"Lose 70 pounds and you get a new knee": The current approach to obesity in patients awaiting total joint arthroplasty

Significant advancements in obesity medicine have led to evidence-based treatments for patients who were once denied care and have provided them with legitimate clinical options for managing both obesity and arthritis.

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ABSTRACT: Obesity is a risk factor for osteoarthritis. Excess weight has been associated with increasing joint symptoms and the need for joint replacement surgery. Obesity is also a contributing risk factor for postoperative complications associated with total joint arthroplasty. Between 2020 and 2021, more than 100 000 total joint arthroplasties were performed in Canada. The demand for total joint arthroplasty among patients with obesity continues to increase. Despite higher complication rates, total joint arthroplasty is a cost-saving measure in patients with obesity. To mitigate the risk of complications, physicians encourage weight loss in patients with obesity before recommending total joint arthroplasty. This often involves just telling patients to "lose weight," which has marginalized and stigmatized a patient population and left them to manage their own health. Obesity medicine—pathophysiology and treatment—has expanded significantly over the last decade. This has led to evidence-based treatments and has allowed patients who were once denied care to be provided with legitimate clinical options for managing both obesity and arthritis. Our system is changing. Our bias is real. The science is undeniable, and the call to action has come to us all.

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Beatrice is a 70-year-old woman with severe bilateral knee pain. She presented to her family doctor with a story of progressive immobility over the last 4 years. Although she was very active during much of her life, over the last decade, her knees began to hurt. She became less mobile. She has taken to swimming instead of walking, and she continues to swim 4 days per week. X-ray showed she has significant arthritis of both knees, and her right knee is worse than her left. She was referred to an orthopaedic surgeon for assessment for possible bilateral total knee replacement. Her X-rays confirmed that her left knee has end-stage osteoarthritis that can benefit from a knee replacement as soon as possible; the right knee is likely soon to follow.

Beatrice's height is 170 cm (5 ft. 6 in.), her weight is 133 kg (293 lb.), and her BMI is 46 kg/m^2. She carries her weight quite uniformly.

An orthopaedic surgeon has told Beatrice if she loses about 33 kg, her knees will be replaced. Her goal weight before surgery needs to be 100 kg.

"Just lose weight" has been the constant refrain we have told patients with obesity for decades. "Just lose weight, and they will replace your hip/knee." "Just lose weight, and they will fix your abdominal hernia." "Just lose weight, and your diabetes/hypertension/sleep apnea will get better." "Just lose weight, and they will list you for a kidney transplant."

When did this shift happen? When did medicine ask patients to bear the sole responsibility for access to life-changing, and in many cases lifesaving, care?

The weight of the matter

Worldwide, more than 240 million people have symptomatic and activity-limiting osteoarthritis of their hips or knees. More than half of patients with osteoarthritis of the knee will undergo a total knee arthroplasty during their lifetime.[1] In Canada, arthritis affects more than 6 million people. More than 23% of women and 17% of men in Canada live with arthritis.[2]

Between 2020 and 2021, 55 300 total hip arthroplasties and 55 285 total knee

arthroplasties were performed in Canada; 8892 total hip arthroplasties and 9093 total knee arthroplasties were performed in BC. In the year prior to the onset of the COVID-19 pandemic (i.e., 2019–2020), 63 496 total hip arthroplasties and 75 073 total knee arthroplasties were performed in Canada; 9945 total hip arthroplasties and 11 469 total knee arthroplasties were performed in BC. Almost 70% of those surgeries were a result of osteoarthritis, and approximately 55% to 56% of patients were women.[3]

More than 650 million people worldwide have obesity,[1] currently defined as a BMI greater than 30 kg/m^2. Obesity is a risk factor associated with the development of osteoarthritis and is overrepresented in patients who present for total joint replacement surgery. Excess weight has been associated with increasing joint symptoms and the need for joint replacement surgery.[4,5]

In Canada, 73% of men with arthritis have increased weight or obesity compared with 59% of men without arthritis. Likewise, 61% of women with arthritis have increased weight or obesity compared with 43% of women without arthritis.[2,3]

Obesity is also a contributing risk factor for postoperative complications associated with total joint arthroplasty. Patients with a BMI greater than 40 kg/m^2 have increased risk for surgical site infection, dislocation, early loosening of prostheses, revision surgery, periprosthetic fracture, and thromboembolism after total joint arthroplasty compared with those patients who have a lower BMI.[4,6-9]

The demand for total joint arthroplasty among patients with obesity continues to increase. Despite a higher risk of complications, patients with obesity experience similar improvements in function and quality of life after total joint arthroplasty as those without obesity.[10,11] Despite higher complication rates, total joint arthroplasty in general and total knee arthroplasty in particular are cost-saving in patients with obesity—even in patients with a BMI greater than 40 kg/m^2.[10,11]

To mitigate the risk of complications, many physicians and surgeons encourage weight loss in patients with severe obesity before recommending total joint arthroplasty. Most hospitals in the US have set a "cutoff" BMI of 40 kg/m^2, above which they refuse to offer arthroplasty due to the preoperative risk. This arbitrary cutoff has been based primarily on a "cost decision," with additional pressure from insurance companies on American hospitals and the need to reduce hospital stay time and complications in order "to stay competitive."[6,12,13]

Only 8% of patients who are denied surgery for a high BMI eventually reach the BMI cutoff and have total joint arthroplasty.[14] Without a reliable pathway for weight loss, should we categorically withhold an operation that improves pain and function for patients in all BMI classes to avoid risk?

Is it risk or is it bias?

All surgeries involve risk. As clinicians, we accept a certain amount of risk in our practices. Should a patient's size be held to a higher standard than other risk factors? Could weight bias be influencing our clinical decision making beyond the data on risk?[14,15]

BMI is a weak risk factor for several reasons, which are discussed below. Despite its flaws, increased BMI is comparable in magnitude to other risk factors that are commonly accepted in all surgeries. In studies with more than 5000 patients, odds ratios for any complication in patients with BMI greater than 40 kg/m^2 range from 1.18 to 1.47. Patients older than 80 years of age have odds ratios of 1.94, and an American Society of Anesthesiologists score greater than 2 gives an odds ratio of 1.49. In terms of specific complications, the data are similar. In total joint arthroplasty, the odds ratio for infection in patients with a BMI between 40 and 50 kg/m^2 is 3.2, which is comparable to an odds ratio of 3.1 in patients with diabetes.[7,8,16,17]

Benefits and risks of total joint arthroplasty

Patients with significant obesity (defined as a BMI greater than 40 kg/m^2) have lower baseline function and mobility. After successful and uncomplicated total joint arthroplasty, they have equal or greater changes in validated outcome scores, improved function scoring, and patient satisfaction relative to patients with BMIs lower than 40 kg/m^2, despite having a higher risk of complication.[10,11,18]

The establishment of an exclusion criterion of a BMI greater than 40 kg/m^2 draws on both a consensus document from a group of leading experts at the American Association of Hip and Knee Surgeons and a body of research that demonstrates that obesity presents an independent risk for complications following total joint arthroplasty. This has created barriers that limit surgery for patients who are most in need of these procedures.[19]

Problem 1: The BMI

Obesity is a disease. But our diagnostic tool (BMI) is antiquated and inappropriate. We measure the height and weight of a patient, look up the numbers on a chart, and boom: disease. Nowhere else (except perhaps in dermatology) has a spot diagnosis been so overused. Other systems, such as the Edmonton Obesity Staging System, have been proposed, but with all of them, we still compare the new system to the BMI.[20]

The BMI was invented sometime between 1830 and 1850, at a time when "social physics" was becoming increasingly popular in the scientific community. Its creator was Adolphe Quetelet, an astronomer and mathematician who never studied medicine. He never intended the BMI to be used in a medical context. "Quetelet's Quotient," as it was first called, was established by taking the heights and weights of soldiers from the Scottish and Flemish armies and using them to create the statistical formulary of *l'homme moyen*—the average man—who, to Quetelet, represented a social ideal. This formulary, unchanged, is what we now refer to as the BMI.[21,22]

It was not until the early 20th century that weight was considered a primary indicator for health. In the late 1940s, American life insurance companies began to compile

tables of height and weight to determine premiums for future policyholders. Like Quetelet's Quotient, these actuarial tables represented only a small set of people: those with the resources to purchase a policy. By the early 1950s, these actuarial tables found their way into mainstream medicine. Dr Ancel Keys used the BMI as a measurement of "the overall size of the population" in his seven-continents study.[20]

From there, the BMI became well established in the clinical setting. In 1985, the National Institutes of Health established the BMI as the clinical tool for diagnosing obesity. Over the next 20 years, the World Health Organization and every other national governing body followed suit, using the BMI to diagnose obesity in adults and children. The basis of this research continued to be unfounded and flawed, but the horse had already left the barn.[21-23]

And so, the use of BMI as an isolated measurement of orthopaedic surgical risk is founded on the very basics of biased evidence.[24]

BMI does not account for age, gender, fat distribution, metabolic risk, muscle mass, body frame size, or fitness, all of which are important when calculating perioperative risk and technical challenges associated with any orthopaedic surgery, especially joint replacement.[24-26]

Problem 2: A knee is not a hip; a man is not a woman

In orthopaedic studies, surgical risk often groups all joint replacements together. We know that a knee is not a hip. Some studies go so far as to group all orthopaedic surgeries together. If a knee is not a hip, certainly neither is a back.

This is further complicated by grouping men and women together in such studies. For example, there is a higher rate of complications related to increased weight in women who have total hip replacements compared with men. Women tend to carry weight around their hips; therefore, the location of fat tissue would impact infection rates.[8,25,27] There is increased surgical risk in this population, but such "broad strokes" in interpretation of the data lead to dangerous sweeping exclusions of patient populations. A more individualized evaluation of each patient's risk and benefit is necessary. Medicine, by its nature, evaluates a risk-benefit ratio at all times. Although an increased risk from total joint arthroplasty in patients with significant obesity has been adopted, there is a volume of data to support significant benefit of the surgery despite the increased risk.[11]

[T]he use of BMI as an isolated measurement of orthopaedic surgical risk is founded on the very basics of biased evidence.

Problem 3: Risk is not absolute—it is nuanced

Hard BMI cutoffs oversimplify preoperative risk assessment. Greater muscle mass mitigates the health effects of BMI, and in knee replacement, thickness of prepatellar fat predicts complications better than BMI.[20,24,27] A much more sophisticated and accurate way to assess preoperative risk is to use risk calculators, which consider BMI, demographics, and other comorbidities.[8]

In all the studies that examined BMI and risk of joint replacement surgeries, patients were grouped together based on a clustering of BMI at every 5 points. The flaw is that most of those studies grouped together all patients with a BMI greater than 40 kg/m^2. Does a patient with a BMI of 41 kg/m^2 have the same risk as one with a BMI of 60 kg/m^2?[26]

In addition to evaluating body habitus and BMI, a preoperative risk assessment should include an evaluation of comorbidities. Addressing a patient's glycemic control, sleep apnea, thrombotic risk, fitness, and hypertension prior to surgery will affect their postoperative complications. These are modifiable risks that can be addressed independent of weight loss.[8,20]

Denying access to care

A BMI cutoff is dangerous medically because BMI does not solely or strongly predict complications.[5,17-19]

In the Veterans Health Administration, enforcing a strict BMI eligibility criterion of 40 kg/m^2 would deny complication-free surgery to 14 patients in order to avoid one complication; for a BMI cutoff of 35 kg/m^2, it would be 16 to 1. To put this into perspective, if you flipped a coin to determine surgical eligibility, it would be 19 to 1.[9]

In short, by grouping together all patients with a BMI greater than 40 kg/m^2, men and women, and hip and knee surgeries, we have oversimplified a complicated risk assessment and thus excluded a population whose risk-benefit ratio of surgery likely favors surgery.[9-11]

The solution

In medicine, there are two approaches to changing risk in the delivery of care: change the disease or change the system.

Option 1: Change the disease

In the last decade, there has been significant improvement in our understanding of energy regulation in the body. An overwhelming amount of research has shown that complex genetic and physiological factors are involved in weight gain and neurohormonal energy dysregulation. This has led to the establishment of evidence-based treatments that offer legitimate, safe, and significant weight loss.

Goals of perioperative optimization: The goal of treatment, or surgical optimization, should consider three factors or surgical risks and address each one:[22,28,29]

- Address malnutrition.
- Reduce comorbidities.
- Focus on weight loss as a percentage of body weight rather than an absolute number.

Address malnutrition: A large percentage of patients with obesity have malnutrition due to an inappropriate starvation response. Patients with obesity have a fourfold

increased likelihood of having hypoalbuminemia.[23,30,31] Low serum prealbumin and albumin are predictors of poor surgical outcomes. Some data suggest that improvement in this marker of malnutrition translates to better surgical outcomes. Therefore, part of the preoperative assessment should include a review of the patient's albumin, vitamin D, and iron levels. Vitamin D and iron can easily be supplemented, and patients can be counseled on protein intake.[28]

Reduce comorbidities: Mitigating perioperative risk has been a cornerstone of surgical optimization since the first preoperative clinics were established by Dr Alfred Lee in the 1940s.[32,33] The benefits of and evidence for preoperative optimization have been well established in literature and practice since 2000. Preoperative clinics are now mainstream across North America and Europe; they focus on assessing and treating cardiovascular risk factors and comorbidities to lower a patient's overall surgical perioperative risk.[29]

Develop a better understanding of weight and weight loss: Our approach to weight loss in the past was ineffective, because we came to it with a primitive understanding of body energy regulation. The entire premise of weight loss prior to the turn of this century was simple. We believed that weight gain occurred as a result of excess caloric intake. Our physiological understanding was wrongly based on the model that human beings, like machines, have a specific rate of energy function—we need a certain amount of "fuel" to function. If we consume more of the fuel than we need, we store it; if we consume less than we need, we burn it. And so, the "calories in, calories out" model persisted, and our approach to treatment of obesity was simple: "Eat less; move more."

We were wrong.[22,34,35]

Volumes of data have confirmed the complex genetic and neurohormonal dysregulation that causes one person to gain weight or maintain a higher body weight and another person not to do so. To date, more than 5500 genes have been implicated in both monogenic and polygenic patterns that predispose people to inappropriate weight gain.[22,36]

It is now well understood that there is a series of hormonal connections between gut, brain, fat, and muscle tissue. The gut produces "hunger" and "satiety" hormones; fat and muscle tissue produce "storage" and "usage" hormones. All of these hormones send feedback to the brain, which then communicates with our tissues to regulate how much we eat, how much we use, and how much we store. Although this is a simplification of highly sophisticated neurohormonal feedback, we now understand that brain function is based on both homeostatic and hedonic drives toward food intake that are rooted in evolutionary biology.[34,35,37] We evolved highly sophisticated endocrine systems to prevent starvation. When the brain thinks it is starving, it will store fat and hunt food.[34]

In short, obesity is a not a function of the simple behavior of excess consumption; it is the result of an inappropriate activation of the physiological starvation response that exists in all mammals and is a function of complex neurohormonal feedback.

Although the physiology of fat tissue regulation is complex, the principal goal of treatment is to essentially "trick" a starving brain into believing it is not starving. When this happens, the body no longer stores fat inappropriately or hunts food inappropriately. Clinically, this translates into weight loss; symptomatically, it translates into less food thought, less hunger, and fewer food cravings.[35]

Weight loss mechanisms: There are potential benefits for patients if obesity is treated before total joint arthroplasty. Studies of diet-induced weight loss before total joint arthroplasty report improved outcomes compared with treatment as usual but tend to include patients with a lower BMI and involve short-term follow-up.[38]

Emerging data have shown that medical management of weight loss is effective and sustained as long as the treatment is continued. We understand this principle in other areas of chronic disease management. Hypertension and dyslipidemia are well managed when patients continue their antihypertensives and statins, respectively.

In Canada, four medications have been approved for treatment of obesity: orlistat, liraglutide, semaglutide, and naltrexone-bupropion. All of the studies on these medications were based on randomized placebo-controlled trials, which supported their use. Their efficacy ranges from 5% to 20% body weight loss.[37]

Orlistat (120 mg three times per day) is a selective inhibitor of pancreatic lipase and was approved as pharmacotherapy for obesity management in Canada in 1999. Although its use has resulted in 3% to 5% body weight loss in patients in randomized controlled trials, it is rarely used in obesity treatment at present.[39]

Liraglutide (3.0 mg subcutaneously daily) is a human glucagon-like peptide 1 (GLP-1) analog that acts centrally on the pro-opiomelanocortin or "fullness" neurons in the hypothalamus. It also increases insulin release and suppresses glucagon during times of glucose elevation. Clinically, liraglutide improves satiety and reduces hunger because there are GLP-1 receptors throughout the brain, liver, and gut that affect both hedonic and homeostatic neurohormonal feedback.

Liraglutide was first approved in Canada in 2010 for the management of type 2 diabetes, and in 2015 for the long-term treatment of obesity. In randomized placebo-controlled trials on liraglutide versus placebo, 63.2% of patients on liraglutide had lost at least 5% of their body weight at 1 year, 33.1% had lost more than 10%, and 15.0% had lost more than 20%. Both the treatment group and placebo group were on dietary management. The amount of weight loss in the liraglutide group was more than double that in the "diet alone" group, which shows the superiority of medication in addition to diet alone.[40]

Semaglutide (2.4 mg subcutaneously once per week) is a human GLP-1 analog. Like liraglutide, semaglutide acts centrally on the hypothalamic pro-opiomelanocortin

neurons as a "fullness signal." Semaglutide increases insulin release and suppresses glucagon during times of glucose elevation. It was approved in Canada in 2018 for the management of type 2 diabetes at a dose of 0.5 or 1.0 mg weekly, and in 2022 at a dose of 2.0 mg weekly. Semaglutide was approved in Canada in 2021 for long-term obesity management at a dose of 2.4 mg weekly in people with or without type 2 diabetes. In a randomized placebo-controlled trial, use of semaglutide 2.4 mg resulted in 14.9% weight loss at 68 weeks compared with 2.4% with placebo in patients with obesity. Both groups had health-behavior modification.[41]

The naltrexone-bupropion (16 mg/180 mg twice per day) sustained-release formulation was approved in Canada in 2018 for long-term obesity management, at a dose of 16 mg naltrexone and 180 mg bupropion twice daily. In a randomized placebo-controlled trial of patients with obesity but not diabetes, use of naltrexone-bupropion 16 mg/180 mg twice per day was associated with weight loss of 6.1% versus 1.3% in the placebo group. In the naltrexone-bupropion treatment group, at least 5% weight loss was recorded in 48% of patients and at least 10% was recorded in 25% of patients, compared with 16% and 7% in the placebo group, respectively.[42]

When the anatomy of a tissue is changed, its hormonal signaling changes. Bariatric surgery was once thought to be effective for weight loss because of its "restrictive and malabsorption" effects. Further understanding of physiology has shown that bariatric surgery is truly a metabolic surgery that causes significant elevations in fullness hormones and changes in bile salts and gut flora. All of these hormonal and metabolic shifts happen because of the alteration in the "gut landscape." Metabolic changes are responsible for the shift in starvation response and significant positive effects on patients' metabolic comorbidities.[43,44]

The bariatric surgical procedures currently performed in Canada are sleeve gastrectomy, Roux-en-Y gastric bypass, biliopancreatic diversion with or without duodenal switch, adjustable gastric banding, single anastomosis gastric bypass, and single anastomosis duodenoileostomy with sleeve gastrectomy. Bariatric surgery offers between 30% and 70% body weight loss depending on the procedure. Most long-term data have been recorded in the population of patients who have had Roux-en-Y gastric bypass.[45,46]

If we, as a medical system, truly want to be better and do better, we need to examine our old ways, dismantle their origins, and use our science to build a better way.

Option 2: Change the system

Beatrice has to lose weight in order to have surgery. What if her surgeon was retrained and better equipped to deal with hips and knees of women with obesity?

A different way to address the issue of risk may be to change the system in which we practise. Obesity, by any definition, is not going away. In addition to broadening the term of perioperative optimization, we can, as a medical community, subspecialize our obesity joint replacement. Recent data point to the importance of surgeon experience in reducing risk in patients with increased BMI who undergo total joint arthroplasty.[47,48]

In a population-based cohort study of 4781 patients, the volume of obesity-specific procedures was associated with fewer major surgical complications after total hip arthroplasty performed in patients with severe obesity (reduction in risk by 35% for every 10 additional patients).[47]

In addition to optimizing the patient, it is clear that there is an opportunity to optimize the system. Increased training in total joint arthroplasty in patients with obesity allows for the development of centres of excellence for patients with a BMI greater than 50 kg/m^2.

Summary

Until now, our view of a patient population has been based on our own weight bias and very little rudimentary data. The science is mounting to support more comprehensive evaluations of risk in patients with obesity and more comprehensive management plans for mitigating risk prior to surgery and in the operating room. This is the beginning of a change in how we approach patients with obesity in the orthopaedic world and how we as a medical community move forward.

If we, as a medical system, truly want to be better and do better, we need to examine our old ways, dismantle their origins, and use our science to build a better way. Never before have we known more about human physiology, disease, and treatment. Never before have we had such technology and advancements to treat disease. Never before has there been such a demand for compassion, inclusivity, and empathy. It is our obligation and our imperative to repair the future by changing our present practice. ■

Competing interests

Dr Zentner is founder of the not-for-profit organization No Fat Shame. She has received honoraria and speaking fees from Bausch Health, Novo Nordisk, Eli Lilly, and Valeant, and an unrestricted educational grant from Bausch Health and Novo Nordisk.

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