Cardiac Diseases and Obesity

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ABSTRACT

Obesity is a raging epidemic with a significant impact on cardiovascular health profile. It harbors the development of both structural [coronary artery disease (CAD), heart failure (HF)] and electrical (atrial fibrillation) cardiac disorders. On a background of low-grade chronic inflammation, the changes in body composition, hemodynamic alterations, and myocardial fat deposition accelerate the associated cardiovascular morbidity. Quite counterintuitively, obese patients having CAD or HF fare better than their normal-weight counterparts; a phenomenon described as the "obesity paradox". An increase in lean mass (LM) and consequent improvement in cardiorespiratory fitness (CRF), partly explains this phenomenon. In the following review, we aim to decipher the links between obesity and cardiovascular diseases (CVDs) along with the evolving concept of "adiposopathy" and current evidence shedding light on the intriguing "obesity paradox". The significance of estimating and improving CRF is also discussed along with current recommendations for managing obesity.

Keywords: Cardiorespiratory fitness, Epicardial fat, Metabolically healthy obese, Obesity, Obesity paradox.

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INTRODUCTION

Being obese is a challenging health concern that affects people from varying demographics with different sociocultural backgrounds. Data from the World Health Organization (WHO) underscores the ever-expanding prevalence of this non-communicable disease. Thirty-nine percent of adults across the world are overweight and 13% are obese.¹

Obesity as defined by WHO is an increase in fat mass (FM) which impairs health. Body mass index (BMI) ≥ 30 kg/m² is the most accepted definition of obesity. It is further classified into:

- Class I: BMI between 30.0 and 34.9 kg/m².
- Class II: BMI between 35.0 and 39.9 kg/m².
- Class III: BMI exceeding 40.0 kg/m².

Overweight is considered when BMI is ≥ 25 kg/m². World Health Organization has suggested modified BMI cutoffs in Asian subjects because they have proportionately more visceral fat for a given BMI. This makes them particularly vulnerable to the adverse effects of obesity. Thus, the modified cutoffs are BMI ≥ 23 kg/m² for overweight and ≥ 25 kg/m² for obesity.²

Cardiac conditions like coronary artery disease (CAD), heart failure (HF), and rhythm disturbances have been linked to obesity.¹ Obesity has also been shown to independently influence cardiac outcomes. But this linear relationship does not hold in the setting of an already established cardiac disease. This means that *before the onset* of a cardiovascular disease (CVD), reducing bodyweight can serve as a primary prevention strategy. But once a cardiac disease has already set-in, then obesity has a paradoxical protective effect for cardiovascular outcomes, a phenomenon termed the "*obesity paradox*".³ This review will highlight the occurrence of this phenomenon in various conditions like HF, coronary atherosclerosis, arrhythmias, etc.⁴

Furthermore, we will discuss the significance of compartmentalizing obesity into FM, lean mass (LM), and fat-free mass (FFM) to improve CVD risk stratification. Current views on the interesting entity called "metabolically healthy obese" and the

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emerging role of epicardial fat affecting CVD will be highlighted. The concept of cardiorespiratory fitness (CRF) as a novel yardstick to define therapeutic targets will be reviewed with a brief discussion about the currently accepted benefits of lifestyle changes, drug therapy, and surgical options in the management of obesity.

EPIDEMIOLOGY

As per the latest epidemiological studies, the overweight adult population is about 1.9 billion while obesity affects almost 6.50 billion adults. Nearly 2.8 million lives are lost annually as a consequence of obesity-related illnesses.⁵ Consequent to the adoption of western dietary habits like the over-consumption of refined carbohydrates and saturated fats coupled with a sedentary lifestyle, the financially challenged healthcare systems of underdeveloped nations are now suffering the toughest impact of obesity. Presently, almost 13.5 crore Indians suffer from obesity. The growing menace of obesity in India has spread to involve 11.8–31.3% of the population; among them, abdominal obesity shows prevalence between 16.9% and 36.3%.⁶

Obesity in south Asians shows certain peculiar features. Compared to western populations, their body fat is comprised of proportionately more visceral fat and there is more prevalence of insulin resistance. The differing body composition compels faster pancreatic beta-cell destruction and early onset diabetes mellitus (DM), thus highlighting the importance of body composition rather than anthropometric indices alone in risk stratifying these patients.⁷

Fit-fat Patient: Metabolically Healthy Obesity

The concept of metabolically healthy obese (MHO) stems from the fact that not all overweight or obese patients have increased CVD risk. Most of the association between adiposity indices and CVD is explained by the *altered levels of intermediate risk factors* and not by the bodyweight *per se*. Such intermediaries include a constellation of metabolic abnormalities, including insulin resistance leading to type 2 DM, atherogenic dyslipidemia, elevated blood pressure (BP), subtle chronic inflammation, and a prothrombotic profile; collectively termed the metabolic syndrome (MS).⁸ Metabolic syndrome is generally characterized by the accumulation of excess fat in visceral depots. For any given level of total body fat, individuals showing a lack of excess abdominal visceral adipose tissue have a lower CVD risk profile than individuals harboring its excess. The concept of MHO has introduced a change in approach to obesity from that of a “one-size-fits-all” approach to an individualistic approach whereby not only total body fat content but also its regional distribution has assumed paramount significance.

BMI and CVD Flummox: Search for Novel Measurements

The inherent limitation of BMI is that it gives no information on fat distribution. In an obese phenotype, the clinician has two choices: to reduce CVD risk by targeting conventional risk factors like low-density lipoprotein cholesterol (LDL-C), BP, and uncontrolled hyperglycemia with pharmacological agents *or* to target weight loss. Randomized trials have established the clinical benefits of targeting the BP, LDL-C and achieving appropriate glycemic control, but no weight-loss drug has clearly shown any benefit in cardiovascular events and mortality, except for some new diabetes drugs.⁹ “Look AHEAD” trial (studying diet and losing weight in obese diabetic subjects) showed that intensive weight-loss interventions

did not result in any reduction in CVD events despite beneficial effects on some CVD risk factors and quality of life.¹⁰

This suggests that instead of reducing total bodyweight, the target should be to reduce the fat that “*troubles the most*”, i.e., the visceral fat. To estimate this entity newer physical assessments like abdominal circumference and waist/hip ratio (WHR) are useful. In males, an abdominal girth ≥ 102 cm defines central obesity and predicts future cardiac events (for females the corresponding cutoff is ≥ 88 cm). A WHR ≥ 0.9 in males (and ≥ 0.85 in females) is another criterion.⁹ For Asian-Indian patients, these cutoffs are revised as:

- A waist circumference of ≥ 85 cm in males (≥ 80 cm for females).
- Waist/hip ratio as ≥ 0.88 for males (≥ 0.81 for females).²

COMPARTMENTALIZING OBESITY

The *regional distribution* of adipose tissue, as discussed earlier, is more significant than the total body adipose *content*. The rise in BMI may be a consequence of an increase in subcutaneous fat, visceral fat, or both, with a concomitant rise or fall in LM. Among these, visceral fat (estimated from waist circumference) is considered the prime culprit. It incites the synthesis of adipokines and various cytokines. These biological compounds with their cardiac-depressant and pro-atherosclerotic properties become the chief predictors of future CV events.¹¹ On the contrary, excess accumulation of fat in the lower body (hips and thighs) may actually reduce the chances of developing CVD or diabetes.⁹

Increased BMI is almost always mirrored by a rise in FFM. Conversely, if FFM decreases in the face of an increased FM, then it is known as the sarcopenic variant of obesity. By compartmentalizing the bodyweight, we can also distinguish the beneficial rise in BMI in an athlete from the sarcopenic and nonsarcopenic variants of obesity (Fig. 1).¹²

Fig. 1: Various phenotypes of increased BMI according to proportion of fat and lean mass. BMI, body mass index; CRF, cardiorespiratory fitness

PATHOGENESIS: THE CONCEPT OF ADIPOSOPATHY

Multiple factors converge to cause obesity including:

- Genetic predisposition, sociocultural, and environmental situations.
- Behavioral and physiological components.
- Epigenetic risk factors.

Hereditary predisposition also arises from epigenetic mechanisms. Research has implicated approximately 140 chromosomal loci with this condition.¹³

Gut microbiota has also been incriminated in the predisposition to obesity. Bacterial lipopolysaccharide triggers an inflammatory cascade. This has effects on behavior control areas of the brain and also leads to derangement of adipose tissue functioning. The final result is weight gain with an enhanced risk of metabolic and cardiac diseases (Fig. 2A).¹³

Adiposopathy is the pathological alteration in the normal functioning of adipocytes that occurs as a consequence of energy intake exceeding energy expenditure and an inactive lifestyle.¹⁴ It is manifested by fat-cell hypertrophy, increased visceral, pericardial, perivascular, and peri-organ adiposity whose nutritional requirements are unmet by the existing vascular supply and resultant ischemia culminates in the release of inflammatory mediators.

Why Specifically Visceral Fat Worsens CV Outcomes?

Plausible explanations for this include:

- Portal vein serving as a conduit to deliver the free fatty acids (FFAs) from hypertrophied adipocytes (in the omentum) *directly* to hepatocytes. This promotes the excess hepatic triglyceride and glucose production.

- Visceral adipose tissue expanding through hypertrophy rather than hyperplasia generates rupture-prone fat cells that incite an inflammatory state.
- An increase in visceral fat may be a reflection of spill-over due to saturation of subcutaneous fat depots (which behaves as a protective “metabolic sink” in healthy individuals).⁹

Apart from fat tissues, two other systems regulate bodyweight: the appetite and satiety centers in the brain, and the endocrine system in the gastrointestinal tract.¹⁵ Peripheral nervous system regulates the heat production by controlling musculoskeletal activities. The autonomic (sympathetic) nervous system also regulates energy expenditure.^{15,16} Gastrointestinal endocrine system is involved in satiety and hunger signaling. This effect is mediated by hormones like leptin, insulin, glucagon-like peptide-1 (GLP-1), peptide YY (PYY), cholecystokinin (CCK), and ghrelin (Fig. 2B). Their signaling pathways converge to relay details regarding the energy equilibrium to the hypothalamus and other cerebral centers. These areas then modulate the urge to consume food by interacting with the reward centers of the brain.¹⁶ Perturbations of the fine balance between hunger, satiety, and energy expenditure knit the pathogenesis of obesity.

EMERGING ROLE OF EPICARDIAL FAT

Epicardial fat occupies the potential space between the visceral pericardium and cardiac musculature. The most notable locations are the confluence sites between the ventricles and the atrioventricular grooves. There is no fascial barrier between this fat layer and the underlying muscles and hence share a common microcirculation. It has been suggested that tumor necrosis factor- α (TNF- α) is secreted by adipocytes embracing the arteriolar origin sites. This cytokine causes inhibition of insulin-dependent engagement of capillaries and thus functions as a “vasocrine” signal. Myocardial blood supply can thus be altered by this epicardial fat.¹⁷

Figs 2A and B: Pathogenic mechanisms in obesity and its impact on cardiovascular diseases. ROS, reactive oxygen species; RNS, reactive nitrogen species; LPS, lipopolysaccharide; TLR, toll-like receptor; CCK, cholecystokinin; GLP-1, glucagon-like peptide-1; PYY, peptide YY; T2DM, type 2 diabetes mellitus; ATH, atherosclerosis; HTN, hypertension

Epicardial fat also has other well-defined physiological roles. It acts as an immunological “fence” to protect the cardiomyocytes and coronary arteries from inflammation-induced damage. Also, there is a mechanical “cushion” provided to the epicardial vessels because it provides space for positive remodeling to occur. This fat also serves as a nutritive depot by providing fatty acids to the heart during a high energy-demand state like exercise.¹⁸ Myocardial triglyceride content is strongly related to epicardial fat depots as shown by magnetic resonance and spectroscopy findings.¹⁹

The physiological properties assume a pathological nature when excess deposition of pericardial fat is not paralleled by a proportionate increase in blood supply. The ensuing ischemia triggers inflammation and sets the stage for CVD.

Data from various studies like Framingham and MESA studies have highlighted a direct association between the magnitude of epicardial fat and the presence and severity of coronary atherosclerosis.^{20,21} Infiltration of this ectopic fat into the interstitium can also alter the diastolic properties of the myocardium and affect the electrical impulse propagation (increasing the risk of AF).²²

How OBESITY “WEIGHS DOWN” ON CARDIOVASCULAR SYSTEM?

Obesity and Atherosclerosis

Several common factors underlie the pathology of obesity and atherosclerotic changes in the arteries. As with atherosclerosis, obesity is also incited against a background of systemic inflammation caused by lipid peroxidation (Fig. 3A). Other common features include a cytokine-induced insulin-resistance, endothelium damage, and a procoagulant milieu.^{23,24} Studies report that an obese profile, sustained for a minimum of 20 years, independently increases the risk of CAD. For every 10 kg increment in weight, CAD risk rises by 12%.²⁵ In young subjects, the risk of developing ST-elevation myocardial infarction (STEMI) also seems to be dependent on excess bodyweight.²⁶ For cerebrovascular outcomes, per unit rise in BMI causes 4 and 6% increment in the risk for ischemic attacks and cerebral bleeds, respectively.²⁷

Obesity and Risk of Heart Failure

Framingham Heart Study reported that per unit rise in BMI augments myocardial dysfunction risk by 5% for males (and 7% for females).²⁸ More than one-third of patients with HF are affected by obesity. Also, in obese patients, there is almost a decade earlier onset of HF than normal-weight patients.²⁹

Obesity-induced morphological and physiological alterations in the myocardium constitute “*obesity cardiomyopathy*” that ultimately causes HF (Fig. 3B).³⁰ Dual mechanisms of renin-angiotensin-aldosterone system (RAAS) activation and sympathetic overdrive act to increase myocardial fibrotic changes, endothelial injury, and increased thrombogenicity.³¹ Eplerenone acting as an inhibitor of RAAS was shown to benefit obese patients having HF with reduced ejection fraction in the EMPHASIS-HF trial.³² Renin-angiotensin-aldosterone system activation and sympathetic drive increase blood volume and thus augment preload. These two systems also raise afterload by causing systemic hypertension. Remodeling process initiated by these changes instigates pathological impairment of both systolic and diastolic phases of the cardiac cycle leading to HF. Proinflammatory cytokines belonging to the interleukin and tumor necrosis factor family, and hormones leptin and adiponectin also promote this dysfunction.³³

Obesity and Cardiac Arrhythmias

Hippocrates observed that “sudden death is more common in those who are naturally fat than in the lean”. Consolidating this statement, various studies have linked obesity with rhythm disturbances, which can sometimes be life-threatening.^{34,35}

Atrial fibrillation (AF) has a significant impact on the quality of life and mortality and poses a significant economic burden. Nearly one-third of arrhythmia-related admissions are attributable to AF. The chances of developing AF increase by 4% per unit rise in BMI.³⁶ Obesity causes left atrial dilation and dysfunction. A 5-mm increase in left atrial diameter increases the chances of paroxysmal AF by 1.39 times.³⁶ Epicardial fat initiates fibrotic changes in atria through its vasocrine signaling. The adipocytes from this fat depot also infiltrate the underlying atrial muscles and further promote inflammation. These factors derail the organized electrical conduction in atrial tissue leading to disharmony between

Figs 3A to C: Detrimental impact of obesity on various cardiovascular entities: (A) Coronary artery disease; (B) Heart failure; (C) Atrial arrhythmias. SMC, smooth muscle cell; CO, cardiac output; SVR, systemic vascular resistance; DM, diabetes mellitus; LVH, left ventricular hypertrophy; LV, left ventricle; PAH, pulmonary artery hypertension; RV, right ventricle; LA, left atrium; HTN, hypertension

epicardial and sub-endocardial potentials and facilitate re-entry arrhythmias (Fig. 3C). Arrhythmogenicity is also promoted by leptin which increases myocyte depolarization time. Obesity leads to ECG changes like increased duration and height of *P* waves, prolongation of PR interval, and terminal deflection of *P* wave.³⁷

Besides AF, ventricular tachyarrhythmias may also be a consequence of obesity. A remodeled ventricle with concomitant fibrosis leads to electrical disruption in impulse propagation. This is further augmented by the effects of epicardial fat as noted above.³⁶ An electrical rearrangement occurs owing to alteration in potassium channel (voltage-gated) functioning and sympathetic overdrive. There is resultant QT and QTc (corrected QT interval) lengthening which promotes arrhythmogenesis.³⁸

OBESITY PARADOX

Although obesity increases the risk of developing CVD, it is noteworthy that once a particular disease phenotype is established then the excess weight actually improves the prognosis. This is termed the obesity paradox which has been noted in a wide range of obesity-related cardiac conditions.^{36,39}

Obesity Paradox in Heart Failure

Clinical outcomes in HF are improved by associated obesity. All-cause and cardiovascular deaths were found to be 16% and 19% lower in overweight patients, respectively, compared with normal-weight subjects. This paradoxical protection is further magnified in obese patients where mortality difference is 33% for all-cause and 40% for cardiovascular causes.⁴⁰ Another meta-analysis has reported that in comparison to non-obese HF patients the risk of all-cause mortality is reduced by a factor of 0.78 and cardiac-specific mortality by a factor of 0.80.⁴¹ This paradox is especially noted in patients belonging to *class I obesity*.⁴²

Obesity Paradox and Coronary Revascularization

Pooled data from BARI, Scottish Coronary Revascularization, and APPROACH registries derived for patients undergoing coronary angioplasty have suggested a reciprocal correlation between BMI and the percutaneous coronary intervention (PCI)-related short-term complications.³⁶

Obesity Paradox and Chronic Inflammation

As already discussed, chronic inflammation is the hallmark of obesity. The phenomenon of obesity paradox peculiarly occurs in the setting of milder grades of systemic inflammation [defined by high sensitivity (hs)-CRP <3 mg/L] (Fig. 4A). This is reflected by the fact that an obese CAD patient having high FM with low hs-CRP has a better prognosis. Thus, the brunt of excess body fat is somewhat offset by the concurrent mild systemic inflammation.⁴³

Concept of Cardiorespiratory Fitness: An Emerging Tool

The capacity of respiratory and circulatory systems to meet the oxygen demand of the musculoskeletal system during sustained exercise is defined as CRF. It is best measured by peak oxygen consumption, i.e., VO_2 max. Higher CRF seen in obesity occurs by virtue of an increase in LM that parallels an increase in FM (Fig. 4B). The obesity paradox is observed only in the setting of low CRF, i.e., when the linear relation between LM and FM is not followed. This paradoxical relationship is not seen in patients having VO_2 max >14 mL/kg/minute or more than four-metabolic equivalents of task (METS).¹² In obese individuals, peak VO_2 when indexed to weight (mL/kg/minute) underestimates CRF.⁴⁴ This shortcoming is addressed by adjusting peak VO_2 to FFM. A threshold of 19 mL/kg LM/minute is now considered a better indicator than the previous peak VO_2 max cutoff of 14 mL/kg/minute.⁴⁴

Figs 4A and B: (A) Survival estimates (Kaplan-Meier) by body fat (BF) and high-sensitivity C-reactive protein (hsCRP) strata: low hsCRP = CRP <3 mg/L; high hsCRP = CRP ≥3 mg/L; low BF = BF <25%; high BF ≥25%. Reproduced from The impact of inflammation on the obesity paradox in coronary heart disease.⁴³ CRP, C-reactive protein; (B) Proposed mechanisms driving obesity to heart failure (HF) and the obesity paradox once HF is diagnosed. The dark blue arrows indicate the potentially detrimental effects of body composition components (fat mass and lean mass) on cardiac function and eventually HF development. The light blue arrows indicate the potential mechanisms by which body composition improves cardiorespiratory fitness. Reprinted from Obesity and heart failure: focus on the obesity paradox.¹² SVR, systemic vascular resistance; CRF, cardiorespiratory fitness; LVH, left ventricular hypertrophy¹⁰

Recently, the American Heart Association has stressed the significance of measuring CRF.⁴⁵ Even after adjustment for other risk factors like BMI or visceral fat, improved CRF independently predicts a better event profile for CVD patients. From this stems, the novel concept that rather than BMI, the target for improving outcomes in CVD patients should be CRF.

Since a cardiopulmonary exercise test may not always be feasible, hence CRF can be estimated from peak exercise at the time of a treadmill test (or cycle ergometer test).⁴⁵ Both isotonic and isometric exercise training are utilized to improve CRF for cardiac rehabilitation.⁴⁶ In obese patients with heart failure and preserved ejection fraction (HFpEF), aerobic exercise done under supervision for 1-hour at least thrice in a week for 20 weeks improves CRF by 1.2 mL/kg/minute. The benefit can be further improved to 2.5 mL/kg/minute through diet modifications.⁴

Besides improved CRF, other explanations cited for the occurrence of obesity paradox include:⁴⁷

- Comorbid conditions like malignancy, multiorgan dysfunction, etc., and advanced age are more common in lean patients.
- In obese patients, CVD is usually diagnosed earlier.
- Raised serum lipoproteins in obesity may neutralize bacterial endotoxins.
- In critically ill, fat depots may serve to maintain energy balance, and therefore prevent wasting.

Investigations of CVS in Obese

Due to the huge impact on the overall CVD profile, obesity even in an asymptomatic patient warrants evaluation of cardiovascular health. According to a meta-analysis, left ventricular hypertrophy (LVH) is four times more likely to develop in obese individuals. Eccentric hypertrophy is more common than the concentric subtype. Also, more than half of obese patients have diastolic dysfunction and this shows a close correlation with abdominal circumference.⁴⁸ Left atrial volume enlargement is also more common in obese. Thus screening tests are warranted to detect the cardiac complications related to obesity at an early stage.

Routine echocardiography derived indices are not a very sensitive tool to detect early deterioration in systolic and diastolic properties of the myocardium. Among emerging noninvasive

modalities, “integrated backscatter” assessed by color Doppler imaging is a novel method to assess the state of compliance of the myocardium, its inotropic properties, and associated interstitial fibrotic changes in very early stages.⁴⁹

CURRENT MANAGEMENT OPTIONS FOR OBESITY

Lifestyle and Dietary Recommendations

Although the excess visceral adiposity and ectopic fat have a genetic basis, diet clearly plays a pivotal role. The weight that is lost in the initial period determines the long-term success of such lifestyle measures. The recommendation is to lose 5–10% weight in the first 6 months.⁵⁰ The recommendations from the International Chair on Cardiometabolic Risk are summarized in Figure 5.⁹

Pharmacological Intervention

Patients with BMI ≥ 30 kg/m² (or ≥ 27 kg/m² for those with one or more obesity-related complications) are candidates for drug therapy to lose weight.⁹ Pharmacotherapy options currently approved in the United States and Europe include:

- Oral phentermine (15–30 mg) which acts as a sympathomimetic.
- Oral orlistat (120 mg thrice daily preceding meals) which is an inhibitor of pancreatic lipase.
- Oral lorcaserin (10 mg twice daily) acts as a selective agonist for the 5-HT_{2C} serotonin receptor.
- Oral phentermine/topiramate (7.5 mg/46 mg or 15 mg/92 mg)—which has a pleiotropic action profile including facilitating sympathetic activity, antagonizing glutamate, modulation of GABA receptors, and inhibiting carbonic anhydrase.
- Naltrexone, which acts by opioid receptor antagonism and is used in fixed-dose combination with bupropion—a noradrenaline and dopamine reuptake inhibitor (32 mg/360 mg orally).
- Injectable liraglutide (given as 3.0 mg dose) acts by facilitating GLP-1 receptor function and is useful for promoting weight loss.⁵¹

No of these drugs have yet demonstrated improved cardiovascular outcomes or efficacy in reducing visceral adipose tissue or ectopic fat.⁵¹ Recently, trials of diabetes drugs empagliflozin and semaglutide (that have an impact on energy

Fig. 5: Lifestyle management recommendations

Table 1: Comparison of various bariatric surgery techniques

	Restrictive procedure		Hybrid procedure	
	Adjustable gastric banding	Sleeve gastrectomy	Roux-en Y gastric bypass	Biliopancreatic diversion with duodenal switch

Mortality rates <30 days	0–0.10%	0.13–0.50%	0.15–1.15%	0.30–1.20%
Weight loss at 1 year	14–30%	20–28%	23–43%	38–52%
Comorbidity resolution at 1 year				
T1DM	23–61%	37–81%	17–93%	59–95%
Dyslipidemia	17%	16–83%	33–47%	33–65%
Hypertension	19–55%	15–82%	20–45%	24–53%
Sleep apnea	78%	52–100%	33–100%	100%

T1DM, type 1 diabetes mellitus

balance) have shown, for the first time, a significant reduction in adverse cardiovascular outcomes.⁹

Bariatric Surgery

There are multiple guidelines suggesting criteria to identify patients who are likely to benefit from bariatric surgery. Guidelines from The American Diabetes Association, International Diabetes Federation, etc., consider weight-loss surgery as the sole modality that provides stable weight loss and ascertains improved prognosis.^{52,53} Bariatric surgery is indicated for patients having BMI above 40 kg/m² (or 35 kg/m² if associated OSA, diabetes, hypertension, etc., are present) when other treatment options have been exhausted.⁹

Bariatric surgeries are of two types *restrictive* or *hybrid*. Hybrid surgery combines restrictive and malabsorptive mechanical re-routing of the gastrointestinal tract. The frequency of complications and death-rate vary with the type of surgical modality, the amount of weight lost as well as its durability, and how promptly comorbid conditions are managed (summarized in Table 1).⁹ Hybrid procedures are *best for weight loss and long-term outcomes*. The 30-day complication rate post-surgery is below 10% (lower with restrictive surgeries) and the death rate is in the range of 0.1–1.2%. Bariatric surgeries have been shown to improve cardiac geometry as well as the diastolic and systolic function of both ventricles.⁵⁴

SUMMARY

Various CVDs are strongly influenced by excess bodyweight. The concept of adiposopathy underlies the common pathogenic mechanism related to these entities. Insights into the underlying mechanisms have unveiled that increased LM drives the better outcomes observed with the obesity paradox. An effective strategy should not only target weight loss but strive to improve the LM through exercise and dietary changes. Physicians should

focus on improving CRF (and thus LM) for better clinical outcomes. We must recognize that the epidemic proportions reached by obesity are a reflection of societal conditions that extend beyond the traditional medical model. As key stakeholders, physicians can advocate for environments that promote human health rather than disease.

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