Managing Obesity and Comorbid Conditions: A New Era of Treatment Strategies

Introduction: The Biology of Weight Regulation

Behavioral Approaches to Weight Loss

Developments in Weight-Loss Management: Medical and Surgical Options

Matching the Patient to the Treatment: Stratifying Risk in Obese Patients

Case and Panel Discussion

Questions and Answers

Post-Test and Evaluation

Faculty

Caroline M. Apovian, MD
Course Director and Program Chair
Professor of Medicine
Boston University School of Medicine
Director, Center for Nutrition and Weight Management
Boston Medical Center
Boston, Massachusetts

Harold E. Bays, MD
Medical Director/President
Louisville Metabolic and Atherosclerosis Research Center Inc. (L-MARC)
Louisville, Kentucky

Donna H. Ryan, MD
Professor Emeritus
Pennington Biomedical Research Center
Baton Rouge, Louisiana

Date of Original Release: January 2013
Expiration Date: December 31, 2013
Estimated Time to Complete Activity: 1.5 hours
Managing Obesity and Comorbid Conditions: A New Era of Treatment Strategies

PROGRAM DESCRIPTION
This supplement represents proceedings of a satellite symposium held at Obesity 2012 on September 22, 2012, in San Antonio, Texas.

PROGRAM GOAL
To update health care professionals on recent developments in the management of patients with obesity, discuss the biologic mechanisms of new antiobesity medications, and stratify patient risk to optimize treatment selection.

LEARNING OBJECTIVES
After completing this activity, participants should be better able to:
• Characterize the population of patients who are overweight and obese, with specific attention to the relationship between overweight/obesity and comorbid conditions
• Design management strategies for patients who are overweight and obese
• Describe risk/benefits of weight-loss medications in treating overweight, obesity, and comorbid conditions.

TARGET AUDIENCE
Physicians and other health care professionals who treat obesity

Brought to you by Continuing Education Alliance®

ACCREDITATION INFORMATION
This activity has been planned and implemented in accordance with the Essential Areas and Policies of the Accreditation Council for Continuing Medical Education (ACCME) through the joint sponsorship of Boston University School of Medicine and Continuing Education Alliance. Boston University School of Medicine is accredited by the ACCME to provide continuing medical education for physicians.

Boston University School of Medicine designates this enduring material for a maximum of 1.5 AMA PRA Category 1 Credit™. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

FACULTY
Caroline M. Aposian, MD
Course Director and Program Chair
Professor of Medicine
Boston University School of Medicine
Director, Center for Nutrition and Weight Management
Boston Medical Center
Boston, Massachusetts

Harold E. Bays, MD
Medical Director/President
Louisville Metabolic and Atherosclerosis Research Center Inc. (L-MARC)
Louisville, Kentucky

Donna H. Ryan, MD
Professor Emeritus
Pennington Biomedical Research Center
Baton Rouge, Louisiana

FACULTY DISCLOSURES
Boston University School of Medicine asks all individuals involved in the development and presentation of Continuing Medical Education (CME) activities to disclose all relationships with commercial interests. This information is disclosed to CME activity participants. Boston University School of Medicine has procedures to resolve any apparent conflicts of interest. In addition, faculty members are asked to disclose when any discussion of unapproved use of pharmaceuticals and devices is being discussed.

This educational activity may contain discussion of published and/or investigational uses of agents that are not indicated by the US Food and Drug Administration.


The Planning Committee for this activity included Mike Burk, Ilana Hardesty, and Jason Worster, MD, of Boston University School of Medicine; and Ruth Cohen and Margaret Inman of Continuing Education Alliance. The members of the Planning Committee have no significant relationships to disclose.

DISCLAIMER
These materials and all other materials provided in conjunction with continuing medical education activities are intended solely for purposes of supplementing continuing medical education programs for qualified healthcare professionals. Anyone using the materials assumes full responsibility and all risk for their appropriate use. Trustees of Boston University makes no warranties or representations whatsoever regarding the accuracy, completeness, currentness, noninfringement, merchantability, or fitness for a particular purpose of the materials. In no event will Trustees of Boston University be liable to anyone for any decision made or action taken in reliance on the materials. In no event should the information in the materials be used as a substitute for professional care.

HOW TO RECEIVE CREDIT
Participants wishing to earn CME credit must:
1. Read the supplement.
2. Relate the content material to the learning objectives.
3. Complete the self-assessment questions and the evaluation form online at: www.cealliance.org/credit/CEE80412

Successful completion of the self-assessment is required to earn CME credit. Successful completion is defined as a cumulative score of at least 70%.

The estimated time to complete this activity is 1.5 hours.

Release Date: January 2013
Expiration Date: December 31, 2013

After login, please select the following code: CEE80412.

For continuing medical education questions, please contact: cme@bu.edu.

For information on Boston University School of Medicine Privacy Policy, please visit: www.bu.edu/cme/policies/privacy_policy.html.
Obesity is a worldwide epidemic and one of the most serious health concerns of the 21st century—perhaps the greatest epidemic in human existence. In the United States, the prevalence of obesity has doubled over 3 decades, and present-day estimates indicate that more than one third of the adults in the United States (35.7%) are obese (body mass index [BMI] ≥30 kg/m²) (Figure 1). Obesity is particularly prevalent among women and racial and ethnic minority populations.

Defined most simply as the presence of excess adiposity, obesity has massive health consequences. It affects every organ system in the body, including the brain (Figure 2). Besides the well-known cardiovascular complications of obesity, which are directly related to the actual weight of fat mass on cardiac output as well as to the metabolic and inflammatory effects of fat tissue, obesity leads to gastrointestinal, respiratory, hematologic, and musculoskeletal disorders. Certain cancers are highly associated with obesity. Its endocrinologic impact is infamous: more than 60% of the prevalence of type 2 diabetes in the United States can be attributed to obesity. Ultimately, obesity increases total and cardiovascular mortality. Physicians in primary care and specialty practices will inevitably encounter overweight and obese patients who have pressing and long-term needs. The clinicians’ ability to identify and address these needs depends on their fundamental understanding of obesity’s biologic causes, clinical consequences, and the expanding armamentarium of weight control methods.

Pathobiology of Obesity

In decades past, many clinicians were taught in medical school that the adipose cell is a vehicle for energy storage and nothing more. Today, there is a different perception of the adipose cell; specifically, it is an active endocrine organ that communicates with gut hormones and a master regulator in the brain to control appetite and satiety. It also exerts pathologic effects on other organs and critical metabolic and immunologic processes.

Four major hormones—ghrelin, insulin, peptide YY (PYY) from the gut, and leptin from fat tissue—participate in appetite and satiety regulation in communication with each other and the central control of energy balance, the arcuate nucleus in the hypothalamus (Figure 3 on page 4). Ghrelin is a short-term appetite hormone—the “hunger hormone”—that brings on feelings of hunger at mealtimes. Secreted in the gastric

---

**Figure 1. Prevalence of Obesity Among Adults Aged ≥20 Years, by Sex and Age: United States, 2009–2010, National Health and Nutrition Examination Survey**

<table>
<thead>
<tr>
<th>Age in years</th>
<th>All*</th>
<th>Men</th>
<th>Women*</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥20</td>
<td>35.7</td>
<td>35.5</td>
<td>35.9</td>
</tr>
<tr>
<td>20–39</td>
<td>32.6</td>
<td>32.2</td>
<td>33.3</td>
</tr>
<tr>
<td>40–59</td>
<td>36.6</td>
<td>37.2</td>
<td>35.8</td>
</tr>
<tr>
<td>≥60</td>
<td>37.2</td>
<td>36.6</td>
<td>42.3</td>
</tr>
</tbody>
</table>

*Significant increasing linear trend by age (P<0.01).

Source: Ogden CL, et al. 2

**Figure 2. Obesity Affects Every Organ System**

NEUROLOGIC/PSYCHOLIGIC
Stroke, depression, idiopathic intracranial hypertension, disordered eating

RESPIRATORY
Hyperventilation (Pickwickian) syndrome, obstructive sleep apnea, asthma, respiratory failure

CARDIOVASCULAR
Congestive heart failure, hypertension, myocardial infarction, dyslipidemia

GASTROINTESTINAL
GERD, NAFLD, NASH, gastroparesis, gallstones, biliary tract disease, pancreatitis, hernias

MUSCULOSKELETAL
Degenerative joint disease, chronic back pain

CANCERS
Breast, uterus, cervix, colon, esophagus, pancreas, kidney, prostate

ENDOCRINE
Diabetes mellitus (type 2), metabolic syndrome, polycystic ovarian syndrome, hypothyroidism, infertility, male hypogonadism

HEMATOLOGIC
Deep vein thrombosis, hypercoagulable state, chronic venous stasis

GERD= gastroesophageal reflux disease; NAFLD = nonalcoholic fatty liver disease; NASH = nonalcoholic steatohepatitis.

Sources: Port AM, et al; 4; Malnick SDH, et al. 5

---
who have had gastric bypass surgery, which make successful weight loss so difficult. However, ghrelin levels decrease in persons during weight loss, which may be one reason it’s so hard to stay on a diet. Levels of PYY are significantly lower in obese versus normal-weight persons, but are elevated after gastric bypass.

Figure 3. Hormones That Influence Body Weight Regulation

The hormone PYY, secreted by the small intestine after meals, acts as an appetite suppressant that counters the appetite stimulant ghrelin.

Leptin was the original “satiety factor,” discovered to much excitement by Friedman and coworkers in 1994. This hormone is produced in adipose tissue in proportion to body fat; the more fat present, the more leptin secreted. Initially, it was hoped that exogenous leptin administration would be a “magic bullet” for curing obesity and establishing the condition as a metabolic disorder instead of a personal failing. Despite its effectiveness in mice with a genetic defect in their leptin molecule, exogenous leptin does not produce meaningful weight loss in obese humans, apparently because the brain becomes resistant to it. However, leptin is one of many neurohormonal pathways that have evolved genetically to prevent starvation and ensure survival. In addition, the hypothalamus contains neurons that can either stimulate or inhibit food intake, and many of the gut hormones exert actions on both sides of the equation. The possibilities for routes to intervention among this wealth of pathways remain promising and rational in the search for weight control treatments. Current thinking now holds that fat tissue is an active participant in weight regulation. In one current theory, it is the basis of adiposopathy, defined by Bays and colleagues as pathogenic adipose tissue whose toxicity may be worsened by fat accumulation and a sedentary lifestyle in genetically susceptible individuals. This dysfunctional tissue releases increased amounts of free fatty acids (FFAs) and abnormal amounts of inflammatory factors such as cytokines from macrophages. Such changes can promote insulin resistance in skeletal muscle and the liver, increased insulin secretion, dyslipidemia, hypertension, and type 2 diabetes—all components of the metabolic syndrome, which increases the risk of atherosclerosis (Figure 4). Muscle biopsies in obese patients show that deposits of fat are stored in liver when fat can no longer be stored subcutaneously, with some cases resulting in steatohepatitis and eventually fibrosis. Excess fat may be associated with androgen elevations in women (or decreases in men); increases in plasminogen-activator inhibitor 1, which encourages thrombosis; and asthma, as a consequence of proinflammatory changes. Even osteoarthritis may be an outcome of increased inflammation, although it is also mediated by the mechanical load of excess weight on the joints.

Muscle biopsies in obese versus normal-weight persons, but are elevated after gastric bypass.
Behavioral Approaches to Weight Loss

Donna H. Ryan, MD
Professor Emeritus, Pennington Biomedical Research Center
Baton Rouge, Louisiana

Behavioral intervention is fundamental to any weight-loss effort no matter what other modalities may be incorporated. Simply put, weight loss depends on behaviors that produce a negative energy balance and sustained weight control. Any clinician advising patients to lose weight for health benefits performs at least one of two basic roles: that of an expert coach motivating and pushing the patient to excel and/or a conductor directing the effort. In any setting, the goal of behavioral modification is to create the required energy deficit through behaviors around food and physical activity.

Roles and Challenges

The primary care practitioner (PCP) has the frontline responsibility for evaluating patients’ weight status, diagnosing overweight and obesity, and communicating to each patient the health risks of obesity and the benefits of weight loss. Most PCPs do not direct weight-loss efforts in their offices, but this may change with a report from the US Preventive Services Task Force (USPSTF) and a decision memo from the Centers for Medicare & Medicaid Services (CMS) that charges primary care clinicians with directing these efforts. The CMS will provide coverage for this service. In addition, a central role of the PCP is the selection of medications for chronic diseases and comorbidities (such as depression and diabetes), with awareness of therapies that produce weight loss or at the very least do not promote weight gain. Additionally, PCPs must skillfully manage these medications (eg, lowering antidiabetic therapy for patients beginning weight loss and adjusting medications after weight loss has been achieved).

For the specialist in obesity care, more complicated cases are the norm. The obesity specialist may be a leader of a medically directed weight-loss program or participate in a team that delivers counseling on diet and physical activity, uses behavioral modification techniques and special dietary techniques, manages complicated medication regimens, and works with surgical teams to deploy weight-loss procedures and devices.

Without question, patients and health care professionals face daunting challenges in establishing successful weight-loss efforts. Many patient-related factors affect the odds of success: motivation, confidence, knowledge, social and emotional support, comorbidities, competing commitments of time, and cost issues. Fundamentally, patient motivation is essential. Confidence generally is related to knowledge about the behaviors that can produce results. Among comorbidities that can sabotage weight loss, diabetes and mobility limitations are both formidable (although not insurmountable) barriers to physical activity. Cost concerns can be major obstacles because medications, lifestyle programs, and surgical procedures for weight loss may not be covered by insurers. However, as the toll of obesity on every aspect of health becomes more obvious, coverage is becoming available. Clinicians need to maintain empathy for their patients’ busy, complicated lives and to plan for ways to address the challenges that thwart successful weight loss.

Practitioners themselves may lack confidence and knowledge in weight-loss efforts for their patients and in counseling them on behavioral modification. They may have more experiences of failure than success, or they may be inadequately trained to implement appropriate behavioral programs and foster motivation. Providers, like their patients, may be confused by the barrage of messages in the media about quick and easy diets of the moment. Clinicians who provide weight-loss management services may face bias from colleagues and/or office staff who hold the same attitude toward obesity as the general culture. These clinicians have the real challenge of trying to fit weight-loss interventions into the usual pattern of primary care practice.

Finally, providers have labored without the benefit of clearly defined protocols to implement behavioral intervention and without an adequate selection of effective medical therapies to support behavioral change. Again, progress is being made with the USPSTF report. Further, the National Heart, Lung, and Blood Institute is updating its 1998 recommendations on identification, evaluation, and treatment of overweight and obesity in adults, which is expected to be released within months.

The Changing Landscape of Reimbursement

Acting upon the USPSTF recommendations on adult obesity, the CMS stated: “The evidence is adequate to conclude that intensive behavioral therapy for obesity, defined as a body mass index (BMI) ≥30 kg/m², is reasonable and necessary for the prevention or early detection of illness or disability and is appropriate for individuals entitled to benefits under Part A or enrolled under Part B and is recommended with a grade of A or B by the USPSTF.” Medicare and Medicaid will cover up to 20 face-to-face visits with a patient over the first year of treatment with intensive behavioral therapy. Reimbursement is dependent upon achievement of at least a 3-kg weight loss in the first 6 months of therapy (see Table 1 on page 6). An important limitation: reimbursement is not available to endocrinologists and other subspecialists.

“Strategies for successful weight loss may not be the same strategies that support maintenance of weight loss.”
Managing Obesity and Comorbid Conditions: A New Era of Treatment Strategies

Table 1. CMS: Coverage for Intensive Behavioral Therapy for Obesity (IBTO)

<table>
<thead>
<tr>
<th>IBTO consists of:</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Screening for obesity in adults using BMI measurement</td>
</tr>
<tr>
<td>• Dietary (nutritional) assessment</td>
</tr>
<tr>
<td>• Intensive behavioral counseling and behavioral therapy to promote sustained weight loss through high-intensity interventions in diet and exercise.</td>
</tr>
<tr>
<td>IBTO is consistent with the 5A framework (Ask, Advise, Assess, Assist, Arrange)</td>
</tr>
</tbody>
</table>

CMS will cover:

| • Screening for obesity in adults using BMI measurement (obese ≥30 kg/m²) |
| • Dietary (nutritional) assessment |
| • One face-to-face visit every week for the first month |
| • One face-to-face visit every other week for months 2–6 |
| • One face-to-face visit every month for months 7–12, if the beneficiary loses 3 kg at month 6. |
| Total: 20 visits over 1 year |

The challenge:

| • Service must be administered by “qualified primary care physician or other primary care practitioner and in a primary care setting.” |

Source: US Department of Health and Human Services, Centers for Medicare & Medicaid Services.

State-of-the-Art Behavioral Modification: Look AHEAD

The CMS interprets intensive behavioral therapy for obesity as consisting of:

- Screening for obesity in adults using BMI measurement
- Dietary (nutritional) assessment
- Intensive behavioral counseling and behavioral therapy to promote sustained weight loss through high-intensity interventions in diet and exercise
- Utilization of the 5A framework (Ask, Advise, Assess, Assist, Arrange)

The best study providing evidence for these concepts comes from the randomized controlled trial known as Look AHEAD (Action for Health in Diabetes), which evaluates a broad range of health outcomes of weight loss in 5,000+ overweight and obese persons with type 2 diabetes.4 In the trial’s induction phase over 6 months, participants who received intensive behavioral therapy (vs less aggressive diabetes support and education in the control group) had weekly contact with interventionists in the form of group sessions (3 per month) and individual sessions (1 per month). The universal weight-loss goal was set at 10%. After the first 6 months, the regimen changed slightly, with the number of required face-to-face visits decreasing to 2 per month (but 3 visits were still encouraged) for the next 6 months. Patients who met their 10% weight-loss goal then entered a 3-year maintenance phase in which monthly face-to-face visits were supplemented by e-mail and telephone contacts. In addition, and crucial for long-term success, was the use of refresher courses or campaigns 2 or 3 times per year. These were periods of 6 to 8 weeks where self-monitoring, meal replacements, medications (some patients took orlistat), incentive, and motivation could be reinstituted and updated for the patients.

Intensive behavioral intervention produced significantly greater weight loss than conventional diabetes support and education for each year of the trial. The mean maximum weight loss of 8.6% in the intensive therapy group was reached at year 1 (Figure 1). Thereafter, despite some weight gain, this group maintained a mean weight loss of 4.7% at 4 years compared with 1.1% in the control group (P<0.001).5

However, these are the “mean” results. Much more can be learned from the most successful “losers” and “maintainers.” The biggest predictor of long-term success in Look AHEAD was the amount of weight lost in the first year of intervention (Figure 2).6 Of participants with an initial loss of ≥10%, 42% sustained this level or better at the end of 4 years. These patients had attended more treatment sessions and reported more favorable activity and food intake at year 4. Look AHEAD has provided solid evidence that weight regain is not inevitable and that some patients do well with some predictability.

National Weight Control Registry

The National Weight Control Registry (NWCR) has provided additional data on the characteristics of successful sustained losers—those who are able to lose weight and keep it off.7 The NWCR has more than 4,000 registrants, 80% of whom are women. They have succeeded in keeping off, on average, about 66 lb for more than 5.5 years. They followed no uniform...
protocols, diets, or medical regimens to achieve the loss: some ate low-fat diets, some reduced the quantity of all types of food consumed, and some used weight-loss medication. To maintain their loss, however, there were notable similarities (Table 2): using low-fat diets, watching total calories, performing high daily levels of physical activity, and monitoring their progress frequently (ie, weighing themselves daily). The best predictor of success at long-term weight loss was the amount of daily physical activity. Successful losers averaged 400 kcal of physical activity per day—about an hour of brisk walking.

**Behavioral Therapy: What Works in the Office**

What worked in Look AHEAD and for the NWCR registrants can also work in the medical office. In all cases, behavioral counseling should be guided by these lessons: (1) strategies for successful weight loss may not be the same strategies that support maintenance of weight loss; (2) diet appears to be very important in losing weight, whereas exercise appears to be very important in sustaining that weight loss; (3) monitoring food intake appears to be important to weight loss, whereas monitoring weight daily appears to be important in sustaining weight loss; and (4) the physician’s role changes from that of an “enforcer” during weight-loss achievement to a “supporter” during maintenance.

In Look AHEAD, essential elements of behavior modification included motivational interviewing (all interventionists in the trial were trained in this technique) and educating patients about self-monitoring (food diaries, records), goal setting, stimulus control, problem solving, and relapse prevention. Above all, patients were educated about the strong biologic forces that drive weight regain to allow each patient to anticipate and try to prevent the tendency to reverse hard-won losses. The practitioner/patient relationship should be one of collaboration. The practitioner should focus on patient accountability in the weight-loss effort; any toughness must be laced with encouragement and support or the patient may choose not to return. The ideal is for the patient to want to be accountable and to arrive for visits with food diary in hand and weight loss to show for it.

Group support can also play an important role in behavior modification by providing the opportunity for patients to receive emotional and social support in the midst of their busy daily lives. They can hear themselves and others discuss “why” they should change their lifestyles, and the groups help establish “norms” and benchmarks that encourage success.

Physicians or practices may choose to refer out some or all patients for behavioral modification to self-help books or the Internet, commercial programs, community-based programs, or a medically supervised program. They may decide to embrace the effort themselves or arrive at a blended approach with some cases referred and some treated in the office.

---

**Table 2. What the NWCR Reveals About Maintaining Weight Loss**

<table>
<thead>
<tr>
<th>Characteristics (≥4,000 registrants)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maintained an average of 66-lb weight loss (range 30–300 lb) for average of 5.5 years</td>
</tr>
<tr>
<td>66% were overweight children</td>
</tr>
<tr>
<td>60% had a family history of obesity</td>
</tr>
<tr>
<td>50% lost weight on their own</td>
</tr>
<tr>
<td>4% used weight-loss medication</td>
</tr>
<tr>
<td>≥4,000 registrants (80% women, 20% men)</td>
</tr>
<tr>
<td>Women currently average age 45 years, weight 145 lb</td>
</tr>
<tr>
<td>Men currently average age 49 years, weight 190 lb</td>
</tr>
</tbody>
</table>

**How they have maintained weight loss**

- Consumed low-fat diets (24% of kcal)
- 50% reduced quantity of food consumed
- Expended 400 kcal/d in physical activity
- Walking most frequently reported

Source: NWCR. National Weight Control Registry: 1-800-606-NWCR

---

**References**


Increases in body fat can contribute to disease processes related to excessive body fat mass (adiposity) and/or pathogenic fat dysfunction (adiposopathy). Thus, an emerging concept in treating the overweight patient is to not only improve the weight of the patient, but to improve the health of the patient. Appropriate nutritional intervention and physical activity are fundamental to successful weight loss and maintenance. Unfortunately, these measures alone are often inadequate in the majority of overweight/obese patients; most require additional support to achieve and maintain significant weight loss. Pharmacologic and/or surgical approaches together with behavioral therapy can help many patients better achieve beneficial weight loss.

In 2012, lorcaserin and phentermine/topiramate extended release (PHEN/TPM ER) were approved by the US Food and Drug Administration (FDA) as weight-loss/maintenance drugs. These were the first such drugs approved since the approval of orlistat in 1999. Contributing to the relative lack of weight-loss therapeutic agents was the history of prior antiobesity drugs, which were withdrawn due to undesirable adverse experiences.

Phentermine is the most commonly prescribed central nervous system (CNS) weight loss agent. It is a sympathomimetic amine that decreases the desire for food through hypothalamic stimulation. Phentermine is a schedule IV drug and a pregnancy category X drug. Orlistat is a gastrointestinal lipase inhibitor. It is unique in that its weight-loss effects are due to impaired energy absorption, which is the result of the malabsorption of fat, and thus impaired intake of energy/calories. Orlistat is also pregnancy category X.

As with most weight-loss/maintenance drugs, the more recently approved agents primarily affect the CNS, with the main therapeutic effect being increased satiety.

**Approved Agents: Lorcaserin and Phentermine/Topiramate ER**

Lorcaserin. Lorcaserin was approved by the FDA in 2012 as an adjunct to appropriate nutrition and physical activity for chronic weight management in obese adults (initial body mass index [BMI] ≥30 kg/m²) and in overweight adults (BMI ≥27 kg/m²), with at least one weight-related comorbid condition (eg, hypertension, dyslipidemia, type 2 diabetes). Lorcaserin is a selective serotonin/5-hydroxytryptamine (5-HT)2C receptor agonist that acts via the hypothalamus in promoting increased satiety.

Approval of lorcaserin was based largely on the findings of studies such as the Behavioral Modification and Lorcaserin for Overweight and Obesity Management (BLOOM) study. This study was a phase III, randomized, controlled trial that evaluated lorcaserin (10 mg twice daily) versus placebo among 3,182 obese adults (BMI 30–45 kg/m²) or overweight adults (BMI 27–29.9 kg/m²) with at least one associated comorbidity (hypertension, dyslipidemia, cardiovascular disease, impaired glucose tolerance, or sleep apnea) over 52 weeks. At 1 year, 47.5% of patients in the lorcaserin group and 20.3% in the placebo group had lost ≥5% of their body weight (P<0.001), corresponding to an average loss of 5.8±0.2 kg with lorcaserin and 2.2±0.1 kg with placebo (P<0.001).

Among patients who continued past year 1 of the BLOOM study, those who were switched to placebo had weight regain similar to the weight of those who took placebo since the start of the study, whereas those who took lorcaserin experienced only a trending toward weight regain (Figure 1).

Equally important as the weight improvement was the improvement in metabolic health parameters. In the BLOOM study, lorcaserin improved diastolic blood pressure and reduced triglycerides by 6.15% versus 0.14% with placebo (P<0.001). From a safety perspective, lorcaserin did not result in a significant increase in cardiac valvular abnormalities, with data extending as long as 104 weeks.

The BLOOM-Diabetes Mellitus (DM) study included patients with type 2 diabetes mellitus (T2DM). In this study of 604 patients with T2DM, lorcaserin (10 mg...
once or twice daily) not only reduced body weight, but significantly reduced glycosylated hemoglobin (A1C) and fasting plasma glucose in the first year (Figure 2). This further validated the efficacy of lorcaserin to improve the weight of patients and improve the health of patients—in this case, glycemic control among patients with T2DM.

The most common adverse effects observed in clinical trials with lorcaserin were nausea, dizziness, fatigue, dry mouth, and constipation. According to the prescribing information, lorcaserin should be discontinued if a weight loss of 5% of body weight is not achieved after 12 weeks. Moreover, therapy should be discontinued and the patient re-evaluated if there are signs or symptoms of valvular heart disease. Lorcaserin has not been scheduled. However, as with all weight-loss/maintenance drugs, lorcaserin is contraindicated during pregnancy (category X).7

PHEN/TPM ER. The other weight-loss/maintenance agent approved in 2012 was a combination of phentermine HCl and TPM ER. Because it is a salt (as opposed to the phentermine resin formulation), phentermine HCl more easily dissociates in the gastrointestinal tract, contributing to short-term satiety. Controlled-release topiramate is intended to produce longer-term satiety, such that the combination would produce short- and long-term weight-loss/maintenance effects.5

Phentermine is an FDA-approved weight-loss/maintenance agent. Topiramate monotherapy is approved for seizure disorders and prevention of migraine headaches. The doses used in PHEN/TPM ER are substantially less than doses used as monotherapy for non-weight-loss goals, with the phentermine dose 40% less than the maximum dose used for weight loss and the topiramate dose 25% less than the maximum dose used for treatment of seizure disorders.10 Because of concerns of an increased risk of cleft palate with topiramate, the drug’s manufacturer has established a Risk Evaluation and Mitigation Strategy (REMS) program to help prevent fetal exposure (see Risk Evaluation and Mitigation Strategy for PHEN/TPM ER).11

With respect to weight-loss effectiveness, the CONQUER study was a phase III trial that included 2,487 overweight/obese adults with two associated comorbidities. In this study, PHEN/TPM ER 75/46 mg/d and 15/92 mg/d significantly reduced body weight (Figure 3). There was a ≥5% loss of body weight in 70% of patients taking PHEN/TPM ER full dose.12 PHEN/TPM ER improved lipid levels, and among patients in CONQUER with T2DM, PHEN/TPM ER significantly (P<0.0001) improved hemoglobin A1C and systolic blood pressure.13

The most common adverse effects associated with PHEN/TPM ER were based on the individual components. They included paresthesias, cognitive dysfunction, disgeusia, metabolic acidosis, and elevated creatinine. Like phentermine, PHEN/TPM ER is a Drug Enforcement Administration (DEA) schedule IV and a pregnancy category X drug (due to being a weight-loss/management drug and the potential risk of cleft palate). PHEN/TPM ER full dose should be discontinued if a 5% loss of body weight has not been achieved after 12 weeks. If PHEN/TPM ER full dose is to be discontinued, it should be done gradually to avoid the risk of seizures.6,10,14

*Weight change for either dose vs placebo, P<0.0001.
LOCF=last observation carried forward; MI=multiple imputation.
Source: Reprinted from The Lancet; Gadde KM, et al,12 copyright 2011, with permission from Elsevier.

More information about the REMS program for phentermine/topiramate can be accessed at: www.qsymiarems.com/Qsymia-HCP-Training-Print or by calling VIVUS Medical Information at 1-888-998-4887.
Expanded the indication for gastric and frequency of metabolic syndrome and weight loss.

Clinical trial and bupropion may have synergistic action unrelated to other antidepressants, which is an antidepressant of the aminoketone class, unrelated to other antidepressants, which is an antidepressant of the aminoketone class.

Naltrexone is an opioid antagonist further study in a cardiovascular outcome trial. Naltrexone is an opioid antagonist.

Clinical trial and bupropion may have synergistic action unrelated to other antidepressants, which is an antidepressant of the aminoketone class,

Liraglutide. Liraglutide is a glucagon-like peptide-1 agonist approved for treatment of T2DM. It also is being evaluated as a potential weight-loss maintenance agent. Liraglutide may have a number of neurologic effects that increase satiety and other effects that may improve fat function.

In a randomized controlled trial of obese patients without diabetes, liraglutide produced significantly greater weight loss compared with placebo or orlistat.

Other effects included reduction in waist circumference, systolic and diastolic blood pressure, and frequency of metabolic syndrome and prediabetes mellitus. The most common adverse events associated with liraglutide include nausea, vomiting, and diarrhea.

**Surgical Interventions**

Bariatric surgery is becoming increasingly safe, especially with the use of advanced laparoscopic surgical techniques. In addition, it is increasingly effective in improving the weight and health of patients.

With advances in reduced invasiveness of procedures, laparoscopic approaches have increased from 20% of bariatric procedures in 2003 to 90% in 2008. In addition, whereas laparoscopic gastric bypass represented 85% of laparoscopic procedures in 2003, that number declined to 55% by 2010. Conversely, laparoscopic gastric band procedures increased from 9% to 40% in the same period, and sleeve gastrectomy (more widely used outside the United States) increased from 0% to 2% of procedures.

**References**


Matching the Patient to the Treatment: Stratifying Risk in Obese Patients

Caroline M. Apovian, MD, Course Director and Program Chair
Professor of Medicine, Boston University School of Medicine
Director, Center for Nutrition and Weight Management, Boston Medical Center
Boston, Massachusetts

Awareness of models for assessing obesity-related risk of comorbid diseases is especially important now that newly approved weight-loss therapies have emerged. There are many approaches to risk assessment. The approaches reviewed in this article make sometimes novel use of key parameters of risk to guide the clinician in matching patients to therapy.

About 15 years ago, the National Heart, Lung, and Blood Institute (NHLBI) published a risk stratification scheme for overweight/obesity (Table 1) and 2 years later published a “practical guide” for treatment of obese patients. Since then, new pharmacotherapies have become available, and surgical techniques have advanced that make the guidelines for treatment obsolete. A new set of guidelines is imminent.

The NHLBI guidelines use body mass index (BMI) and waist circumference as major determinants of risk. BMI is a useful population-based tool to classify adiposity and estimate its prevalence. However, at the individual level, BMI is limited in that it cannot directly distinguish between lean and fat tissue. Thus, a given BMI, substantial variation in adiposity can occur. Furthermore, neither BMI nor waist circumference directly reflects the presence of underlying obesity-related comorbidity, reduced quality of life, or diminished functional status—elements that are critical in stratifying risk in overweight/obese patients.

Alternatives—or adjuncts—to BMI and waist circumference for risk stratification are needed. Scoring systems that incorporate assessments of comorbidity have been proposed as alternatives. For example, the Edmonton Obesity Staging System (EOSS), which was developed over several years and validated in 2011, assigns risk according to the patient’s medical profile—BMI, waist, comorbidities, and so on, but also considers mental health status (eg, depression, anxiety) and functional parameters (activities of daily living) as indicators of overall well-being (Figure 1).

Table 1. 1998 NHLBI Classification of Overweight and Obesity by BMI, Waist Circumference, and Associated Disease Risk

| Disease Risk* Relative to Normal Weight and Waist Circumferencea |
|------------------|------------------|
| BMI (kg/m²)      | Obesity Class    |
|                  | Men ≤102 cm (<40 in) | Women ≤88 cm (<35 in) | >102 cm (>40 in) | >88 cm (>35 in) |
| Underweight      | <18.5            |
| Normal           | 18.5–24.9        |
| Overweight       | 25.0–29.9        | Increased             | High            |
| Obesity          | 30.0–34.9        | I                     | High            |
| Obesity          | 35.0–39.9        | II                    | Very high       |
| Extreme obesity  | ≥40.0            | III                   | Extremely high  |

*Risk for type 2 diabetes, hypertension, and cardiovascular disease.

In an analysis using data from National Health and Nutrition Examination Surveys (NHANES), the EOSS was found to be a strong predictor of increased mortality in both the overall population of 8,000+ overweight or obese adults and a subset of individuals considered eligible for bariatric surgery. The predictive strength of the EOSS scores held independently of BMI, waist circumference, or presence of the metabolic syndrome. Compared with BMI findings alone, EOSS scores differentiated mortality curves among risk categories much more clearly (Figure 2 on page 12).

The clinical implication is that whereas BMI range from a score of 0 for a patient with elevated BMI but good function and no comorbid conditions to a score of 4, indicating severe obesity, comorbidities, and impairment of well-being.

Figure 1: The Edmonton Obesity Staging System

Figure 2: The Edmonton Obesity Staging System

Source: Padwal RS, et al.4

Stages of risk
Updates and New Developments in Risk Assessment
Ideas developed at a 2006 conference of the International Chair on Cardiometabolic Risk6 have led to a re-examination of metabolic syndrome as a risk predictor in obesity. Some maintain that whereas metabolic syndrome was a significant milestone in the search for accurate cardiovascular disease (CVD) risk indicators, an “all or none” approach to its assessment does not capture global CVD risk (or cardiometabolic risk). Novel new algorithms acknowledge that the most prevalent form of metabolic syndrome occurs in viscerally obese patients with insulin resistance. Therefore, biomarkers of visceral obesity and insulin resistance might bring greater strength to risk predictions, and assessment of “hypertriglyceremic waist” (increased girth and triglycerides) may more directly and simply represent metabolic syndrome. These ideas are in the research stage, but they reflect the acknowledged need to improve current risk assessment models.

Although the predictive power of metabolic syndrome remains under scrutiny, the NHLBI and five other guideline bodies7 have agreed on a set of cutpoints to better define the syndrome by its constellation of symptoms, including visceral fat, elevated triglycerides, hypertension, low-density lipoprotein cholesterol, and raised fasting blood glucose. Their report did not publish cutpoints for waist circumference, as no agreement was reached on this parameter. Nonetheless, central obesity is arguably the key to obesity-related risk, perhaps more than is widely appreciated. Obese persons who are unable to store excess fat elsewhere (eg, the thigh regions or subcutaneously) and who end up storing the fat wherever they can—in the liver, around the heart, in the abdominal cavity and wall, and in muscle—are at greater risk of deleterious effects on their health, despite total adiposity.

In a new analysis of NHANES-III data, reported at the 2012 European Society of Cardiology congress,8 individuals with a normal BMI but central obesity (as defined by a high waist-to-hip ratio) were found to have the highest cardiovascular and all-cause mortality risks among the patient subgroups studied (normal, overweight, and obese). The authors concluded: “Our research shows that if a person has a normal BMI, this by itself should not reassure them that their risk for heart disease is low.”9

The message to providers who are in the trenches trying to apply the most appropriate treatment modality for weight loss is that clinical judgment is still a factor in assessing an individual patient’s risk. There are no sophisticated risk engines or algorithms to quantitate an individual’s risk over the short or long term. Thus, whereas BMI may serve as a guide, the patient’s health profile is paramount in driving clinical decision making.

References
Cece, a Middle-Aged Obese Woman With Multiple Comorbidities

Caroline M. Apovian, MD
Harold E. Bays, MD
Donna H. Ryan, MD

Cece is a 56-year-old white woman who has gained weight steadily for the last 6 years. She blames the start of her recent weight gain on depression triggered by her divorce. She sees a psychotherapist weekly for the depression. Cece’s history is also significant for hypertension, dyslipidemia, and knee and toe osteoarthritis. She has joined exercise programs, but dropped out several times in the last 3 years. Eventually, Cece lost 18 lb through a commercial program and weekly swims. In the last year, she has gained 23 lb and returned to eating larger portion sizes at mealtimes and drinking sweetened beverages at her desk during the day. Cece is considering knee replacement surgery, but would prefer to shed at least the 23 lb she recently gained.

**Physical Examination**

- **Blood pressure (BP):** 149/87 mm Hg
- **Pulse:** 76 bpm
- **Weight:** 251 lb
- **Height:** 68.5 in
- **Body mass index (BMI):** 37.6 kg/m²
- **Review of systems:** history of snoring
- **Moderate swelling of right knee**
- **Remainder of physical examination is normal.**

**Laboratory Evaluation**

- **Random glucose:** 105 mg/dL
- **A1C:** 6.7%
- **Triglycerides:** 168 mg/dL
- **Total cholesterol:** 190 mg/dL
- **Low-density lipoprotein cholesterol (LDL-C):** 115 mg/dL
- **High-density lipoprotein cholesterol (HDL-C):** 48 mg/dL
- **High-sensitivity C-reactive protein (hs-CRP):** 3.10 g/L (normal <1)
- **Tests of creatinine, liver function, thyroid function, and urine microalbumin are normal.**

**Current Medications**

- Escitalopram oxalate 20 mg/d
- Rosuvastatin 10 mg/d
- Hydrochlorothiazide 20 mg/d
- Ibuprofen for knee and ankle pain
- Ibuprofen for knee and ankle pain
- Rosuvastatin 10 mg/d
- Hydrochlorothiazide 20 mg/d
- Escitalopram oxalate 20 mg/d

**Panel Discussion**

**Dr. Apovian: Dr. Bays, how would you characterize Cece’s cardiometabolic risk?**

**Dr. Bays:** Cece’s cardiovascular risk status is a serious concern, considering her current clinical profile. The influence of obesity on Cece’s cardiometabolic function is of paramount importance to her long-term health. With her excess weight, high triglycerides, elevated glucose, and hypertension, she meets the criteria for metabolic syndrome.1

If we accept that adipose tissue and adipocytes are active from an endocrine and an immune standpoint and that these dysfunctions contribute to metabolic disease and inflammation, then Cece is like many of the patients we see in clinical practice every day—her poor cardiometabolic health is a direct result of adipose tissue dysfunction.

**Dr. Apovian:** Note that Cece’s waist circumference was not recorded as a part of her physical findings. Dr. Ryan, wouldn’t this be an important component in assessing her risk?

**Dr. Ryan:** Of course, waist circumference as an indicator of central obesity is a pivotal measure in risk assessment and weight control management. However, for a patient like Cece, with a BMI above 35 and documented comorbidities, information on waist circumference adds little to the clinical picture. Accurate measurement requires the patient to change into a gown and an appropriately trained staff member must be present. However, it may be difficult to get an accurate reading in the presence of pannus. Cece knows she is obese. We know she is obese. We know she is at high risk because of her weight and comorbidities. To insist on waist measurement may create a barrier to an open, productive relationship with the patient.

However, waist measurement is highly relevant for people whose BMI is 25 to 35 and whose cardiovascular risk status is not clear from other clinical and historical findings. Waist measurement also may be particularly important if the patient is participating in a clinical trial.

**Dr. Apovian:** I agree. When you have a patient like this, she already has type 2 diabetes, hypertension, hyperlipidemia, BMI is 38, you know she’s at high risk.

Moving on, we know that weight loss of as little as 5% to 10% would likely improve all of Cece’s signs and symptoms.8 How do we help her on this path?

We must further assess her using a “global,” more comprehensive approach to identify factors that are going to thwart an effective treatment plan. These factors include, but are not limited to, sleep problems (such as sleep apnea, which may increase insulin resistance); digestive problems (eg, reflux disease); symptoms of urinary stress incontinence; mobility and physical limitations; and, importantly, concurrent medications that may promote weight gain. In Cece’s case, escitalopram oxalate promotes weight gain, and if she is treated for diabetes with a sulfonylurea or thiazolidinedione, she will likely gain more weight. A weight-neutral medication, such as a dipeptidyl peptidase-4 inhibitor or a weight-loss–promoting agent such as a glucagon-like peptide-1 analog, would be a more prudent choice.
Introduction: The Biology of Weight Regulation

References


**Questions**

**In a patient who has successfully lost the desired body weight, do weight-loss drugs have a place in weight maintenance programs?**

Yes, but adherence can be a challenge. Patients may be happy to take the medication in the first 6 months when the most weight loss takes place, but when weight plateaus, the patient may believe the drug is no longer working. Patients don’t associate weight-loss medications with long-term weight control. One solution is to try to get patients to commit to 1 year of therapy and then to commit to a strategy of intermittent therapy. If their weight increases by 2%, therapy will be reinitiated. The patient should go back on medication, back on two meal replacements a day, and back on a more vigorous exercise program. Of course, the clinical studies have demonstrated that weight-loss maintenance persists for up to 2 years for lorcaserin and phentermine/topiramate, but we don’t have data after that. Orlistat is US Food and Drug Administration (FDA) approved and has shown efficacy in weight maintenance for up to 4 years.

**Were the different weight-loss trajectories predictable by initial body mass index (BMI) in the Look AHEAD trial?**

Participants with a BMI of more than 40 lost the same percentage of weight as those with a BMI of 25 to 30, 30 to 35, and 35 to 40. Of course, they lost more kilograms because they started at more kilograms, but the proportion (%) of weight loss was the same across different BMI categories. Remember, the participants in Look AHEAD were obese when they started and most were obese when they finished their weight loss, achieving about 9% loss on average at 1 year. Analysis of the effect of 2.5% to 5% weight loss, 5% to 10%, 10% to 15%, and so forth showed a cardiometabolic benefit with even modest weight loss. The greater the weight loss, the greater the benefit, but for most risk factors even a small reduction in weight produced risk factor and symptom benefit. The message for our patients is that even if they don’t achieve a BMI of 25, they can still be much healthier.

**How do clinicians choose between lorcaserin and other existing and recently approved weight-management drugs?**

Because phentermine/topiramate and lorcaserin have not been tested in a head-to-head clinical trial, it’s difficult to facilitate the clinician’s choice of weight-management drug. In the placebo-controlled clinical trials that led to approval of these agents:

- **Lorcaserin** yielded an average weight loss that was 3% to 3.7% greater than placebo. After taking lorcaserin for 1 or 2 years, about 47% of participants without diabetes lost at least 5% of their body weight. Only 23% of patients taking an inactive placebo lost this much weight.

- **Participants taking phentermine/topiramate for up to 1 year** had an average weight loss of 8.9% over those taking an inactive placebo. Therapy with phentermine/topiramate resulted in a loss of at least 5% of body weight in 70% of patients. Only 20% of patients taking an inactive placebo lost that much weight.

These numbers cannot be used to compare the two drugs, as the clinical trials had different designs. While both medications have a favorable tolerability profile, it looks as though the tolerability was somewhat better with lorcaserin. In addition, the Risk Evaluation and Mitigation Strategy (REMS) program required with phentermine/topiramate should be considered, which is intended to inform clinicians and patients about the possibility of cleft palate and limits the availability of the agent to special pharmacies.

**Are lorcaserin and phentermine/topiramate Drug Enforcement Administration (DEA)-scheduled drugs?**

Phentermine/topiramate is a schedule IV substance because any formulation that contains any quantity of phentermine is classified as a schedule IV drug. Lorcaserin is expected to be classified by the DEA as a schedule IV agent. This status does not necessarily reflect a heightened potential for abuse or dependence; the FDA wants antiobesity agents to be handled responsibly, not indiscriminately.

**If a woman becomes pregnant while taking full-dose phentermine/topiramate, how should she discontinue use?**

Women of childbearing potential, and especially those planning on becoming pregnant, are not good candidates for weight-loss medications in general, and particularly phentermine/topiramate; that is why there is a REMS in place for it. In general, the dose of phentermine/topiramate should be tapered to every other day for a week and then stopped to avoid the risk of seizures. This does not address the risk of cleft palate in the fetus. Is it better just to abruptly discontinue phentermine/topiramate and risk seizure? The answer is not clear, but weighing the risks, because obese patients are not especially seizure prone, most physicians would stop the medication immediately or at the very least do a rapid taper.

**Is the positive effect on cardiometabolic parameters seen with these agents an effect of the drug or the weight loss itself?**

Weight loss itself is very important; dietary and lifestyle modification that produces even modest weight loss can improve cardiometabolic health. However, the Behavioral Modification and Lorcaserin for Overweight and Obesity Management (BLOOM), BLOOM-Diabetes Mellitus for lorcaserin, and CONQUER for phentermine/topiramate studies show greater improvement (albeit with greater weight loss) compared with placebo in parameters such as waist circumference and lipids and, in patients with type 2 diabetes, A1C and fasting blood glucose. The same is true of the glucagon-like peptide-1 agonist liraglutide, which does not have a weight-loss indication. The question of independent effect cannot be answered from these study designs.

---

**Answers**

Managing Obesity and Comorbid Conditions: A New Era of Treatment Strategies • www.globalacademyne.com/primarycare
Managing Obesity and Comorbid Conditions: A New Era of Treatment Strategies CME Post-Test Answer Sheet and Evaluation Form

THE SELF-ASSESSMENT QUESTIONS AND EVALUATION ARE PROVIDED HERE FOR PREVIEW PURPOSES ONLY.

HOW TO RECEIVE CREDIT: Participants wishing to earn CME credit must: 1. Read the supplement. 2. Complete the self-assessment questions and the evaluation form online at: www.ceainitiatives.org/certify/GEE0412

CME QUESTIONS: For each question or incomplete statement, choose the answer or completion that is correct. Circle the most appropriate response.

1. The proportion of type 2 diabetes prevalence attributable to obesity is estimated to be:
   A. 5%
   B. 10%
   C. 25%
   D. >60%

2. Ghrelin, leptin, peptide YY, and insulin regulate appetite via communication with the:
   A. Adrenal cortex
   B. Gastrointestinal mucosa
   C. Hypothalamus
   D. Liver

3. The satiety hormone leptin is produced in:
   A. Beta cells
   B. Fat cells
   C. Gastrointestinal mucosa
   D. The liver

4. In the Action for Health in Diabetes (Look AHEAD) study of intensive behavioral therapy, the strongest determinant of long-term weight loss was:
   A. Amount of first-year weight loss
   B. Baseline activity level
   C. Female gender
   D. Frequency of first-year group therapy

5. “Successful losers” in the National Weight Control Registry (NWCR) were very active, performing activity that amounted to:
   A. 400 kilocalories per day (about 1 hour of brisk walking)
   B. 1200 kilocalories per week
   C. Twice-weekly lap swims
   D. 2 brisk 10-minute walks every other day

6. Lorcanserin and phentermine/topiramate are both:
   A. DEA schedule III
   B. Pregnancy category C
   C. Pregnancy category X
   D. Required to have a Risk Evaluation and Mitigation Strategy (REMS)

7. Patients in the Behavioral Modification and Lorcanserin for Overweight and Obesity Management (BLOOM) study who continued lorcanserin therapy post year 1:
   A. Continued to lose weight at a moderate rate
   B. Became indistinguishable from placebo-treated patients
   C. Had some trending of weight gain in year 2, but maintained greater weight loss than placebo groups
   D. Fluctuated widely in weight for 6 months

8. Doses of phentermine in the combined formulation of phentermine/topiramate are:
   A. The same as doses used for monotherapeutic indications
   B. More than twice the doses used for monotherapy
   C. Less than half the doses used for monotherapy
   D. Titrated according to meal size

9. Naltrexone/bupropion is:
   A. A combination antiobesity agent whose components are approved for seizures (bupropion) and depression (naltrexone)
   B. An investigational agent undergoing cardiovascular safety testing
   C. Approved for obese patients with a body mass index (BMI) >30 mg/kg²
   D. Currently approved for type 2 diabetes

10. Which of the following statements about bariatric surgery is correct?
    A. Gastric bypass procedures account for 10% of bariatric procedures
    B. Gastric bypass accounts for 10% of bariatric procedures
    C. Laparoscopic procedures account for 90% of bariatric procedures
    D. Sleeve gastrectomy is now the most popular bariatric procedure

11. In the classification scheme of the National Heart, Lung, and Blood Institute (NHLBI), the 2 pivotal factors used to determine cardiometabolic risk in overweight and obese adults are:
    A. BMI and family history
    B. BMI and waist circumference
    C. Hypertension and waist and functional status
    D. Waist circumference and fasting blood glucose

12. Components of the metabolic syndrome that may be predictive of risk in overweight/obese patients include all of the following except:
    A. Hypertension
    B. High-density lipoprotein cholesterol
    C. Elevated triglycerides
    D. Visceral fat

13. Measurement of waist circumference:
    A. Adds little information beyond BMI about risk
    B. Is easily performed by a nurse in the examining room
    C. Is an essential component when assessing cardiometabolic risk in an obese patient
    D. Is especially relevant for people whose BMI is 25 to 35 mg/kg² and whose cardiovascular risk status is not clear from other clinical and historical findings

14. Medications that may impede weight loss include:
    A. Antidepressants such as escitalopram
    B. Antidiabetics such as exenatide
    C. Antidiabetics such as liraglutide
    D. Antihypertensives such as hydrochlorothiazide

15. After a patient taking an antiobesity agent has successfully lost body weight, it is recommended that he or she:
    A. Discontinue the drug immediately
    B. Never resume these medications as they cannot be continued after 1 year of use
    C. Resume therapy if body weight increases by 2%
    D. Start on a low-carbohydrate diet

16. Components of the metabolic syndrome that may be predictive of risk in overweight/obese patients include:
    A. Diabetes
    B. Hypertension
    C. Hyperlipidemia
    D. All of the above

17. I would recommend this activity to others.
    A. Yes
    B. No
    C. Not applicable

18. This activity will assist in the improvement of my:
    (check all that apply)
    A. Competence
    B. Performance
    C. Patient outcomes
    D. Other

19. I plan to make changes to my clinical practice as a result of this activity.
    A. Yes
    B. No
    C. Not applicable

20. If you answered Yes for Question 19, what is your level of commitment to making the changes stated above?
    A. Very committed
    B. Somewhat committed
    C. Not very committed

21. What are the barriers you face in your current practice setting that may impact patient outcomes? (check all that apply)
    A. Lack of evidence-based guidelines
    B. Guidelines not applicable to my current practice/patients
    C. Lack of time
    D. Organizational/institutional
    E. Insurance/financial
    F. Patient adherence
    G. Treatment-related adverse events
    H. Other

22. Please indicate topics for future activities:

© 2013 Global Academy for Medical Education. All Rights Reserved.