horn L *et al*. Popular dietary patterns: alignment with American Heart Association 2021 Dietary guidance: a scientific statement from the American Heart Association. *Circulation* 2023;**147**:1715–1730.
70. Mann T, Tomiyama AJ, Westling E, Lew AM, Samuels B, Chatman J. Medicare's search for effective obesity treatments: diets are not the answer. *Am Psychol* 2007;**62**:220–233.
71. Oppert JM, Bellicha A, Ciangura C. Physical activity in management of persons with obesity. *Eur J Intern Med* 2021;**93**:8–12.
72. Oppert JM, Ciangura C, Bellicha A. Physical activity and exercise for weight loss and maintenance in people living with obesity. *Rev Endocr Metab Disord* 2023;**24**:937–949.
73. Castelnuovo G, Pietrabissa G, Manzoni GM, Cattivelli R, Rossi A, Novelli M *et al*. Cognitive behavioral therapy to aid weight loss in obese patients: current perspectives. *Psychol Res Behav Manag* 2017;**10**: 165–173.
74. Grilo CM, Masheb RM, Wilson GT, Gueorguieva R, White MA. Cognitive-behavioral therapy, behavioral weight loss, and sequential treatment for obese patients with binge-eating disorder: a randomized controlled trial. *J Consult Clin Psychol* 2011;**79**:675–685.
75. European Medicines Agency. Xenical. https://www.ema.europa.eu/en/medicines/human/EPAR/xenical (26 January 2024).
76. European Medicines Agency. Mysimba. https://www.ema.europa.eu/en/medicines/human/EPAR/mysimba (26 January 2024).
77. European Medicines Agency. Saxenda. https://www.ema.europa.eu/en/medicines/human/EPAR/saxenda (26 January 2024).

78. European Medicines Agency. Wegovy. https://www.ema.europa.eu/en/medicines/human/EPAR/wegovy (26 January 2024).
79. European Medicines Agency. Victoza. https://www.ema.europa.eu/en/medicines/human/EPAR/victoza (26 January 2024).
80. Abdul Wahab R, le Roux CW. A review of the evidence on cardiovascular outcomes from obesity treatment. *Obes Pillars* 2023; **7**:100071.
81. Pi-Sunyer X, Astrup A, Fujioka K, Greenway F, Halpern A, Krempf M *et al*. A randomized, controlled trial of 3.0 mg of liraglutide in weight management. *N Engl J Med* 2015;**373**:11–22.
82. Kalra S, Sahay R. A review on semaglutide: an oral glucagon-like peptide 1 receptor agonist in management of type 2 diabetes mellitus. *Diabetes Ther* 2020;**11**:1965–1982.
83. Rubino DM, Greenway FL, Khalid U, O'Neil PM, Rosenstock J, Sørrig R *et al*. Effect of weekly subcutaneous semaglutide vs daily liraglutide on body weight in adults with overweight or obesity without diabetes: the STEP 8 randomized clinical trial. *JAMA* 2022;**327**: 138–150.
84. Davies M, Færch L, Jeppesen OK, Pakseresht A, Pedersen SD, Perreault L *et al*. Semaglutide 2.4 mg once a week in adults with overweight or obesity, and type 2 diabetes (STEP 2): a randomised, double-blind, double-dummy, placebo-controlled, phase 3 trial. *Lancet* 2021;**397**: 971–984.
85. Wilding JPH, Batterham RL, Calanna S, Davies M, Van Gaal LF, Lingvay I *et al*. Once-weekly semaglutide in adults with overweight or obesity. *N Engl J Med* 2021;**384**:989–1002.
86. Lincoff AM, Brown-Frandsen K, Colhoun HM, Deanfield J, Emerson SS, Esbjerg S *et al*. Semaglutide and cardiovascular outcomes in obesity without diabetes. *N Engl J Med* 2023;**389**:2221–2232.
87. Borlaug BA, Kitzman DW, Davies MJ, Rasmussen S, Barros E, Butler J *et al*. Semaglutide in HFpEF across obesity class and by body weight reduction: a prespecified analysis of the STEP-HFpEF trial. *Nat Med* 2023;**29**:2358–2365.
88. European Medicines Agency. Imcivree. https://www.ema.europa.eu/en/medicines/human/EPAR/imcivree (26 January 2024).
89. Di Lorenzo N, Antoniou SA, Batterham RL, Busetto L, Godoroja D, Iossa A *et al*. Clinical practice guidelines of the European Association for Endoscopic Surgery (EAES) on bariatric surgery: update 2020 endorsed by IFSO-EC, EASO and ESPCOP. *Surg Endosc* 2020;**34**: 2332–2358.
90. U.S. Food and Drug Administration. FDA approves new medication for chronic weight management. November 8, 2023. https://www.fda.gov/news-events/press-announcements/fda-approves-new-medication-chronic-weight-management (26 January 2024).
91. Eisenberg D, Shikora SA, Aarts E, Aminian A, Angrisani L, Cohen RV *et al*. American Society of Metabolic and Bariatric Surgery (ASMBS) and International Federation for the Surgery of Obesity and Metabolic Disorders (IFSO) indications for metabolic and bariatric surgery. *Obes Surg* 2022;**2023**:3–14.
92. American Society for Metabolic and Bariatric Surgery. Bariatric surgery procedures. https://asmbs.org/patients/bariatric-surgery-procedures (26 January 2024).
93. Aminian A, Zajichek A, Arterburn DE, Wolski KE, Brethauer SA, Schauer PR *et al*. Association of metabolic surgery with major adverse cardiovascular outcomes in patients with type 2 diabetes and obesity. *JAMA* 2019;**322**:1271–1282.
94. Sundström J, Bruze G, Ottosson J, Marcus C, Näslund I, Neovius M. Weight loss and heart failure: a nationwide study of gastric bypass surgery versus intensive lifestyle treatment. *Circulation* 2017;**135**: 1577–1585.
95. Ryan DH, Yockey SR. Weight loss and improvement in comorbidity: differences at 5%, 10%, 15%, and over. *Curr Obes Rep* 2017;**6**:187–194.
96. Embody Health London. Diet culture throughout history. September 13, 2022. https://embodyhealthlondon.com/diet-culture-throughout-history (26 January 2024).