**COURSE OBJECTIVE:** The purpose of this course is to enable learners to recognize and manage metabolic syndrome in patients using evidence-based information about common risk factors, assessment measures, diagnostic and monitoring tests, pharmacology and treatment guidelines, complications, and therapeutic lifestyle changes associated with the syndrome.

**LEARNING OBJECTIVES**
Upon completion of this course, you will be able to:

- Define metabolic syndrome.
- Identify characteristics of patients at risk for metabolic syndrome.
- Describe the assessment and screening criteria used to diagnose metabolic syndrome.
- Review the current treatment guidelines and options for patients with metabolic syndrome.
- Describe the components of a comprehensive plan of care and monitoring for patients with metabolic syndrome.
- Explain the necessary lifestyle changes for managing metabolic syndrome.
- Identify serious complications associated with metabolic syndrome and their effective treatment interventions.
- Discuss prevention strategies for metabolic syndrome.

Metabolic syndrome is the name for a particular cluster of health problems first identified in 1988 by Gerald Reaven, a Stanford University endocrinologist, in a lecture to the American Diabetes Association (Reaven et al., 2000). At various times, this syndrome has been called dysmetabolic syndrome, insulin resistance syndrome, obesity dyslipidemia syndrome, and syndrome X. (Now simply known as metabolic syndrome, the “X” was dropped because of confusion with a cardiac syndrome having a similar designation.)
At its core, metabolic syndrome is characterized by the presence of obesity and insulin resistance, a reduced responsiveness of the body tissues to insulin. Associated disorders of metabolic syndrome are high blood pressure and two lipid problems: high blood levels of triglycerides and low blood levels of high-density lipoprotein cholesterol (HDL).

Metabolic syndrome has become increasingly common in the United States, and it is a major public health problem. According to the American Heart Association (AHA), an estimated 34% of the adult population is affected (AHA, 2014). Metabolic syndrome increases the risk of cardiovascular disease, diabetes, stroke, and coronary artery disease.

The primary causes of metabolic syndrome include obesity, physical inactivity, and possible genetic factors (AHA, 2014). The primary treatment of choice for metabolic syndrome includes lifestyle modifications such as increasing physical activity; eating a heart-healthy diet; and lowering blood glucose, blood cholesterol, and blood pressure (AHA, 2014).

**WHAT IS METABOLIC SYNDROME?**

**The Phenomenon of Insulin Resistance**

In the early 1920s, the Canadian surgeon Frederick Banting and his assistant Charles Best, a medical student, extracted from the islets of Langerhans in the pancreas a compound that they named insulin. When injected into diabetic dogs, this compound decreased the level of sugar in the dogs’ blood and reduced the amount of sugar in the dogs’ urine. Before the discovery and purification of insulin, childhood diabetes was a fatal disease; after Banting and Best’s work, diabetes became a chronic illness.

As early as the 1930s, clinicians found that people with diabetes could be divided into two classes according to how they reacted to an injection of insulin. “Insulin-sensitive” diabetics (who tended to be young and prone to developing ketosis, a build-up of ketone bodies in body tissues and fluids, leading to nausea, vomiting, and stomach pain) easily disposed of an oral dose of glucose when they also received a subcutaneous insulin injection. In contrast, “insulin-insensitive” diabetics (who were usually middle-aged and who did not have ketotic episodes) did not significantly reduce their blood glucose levels after receiving the same amount of insulin.

Today, insulin-sensitive diabetics are categorized as type 1 diabetics. In **type 1 diabetes**, the pancreas produces little or no insulin because the beta cells in the islets of Langerhans of the pancreas are not functioning. Type 1 diabetes shows up most commonly in young people, although it can occur in any age group.

On the other hand, insulin-insensitive diabetics are categorized as type 2 diabetics. **Type 2 diabetes** usually shows up in older adults, but it can occur at any age. A distinguishing feature of type 2 diabetes is that, even when there is a normal amount of circulating insulin, body tissues do not take up glucose as readily as normal. This is called insulin resistance, a condition in which normal concentrations of insulin in the blood produce less than the normal effects in the body, resulting in hyperinsulinemia, or high levels of insulin in the bloodstream.
At first, insulin resistance was thought of in terms of diabetes. Later, however, insulin resistance began to be recognized in patients who did not have diabetes. Many of these insulin-resistant people also had certain other systemic problems—notably obesity, hypertension, lipid disorders, and coronary heart disease. Insulin resistance was also found to be common in women with polycystic ovarian syndrome (PCOS), a disorder of the ovaries caused by numerous small cysts in both ovaries and characterized by absent menstruation, sterility, obesity, and a distribution of body hair more characteristic of men.

In the 1980s, researchers stepped back from their focus on diabetes and realized that insulin resistance frequently occurred as part of a particular cluster of systemic metabolic disorders. This cluster includes:

- Intra-abdominal obesity
- Insulin resistance
- High blood levels of triglycerides
- Low blood levels of high-density lipoprotein (HDL) cholesterol
- High blood pressure

Looking back, researchers discovered that a similar cluster of metabolic problems had already been identified as a special health risk in the 1920s. Putting all the data together in the late 1980s, clinicians proposed that it would be useful to call this cluster of metabolic disorders a syndrome and give it a name.

**Is Metabolic Syndrome a Single Disease?**

The group of problems collected under the heading *metabolic syndrome* does not seem to have one unique cause and does not involve a clear-cut target organ or system. For this reason, some scientists have wondered whether it is necessary to separate out this particular cluster of disorders and give it the status of a syndrome. Most clinicians, however, believe that the formal recognition of metabolic syndrome is justified.

Proponents point out that identifying metabolic syndrome is a strong warning of future health problems in an individual. In particular, if metabolic syndrome remains untreated, the patient is likely to develop serious cardiovascular disease and type 2 diabetes. An important feature of the clinical definition of metabolic syndrome is that it uses a small set of direct measurements—waist circumference; blood pressure; and the blood levels of triglycerides, high-density lipoproteins, and glucose—that allows the syndrome to be diagnosed simply and objectively.

The medical problems in a person with metabolic syndrome are complex and interrelated. Nonetheless, the definition of metabolic syndrome offers a clear therapeutic path for lessening a person’s chance of developing coronary heart disease and diabetes. All components of metabolic syndrome can be treated by exercising regularly, improving eating habits, and staying thin. In those cases where therapeutic lifestyle changes are insufficient, the definition of metabolic

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Metabolic syndrome gives clinicians a specific set of disorders (obesity, blood pressure, dyslipidemias, and hyperinsulinemia) to treat individually.

Establishing metabolic syndrome as an entity also focuses attention on certain key malfunctioning processes in the bodies of people who are on the road to serious health problems. Identifying the five disorders that compose metabolic syndrome gives scientists important targets for basic research, drug development, and therapeutic innovation.

The creation of a single entity called metabolic syndrome has many practical benefits. At the same time, doctors caution that metabolic syndrome is a creation of convenience. As currently defined, metabolic syndrome is a good but not an ideal predictor of the chance of developing serious health problems. Research continues, and it is likely that the definition of metabolic syndrome will be modified in the future.

**A Formal Definition**

Metabolic syndrome is not a disease in the usual sense. Instead, it is a condition, a collection of problems impacting the body’s ability to maintain circulation of useful but not excessive levels of energy molecules (i.e., glucose and lipids) in the bloodstream. Initially, when these problems arise, they interact to worsen each other. Eventually, the set of problems becomes severe enough to lead to serious health consequences. At this point, clinicians say that a person has metabolic syndrome.

It is still not clear whether any one of the individual problems of metabolic syndrome is the primary cause. More likely, two or more of the problems develop independently and then set off the condition. In any case, current definitions are not based on cause. Instead, they have been developed by looking for clusters of signs and symptoms in people who later developed coronary heart disease or type 2 diabetes.

From studies of large populations of people, the most common cluster of signs has been found to include obesity, insulin resistance, dyslipidemia (either too much or too little fat in the blood), and hypertension.

### DEFINITION OF METABOLIC SYNDROME

According to the guidelines from the National Heart, Lung, and Blood Institute and the American Heart Association, a diagnosis of metabolic syndrome is made if at least three of the following are present:

- Abdominal obesity: Waist circumference >102 cm (>40 inches) in men, >89 cm (>35 inches) in women
- Hypertriglyceridemia: Blood triglycerides >150 mg/dl (or on triglyceride-lowering medication)
• Low high-density lipoprotein cholesterol: Blood HDL-C <40 mg/dl in men, <50 mg/dl in women
• High blood pressure: BP >130/85 mmHg or already diagnosed with hypertension
• High fasting glucose: Blood glucose >100 mg/dl


DEMOGRAPHICS

Are demographics helpful in recognizing metabolic syndrome? The race or gender of a person who walks into the clinic is no help in determining whether that person has metabolic syndrome. The person’s age is not much help, either. Older people are more likely than young people to have the disorder, but 1 in 20 teenagers have metabolic syndrome. Prevalence does vary in different populations, but the syndrome is too common everywhere to use demographics as a discriminator in clinical practice.

Adolescents (12–19 years)

In the United States, the most recent National Health and Nutrition Examination Survey (NHANES) data from 1999–2006 estimate that 8.6% of adolescents have metabolic syndrome. It is more common in adolescent boys (10.8%) than in adolescent girls (6.1%), and in Hispanic (11.2%) and non-Hispanic white adolescents (8.9%) than in African-American adolescents (4.0%). Among overweight or obese adolescents, the prevalence of metabolic syndrome is much higher: 44% (Lipshultz et al., 2012). The criteria for diagnosis of metabolic syndrome in adolescents and children are adjusted for age.

Overweight teenagers who do not have metabolic syndrome remain at risk for developing it. Obesity—especially excess intra-abdominal fat—in childhood and adolescence foreshadows higher than normal blood levels of insulin, triglycerides, and LDL cholesterol; low blood levels of HDL cholesterol; and high blood pressure in adulthood.

Adults (19 years and older)

The incidence of metabolic syndrome in adults is reported to be around 23% (Beltran-Sanchez, et al., 2014). The syndrome tends to be more common in older adults. Metabolic syndrome affects 42% of Americans aged 60 years and older (Sanchez Silveira et al., 2013).

Prevalence of metabolic syndrome varies markedly from country to country. This seems to be caused by two factors: 1) variations in lifestyle (especially, diet, smoking, and level of physical exercise) between countries, and 2) variations in ethnicity. In all settings and all populations, the prevalence of metabolic syndrome increases with age.
Metabolic syndrome occurs more frequently in African American women and Mexican American women than in men of the same ethnic groups. Some racial and ethnic groups in the United States are at higher risk for metabolic syndrome than others. Mexican Americans are found to have the highest rate of metabolic syndrome, followed by whites and African Americans (NHLBI, 2011b).

From a global perspective, certain ethnic groups, such as South Asians, are at increased risk for metabolic syndrome.

Individuals at increased risk for metabolic syndrome include:

- People with a family history of diabetes
- People with a personal history of diabetes
- Women with a personal history of polycystic ovarian syndrome

CAUSES OF METABOLIC SYNDROME

In some diseases, the initiating events act on a common target. For example, most of the causes of type 1 diabetes act, ultimately, to disable the beta cells in the pancreas. In contrast, metabolic syndrome appears to result from the interaction of a number of disorders that can be initiated separately.

Currently, scientists believe that both obesity and insulin resistance are critical problems underlying metabolic syndrome and that the two problems can develop independently. It is still unclear whether the dyslipidemias and the hypertension of metabolic syndrome can trigger the syndrome or are, instead, consequences of the interaction of obesity and insulin resistance.

Although the specific chain of events leading to the appearance of metabolic syndrome is still not clear, much is known about the development and interactions of its separate components. Here is a summary of the causes of the individual components of metabolic syndrome.

Intra-Abdominal Obesity

GENETICS AND LIFESTYLE

The tendency to be obese is heritable, and genes are usually part of the cause of a person’s obesity. In rare cases, a single gene can cause obesity; in most cases, obese people have more than one contributory gene.

In addition to an inherited metabolic tendency to be overweight, eating patterns that a person develops are key causes of excess weight gain. Aspects of a person’s eating patterns are learned, but other parts are inborn and probably genetic. Normally, a number of proteins, hormones, and neural signals communicate with the hunger and satiety centers in the brain. These biochemical
cues are triggered by fullness of the stomach, the presence of food in the small intestine, and the levels of fat and glucose in the blood. In many obese people, the food signals do not work properly, and these people’s brains do not recognize when they have eaten a sufficient meal. This “satiety blindness” leads to overeating and weight gain.

Regular consumption of sugar-sweetened beverages is another risk factor for diabetes and metabolic syndrome. In a meta-analysis of eleven studies involving more than 310,000 individuals evaluated for diabetes and more than 19,000 for metabolic syndrome, those who drank one to two 12-ounce servings per day of sugar-sweetened beverages increased their risk of developing type 2 diabetes by 26% and of developing metabolic syndrome by 20%, compared to those who drank less than one serving per month (Malik et al., 2010).

NON-GENETIC FACTORS

• **Prenatal:** The non-genetic contributions to a person’s obesity can start in the womb. For example, a fetus who is undernourished in the first two trimesters of pregnancy will have a higher than normal chance of becoming an obese adult.

• **Psychological:** Depression, especially when part of bipolar disorder, can lead to excess eating and weight gain. Emotional, physical, and sexual abuse can also lead to obesity.

• **Pharmacologic:** Many medications have weight gain as a side effect, and it is important to monitor persons taking these medications. They include:
  - Psychiatric drugs (e.g., lithium, “atypical” antipsychotics such as clozapine and olanzapine, and antidepressants such as the tricyclics)
  - Neurologic drugs (e.g., antiepileptic drugs such as valproate)
  - Steroids (e.g., hormonal contraceptives and prednisone)
  - Anti-diabetic drugs (e.g., insulin)
  - Antihistamines
  - Beta-blockers

• **Diseases:** Diseases cause less than 1% of the obesity in the United States. The most common of these diseases are endocrine disorders, such as Cushing disease.

### CASE

Judy is a 52-year-old white woman with no previous history of diabetes or metabolic syndrome who presents to the clinic with mild hyperglycemia (152 mg/dL) and low HDL cholesterol (33 mg/dL). Judy appears to be overweight, and the nurse, Robert, calculates her BMI to be 32 kg/m². Judy also reports a lack of energy and that her weight has been slowly increasing over the past eight years. Judy is already on a beta-blocker for high blood pressure that was diagnosed two years previously.

Robert continues the assessment by asking Judy about any classic symptoms or complications of diabetes, such as weakness, fatigue, blurred vision, headache, dizziness, or dehydration.
Robert also asks Judy about her family history of diabetes and discovers that her mother has also been diagnosed with type 2 diabetes.

Robert suspects that Judy may have either diabetes or metabolic syndrome based on the laboratory findings of elevated blood pressure, blood sugar, and low HDL levels as well as Judy’s physical assessment and family history. After discussing Judy’s case with her primary care physician, a full medical workup is initiated.

Insulin Resistance

Insulin triggers the mechanisms that cells use to take up glucose from their surroundings. In addition, insulin tells cells to:

- Use their internal glucose for generating energy
- Store any excess internal glucose in the form of glycogen
- Stop releasing internal stores of glucose into the circulation

The body’s cells have specialized roles in metabolism. Most of the body’s glucose uptake, oxidation, and storage are carried out in skeletal muscle cells and fat cells. On the other hand, most of the release of stored glucose into the circulation comes from liver cells. Insulin is the signal to all these cells.

Insulin molecules remain outside cells, and they work by interacting with specific receptors on (and in) a cell’s membrane. Once activated, the receptor molecules encourage glucose transport into the cell. The insulin receptors also set off a cascade of events inside the cell. This internal cascade regulates the cell’s oxidation of glucose and lipids, storage and release of glucose, and a host of other processes, including the transport and metabolism of amino acids, protein synthesis, cell growth, cell differentiation, and even cell death.

In a person with insulin resistance, a normal amount of circulating insulin produces:

- Less than the normal amount of glucose transport into cells
- Reduced use (metabolism) of intracellular glucose
- Reduced storage of excess internal glucose in the form of glycogen
- Increased glucose release into the circulation (mainly, by the liver)

In insulin resistance, the basic problem lies in the responding cells, specifically in the mechanisms by which these cells recognize insulin and then produce the intracellular effects of the insulin signal.

The insulin receptor molecule in the membrane of the responding cell is a complex structure with a number of subunits. The malfunctioning or mutation of any of the receptor subunits can make them work inefficiently or make them insensitive to insulin, leading to insulin resistance.
Resistance can also be caused by the malfunctioning of any of the components of the intracellular cascade that connects the insulin receptors in the cell membrane to the glucose-processing machinery inside the cell.

**GENETICS**

As with many pathologic processes, insulin resistance develops most readily in people with a genetic predisposition for it. In predisposed people, it is possible that certain genes produce poorly functioning insulin receptor subunits or other molecules in the intracellular chain leading from the receptor to the actual glucose utilization machinery. It is still not clear, however, if any of these potential problems are common causes of the genetic predisposition to develop insulin resistance.

**EXCESS VISCERAL FAT**

Intra-abdominal fat is strongly associated with insulin resistance—more so than is extra-abdominal (subcutaneous) fat. Intra-abdominal fat is largely visceral fat, and an overabundance of visceral fat cells will cause insulin resistance.

Visceral fat cells are more responsive than other fat cells to signals from the sympathetic nervous system, which causes the breakdown and release of intracellular fat stores. At the same time, visceral fat cells are less responsive than other fat cells to insulin, which signals them to slow or stop the breakdown and release intracellular fat stores. In other words, visceral fat cells are easy to turn on and difficult to turn off.

The effect of too many visceral fat cells is too much free fatty acid in the bloodstream. High levels of free fatty acids stimulate the liver to release excess glucose into the bloodstream. High levels of free fatty acids also reduce the amount of glucose taken up by cells throughout the body, even when there is sufficient insulin available. The result of both of these effects is hyperglycemia. The pancreas responds to hyperglycemia by secreting more insulin, so at least temporarily, hyperglycemia always leads to hyperinsulinemia.

If it had been subcutaneous fat cells that were releasing the excess fatty acids, the newly released insulin would turn off the spigot by slowing or stopping the fatty acid release. Visceral fat cells, however, are less sensitive to insulin signals, and the feedback circuit is not very effective when visceral fat cells are the culprits. When visceral fat is the source of excess free fatty acids, the natural balancing mechanisms do not work well, and the hyperinsulinemia persists. This persistent hyperinsulinemia is a direct cause of insulin resistance.

This sequence of events leads to insulin resistance:

1. Persistent elevation of circulating free fatty acids causes hyperglycemia.
2. Persistent hyperglycemia causes hyperinsulinemia.
3. Persistent hyperinsulinemia causes insulin resistance.
Insulin resistance can be triggered by anything that causes high blood levels of free fatty acids, glucose, or insulin. Conditions that lead to insulin resistance through this mechanism include high levels of glucocorticoids (e.g., Cushing disease or long-term treatment with prednisone), nonalcoholic fatty liver disease, and treatment with protease inhibitors (e.g., for HIV).

**Dyslipidemias**

In the bloodstream, most lipids are carried in lipoproteins, a group of conjugated proteins in which at least one of the components is a lipid. The surface of a lipoprotein is made up of the more water-soluble lipids, cholesterol and phospholipids. The least soluble lipids, cholesteryl esters and triglycerides, are carried in the centers of the lipoproteins. This spherical package is held together by apolipoproteins, which are specialized fat-carrying proteins.

Lipoproteins come in five sizes. From the largest to the smallest, they are:

1. Chylomicrons
2. VLDL (very low density)
3. IDL (intermediate density)
4. LDL (low density)
5. HDL (high density)

Each size of lipoprotein has its own characteristic balance of lipids. The largest lipoproteins—chylomicrons and VLDL—are especially rich in triglycerides, while 70% of all blood cholesterol is contained in the LDL lipoproteins.

HDL lipoproteins are the second main carriers of cholesterol. HDL particles have a special role: they are cholesterol scavengers. HDL particles remove cholesterol from nonliver cells, such as fibroblasts and macrophages, and transport the cholesterol back to the liver. Because the cholesterol in HDL particles is unavailable for new atherosclerotic plaque formation, HDL lipoproteins can slow or even reverse cholesterol buildup throughout the vascular system.

Dyslipidemia is an unhealthy amount of lipid circulating in the bloodstream. The specific dyslipidemias of metabolic syndrome include an increase in blood triglycerides and a decrease in blood HDL lipoproteins.

**GENETICS**

A number of different genetic mutations that affect fat cells will cause the dyslipidemias of metabolic syndrome. In addition, certain genetic mutations of apolipoproteins (e.g., familial combined hyperlipidemia) will cause high blood levels of triglycerides and low blood levels of HDL cholesterol.

Beyond direct genetic causes, the dyslipidemias of metabolic syndrome can result from a variety of problems.
METABOLIC DISORDERS

The most common causes of dyslipidemias are other metabolic problems. For example, glycogen storage diseases and hypothyroidism each elevate the levels of blood triglycerides. Insulin resistance and excess visceral fat elevate blood triglycerides and lower blood HDL cholesterol, and type 2 diabetes and poorly controlled type 1 diabetes will do the same.

LIFESTYLE

The same habits that tend to make a person obese will also cause lipid problems. Dyslipidemias can result from insufficient physical activity and a high-calorie diet with excess carbohydrates and too many saturated fats.

KIDNEY PROBLEMS

Patients with chronic renal failure develop high blood triglycerides and decreased levels of blood HDL cholesterol. Later, if they receive kidney transplants, patients are put on immunosuppressive drugs, typically glucocorticoids and cyclosporine; these drugs also raise blood triglycerides and reduce blood HDL cholesterol.

PHARMACOLOGIC CONSIDERATIONS

Blood triglycerides are increased by retinoic acid, estrogens, and thiazide diuretics. Corticosteroids, immunosuppressive drugs, and beta-blockers increase triglyceride levels and also lower HDL cholesterol levels.

Hypertension

Blood pressure depends on two factors: cardiac output (how much blood is ejected with each heartbeat) and vascular resistance (how much opposition the bloodstream encounters from arteries and veins).

The first factor, cardiac output, depends on blood volume, which is normally regulated by the kidneys. The second factor, vascular resistance, is normally regulated by a balance of vasoconstrictors (e.g., angiotensin II, sympathetic nervous system activity) and vasodilators (e.g., prostaglandins, nitric oxide). High blood pressure can be caused by an increase in cardiac output, an increase in vasoconstriction, or a combination of the two factors.

Among the individual problems that constitute metabolic syndrome, obesity and insulin resistance directly cause hypertension. In addition to weight gain and excess salt intake, stress, smoking, and physical inactivity can cause or worsen hypertension.
GENETICS

The tendency toward hypertension is inherited. In most cases, this predisposition seems to depend on the interaction of a number of different genes.

LIFESTYLE

Weight Gain

The chance of having high blood pressure increases continuously as one gets heavier. At least two features of obesity lead directly to hypertension. First, obese people have a larger volume of blood, and therefore they have a higher than normal cardiac output. Second, obese people have overactive sympathetic nervous systems. This causes water and salt retention, which increases the blood volume. An overactive sympathetic nervous system will also increase the constriction of peripheral arteries, which increases vascular resistance. Obesity can also cause sleep apnea, which causes hypertension.

Salt Intake

For genetically predisposed people, excess salt in their diets will cause hypertension.

KIDNEY DISORDERS

The kidneys affect blood pressure by regulating both cardiac output and vascular resistance. Kidneys can adjust the blood volume by changing the reabsorption of sodium and water, and they can adjust the level of vasoconstriction through the renin-angiotensin system.

Hyperinsulinemia causes hypertension by way of the kidneys. Hyperinsulinemia increases the kidneys’ reabsorption of sodium and water and, in this way, increases the blood volume and the potential for hypertension.

Other Risk Factors

PREGNANCY

For most pregnant women, a healthy pregnancy modestly increases the risk of metabolic syndrome even after accounting for excess weight gain and reduced physical activity. For women who have a history of gestational diabetes, the lifetime risk of developing metabolic syndrome is higher than that in non-child-bearing women; women who have a history of gestational diabetes are also more likely to develop diabetes later in life (Gunderson, 2014).
PROSTATE DISEASES

Men with mild to severe lower urinary tract symptoms (LUTS) are at increased risk for developing metabolic syndrome compared to those without such symptoms. Recent data also show a connection between metabolic syndrome and the development and progression of prostatic diseases such as benign prostatic hyperplasia (BPH) and prostate cancer. Evidence for a cause-and-effect relationship is still lacking, however patients with prostate disorders along with related metabolic syndrome characteristics should be assessed for the disorder (De Nunzio et al., 2011).

DIAGNOSING METABOLIC SYNDROME

Medical History

Diagnosing metabolic syndrome requires a physical examination and blood tests. Nonetheless, the medical history offers important information that can confirm the diagnosis and help determine the extent of the problem.

A person who has metabolic syndrome may already have been diagnosed with some components of the syndrome, such as obesity, hypertension, or dyslipidemia. A major complication of the syndrome (atherosclerotic artery disease, ischemic heart disease, diabetes) may also be present.

In addition, the person may come with a diagnosis (or the signs and symptoms) of one of a number of other medical problems that occur especially frequently with metabolic syndrome. Diseases that are often found with metabolic syndrome include:

- Obesity
- Polycystic ovary syndrome (PCOS)
- Cardiovascular disease
- Non-alcoholic fatty liver disease
- Chronic kidney disease (NDIC, 2014)

Any of these problems should alert one to the possibility of metabolic syndrome.

Two Key Physical Characteristics

When screening for metabolic syndrome, two physical measurements must be included: waist circumference and blood pressure.
INTRA-ABDOMINAL FAT

Today, the standard physical examination of a patient includes height and weight but it does not usually include a measurement that is essential for diagnosing metabolic syndrome: the patient’s waist circumference. The specific aspect of obesity that best warns of future cardiovascular problems is the amount of fat concentrated inside the abdomen (AHA, 2014), and waist circumference is a good measure of intra-abdominal fat.

Measuring Obesity

Obesity is a condition identified with having more stored body fat than is considered normal. Clinically, obesity is measured indirectly. The simplest obesity tables compare two external physical measurements—height and weight—and obese is then defined as “more than the normal weight for a given height.”

The most commonly used measure of obesity is the body mass index (BMI). This is measured using the formula:

\[
\text{BMI} = \frac{\text{weight in kilograms}}{\text{height in meters squared}}
\]

or

\[
\text{BMI} = \frac{\text{weight in pounds} \times 703}{\text{height in inches squared}}
\]

BMI has been shown to be a good indirect indication of the percentage of body fat, and it is the most commonly used measure of total body fat. The BMI obesity definitions for adults are as follows:

<table>
<thead>
<tr>
<th>OBESITY DEFINITIONS: BMI (kg/m²)</th>
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</thead>
<tbody>
<tr>
<td>Normal</td>
</tr>
<tr>
<td>Overweight</td>
</tr>
<tr>
<td>Obese</td>
</tr>
<tr>
<td>Class 1</td>
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<tr>
<td>Class 2</td>
</tr>
<tr>
<td>Class 3 (extreme obesity)</td>
</tr>
</tbody>
</table>

Obese people are more likely than people of normal weight to suffer from certain medical problems, including diabetes, hypertension, dyslipidemias, polycystic ovarian syndrome, degenerative joint disease, sleep apnea, cancer (specifically, breast, colon, endometrial, prostatic), gastroesophageal reflux disease, fatty liver disease, and gallstones. For class 3 (extremely) obese people, the list is longer.

All overweight people have an increased risk of developing metabolic syndrome. In overweight and class 1 obese people, the risk of having or developing metabolic syndrome is much greater if their excess fat is located inside the abdomen (i.e., visceral).
When excess fat is concentrated in the abdomen, a person will have a round, apple shape. This is called **android obesity**, and, of all shapes, it is the most strongly predictive of metabolic syndrome–related conditions such as diabetes, hypertension, dyslipidemias, and atherosclerotic cardiovascular disease. (Another common shape of obesity has excess fat concentrated lower on the body, in the hips and thighs. This gives a person a pear shape and is called **gynecoid obesity**.)

**Measuring Waist Circumference**

Many large studies have shown that simply measuring a person’s waist circumference gives a good indication of the amount of excess body fat that is located inside the abdomen. A waist circumference of >94 cm (37 inches) in men and >80 cm (31.5 inches) in women is considered a warning sign, and a circumference of >102 cm (40 inches) in men and >88 cm (35 inches) in women puts the person in the high or very high risk category for developing metabolic syndrome and its serious health consequences.

In addition to being an indicator for metabolic syndrome, increased waist circumference is correlated with other health problems. These include decreased pulmonary functioning, lessened quality of life, increased disability in older adults, increased osteoarthritis in the knees, increased likelihood of asthma, increased risk of colon cancer, and increased risk of age-related macular degeneration.

The waist is the narrow band of the abdomen below the lowest margin of the ribs and above the top (iliac crest) of the hipbones.

To measure the waist:

- Place the tape measure around the abdomen, just above the hip bone.
- Hold the tape measure snug to the skin and parallel to the floor.
- Measure with the patient relaxed and breathing normally.
CASE

Sharon, Age 52
Sharon is a 52-year-old woman who has come to the office for her first appointment to manage her hypertension and joint pain. Sharon appears to be obese, with an android shape, prompting the nurse, Jennifer, to measure her waist circumference, which is 92 cm. After weighing the patient and measuring her height, Jennifer calculates the patient’s BMI as 35.2 kg/m². Her blood pressure is measured at 187/93 mm Hg today in the clinic after resting for 5 minutes.

Jennifer reviews Sharon’s pre-ordered laboratory tests, draws her blood, and sends the samples to the lab for a fasting glucose level and total lipid panel. Jennifer goes on to ask Sharon about her recent medical history, and the patient reports having pain in her joints, occasional difficulty in breathing, excessive thirst, and having to get up several times during the night to urinate.

Sharon also reports that her mother has a diagnosis of type 2 diabetes and her father has heart disease; both of her parents have high cholesterol and are on medications. She states that for the past 10 years, she has had increasing problems keeping her weight under control, and even more so now that she has gone through menopause. She states that she would like to exercise more, but with the excess weight and joint pain, she has not been able to do any regular exercise.

Jennifer suspects that Sharon may have metabolic syndrome, with possible coexisting hypertension, osteoarthritis, and asthma, and discusses this with Sharon’s physician. They also discuss the patient’s desire to incorporate exercise and include an initial appointment with a physical therapist to evaluate Sharon for recommendations for exercise in the setting of potential osteoarthritis.

Sharon is scheduled for a return visit to review the results of her blood tests to discuss possible therapeutic recommendations, including both lifestyle changes and medications to control her blood pressure and possibly dyslipidemia.

(continues)

HIGH BLOOD PRESSURE

The second component of metabolic syndrome that can be picked up in a physical exam is high blood pressure. To be used as a diagnostic condition for metabolic syndrome, a person’s blood pressure must be >130/85 mm Hg. (If a person is already taking antihypertensive medication, it is assumed that his or her blood pressure would normally be >130/85 mm Hg.)

Hypertension is defined as blood pressure of >140/90 mm Hg. As with most clinical measurements, the blood pressure values found in people grade smoothly between the healthy and the unhealthy ranges. The boundary of 140/90 mm Hg was chosen because it is at this value that, on average, the benefits of treatment outweigh the risks. The risks of living with hypertension include stroke, myocardial infarction, heart failure, peripheral vascular disease, aortic dissection, and chronic renal failure.
Hypertension has been referred to as a silent killer because, unless they are told their blood pressure measurements, people are usually unaware of any problem. Hypertension is a chronic illness that is largely asymptomatic until it leads to heart, brain, or kidney damage.

**Prehypertension** is the borderline region of blood pressures of 120 to 139 mm Hg systolic and 80 to 89 mm Hg diastolic (NHLBI, 2011a). Prehypertension warns of future health risks; for example, people with prehypertension are twice as likely to progress to hypertension when compared to people with lower blood pressures.

### BLOOD PRESSURE RANGES (mm Hg)

<table>
<thead>
<tr>
<th>Level</th>
<th>Systolic</th>
<th>Diastolic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Optimal</td>
<td>&lt;120</td>
<td>&lt;80</td>
</tr>
<tr>
<td>Prehypertensive</td>
<td>120–139</td>
<td>80–89</td>
</tr>
<tr>
<td>Hypertensive</td>
<td>&gt;140</td>
<td>&gt;90</td>
</tr>
</tbody>
</table>

Epidemiologic studies have shown that prehypertension (specifically, >130/85 mm Hg, or being on medication to lower blood pressure) is a sufficient criterion for making a diagnosis of metabolic syndrome (NHLBI, 2011b).

**Measuring Blood Pressure**

The basic rule is: “Measure on more than one occasion.” Blood pressure varies dramatically throughout a 24-hour period, and blood pressure will be raised by stress, recent meals, and recent physical activity. To take variation into account, two or more readings on two or more different days are needed to estimate a person’s usual blood pressure.

Other basic rules for measuring blood pressure include:

- **Setting.** The patient should have had no caffeine or tobacco for at least 30 minutes and must have been sitting or lying quietly for at least five minutes. When blood pressure readings are taken, the patient should be sitting or lying with the back supported. The arm from which the reading is taken should be resting and supported, and it should be positioned horizontally at the level of the patient’s heart.

- **Cuff size.** Large or overweight adults need a large-size adult cuff. The inflatable part of the blood pressure cuff should cover about 80% of the circumference of the patient’s upper arm. The cuff should cover two thirds of the distance from the patient’s elbow to shoulder.

- **Technique.** Begin by palpating the radial artery as the cuff is inflated. The radial pulse will disappear at the systolic value. Continue to inflate the cuff 20 mm Hg beyond that point. Put the bell of the stethoscope lightly over the brachial artery next to the lower edge of the cuff. Deflate the cuff slowly, at a rate of 3 to 5 mm
Hg/sec, noting the pressure at which the first sound is heard (the systolic value) and at which the last sound disappears (the diastolic value).

Take two or more readings during an exam, separated by at least five minutes of rest. Then take the pressure in the other arm.

- **Differences in readings between the two arms.** Small differences in blood pressure readings between the two arms are quite common. If a patient has a higher reading consistently on one side, the side that reads highest should be used for comparison from week to week. However, if a patient has a difference of 10 to 15 mm Hg for systolic pressure on a regular basis, it could indicate that the patient is at higher risk for vascular or cardiovascular disease (Mayo Clinic, 2012).

### CASE

**Sharon, Age 52 (continued)**

Sharon returns for a follow-up appointment. At her previous appointment, her blood pressure was found to have been 187/93 mm Hg. The nurse, Jennifer, measures the patient’s blood pressure once again; this time the reading is 189/94 mm Hg.

At this time, Sharon’s blood test results have come in, and they show blood triglycerides of 155 mg/dl and an HDL cholesterol level of 43 mg/dl. Her fasting blood glucose level is 142 mg/dL.

Based on the previous visit’s information of an abnormal waist measurement and hypertension, the nurse suspects that the patient has metabolic syndrome. The physician confirms the diagnosis of metabolic syndrome and outlines a treatment recommendation for Sharon, including appropriate diet and exercise as well as adhering to her prescribed medication regimen to control her blood sugar, lipids, and blood pressure. The physician provides referrals to a dietitian for nutrition recommendations as well as a physical therapist for an individual exercise program.

Jennifer reviews recommendations for following a Mediterranean diet model as outlined by the dietitian as well as reinforcing recent exercise recommendations outlined by the physical therapist. Sharon is also scheduled to see an exercise specialist, who will continue to monitor her progress through the next four weeks. Sharon is scheduled for a follow-up visit in one month to monitor her symptoms and progress.

### Laboratory Tests

It is important to look at two factors that contribute to metabolic syndrome: insulin resistance and dyslipidemias. In evaluating a patient, laboratory information should include both fasting glucose levels and fasting lipid profiles.
ASSESSING INSULIN RESISTANCE

Among the various measurements of the body’s ability to produce and use glucose, the blood level of glucose after an 8-hour fast is probably the simplest. Fasting glucose levels are a well-calibrated standard that is now widely used to screen for insulin resistance, a common cause of diabetes.

**Diabetes**

Diabetes mellitus is an endocrine disease that disrupts the body’s energy metabolism. In diabetes there is an insufficient amount of insulin available to the cells, and therefore glucose is not used efficiently throughout the body. One cause of the insulin insufficiency can be a reduced production of insulin by the beta cells in the pancreas (i.e., type 1 diabetes); another cause can be a reduced effect of the available insulin, known as insulin resistance (i.e., type 2 diabetes). Both causes can occur in the same person.

Without sufficient effective insulin, body tissues cannot take up all the glucose that is circulating in the bloodstream, and a hallmark of diabetes is hyperglycemia, the presence of more than the normal amount of glucose in the blood. After an 8-hour fast, the body should maintain blood glucose levels at <110 mg/dL, typically in the range of 95–100 mg/dL.

The health problems of diabetes are caused directly from hyperglycemia, and the medical diagnosis of the disease is not based on its cause but rather on evidence of persistent high plasma glucose levels, regardless of the cause. Diabetes is diagnosed when any one of the following hyperglycemic conditions is present:

- Fasting blood glucose level is found to be ≥126 mg/dL*
- Hemoglobin A1C level (an index measuring the amount of glucose sticking to hemoglobin inside red blood cells, and which indicates a person’s average blood glucose level over the past two to three months) is ≥6.5%*
- Two-hour plasma glucose level is ≥200 mg/dL in an oral glucose tolerance test (OGTT)*
- Random plasma glucose level is ≥200 mg/dL, accompanied by classic symptoms of hyperglycemia or hyperglycemic crisis

* In the absence of unequivocal hyperglycemia, results should be confirmed by repeat testing (ADA, 2011).
**GLUCOSE METABOLISM**

<table>
<thead>
<tr>
<th>Category</th>
<th>Fasting Blood Glucose (mg/dL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>&lt;100</td>
</tr>
<tr>
<td>High normal</td>
<td>101–109</td>
</tr>
<tr>
<td>Hyperglycemia (impaired maintenance of glucose levels)</td>
<td>110–125</td>
</tr>
<tr>
<td>Diabetes</td>
<td>&gt;126</td>
</tr>
</tbody>
</table>

**Hyperglycemia**

Hyperglycemia, a fasting glucose level of >110 mg/dL, can result from a variety of causes. The two most common causes are decreased secretion of insulin and insulin resistance. Less common causes include a generalized increased activity of the sympathetic nervous system (e.g., during stress) or an increased secretion of glucagon (a pancreatic hormone), epinephrine (an adrenal hormone also known as adrenaline), cortisol (a steroid hormone produced by the adrenal cortex), or growth hormone (a pituitary hormone that stimulates growth of the body and that influences metabolism of proteins, carbohydrates, and lipids).

Although insulin resistance and not hyperglycemia is considered a key problem in metabolic syndrome, the biochemical diagnosis of insulin resistance is technically difficult. Large epidemiologic studies have shown that the presence of fasting hyperglycemia can be used as a surrogate for demonstrating the insulin resistance of metabolic syndrome. Both the NCEP/ATP III and the IDF definitions of metabolic syndrome agree that a fasting blood glucose of >110 mg/dL can be used as one criterion for diagnosing the syndrome.

**ASSESSING DYSLIPIDEMIAS**

The four main classes of lipids (fats) found in the bloodstream are fatty acids, triglycerides, phospholipids, and cholesterol. By themselves, lipids do not dissolve in blood. In the circulation, lipids are transported in lipoproteins, soluble packets built around special protein carrier molecules.

Dyslipidemias are conditions in which the bloodstream contains unhealthy amounts of lipids. The dyslipidemias of metabolic syndrome are: 1) elevated blood levels of triglycerides, and 2) reduced blood levels of high-density lipoproteins (HDL). Metabolic syndrome is often accompanied by additional dyslipidemias, although these abnormalities are not necessary for the diagnosis of the syndrome.
Metabolic syndrome is characterized by fasting blood triglycerides >150 mg/dL and fasting blood HDL cholesterol <40 mg/dL in men and <50 mg/dL in women.

### CLASSIFICATION OF BLOOD LIPID LEVELS

<table>
<thead>
<tr>
<th>Type</th>
<th>Blood Concentrations (mg/dL) (Measured after an 8-hour fast)</th>
</tr>
</thead>
</table>
| Triglycerides   | • Normal: <150  
|                 | • Borderline high: 150–199  
|                 | • High: >200 |
| HDL cholesterol | • Low: <40  
|                 | • High: >60 |
| LDL cholesterol | • Optimal: <100  
|                 | • Borderline high: 130–159  
|                 | • High: >160 |

CASE

George is a 45-year-old male who arrives to the clinic for his annual physical. After stepping onto a scale, he is found to have gained 10 pounds over the previous year. His blood pressure has gradually been increasing over the past two years as well, with a current measurement of 145/88 mm Hg.

As his medical and family history is taken, George mentions that his mother has type 2 diabetes and that his uncle was diagnosed with heart disease after suffering a heart attack at age 55. The nurse takes a measurement of his waist circumference, which is 105 cm (41 inches).

After discussing the clinical picture with the primary care physician, a lipid panel is ordered. Three days later, the results of George’s blood test show blood triglycerides of 156 mg/dL and an HDL cholesterol level of 38 mg/dL.

George is diagnosed with metabolic syndrome; he is started on appropriate therapy and instructed on incorporating lifestyle interventions (e.g., diet, exercise) and given a referral to a dietitian at his request. A follow-up appointment is scheduled for three months later to assess how he is doing with initial management.

When George returns for his follow-up visit, he reports that he has been following his diet and exercise plan and feels that this has made a difference in how he is feeling. He has lost 8 pounds, his blood pressure is now 124/68, his triglycerides have improved to 130 mg/dL, and his HDL cholesterol has increased to 52 mg/dL.

George continues to be motivated to make changes in order to improve his health and states that he feels better than ever. He adds that his wife has been very supportive—together they joined the local Weight Watchers to support a healthy diet and weight loss, and they are exercising on a regular basis.
Two Possible Coexistent Diagnoses

Patients with intra-abdominal obesity, high fasting glucose levels, high blood pressure, high blood levels of triglycerides, and low blood levels of HDL cholesterol have metabolic syndrome and should be treated. Yet it is important to remember that a patient may simultaneously have other diseases with similar or overlapping symptoms. Two specific disorders to keep in mind are Cushing syndrome and hypothyroidism.

CUSHING SYNDROME

Cushing syndrome is caused by excess glucocorticoid (any of a group of steroid hormones that are produced by the adrenal cortex and are involved in protein, carbohydrate, and fat metabolism)—either excess intrinsic cortisol (as is produced by the adrenal glands in Cushing disease) or excess extrinsic glucocorticoids (e.g., prednisone), which might have been prescribed to treat another disorder. Typically, a person with Cushing syndrome has weight gain, skin striae (stretch marks), hirsutism, and proximal muscle weakness.

As in metabolic syndrome, Cushing syndrome leads to central (as opposed to peripheral) obesity, although the fat in Cushing syndrome tends to be most noticeable on the back of the neck, upper shoulders, and in the cheeks. Cushing syndrome also includes hypertension, elevated blood glucose levels, and dyslipidemias, including an elevated level of blood triglycerides. Moreover, patients with Cushing syndrome are more susceptible to cardiovascular disease.

HYPOTHYROIDISM

Hypothyroidism is caused by a decreased secretion of thyroid hormone from the thyroid gland, slowing metabolic processes throughout the body. People who have hypothyroidism are typically slow talking, slow to respond, tired, and depressed. Their skin is cool and dry, they look apathetic, they have slow reflexes, and they are constipated. Often, they have an enlarged thyroid gland.

As in metabolic syndrome, people with hypothyroidism tend to be overweight and inactive. They also have dyslipidemia and, sometimes, mild hypertension. Moreover, patients with hypothyroidism are more likely than normal to have cardiovascular disease. On the other hand, unlike metabolic syndrome, low blood glucose levels are typical of hypothyroidism.

CASE

Robert is a 55-year-old male recently diagnosed with metabolic syndrome. The nurse, Monica, has not previously met the patient, but when he arrives in the office, she immediately notices several physical characteristics indicative of his condition. The patient is obese and is carrying excess fat in his cheeks, trunk, upper shoulders, and back of the neck.

Monica introduces herself and starts to ask a few questions. Robert appears fatigued and is slow to respond to her questions. When asked about recent symptoms, Robert reports that he has had...
more weakness in his legs recently and feels “completely worn out.” He also reports recent weight gain despite changing his diet. When asked if he has any other concerns, Robert shows her the pigmented stretch marks on his abdomen and questions why he has developed these.

The nurse suspects that Robert may have Cushing syndrome and possibly hypothyroidism and discusses this with his primary care physician. Robert is then scheduled to have additional tests to rule out both conditions.

HEALTH CONSEQUENCES OF METABOLIC SYNDROME

People who have metabolic syndrome tend to have many associated health problems, although it is not always known whether the person’s metabolic syndrome is the direct cause. Two serious medical problems that are the direct result of long-term metabolic syndrome are coronary heart disease and type 2 diabetes.

Coronary Heart Disease

The most striking risk posed by metabolic syndrome is coronary heart disease (also known as coronary artery disease or atherosclerotic cardiovascular disease). By themselves, the dyslipidemias of metabolic syndrome (i.e., high triglycerides and low HDL cholesterol levels) encourage plaque to form along the walls of arteries. When combined with the other components of metabolic syndrome, these atherogenic dyslipidemias (i.e., those that tend to cause atherosclerotic plaque) put a person at high risk for developing serious atherosclerotic vascular disease with coronary artery blockage.

People who have metabolic syndrome often also have low-level inflammation throughout the body and blood clotting defects that increase the risk of developing blood clots in the arteries. These conditions contribute to increased risk for cardiovascular disease (NDIC, 2014).

Metabolic syndrome also worsens heart failure, and even when no heart disease is apparent, metabolic syndrome makes a person more likely to develop certain arrhythmias (notably, paroxysmal atrial fibrillation or flutter).

Type 2 Diabetes

Metabolic syndrome is a precursor to type 2 diabetes. The mechanism is as follows: The insulin resistance of metabolic syndrome forces the pancreas to secrete higher than normal amounts of insulin. Meanwhile, some hyperglycemia persists even with the excess circulating insulin. The continuous hyperglycemia and hyperinsulinemia are toxic to the beta cells in the pancreas, and over time these cells sicken and the amount of insulin that they produce decreases. Eventually, the pancreas cannot cope with hyperglycemia, and the patient develops diabetes.

A person with diabetes is at risk for serious health problems. Diabetes causes end-stage renal disease. Diabetes is also the most common cause of nontraumatic amputations and is a major
cause of blindness in adults. Nerve damage (diabetic neuropathy) occurs in 60% to 70% of people with diabetes. In addition, people with diabetes are approximately 2 to 4 times more likely to have heart disease and stroke than those without diabetes (CDC, 2011a).

Other Disorders Associated with Metabolic Syndrome

People with metabolic syndrome are at risk for a long list of health problems. It is not always clear whether metabolic syndrome is the cause or whether the related disorders share common causes with the components of metabolic syndrome. In all cases, however, the presence of metabolic syndrome indicates a higher than normal risk that a person will also have:

- Renal disease and microalbuminuria
- Atherosclerotic plaques in the carotid arteries
- Left ventricular hypertrophy
- Polycystic ovarian syndrome
- Nonalcoholic fatty liver disease
- Erectile dysfunction
- Venous thromboemboli
- Periodontal disease
- Low testosterone levels in men
- Hyperuricemia
- Pancreatitis

TREATMENT

The individual components of metabolic syndrome would not always be treated if found in isolation. For example, these values are below the levels that mandate treatment if the person has no other risk factors:

- Blood pressure: 130–139/85–89 mm Hg
- Fasting blood glucose: 110–125 mg/dL
- Blood triglycerides: 150–199 mg/dL

When found together with other risk factors, and especially when they are found to be part of metabolic syndrome, these values signal the need for treatment. Metabolic syndrome lowers the threshold for the treatment of its components.
Treatment for metabolic syndrome consists of the following main therapeutic strategies:

1. Weight loss and increased physical activity focused on reversing the direct causes of the condition
2. Medications designed to treat the various components, such as dyslipidemia, hypertension, prothromic conditions, and insulin resistance
3. Dietary management focused on lowering cholesterol and restricting calories from simple carbohydrates (emphasis on low-fat dairy, whole grains, and fresh fruits and vegetables)
   (Codario, 2011)

Treatment of the components of metabolic syndrome begins with lifestyle changes. Because lifestyle changes are easy to prescribe but difficult to carry out, often medications must be added to ensure that the treatment regimens succeed.

**Therapeutic Lifestyle Changes**

Therapeutic lifestyle changes, such as increased physical exercise, improved diet, and weight reduction, are the cornerstones of the treatment of obesity, hypertension, insulin resistance, and most dyslipidemias. Reducing dietary calories and fats (especially saturated fats) and increasing exercise can significantly reduce the risk of developing diabetes and atherosclerotic cardiovascular disease.

**EXERCISE**

All aspects of metabolic syndrome benefit from increased physical activity. Physical exercise helps in losing weight and in maintaining weight loss, and it has additional independent metabolic effects that directly reduce insulin resistance. Physical activity is usually a safe and beneficial treatment for people with metabolic syndrome and its consequences, atherosclerotic cardiovascular disease and type 2 diabetes. Even patients with end-stage renal disease can benefit from physical training programs.

Both aerobic and resistance exercise are effective therapies. For most people, the recommendation is to do moderately intense activity for 2 hours and 30 minutes a week, or vigorously intense activity for 75 minutes a week, or an equivalent combination of moderately and vigorously intense aerobic physical activity. Aerobic activity should be performed in episodes of at least 10 minutes, preferably spread throughout the week (Kaur, 2014).

For high-risk patients with comorbidities who are deconditioned or have had recent cardiac events, careful supervision of physical rehabilitation is recommended. Referral to a physical therapist or exercise physiologist to evaluate, plan, and monitor the patient’s progress with his or her exercise program is an important consideration (Kaur, 2014).
Although a program of regular exercise does not typically reduce LDL cholesterol levels to a significant degree, it will reduce insulin resistance and blood levels of triglycerides, and it will increase blood levels of HDL cholesterol.

**DIETARY MODIFICATIONS**

Exercise alone rarely leads to significant weight loss. A reduced-calorie diet is usually necessary, and dieting is the second critical part of the initial treatment of metabolic syndrome. Overweight people with metabolic syndrome must reduce the number of calories they eat each day.

Even a modest weight loss makes a difference for an overweight or obese person, and losing 5% to 7% of the original weight and keeping the weight off is a realistic goal. The ADA (2014) recommends that patients aim for a weight loss of 7% of body weight, noting that a small but consistent weight loss of 1/2 to 2 pounds per week is the safest way to accomplish this.

Simply reducing the overall calories in the diet will improve the lipid profile. Reducing the amount of fat improves the lipid profile even further. It is especially important to remove foods that are high in simple carbohydrates, refined grains, and saturated fats, such as:

- Fatty meats (e.g., bacon, sausage)
- Chicken or turkey eaten with the skin
- Egg yolks
- Butter
- Cream, half-and-half, and ice cream
- Cookies, cakes, muffins, breads, and pastries

(Unger, 2013)

Fat-rich foods should be replaced with foods that have high water and fiber content, such as whole grains, fruits, vegetables, legumes, lean meats, seafood, nuts, seeds, and low-fat dairy products (NHLBI, 2011b).

**MEDITERRANEAN DIET**

A recent meta-analysis of 50 research studies involving more than 500,000 participants found that adherence to the so-called Mediterranean diet reduced the risk of metabolic syndrome by nearly onethird (31%). The Mediterranean diet consists of:

- High consumption of monosaturated fatty acids (primarily from olives and olive oil)
- Daily consumption of fruits, vegetables, whole grain cereals, and low-fat dairy products
- Weekly consumption of fish, poultry, tree nuts, and legumes
• A relatively low consumption of red meat (approximately twice per month)
• Moderate daily consumption of alcohol (normally with meals)

Additionally, the studies suggested that adherence to the Mediterranean diet can positively affect individual components of metabolic syndrome such as waist circumference, dyslipidemia, hypertension, and hyperglycemia. These findings are of considerable public health importance, according to the investigators, because this dietary pattern can be easily adopted by all population groups and various cultures.

Source: Kastorini et al., 2011.

CASE

The nurse, Kathy, enters the examination room to check the blood pressure and take a blood sample from Judy, a 52-year-old female patient being treated for metabolic syndrome. While applying the blood pressure cuff to the Judy’s arm, Kathy asks how well she has been controlling her weight, and the patient replies that she has been having difficulty keeping the pounds off. A discussion of diet and exercise ensues, during which Judy reveals that she has continued to consume fried, fatty foods and few fruits and vegetables and has not been exercising regularly.

Kathy discusses the benefits of the Mediterranean diet in managing the various components of metabolic syndrome. While reviewing the various components of the diet, they look together at an educational booklet that outlines how to follow the Mediterranean diet model, with practical menu suggestions and a baseline assessment about knowledge of the healthy food choices included in the model. Judy mentions that the diet seems easier than she imagined to follow and states that she will start to shop and plan her meals better with this information. Kathy helps the patient make an appointment with a registered dietitian with the aim of establishing an individualized diet and exercise plan based on Judy’s needs. Kathy also makes an appointment with an exercise specialist who can create an individualized activity plan to increase Judy’s strength and endurance. They plan to have a follow-up visit in six weeks to check-in together to monitor progress.

NUTRITIONAL SUPPLEMENTS

Over the last two decades, numerous studies have focused on nutritional supplementation to prevent or reduce the impact of metabolic syndrome, thereby potentially protecting against cardiovascular disease and its associated complications. A number of research-supported nutrients are available as dietary supplements. Such nutrients include:

• Policosanol, which has been shown to reduce LDL and total cholesterol (TC) while raising HDL cholesterol
• Plant sterols and stanols, which have been found to inhibit the absorption of cholesterol in the small intestine and reduce LDL
• Soy proteins and isoflavones such as genistein and daidzein, which lower TC, triglycerides, and LDL

• Omega-3 fatty acids, which may reduce the risk of cardiac events (e.g., death, heart attack, and stroke); restenosis (re-narrowing of a coronary artery) after angioplasty; and blood pressure, with some studies suggesting they may also lower TC and triglycerides

SMOKING CESSATION

When associated with metabolic syndrome, smoking increases the chance of developing insulin resistance, type 2 diabetes, and dyslipidemias. In addition, smoking contributes to the development of a variety of cancers, atherosclerotic cardiovascular diseases, lung diseases, gastrointestinal diseases, reproductive problems, osteoporosis, cataracts, age-related macular degeneration, and hypothyroidism.

DENTAL MAINTENANCE

Oral health problems can indirectly increase the risk of developing cardiovascular disease. People with problems such as cavities, sore gums, periodontal disease, and missing teeth prefer to eat soft foods, which are low in fiber. Dental care and tooth replacement are an often-forgotten part of improving a patient’s diet.

Medications

HYPERTENSION

When lifestyle changes in diet and exercise are insufficient, persistent hypertension requires medication. For metabolic syndrome, antihypertensive drug therapy usually begins with an angiotensin-converting-enzyme (ACE) inhibitor or an angiotensin II receptor blocker (ARB). These drugs can also slow the progression of diabetic nephropathy. Beta-blockers are avoided in metabolic syndrome because they tend to cause weight gain, increased triglyceride levels, and reduced HDL cholesterol levels.

In patients with hypertension and diabetes, two or more drugs are usually needed to meet the target goal blood pressure of <130/80 mm Hg. In these cases, the second drug is typically a thiazide diuretic for those with an estimated glomerular filtration rate (eGFR) ≥30 ml/min/1.73m² or a loop diuretic for those with an eGFR <30 ml/min/1.73m² (ADA, 2011).

HYPERGLYCEMIA

The oral medicines for treating diabetes fall into two classes: those that increase insulin secretion (insulin secretagogues) and those that decrease insulin resistance (insulin sensitizers). Insulin sensitizers are used for metabolic syndrome.
Metformin (Glucophage) is the standard insulin sensitizer. Metformin counteracts insulin resistance by reducing the amount of glucose released by the liver and, to a lesser extent, by improving the ability of muscle to extract glucose from the circulation. When metformin alone is not sufficient, the combination of metformin and a sulfonylurea—tolbutamide (Orinase), chlorpropamide (Diabinese), tolazamide (Tolinase), glipizide (Glucotrol), glyburide (Micronase), or glimepiride (Amaryl)—is especially effective in reducing hyperglycemia (Codario, 2011).

Thiazolidinediones, including rosiglitazone (Avandia) or pioglitazone (Actos), are another commonly used class of insulin sensitizers. These drugs will also reduce the blood level of circulating fatty acids and increase the blood level of HDL cholesterol.

### INSULIN SENSITIZERS

<table>
<thead>
<tr>
<th>Biguanides</th>
<th>Thiazolidinediones</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metformin (Foramet, Glucophage, Glucophage XR)</td>
<td>Pioglitazone (Actos)</td>
</tr>
<tr>
<td>Typically taken with meals, acts over 24 hours</td>
<td>Typically taken once daily with breakfast, acts over 24 hours (patients with heart failure should not take pioglitazone)</td>
</tr>
<tr>
<td>Possible side effects include nausea, diarrhea, lactic acid buildup (rare)</td>
<td>Possible side effects include liver disease, stroke, heart failure, myocardial infarction</td>
</tr>
<tr>
<td>Rosiglitazone (Avandia)</td>
<td>Typically taken once daily with breakfast, acts over 24 hours</td>
</tr>
</tbody>
</table>

Source: Burant & Young, 2012.

### DYSLIPIDEMIA

The dyslipidemias of metabolic syndrome have two characteristics: high blood levels of triglycerides and low blood levels of HDL cholesterol. Both of these problems can lead to atherosclerotic cardiovascular disease. Metabolic syndrome is often worsened by the presence of another dyslipidemia, hypercholesterolemia (high blood levels of LDL cholesterol), which by itself is a major contributor to the development of coronary (atherosclerotic) heart disease.

When a 3- to 6-month trial of therapeutic lifestyle changes does not sufficiently improve these heart-threatening features of a patient’s lipid profile, medications are added. Drug therapy usually involves statins, fibrates, or niacin.

### Statins

Statins are drugs that interrupt the synthesis of cholesterol inside cells. They include rosuvastatin (Crestor), atorvastatin (Lipitor), simvastatin (Zocor), lovastatin (Mevacor), fluvastatin (Lescol), and pravastatin (Pravachol). Statins are effective at reducing the
blood level of LDL cholesterol. They also lower blood triglyceride levels modestly and raise HDL cholesterol levels. Statins are the first-line dyslipidemia drugs for preventing cardiovascular disease in those patients with type 2 diabetes who also have high levels of blood cholesterol.

**Fibrates**

Fibrates provide a more direct attack on the dyslipidemias of metabolic syndrome. Fibrates are fibric acid derivatives—gemfibrozil (Lopid) or fenofibrate—that speed up the conversion of triglycerides to fatty acids and raise the blood levels of HDL lipoproteins. Fibrates are second-line drugs for treating high LDL cholesterol levels, and they can be used in combination with a statin.

**Niacin**

Another medication that improves the dyslipidemias of metabolic syndrome is the B vitamin niacin, or nicotinic acid. In doses much higher than the normal recommended daily allowance, niacin lowers the production of VLDL lipoproteins (which are precursors to LDL lipoproteins), reduces blood levels of triglycerides, and increases the blood levels of HDL lipoproteins. Niacin should be used in caution with patients with diabetes, as it has been shown to increase levels of blood glucose. Using niacin in the extended-release formulation is associated with fewer side effects (Codario, 2011).

**OBESITY**

Therapeutic lifestyle changes and counseling are the first steps in treating the obesity of metabolic syndrome. When these steps do not lead to sufficient weight loss, medications can be tried. One used for obesity is the appetite suppressant sibutramine (Meridia), which works in the brain to make a person feel full earlier in a meal. Other central nervous system medications that have been used for obesity are fluoxetine (Prozac), diethylpropion, and bupropion (Wellbutrin, Zyban) (Codario, 2011).

The anti-obesity drug orlistat (Xenical) works differently. Orlistat is a malabsorption agent that inhibits intestinal lipase and reduces the intestine’s absorption of fat; therefore, it is only helpful for meals containing fat. A frequent side effect of this drug and its recent over-the-counter (OTC) variant (Alli) is sudden bouts of diarrhea. Patients should be made aware of this when the medication is prescribed.

Weight that has been lost with the aid of medications is typically regained when the medicine is stopped. For this reason, drug therapy works best when it is part of a treatment plan that includes therapeutic lifestyle changes and counseling.
**XENICAL/ALLI WARNING**

Notably, in May 2010 the U.S. Food and Drug Administration (FDA) approved revised labeling for Xenical and Alli to include new safety information about thirteen cases of severe liver injury, which resulted in two deaths from liver failure and an additional three liver transplants. Providers should weigh the benefits of weight-loss with Xenical and Alli against the potential risks when determining if these medications are appropriate for patients. The labeling also asks patients to report any symptoms of liver dysfunction such as anorexia, pruritus, jaundice, dark urine, light-colored stools, or right-upper-quadrant pain when using these medications. If liver injury is suspected, the medications should be discontinued immediately and liver function tests and ALT and AST levels obtained.


**PROTHROMBOTIC STATE**

Metabolic syndrome is usually accompanied by a prothrombotic state, an increased tendency of the blood to form clots. Treating the prothrombotic state will reduce the risk of coronary heart disease and stroke, and many clinicians prescribe daily low-dose aspirin as part of the therapy for metabolic syndrome (Codario, 2011).

**Psychological Counseling**

Changing one’s lifestyle requires guidance and determination. Losing weight, for example, takes encouragement, monitoring, and practical advice, even for people who are only slightly overweight. Moreover, time works against lifestyle changes. After the initial enthusiasm diminishes, exercise programs can be difficult to maintain. Likewise, lost weight is notorious for reappearing: people who diet on their own tend to regain the lost weight in less than a year.

Organized fitness programs can help a person to continue exercising over longer periods, and organized balanced dieting programs such as Weight Watchers, Jenny Craig, and Medifast can help mildly overweight (and motivated) patients to maintain lower weights. Low-fat and low-carbohydrate diets may also be effective but may require close monitoring and additional nutritional supplementation. For patients who are moderately or severely overweight, however, professional behavioral therapy is usually necessary (Codario, 2011).

**Surgery**

Therapeutic lifestyle changes and medications work least often in severely obese patients. For these patients, bariatric surgery is an option. Surgery is considered if the patient has tried monitored dieting, exercise regimens, and medications.

- Typically, surgery is only recommended for extremely obese (class 3) patients, that is, patients with a BMI >40 kg/m² (Codario, 2011).
• Class 2 patients—those with a BMI of 35.0 to 39.9 kg/m²—are considered for surgery when they have sleep apnea or components of metabolic syndrome (Codario, 2011).

• Although some small trials have suggested that bariatric surgery produces a glycemic benefit in less overweight patients with diabetes—those with a BMI of 30–35 kg/m²—there is insufficient evidence at this time to generally recommend surgery in patients with a BMI under 35 kg/m² unless they are part of a clinical study (ADA, 2011).

The best hospitals for bariatric surgery are those that perform a significant number of the surgeries and that use a team (physician, psychologist, physical and occupational therapists, and dietitian) to treat patients. Patients making their decision to have surgery should be aware of quality and standards for centers that perform bariatric surgery.

BARIATRIC SURGERY ACCREDITATION

The American College of Surgeons and the American Society for Metabolic and Bariatric Surgery combined their respective national bariatric surgery accreditation programs into a single unified program to achieve one national accreditation standard for bariatric surgery centers: the Metabolic and Bariatric Surgery Accreditation and Quality Improvement Program (MBSAQIP). MBSAQIP accreditation is important because it provides an objective and measurable way in which a center demonstrates that it offers high-quality care to patients in the setting of a multidisciplinary team approach.

Source: MBSAQIP, 2014.

In a meta-analysis of studies evaluating bariatric surgery and its effect on type 2 diabetes, 78% of people with diabetes saw their disease completely disappear after having the surgery, and in 86%, the disease was resolved or improved (ADA, 2011). Even in the best of circumstances, however, slimming down to a person’s ideal weight is not a realistic goal. With optimal treatment, bariatric patients are likely to lose 50% to 60% of their excess weight.

Surgery can be problematic because it does not remove the reason for the obesity in the first place. This can often be addressed only when the individual commits to both reduction in food intake and increased physical activity. Thus, after surgery, life-long lifestyle support and medical monitoring is necessary (ADA, 2011).

TYPES OF BARIATRIC SURGERY

Bariatric surgery assists with weight loss in two ways: restriction of the amount of space in the stomach (limiting intake of food) and malabsorption by shortening or bypassing the small intestine (reducing absorption). There are four types of bariatric surgery:

• **Roux-en-Y gastric bypass** is one of the most common bariatric surgical procedures, in which the surgeon creates a small pouch at the top of the stomach and attaches a narrow portion of the small intestine directly to the pouch, limiting the amount of food a person can eat as well as the amount of calories and nutrients absorbed.
• **Adjustable laparoscopic gastric banding** is a procedure that involves placing a band with an inflatable balloon around the upper part of the stomach. The band restricts the size of the stomach as well as narrows the opening to the rest of the stomach. A port placed under the skin in the abdominal area is connected and used to inflate or deflate the band to adjust the size. This procedure restricts the amount of food intake, with an early feeling of fullness.
• **Sleeve gastrectomy** is a procedure involving the surgical removal of a section of the stomach. The remaining part of the stomach is formed into a smaller tube-like structure. The smaller stomach restricts the amount of food intake and decreases the production of ghrelin (a hormone that regulates the appetite).

• **Duodenal switch with biliopancreatic diversion** begins with the removal of a large part of the stomach, leaving the connection to the first part of the small intestine (duodenum). The middle section of the small intestine is closed off and reattached to the end of the intestine, allowing the bile and pancreatic juices to flow normally. As a result, the patient has a smaller stomach, restricting food intake as well as limiting absorption because food bypasses most of the small intestine (Mayo Clinic, 2013).

**POST-SURGICAL CARE**

Clinical guidelines have been developed for nutrition care after bariatric surgery, with an emphasis on detection and management of complications such as vitamin and mineral deficiencies, osteoporosis, and hypoglycemia. The goals of nutrition care after surgery are to provide adequate energy and nutrition to support lean body mass during extreme weight loss, support tissue healing, and encourage foods and liquids that maximize weight loss and promote weight maintenance while minimizing side effects of reflux, dumping syndrome, and early satiety (Franz & Evert, 2012).

After surgery, life-long lifestyle support and medical monitoring is necessary. Physical therapists and occupational therapists are an integral part of the rehabilitation team supporting patients in the post-operative and recovery period. Early mobilization, with assistance from occupational therapists who teach activities of daily living and physical therapists who create and monitor a regular exercise and strengthening program, is an important part of long-term recovery (ADA, 2011).

**PREVENTION OF METABOLIC SYNDROME**

At each stage of life, strategies can be implemented to reduce the chance of developing metabolic syndrome, even for those individuals who have inherited a predisposition to it.

**Prenatal**

Malnutrition of mother and child during pregnancy leads to low birth weight of the infant. Such a child will have a higher than normal risk of developing hypertension, abnormal glucose tolerance, and cardiovascular disease as an adult. A pregnant mother who gets good prenatal care and who eats a healthy diet will reduce her baby’s chances of developing metabolic syndrome later in life.
Children and Adolescents

In the United States, approximately 17% (or 12.5 million) children and adolescents aged 2–19 years are obese (CDC, 2011b). Obesity in children increases the chance that they will have metabolic problems, high blood pressure, kidney problems, and cardiovascular disease as adults. It is therefore important that children be given guidance and encouragement to eat a healthy diet.

In addition, having a low level of physical exercise increases the chances that a child will develop metabolic syndrome as an adult even for children who are not overweight. Thus, children should be encouraged to be active: sedentary pastimes, such as television-watching and video/computer games, should be limited.

Adults

SCREENING

General education programs can reduce the incidence of metabolic syndrome by making everyone aware of the benefits of staying slim and exercising. People who already have metabolic syndrome can prevent many of the serious health problems by losing weight, eating a balanced, healthy diet, and exercising more. The first steps are to identify patients and then to advise them on the lifestyle changes that may benefit the condition. Carefully monitoring their condition over time is also important.

When patients come to doctors, clinics, and hospitals for any reason, healthcare providers should be aware of those who might have metabolic syndrome. Beyond this, some clinicians suggest that it would be worthwhile to institute screening programs that measure waist size, blood pressure, and blood lipid and blood glucose levels.

EDUCATION

Without treatment, metabolic syndrome poses worsening risks with age. On the other hand, young adults with metabolic syndrome who lose weight and then maintain a stable weight can avoid the higher incidence of serious health problems that would have come with advancing age.

Maintaining a reasonable body weight is a key technique for preventing metabolic syndrome. Overweight people should be advised to maintain a healthy weight. The best way to lose weight is to eat fewer calories, but this simple advice is not easy to carry out. People usually have the most success losing weight when they are part of a formal program that provides monitoring and counseling for continued support.

A second key preventive step is increasing physical activity. Although regular exercise can help a person lose weight, the most important benefits of physical activity are metabolic. Moderate exercise for >30 minutes four times a week can actually change the balance of biochemical processes in a person’s body, reducing insulin resistance, lowering triglycerides, and lowering blood pressure.
Lifestyle change strategies that include setting reasonable goals, raising awareness, identifying barriers to change, managing stress, preventing relapse, and providing ongoing support are the keys to long-term success in managing metabolic syndrome.

QUESTIONS PATIENTS MAY ASK ABOUT METABOLIC SYNDROME

Healthcare professionals who are available to consult with patients should know straightforward answers to basic questions that patients and their families might ask. Here are a few important questions and answers about metabolic syndrome.

Informational Questions

Q: What is metabolic syndrome?

A: To say someone has metabolic syndrome means that they have a specific group of health problems: they may have excess abdominal fat, high cholesterol, excess sugar circulating in their bloodstream, and high blood pressure. Having all these problems together makes a person more likely to get heart disease and diabetes.

Q: Is metabolic syndrome different from diabetes, high blood pressure, or high cholesterol?

A: Yes. Although these problems are all closely related, a person can have diabetes, high blood pressure, or high cholesterol without having metabolic syndrome.

Q: What will metabolic syndrome do to me?

A: When metabolic syndrome is not treated, you are more likely to get diabetes, heart and kidney disease, clogged arteries, and strokes. If you have chronic illnesses, metabolic syndrome tends to make them worse.

Q: Is metabolic syndrome contagious?

A: No.

Q: Will my children get metabolic syndrome?

A: Your children may inherit a tendency to develop the same problems that you have. You can help to protect them from metabolic syndrome by teaching them to eat healthy. Provide a diet focused on fruits, vegetables, low fats, and whole grains. Limit fast food, sugary desserts, and foods made with solid fats like butter and trans-fats. In addition, encourage your children to be...
active. Limit their TV, smart phone, and video game time, and encourage programs such as
dance and sports.

Q: What treatment will a doctor suggest?

A: A doctor may recommend losing weight, getting more exercise, and improving your diet. If
these steps do not improve your metabolic syndrome, a doctor may prescribe drugs, such as high
blood pressure medicine or cholesterol-lowering medicine.

Advice and Triage Questions

Q: Is metabolic syndrome an emergency?

A: Discovering that you have metabolic syndrome is not an emergency—it is a warning.
Having metabolic syndrome means that you may need to change your eating habits and increase
your physical activity; in addition, your doctor may need to adjust your current medicines or to
prescribe additional medicines. The longer you have metabolic syndrome without treatment, the
higher your chances for developing life-threatening problems such as diabetes and heart and
artery disease.

Q: What kind of doctor or clinic should I go to?

A: Start with your personal physician or a medical clinic that you normally use. A family
physician or a doctor specializing in internal medicine will be able to treat metabolic syndrome.

Q: What can I do on my own for metabolic syndrome?

A: Here are four things that are safe and effective.

1. Weight loss. Aim for a 5% to 7% weight loss in a year. Losing weight is hard to do on
your own. You may want to seek the help of an organized program.

2. More physical activity. Try to get in the habit of doing regular exercise for 30 minutes
each day. Consider walking, swimming, bicycling, or dancing. Organized fitness
programs can help you maintain healthy exercise habits.

3. Diet changes. Change your eating habits. Eat fewer fatty foods and more fruits,
vegetables, and whole grains. Eat at regular times and have healthy snacks available, such
as vegetables, nuts, and seeds.

4. If you do smoke, seek support to stop smoking.
CONCLUSION

Metabolic syndrome is the combination of:

- Insulin resistance
- Excess intra-abdominal fat
- Unhealthy levels of fats in the blood (too much triglyceride and too little HDL cholesterol)
- High blood pressure

Having metabolic syndrome makes a person more likely to develop diabetes and cardiovascular disease, especially men >45 years of age and women >55 years of age. Metabolic syndrome is a common health problem, and it is becoming increasingly common in those parts of the world where obesity is on the rise.

If they occur in isolation, some of the individual disorders that make up metabolic syndrome may not be in the range mandating treatment; on the other hand, when these disorders are found together, they should always be treated.

Treatment of metabolic syndrome begins with therapeutic lifestyle changes. Weight loss, improved diet, and regular physical exercise are the elements of the initial treatment program. Drugs are used to treat those components of metabolic syndrome that do not improve sufficiently with therapeutic lifestyle changes alone.

RESOURCES

IDF Worldwide Definition of the Metabolic Syndrome (International Diabetes Federation)
http://www.idf.org/metabolic-syndrome

Metabolic Syndrome (American Heart Association)
http://www.heart.org/HEARTORG/Conditions/More/MetabolicSyndrome/Metabolic-Syndrome_UCM_002080_SubHomePage.jsp

Metabolic Syndrome (Mayo Clinic)
http://www.mayoclinic.org/diseases-conditions/metabolic-syndrome/basics/definition/con-20027243

Metabolic Syndrome (MedicineNet.com)
http://www.medicinenet.com/metabolic_syndrome/article.htm

Metabolic Syndrome (National Library of Medicine)

What Is Metabolic Syndrome? (National Heart, Blood, and Lung Institute)
http://www.nhlbi.nih.gov/health/health-topics/topics/ms/
REFERENCES


Metabolic Syndrome


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1. Type 1 diabetes is characterized by:
   a. Insulin resistance.
   b. Onset generally in older adults.
   c. Insulin sensitivity.
   d. Pancreatic overproduction.

2. A 50-year-old male patient with a history of obesity and hyperlipidemia is diagnosed with type 2 diabetes. Which characteristic finding supports the patient’s diagnosis?
   a. Insulin insensitivity
   b. Rapid intake of glucose in the tissues
   c. Low blood pressure
   d. First appearance at an early age

3. Characteristics of metabolic syndrome include intra-abdominal obesity:
   a. Hypoglycemia, and high HDL.
   b. Hypertension, and low HDL.
   c. Pulmonary hypertension, and insulin sensitivity.
   d. Hypotension, and low LDL.

4. In assessing a male patient, the clinician considers which finding to be a possible indicator of metabolic syndrome?
   a. Waist circumference >102 cm (>40 inches)
   b. Blood triglycerides >100 mg/dl
   c. Blood HDL-C <50 mg/dl
   d. Blood glucose >75 mg/dl

5. Following the AHA/NHLBI definition of metabolic syndrome, the clinician can rule out the syndrome if a patient has normal blood pressure and:
   a. Elevated triglyceride levels.
   b. Normal total cholesterol levels.
   c. Normal fasting glucose levels.
   d. Low HDL levels.
6. Among adolescents, metabolic syndrome is more common in which subgroup?
   a. Boys
   b. Girls
   c. African Americans
   d. Hispanics

7. Which is a true statement about one of the causes of obesity?
   a. Obesity is usually caused by an endocrine disorder.
   b. Genetics plays a very small role in a person’s becoming obese.
   c. There is no proven link between depression and weight gain.
   d. Anti-diabetic drugs may cause weight gain.

8. Which health condition directly causes insulin resistance?
   a. Ovarian cancer
   b. Coronary heart disease
   c. Excess visceral fat
   d. Poor hygiene

9. What is the role of HDL lipoproteins in maintaining the balance of lipids in the body?
   a. Providing a process to carry carbohydrate and protein within the body
   b. Assisting in the transfer of proteins to the muscles
   c. Slowing or reversing the plaque formation in the vascular system
   d. Increasing the amount of cholesterol in nonliver cells

10. A nurse is assessing a female patient with new-onset high blood pressure. Which other physical finding or health condition causes the nurse to classify the patient as high risk for metabolic syndrome?
    a. Hyperinsulinemia
    b. No history of having previously given birth
    c. Chronic asthma
    d. Osteoarthritis

11. A nurse is assessing a patient who is being evaluated for high blood pressure and notes excess weight in the patient’s chest and abdomen. The nurse documents the patient as having which body shape?
    a. Gynecoid (pear-shaped)
    b. Ovoid (egg-shaped)
    c. Android (apple-shaped)
    d. Droid (star-shaped)
12. Which clinical measurement is directly useful for diagnosing metabolic syndrome?
   a. A blood urea nitrogen (BUN) level
   b. A coronary angiogram (catheterization)
   c. A chest x-ray
   d. A waist circumference measurement

13. What is the minimum blood pressure level for making a diagnosis of metabolic syndrome?
   a. >120/80 mm Hg
   b. >130/80 mm Hg
   c. >130/85 mm Hg
   d. >140/90 mm Hg

14. A patient is suspected of having elevated blood pressure. To document the most accurate level, the clinician’s action is to measure the patient’s blood pressure:
   a. While the patient is standing.
   b. On more than one occasion.
   c. Early in the day.
   d. Both before and after exercise.

15. Hyperglycemia means blood with more than the normal amount of:
   a. Insulin.
   b. Glucose.
   c. Triglycerides.
   d. Cholesterol.

16. A formal diagnosis of diabetes is based on persistent:
   a. Higher-than-normal plasma glucose levels.
   b. Higher-than-normal urinary albumin levels.
   c. Symptoms of polyuria, polydipsia, and weakness.
   d. Symptoms of retinopathy, neuropathy, or nephropathy.

17. A reading above which fasting blood glucose value would suggest possible metabolic syndrome?
   a. 45 mg/dL
   b. 60 mg/dL
   c. 100 mg/dL
   d. 110 mg/dL
18. Which fasting blood triglyceride value suggests metabolic syndrome?
   a. 100 mg/dL
   b. 120 mg/dL
   c. 140 mg/dL
   d. 160 mg/dL

19. Which fasting blood HDL cholesterol level suggests metabolic syndrome in a female patient?
   a. 40 mg/dL
   b. 70 mg/dL
   c. 90 mg/dL
   d. 110 mg/dL

20. “Atherogenic” is a term referring to the tendency to:
   a. Cause blood clots.
   b. Cause atherosclerotic plaque.
   c. Break up blood clots.
   d. Reduce atherosclerotic plaque.

21. Which pair of serious health problems are the most common results of metabolic syndrome?
   a. Colon cancer and stroke
   b. Coronary heart disease and type 2 diabetes
   c. Pulmonary hypertension and portal hypertension
   d. Breast cancer and prostatic cancer

22. Which is a true statement about treatment for metabolic syndrome?
   a. Glucose levels can be ignored until other risk assessment components are present.
   b. Triglyceride levels can be ignored until other risk assessment components are present.
   c. Metabolic syndrome lowers the threshold for the treatment of all its components.
   d. Only blood pressure levels >140/90 mm Hg need to be treated.

23. Physical exercise provides which important benefit for patients with metabolic syndrome?
   a. Increased blood levels of LDL cholesterol
   b. Reduced blood levels of HDL cholesterol
   c. Reduced insulin resistance
   d. Increased blood pressure
24. Statins are the first choice drugs for lowering blood levels of:
   a. LDL cholesterol.
   b. HDL cholesterol.
   c. Fatty acids.
   d. Glucose.

25. A patient with metabolic syndrome is found to have an accompanying prothrombotic state. Which therapy is initially prescribed for the patient?
   a. Heparin injections, daily
   b. Warfarin (Coumadin)
   c. Vitamin K
   d. Low-dose, daily aspirin

26. Self-care measures to prevent metabolic syndrome include:
   a. Increasing calories and adding high fats to the diet.
   b. Drinking fruit juices and taking a multivitamin.
   c. Maintaining a healthy weight and regular exercise.
   d. Avoiding fad diets and weight training.