Transcript Details

This is a transcript of a continuing medical education (CME) activity. Additional media formats for the activity and full activity details (including sponsor and supporter, disclosures, and instructions for claiming credit) are available by visiting:
https://reachmd.com/programs/cme/treating-obesity-as-a-chronic-disease/16389/

Time needed to complete: 60 minutes

ReachMD
www.reachmd.com
info@reachmd.com
(866) 423-7849

Treating Obesity as a Chronic Disease

Announcer Open:
Welcome to CME on ReachMD. This activity titled, Treating Obesity as a Chronic Disease is provided by Clinical Care Options LLC in partnership with Practicing Clinicians Exchange, ProCE LLC, and Q Synthesis, and is supported by an educational grant from Lilly. Prior to beginning the activity, please be sure to review the faculty and commercial support disclosure statements, as well as the learning objectives.

Dr. Tiso:
Hello and welcome to this presentation on treating obesity as a chronic disease. My name is Dr. Susan Tiso, I'm a Family Nurse Practitioner and retired Professor from the University of California Irvine. I'm excited to have esteemed faculty with me here today, Caroline Apovian, Professor of Medicine at Harvard Medical School and co-Director of the Center for Weight Management and Wellness at Brigham and Women's Hospital in Boston, Massachusetts. And Donna Ryan, Professor Emerita at Pennington Biomedical Research Center in New Orleans, Louisiana.

And the correct answer is: Use shared decision-making strategies to discuss weight options for weight management, or weight loss, including pharmacotherapy.

And the correct answer would be C; a 32-year-old woman with a BMI of 30 and no comorbidities.

And now I'm going to hand it over to Dr. Donna Ryan.

Dr. Ryan:
Thank you so much, Susan. You know, if obesity is a chronic disease, it's got an etiology, right? And that's what we're going to talk about first. Here's what's going on in the United States, we're all aware of this from our practices. But if you look at the dotted line in orange, that's women with a BMI of 30 or higher, and this dotted line is men with a BMI of 30 or higher. You can see that there's an uptick that began after that 1980 INHANES survey, and it's gone up, up, up ever since. So, at the last INHANES survey, there are 42.4% of US adults who have a BMI over 30. And if you look at the very bottom of this chart, you can see that there's also been an increase in BMI of 40 or higher, and that is currently 9.2% of the US population. So, we've got a major problem on our hands. What is driving this obesity epidemic?

Well, the first thing to understand about obesity is that our genes are very important in driving risk for developing excess abnormal body fat in an obesogenic environment. So, what I'm showing you here on the bottom are monozygotic twins. These are identical twins. They share 100% of the same DNA. And I want you to notice how much concordance there is in body habitus among each member of that twin pair. But then look at the dizygotic twins on the top picture. They share 50% of the same DNA and look at how much discordance there is in body habitus among each member of the twin pair. So, what these genes are, they're common polymorphisms. Over 200 have been associated with BMI, over 100 with waist circumference, and what they do is, as we aggravate them, they put us at greater risk for developing obesity.

But, you know, our genes haven't changed. What has changed is the environment. And so, this obesogenic environment is what is driving that obesity epidemic that I showed on the first slide. So yeah, genetic susceptibility combined with an obesogenic environment gives rise to the phenotype of obesity. It's not quite as simple as it seems because there are many, many factors that drive risk for

obesity, including some of the ones shown here.

So, stress is important, financial stress, emotional stress can drive weight gain in susceptible individuals. Medications we're prescribing for mental health disorders, for contraception, for chronic hypertension, those medications can drive weight gain. So, there're many, many factors that give rise to this obesity epidemic on a population basis. But you know, on an individual basis, it's very important to recognize the social determinants of health because these play a major role in risk for obesity. So, individuals who have low socioeconomic status, low levels of education, low income are at increased risk for obesity. Those who live in an environment where there's a lot of food insecurity, where there is lack of access to healthy foods, where there's lack of access to an environment where you can be physically active safely, those drive risk for obesity. So, also, minority status can drive risk for obesity.

So, obesity is associated with environmental factors, with iatrogenic environment, the things that we're prescribing that drive weight gain, and also many social factors. So, to show you the power of obesogenic environment, I want you to look at this slide, and look on the left. What this is, is it's a survey of young adults who were followed for 18 years, so they were age 20 when they started the survey, and then they were age 38 when they stopped. And they self-reported height and weight and from that we calculated BMI and followed what happened to their weight over that time period. And as you can see for men and for women, there's this slow steady weight gain over young adulthood. That is the power of this obesogenic environment. So, what's going on there is there's more than 20 pounds of weight gain that's occurring in that period on average. But what's important is that almost everybody is susceptible to some degree of weight gain. Ninety-five percent of individuals will gain at last one BMI unit during this period. Some individuals gain a lot more, they're more susceptible to those environmental forces.

So, another way of looking at it is with the NHANES surveys. Here, I'm showing you women in orange and men in black, and as you can see between the survey done in 1999-2000, to that in 2015-16, there's an increase in average BMI for both men and for women. That is the power of the obesogenic environment. You know, so that's great for the population, but you know we're not treating populations, we're treating individuals. And so, when we look at weight across the lifespan, we generally see something like this: we gain weight due to multiple factors, not working the night shift. Pregnancy is associated with weight retention and can be severe in some women. Antidepressants, depression, stress, all these things can drive weight gain.

This individual had episodes of weight loss, but they were almost always followed by regain of that weight, and over the lifespan a trajectory that shows weight gain. So, what's going on there? The reason that obesity is a chronic disease is that the body is defending its highest fat mass, so as we gain weight, our body defends this new weight as a new normal weight. As we gain a little more weight, our body defends that weight as another new normal weight. So, when we try to lose weight, we are going to face metabolic and biologic adaptations. So, Caroline, why don't you just say a bit about the tug-of-war about weight management.

Dr. Apovian:
Thank you, Donna. And that was a great introduction to this whole reason for the increase in prevalence of obesity that Dr. Ryan has talked about that's happened really since the 1960s. What is going on here? Well, what's going on is that, you know, our metabolic center in the brain, which is in the arcuate nucleus, protects our body fat mass and its very tightly regulated. And if it's so tightly regulated, why is it that we're all gaining weight in this environment? Something happened to create a dysfunction and we think it is something in the environment, the ultra-processed food, the high sugar high fat content of the food, the palatability of the food, maybe a preservative in the food that caused inflammation so that the brain can't tell, can't get the signals that you're full. So, for whatever reason, the brain is defending more fat than is healthy for each individual. And when we think about the tug-of-war – so if – and many of us do try to lose weight with dieting, which reduces calorie intake, and increased activity. So, when you start to reduce your calorie intake and increase your activity, you certainly do lose weight. We typically say if you reduce your calorie intake by 500 to 1,000 calories a day, you will lose 1 to 2 pounds of weight per week. But that doesn't say anything about what's happening with the tug-of-war, because if you do that you should lose weight and keep it off. But what happens is your body changes its gut hormones that basically think you're starting to starve, because the brain is saying well, you know, we're reducing body fat here, lectin levels are dropping. And what does the body try to do when it thinks you're starving, it decreases metabolism. How does it do that? It decreases basically the calories you burn with low-level work from muscle. So, it decreases metabolism by increasing the efficiency of muscle doing low-level work, like walking around and we walk around every day, all the time.

And so, we're burning less energy. Metabolism drops, and what also happens is we get an increase of the hunger hormone, which is called ghrelin, which is secreted from cells in the stomach, and it makes you hungry right before you eat. It's a short-term hormone that makes you eat. We also see a decrease in the fullness hormones, leptin, PYY, GLP-1, and GIP and oxyntomodulin. We know all of the fullness hormones. We know there must be more hunger hormones, we haven't discovered them yet. So, the reduction in those hormones usually tell the brain it's time to stop eating, and now those fullness hormones have been reduced.

And so, this is the tug-of-war. You try to lose weight and then the factors, the counter-regulatory hormones, change and you regain the

weight back. So, what are the alterations in appetite regulation? An increase in ghrelin, decrease in GLP-1, GIP, CCK, PYY, insulin and amylin, and decrease in resting total energy expenditure from an increase in muscle efficiency, which is related to leptin levels. That is what happens.

Okay, so we really need to change our way of thinking because of all this research that has taught us about the strong biologic forces making weight loss difficult and regain easy. It's not about willpower. If you understand the tug-of-war, it's not about willpower. Are there people who can lose weight and keep it off? Yes, there are. We know that from the weight loss registry. Ten thousand patients who've kept 50 pounds off for at least 10 years, what do they have in common, they're basically vigilant about every calorie they put in their mouth, and they exercise religiously, eat breakfast every morning, and weigh themselves every day. Most people don't have the time to do that, and it requires a vigilance to overcome these biologic forces, so we can't expect people to do that all the time. It's not about willpower.

Dr. Ryan:
That's right, Caroline. And I think it's our duty to explain to patients these physiologic adaptations that occur when they try to lose weight and to explain to patients that it's not their fault, its their biology that makes it so difficult to achieve and sustain weight loss. So, it's absolutely one of the foundations in understanding obesity as a chronic disease.

So, let's talk about how we diagnose obesity. You know, on a population basis, BMI is a really good measure. It corresponds well to body fat. And so, when we look at populations, we have a cut point of the BMI of 25, that's the overweight category, and a BMI of 30 or higher as the obesity category. So, those are good cut points on a population basis because we see risk increase for cardiovascular diseases, for all-cause mortality. It starts to increase at a BMI of 25 and it increases more rapidly as the BMI exceeds 30. The waist circumference is another good cut point, 35 inches for women, 40 inches for men.

Now, some populations require different BMI cut points and so the BMI cut points for Asians are lower, because individuals of Asian descents have a phenotype where they tend to store fat less well subcutaneously. They tend to store excess fat viscerally and have a lot more cardiometabolic problems due to fat. So, their cut points are BMI 23 for overweight, and 25 for obesity. And similarly, the waist circumference cut points are lower than in individuals of European descent.

So, the way we use this BMI on a population level, is we use it as a guidance for how to apply our treatments. So, we're going to apply treatments that have more risk to our individuals who have higher health risk. So, a BMI of 30 or higher is the cut point for eligibility for bariatric surgery. So, it's a BMI of 30 or higher with a complication like diabetes, or a BMI of 35 without complications, that make one eligible for bariatric surgery. And our medication cut points are 27 with a comorbidity, or 30 or higher.

So, you know, this BMI has come under fire lately and there are some valid criticisms of it when we try to apply these cut points to individuals because we do run into individuals who have a BMI that's over 25, or over 30, who don't have excess abnormal body fat. They may be perfectly healthy, have normal glycemia, no cardiovascular risk factors, a normal waist circumference. We see this frequently in body builders and in athletes. So, that's one instance where the BMI does not correlate well with body fat and it's not a good measure for making a clinical diagnosis for obesity.

And similarly, we run into individuals who have low muscle mass, but excess fat mass, and this would be normal weight, but obesity because they have abnormal body fat, increased waist circumference, and they have cardiometabolic complications of obesity.

So, to define obesity, the definition of this chronic disease according to the World Health Organization is that it is excess abnormal body fat that is impairing health. So, look, as clinicians, we are not using the BMI as the only diagnosis. Yes, I know, it's in the ICD-10 codes. It's also in the ICD-11 codes that are coming. But we must apply our clinical judgement, and so, we use that BMI as a screening tool and then we evaluate the patient with a waist circumference, with cardiometabolic risk factors for assessment of other complications of obesity. The clinical diagnosis of obesity is based upon using BMI as a screener and then using our clinical judgement to assess that the patient has excess abnormal body fat that can impair their health.

You know, I think the way to think about this was so beautifully illustrated by Stephen O'Rahilly. He won the Banting Lecture Award from the ADA several years ago and he talked about the soggy bathroom carpet model of overnutrition-related metabolic disease. I love it. So, what he – you know, we all need some fat – so his metaphor for storing body fat is the bathtub.

And, you know, our energy intake is in the faucet, and the drain is the energy expenditure. And we need to have a health mass of fat to fill our bathtubs, but if we take in too much energy, we're going to overflow the bathtub and we're going to get the soggy bathroom carpet, which is cardiometabolic disease. These people with the South Asian phenotype have limited capacity to store fat in healthy depots, like subcutaneous adipose tissue depots, they have a smaller bathtub. So, I think the ability to store fat in healthy depots is genetically determined, is hormonally determined – after menopause women tend to lose the fat in their hips and thighs and get more visceral adiposity. But it's really the key to understanding how obesity drives risk for many diseases, because this ectopic abnormal body

fat looks different under the microscope than subcutaneous adipose tissue. It's full of very large adipocytes, dying adipocyte. It's infiltrated by macrophages, and it's got a much worse profile. It's more atherogenic, it's more prothrombotic, it promotes insulin resistance. It's this ectopic abnormal fat stores that are really driving all the complications of obesity.

So, obesity can affect every organ system, more than 200 diseases have been associated with obesity. How does it produce all of these things? We know there's type 2 diabetes cardiovascular diseases, GERD. There seem to be many, many complications of obesity. How is this excess body fat driving all of these? Well, in some cases it's just the burden of the fatness itself. And that's true in GERD. So, increased intraabdominal pressure promotes gastroesophageal reflux, and urinary incontinence, and around the neck, obstructive sleep apnea. But most of the time, it's the lipotoxic effects of that excess abnormal body fat that's driving risk for cardiovascular disease, diabetes, 13 types of cancers. So, this abnormal body fat produces cytokines and chemokines, adipokines that are prothrombotic, proinflammatory. There's immune dysfunction. It promotes blood pressure elevation. It produces angiotensinogen. It promotes insulin resistance. So, the limitless lipotoxic environment is very, very much involved in the promotion of cardiometabolic risk with obesity.

It's not just that there's a systemic milieu, because there is. There's a systemic milieu of proatherogenic, prothrombotic, proinflammatory factors, but there's also the location of that body fat that's really, really important. So, epicardial adipose tissue is an example I like to use. That fat is right where the coronary arteries are coursing through and it's right on top of the myocardium. So, those blood vessels in the myocardium are subjected to all of those lipotoxic effects, they're right there. And it's producing abnormalities.

So, that's the pathophysiology of this chronic disease, and it's important that we understand that if we're going to help our patients. But look, we don't need to get our patients to a normal weight loss. Modest weight loss can help patients really improve a lot of their cardiometabolic symptoms.

Caroline, would you like to talk a little bit about this?

Dr. Apovian:
Yes. So, the question is, why is modest weight loss so beneficial? You know, even bariatric surgery for patients who weigh 400 pounds, we can get them down to 300, but we don't usually get them down to a quote-unquote normal body weight. Why do we see resolution of diabetes and sleep apnea? It's because each organ system has its own differential improvement in risk factors based on the body.

So, first of all, when you achieve a 10% weight loss, there is a 30% visceral adipose tissue loss, which is – and we know this from animal models but making them gain weight and lose weight. When they gain weight, they first gain visceral fat and then adipose, and when they lose weight, they first lose visceral fat that they have. So, we're going to get more of a bang for your buck with that 10% weight loss because most of it is going to be visceral fat loss. So, we get a benefit right away in insulin sensitivity. Blood hyperinsulinemia drops because we're eating less, so less insulin is coming from the pancreas, blood glucose drops. And then we get the risk factors for thrombosis and inflammatory markers drop later, endothelial function later. But right away, we're going to get an improvement in insulin sensitivity and all of the improvements that you see with that.

So, this is good news because it's not necessary to achieve a normal body weight to achieve major health benefits. Moderate weight loss is always more achievable and more sustainable than more aggressive weight loss. More aggressive weight loss is usually obtained with bariatric surgery procedures. Ectopic and visceral body fat is mobilized preferentially because the body is doing what's right, what's health for the body.

Alright, so, what do we know about percent weight loss and therapeutic complication reduction? So, as little as a 3 – 5% weight loss has been shown to improve the risk of developing diabetes if you already have prediabetes. And we see the maximum benefit of diabetes prevention at about a 15% weight loss. For hypertension, we need to see a little more weight loss than that, maybe 5%, but even more than 15% keeps decreasing that blood pressure. Dyslipidemia, because weight loss really drives a reduction in triglyceride levels, and triglyceride levels are related to prediabetes, we see as little as 3%, but the triglycerides are still dropping in greater than 15% weight loss. Hyperglycemia, same idea, 3 to greater than 5%, about 15%. Now, when we're looking at fatty liver disease, we don't see a reduction quite so early. We really need to get to 10% weight loss, which does improve the steatosis inflammation and mild fibrosis. Sleep apnea, you also need to see at least 10% because we see little benefit with 5%. Osteoarthritis and stress incontinence, these are the more anatomic dysfunctions from obesity. You really have to get some weight off, 5 to 10%, to see the improvement in joint stress mechanics and incontinence from the sheer stress of the excess fat around the bladder.

What about GERD? Well, GERD is different in men and women. To improve GERD in males, you really have to see 10%. Why is that? There's more GERD in males, they have more abdominal fat, you really have to see 10%. For women, it's a little different because they have less push on their hiatal hernia and on their abdominal area pushed into the esophagus, so you can see as little as 5% improving that. And with PCOS, which affects about 7% of women in the United States, we see a good result starting at 5%. Why 5%? That's what you need to lower antigens and improve ovulation, and also, again, increase in insulin sensitivity.

So, we do see a differential here in terms of therapy from weight loss. Now, what do we need to do at the primary care level to treat obesity and prevent complications? We need to treat the weight first. So, we're starting to change this paradigm. The old treatment paradigm is to monitor and treat the dyslipidemia, the hypertension, impaired glucose tolerance. How do we do that? We monitor lipid panels, blood pressure, blood glucose. We provide diets for, in particular, the dyslipidemia – decreased fat diet, decreased cholesterol. For hypertension, decreased salt in your diet. For impaired glucose tolerance, decreased sugar. And then, primary cares know how to treat these three diseases with medications. Statins and fibrates, with antihypertensives, and when it comes to impaired glucose tolerance, we used to treat right away with sulfonylureas, glitazones, and even insulin.

People are still doing this today, they've got 10 minutes per patient, they're already on these medications, they're just basically monitoring the blood pressure, hemoglobin A1C and lipids, and then just re-prescribing these medications. And maybe at the end, primary cares will look at weight, BMI, and talk about diet and exercise, and maybe even think about some of the medications. But primarily, they're treating the complications of obesity. Where if we adopt the new treatment paradigm, which is treat the weight first with diet and exercise, and even some of the medications that we're starting to use for obesity, the new ones, the GLP-1's, then we can see after some weight loss what's going on with the lipids, the hypertension and impaired glucose tolerance. And we may not need to promote the use of these medications because we've treated the obesity, which started the complications we're talking about.

Alright, so there's a significant unmet need in obesity management. Why am I saying that? So, we have 42% of Americans in the United States who have obesity. We are not even putting the diagnosis of obesity in our electronic medical records. You know, when I see the patient, of course, I'm putting the diagnosis in because that's what I do, but it's not already in the record. So, that's a problem. Out of 100% of patients living with obesity, less than 40% have a diagnosis of obesity in the record. Who's getting treatment, anti-obesity treatment? Less than 20% of those 100%. And then, people prescribed an anti-obesity medication from that 100%, about 1 to 2%. If you said this about diabetes, hypertension, or heart disease, you would say this is negligence, but that's what's happening.

So, let's talk about assessment and management. How do you start treating obesity in your offices? You want to determine the degree of obesity. The best, easiest, most convenient measure we have is a height and weight and determining the BMI. We also use the waist circumference. Anything over 40 inches in men, over 35 in women, is considered an abnormal distribution of visceral body fat. Beyond the BMI, the waist circumference – so, BMI is over 25 considered overweight, BMI over 30 is obesity. Assess the comorbidities. How severe are they? Do they need to be treated in addition to weight loss or can we use the weight loss to reduce those complications? Look for medications that promote weight gain. And that in total will help you assess the risk of this patient's comorbidities.

What do you look for in your physical exam? Get the right cuff to measure the blood pressure. If the cuff is too small, you're going to get a falsely low blood pressure. Look at the back of the neck for acanthosis nigricans and skin tags to alert you to the fact that the patient has insulin resistance and may be at risk for prediabetes or diabetes. Look at your thyroid. Do an exam on your thyroid. Check the TSH. If you have any indication that there's something awry there, we always check the TSH. Sometimes we find undiagnosed hypothyroidism. And look for signs of PCOS. You've got to talk to the patient about irregular periods and maybe do some labs.

So, what labs are you going to do? Blood sugar, look for impaired fasting glucose. Do your lipids, look for elevations in triglyceride and low HDL. LFTs, abnormalities may suggest nonalcoholic hepatitis, but you can do a FIB-4 score that uses the age, platelet count, AST and ALT to decide whether or not you should do a FibroScan to look for undiagnosed NAFLD. You can do microalbumin levels in urine. The A1c, important. TSH, important. And most of the time when you have extreme obesity, your ultrasensitive C-reactive protein is going to be quite elevated. Take an obesity-focused history assessing the patient preparedness, reasons for motivations. You know, a lot of times motivational interviewing is very important for assessing if the patient is ready to undergo this journey with you.

What do you want to know about your patients? What are the patients' goals and expectations? What has the patient found hard beforehand? Understanding risk and benefits of treating the obesity now. Talk about physical activity and how much time does the patient have to embark on this journey.

Donna let's talk about treatment options.

Dr. Ryan:
Okay. Let me take it away here. Okay, so, to repeat, what we're going to do is we're going to use that BMI as a screening tool. We are going to use our waist circumference to help assess whether the patient has evidence of excess abnormal body fat that's impairing health, and if that is the case, if we make a clinical diagnosis of obesity, we want to achieve weight loss. We want all the benefits that weight loss can bring. So, let's talk about some of those treatment approaches.

This is our overall scheme. You know, as patients get more severe disease, we tend to use interventions that are riskier. Foundational to all treatments for obesity is recommendations around lifestyle, around diet, physical activity, and behaviors that can support weight loss. We can add pharmacotherapy if patients have a complication and a BMI of 27 and up, or have, with no complications, a BMI of 30

and up. For surgery, surgery is currently recommended with a BMI of 30 and type 2 diabetes as a complication, or a BMI of 35 and up with no comorbidities.

So, that's our overall scheme for how we're going to approach patients. And then, you know, I made my career based on lifestyle intervention, but most of the time all we can get with lifestyle intervention is about 5 to 10% weight loss and many patients cannot even achieve 5% weight loss. So, for patients who have complications, who have more severe comorbidities associated with excess abnormal body fat, we need to up our game. Fortunately, we're getting some medications that can help us treat this chronic disease, that can help us achieve and sustain enough weight loss to improve all of those complications associated with obesity. But in general, for patients who have more severe complications, we're going to use medications and we're going to consider bariatric surgery procedures. On average, we get about 25% weight loss with gastric sleeve, and a little bit more with Roux-en-Y gastric bypass. Gastric bands are rarely used nowadays, but sometimes we'll use devices along with medications to get enough weight loss to sustain health benefits.

I think the most important thing in setting these goals for how much weight we're trying to lose is to use percentage not pounds, because across all the BMI spectrum, the same percentage weight loss will produce the same health benefits. And we need to be upping our game for patients who have more complications. We need to be adding medications, we need to be considering bariatric surgery sooner.

So, lifestyle intervention is the foundation of any intervention. So, if you want to lose weight, you must create a negative energy balance. So, we're trying to create that negative energy balance with a diet meal-plan, but we also want to move our patients to a healthier eating pattern. You know, there's a benefit not just to weight loss, there's a benefit to a healthier diet. So, we really want to do a two-fold approach there. Physical activity is critical. It's less important in the weight loss phase, and more important in that weight loss maintenance phase. But we generally want to introduce physical activity during the weight loss phase. We want to promote the four pillars of health, healthy behavior habits, good sleep, limited alcohol consumption, stress reduction, good mental health approaches. So, the components of success are the healthy reduced calorie meal-plan, aerobic and resistance exercise, and behavior change interventions.

You know, what we're really doing when we're giving lifestyle counseling is we're helping our patients with skills training to give them the mechanisms to achieve and sustain that negative energy balance. You know, when you're losing weight, you're losing not just fat, you're losing lean mass. And that's why it's so important that we include physical activity with every one of our interventions. And I make a special point of it in these newer anti-obesity medications because when you achieve more weight loss, it's more important to preserve that lean mass.

Dr. Apovian:
Okay. So, let's talk about the various therapies. So, we have on the left, here, simple advice, advice from a dietician, internet program, self-help, towards structured programs, like Weight Watchers, multidisciplinary programs. All of these are intensifications of the lifestyle, and then we even have physician-driven individualized structured programs.

So, what is the best diet to put patients on? Well, we've had many, many studies that have shown – this is one of the first, Foster et al, low fat verses low carb. Maybe there's a little bit of difference at 6 months favoring low carb, but at the end of 12 months, and then 24 months, it looks like we're getting weight gain with both, but more weight gain with the low carb. So, we get basically equal weight loss on a low fat or a low carb diet. And then we've even found that it really doesn't matter which diet the patient's on, it matters what they can adhere to and that's what predicts success.

Reduced calorie meal-plans provide structure, but the patient needs to be happy with the reduced calorie meal-plan. So, that means a plant-based diet which is high, low glycemic index. But if the patient finds that it's very hunger-producing, then maybe a low carb diet is best. Physical activity, we always recommend 150 to 300 minutes of moderate intensity aerobic activity, but that's not going to be enough, you must engage in muscle strengthening exercise 2 to 3 days a week, especially as you get older to protect your lean mass. As you lose weight, you lose muscle and fat and the only way to decrease that total energy expenditure is building muscle. The more intense the physical activity, the more weight loss and weight maintenance you can achieve.

Intervention support can be done by either individually or in groups. Anybody who has joined a group and been successful realizes that as long as you keep going to the group, you can keep that weight off.

Alright, weight losses with a year, here, with different lifestyles. As you can see, you can get most of the time, 5% weight loss, and more rarely, 10% weight loss. But adding pharmacotherapy will increase that weight loss. That's the whole point. Let's now talk about a few cases.

Dr. Tiso:

Yeah. Thank you, Caroline. So, we're going to start with a patient case. We have George here. He's a 34-year-old male with a physically demanding job and he presents for his annual follow-up. He reports no health issues, but he failed to meet the weight requirements for his National Guard biannual checkup. Past medical history does include hypertension. His vitals reveal a BMI of 31 and a BP of 126/76. Current meds: he's on losartan 100 mg daily. Physical exam: He has a bodybuilder physique. His waist circumference is 37 inches. His labs show us an A1C of 5.4%, LDL cholesterol of 120, HDL of 44, triglycerides of 150. And his other labs are all normal.

So, for Donna and Caroline, how would you approach a patient like George? What do you think we would suggest for him?

Dr. Ryan:
You know, this is characteristic phenotype of metabolically health obesity. He does not have excess abnormal body fat that is driving ill health, he's got a lot of lean mass. Look at that waist circumference, 37. That's perfectly normal. And you know, there's absolutely no evidence that has got metabolic complications of obesity. Hypertension is fairly common. It is not always related to obesity, and I think that's the case here. But you know, we worry as this patient goes through his life, he's going to fall under the same obesogenic environment that we are all on. So, he's protected now by his job that requires a lot of physical activity, but when he retires, he's going to be at increased risk. So, we need to follow him over time to make sure that he doesn't gain fat and lose lean.

Dr. Tiso:
So, our next case is for Mona. She's a 56-year-old female who is seeing you for the first time. She reports multiple attempts at dietary management for her weight, and her husband has been complaining about her loud snoring at night. She's also noticed increased fatigue and lethargy. Her past medical history includes hypertension, depression that was diagnosed at age 48, weight gain after three pregnancies and menopause. She has bilateral knee arthritis. Prediabetes for two years. Her vitals show us a BP of 150/90, heart rate of 60. Her weight is 175, she is 5 feet 2 inches, yielding a BMI of 31. And her waist circumference is 40 inches. Her current meds include atenolol 50 mg daily, hydrochlorothiazide 25 mg daily, and paroxetine 10 mg daily. Her physical exam reveals 2 plus edema upper ankles and her labs show fasting glucose of 135 with an A1C of 6.4%. Her social history includes a diet high in sugar, high fat foods with nighttime eating, no breakfast, minimal exercise due to patellar pain. She did quit smoking three years ago, but previously smoked two packs per day, and she denies alcohol use.

So, Caroline, what's your impression of Mona? What would you do with her?

Dr. Apovian:
Well, let's go back to her factors, because here we really need to do something. She already has a BMI of 31, which is class one obesity, a waist of 40 inches, which signifies visceral body fat. But we already know that because she's got hypertension and weight gain after her pregnancies and menopause, knee arthritis and she's already had prediabetes. She's also on two drugs that promote weight gain, atenolol and paroxetine. There's no reason for someone with hypertension alone, without congestive heart failure or status post MI to be on atenolol, pick another drug. She's, I mean, basically she has diabetes. Her hemoglobin A1C is one point away from it. And on top of that, we need to talk to her about diet and exercise. She's got a pattern including nighttime eating with no breakfast, and also minimal exercise. She quit smoking and probably instead of smoking, started to overeat, and that's why she's gained all this weight. So, we need to do items that I would argue are best done by an MD. An MD in a weight management program who can put her on better drugs for her depression and hypertension. So, atenolol should be switched out for lisinopril or an ARB, and paroxetine should be switched out for Lexapro or one of the other SSRIs. She should also, and now the dietician can help put her on a better diet than what she's on, but I would argue that she needs an anti-obesity agent to help her make better choices than what she's doing right now. And she also needs to embark on an exercise program without hurting herself, and that may mean starting to swim, or water aerobics, or losing 10 or 20 pounds as soon as possible in order to reduce the pain in her knee so that she can walk and get on a bike and treadmill.

That's what I would do. Now, the other answers here are not necessarily wrong. You want to refer to dietician, you want to recommend the patient to increase exercise, you may want to start metformin. But I think the best option would be weight management, including pharmacotherapy. So, really you want to do all of these things.

Dr. Tiso:
So, following up with Caroline's suggestion, we did start her on a low glycemic eating plan. Atenolol was tapered and then ramipril 5 mg daily was started. Metformin 500 mg daily was started. She described increase fullness at two weeks the met was increased to twice daily. At visit two, bupropion 100 mg daily was started, and the paroxetine was decreased to 10 mg daily. Her mood is the same, a little more energy. The bupropion was eventually increased to 100 mg twice daily and her appetite and craving improved as well. At 6 weeks, she lost 15 pounds, her snoring and fatigue improved, her sleep study was not considered necessary at this time. A course of physical therapy prescribed to improve her knee strength, beginning with a walking program. And over 8 months, she lost 40 pounds. Her weight is now 135 pounds with a BP of 122/74, A1C of 5.7, and a glucose of 93. Her nighttime eating is under control, and her current medications, again, she's on ramipril 2.5 mg daily, met 500 mg twice daily, and bupropion 100 mg twice daily, and paroxetine 10 mg

daily. So, we really did, kind of, a lot of interventions for this patient that were successful.

Dr. Apovian:
Alright. How do you monitor progress? Frequent patient follow-up is key. We do not send patients home with a diet exercise program, and you know, semaglutide and have them come back in 6 months. We need to see them at least monthly for the first months. It's covered by Medicare and Medicaid, then at least every 3 months for follow-up. For follow-up of side effects, maybe they didn't like the diet that you put them on, maybe they hurt themselves exercising. You need to know this. You need to know side effects of medications to assess how your treatment is going. This is a chronic disease with chronic management. Regular support with long-term treatment can be very effective that way.

So, just to summarize here, obesity is a chronic disease, not a matter of willpower. It needs to be chronically managed. You must talk to patients about why it's a disease. When they understand that your body is regulating your body weight, and because of the environment, your set point is higher than is healthy, people will understand, and understand that they need help, number one, and that it's not their fault. Windows of opportunity; when patients will engage in more intensive management efforts, when they get new diagnoses or they had an MI, or they've been told that if they don't do something they're going to have to be on insulin for their diabetes uncontrolled risk factors. Feeling and function; obesity can cause a lot of dysfunction and functionality issues. No diet is superior to others. Using shared decision-making with the patient to choose which diet is optimal for the patient is the way to go. And setting achievable goals. What's achievable? Five to 10%, and now up to 15% with our newer medications, most notably, semaglutide. And go up from there based on targeted health improvements.

Encouraging patients to return by setting realistic expectations and providing support with the understanding that they have a disease will ensure that they keep coming back and get to their goals.

Dr. Tiso:
And yeah, so using shared decision-making strategies to discuss options for weight loss including pharmacotherapy is going to be our best option for this patient, and we've got lots of details on the rationale here for you to take a look at if you need further clarification.

And your best option is going to be C; the 32-year-old woman with a BMI of 30 and no comorbidities. And again, we've got – this was reviewed in the slides that we've covered, and we have rationale detail here for – to explain why that's our best option.

Thank you so much for participating and thank you so much to our wonderful faculty, Dr. Ryan and Dr. Apovian, for your really cutting-edge insight and information that you shared with us.

To earn CME and CE credit for this activity, please click on the button below.

Announcer Close:
You have been listening to CME on ReachMD. This activity is provided by Clinical Care Options LLC in partnership with Practicing Clinicians Exchange, ProCE LLC, and Q synthesis, and is supported by an educational grant from Lilly. To receive your free CME credit, or to download this activity, go to ReachMD.com/CME.

Thank you for listening.