

A practical guide to metabolic syndrome

Clinical Advisor

February 2012

Copyright 2012 Haymarket Media. All Rights Reserved



Section: FEATURE; Clinical Feature; CME/CE

Length: 6780 words

Byline: Jacinta Thomas, APRN-C, and Deborah K. Walker, DNP, CRNP, AOCN

Highlight: With obesity on the rise, more patients are at risk for metabolic syndrome, a diagnosis given to a set of simultaneous disorders.

Body

HOW TO TAKE THE POST-TEST: [Click here](#) after reading the article to take the post-test on [myCME.com](#).

At a glance

In the United States, approximately 34% of adults carry the diagnosis of metabolic syndrome.

Obesity, lipid levels, BP, and insulin resistance should be considered when diagnosing metabolic syndrome.

Screening should include a physical examination, a dietary history, and laboratory workup.

The goal of treatment is to delay or prevent CVD and diabetes.

The National Cholesterol Education Program (NCEP) Adult Treatment Panel III (ATP III) report identified a constellation of factors that increased an individual's risk of developing cardiovascular disease (CVD).¹ Metabolic syndrome is a general diagnosis given to a set of disorders that a patient experiences simultaneously, including hyperglycemia, elevated BP, dyslipidemia and abdominal obesity.

In the United States, approximately 34% of adults have been diagnosed with metabolic syndrome.² As [obesity rates increase](#), the incidence of metabolic syndrome is also expected to rise. The damaging effects of metabolic syndrome may place affected patients at a higher risk of developing CVD and type 2 diabetes mellitus (T2DM).³

The exact societal cost of the syndrome is unknown because the disorder can vary so much. However, given that the annual projected costs associated with hypertension, diabetes, cholesterol disorders and obesity are escalating, one can expect that the annual cost associated with metabolic syndrome will be astronomical. Sullivan and colleagues estimated the loss in productivity resulting from metabolic risk factors was \$17.3 billion annually.⁴

A practical guide to metabolic syndrome

Boudreau et al studied the health-care utilization of patients with metabolic syndrome and found that the cost of care for patients with diabetes who were obese and had dyslipidemia and hypertension was almost twice that of patients with prediabetes who had the same risk factors (\$8,067 compared with \$4,638).⁵

Major organizations, such as the American Heart Association (AHA) and the American Diabetes Association (ADA), do not agree on the usefulness of metabolic syndrome in clinical practice. Several studies have testified to the significance of the syndrome as a diagnostic predictor of CVD.^{6,7} Yet many highly acclaimed scientists refute the validity of the syndrome to predict cardiovascular or diabetes risk as postulated by others in the medical community.^{8,9} Reaven adamantly believes the syndrome is a "pathological process" and challenges the scientific world to stop "spinning" the notion that it is anything more.⁹

Despite the rather intense battles about the utility of a diagnosis of metabolic syndrome in clinical practice, most parties agree that the risk factors associated with the syndrome are problematic for the patient, public health, and health-care providers.¹⁰⁻¹²

Components of metabolic syndrome

While not all organizations agree on the requirements for diagnosing metabolic syndrome (*Table 1*), there is consensus about the factors that should be considered. These include whether the patient is obese or overweight, lipid levels, BP and the presence or absence of insulin resistance.

Obesity. In women, the metabolic syndrome affects 33.1% of overweight persons and 56.1% of obese and extremely obese individuals; slightly more than 10% of normal-weight and underweight persons have the syndrome.² In men with metabolic syndrome, only 6% were in the normal-weight category, with more than 93% in the overweight or obese category. Metabolic syndrome is linked more to central visceral obesity than to overall obesity. Several studies cite central obesity as the pivotal component in development of the syndrome.^{13,14}

The proposed connection between obesity and the risk factors for metabolic syndrome are complex. Increase in visceral fat leads to central obesity and has been linked to increased insulin resistance as a result of hormonal influences.¹⁵ Also, cortisol has been widely studied as a culprit in stimulating the appetite for high-carbohydrate, fatty foods that lead to increased deposition of fat around the abdomen.

The hormones thought to play the greatest role in the development of obesity-related metabolic syndrome are the adipokines, leptin, and adiponectin.¹⁶ The hormone leptin helps regulate appetite and storage of fat and influences thermogenesis to burn calories. Sudden increase in weight or extreme weight gain disturbs the leptin regulation, thereby allowing deposition of fat in the visceral areas as well as inciting an increase in triglyceride storage in vital organs, such as the heart, muscles and liver.¹³

Dyslipidemia. In obese patients with metabolic syndrome, deregulation of the hormonal system often leads to dyslipidemia. Levels of the hormone adiponectin were found to be inversely related to visceral fat and lower in patients with coronary artery disease.¹⁴ Adiponectin promotes insulin sensitivity and has an antiatherogenic effect.¹⁷ The patient with metabolic syndrome usually has normal levels of LDL, although the lipoproteins themselves are believed to be denser and smaller in nature, which makes them more atherogenic. Levels of triglycerides and HDL are often elevated in the patient with metabolic syndrome.¹²

Insulin resistance. In insulin resistance, the body produces insulin, but it is not used properly. This cyclic dysfunction leads to higher glucose levels in the bloodstream, which causes more insulin production (hyperinsulinemia).

An increase in total body fat is independently related to insulin resistance. Scientists believe that adipose tissue releases additional adipokines that are insulin antagonists, such as tumor necrosis factor-alpha (TNF-greekalpha), interleukin-6 (IL-6), and resistin.¹⁷ These inflammatory factors are responsible for insulin resistance, the production of C-reactive protein (CRP), and increased adhesion of WBCs and molecules to endothelial cells.¹⁸

A practical guide to metabolic syndrome

Hypertension. Hyperinsulinemia and obesity in metabolic syndrome raises patients' risk for [elevated BP](#). Elevated insulin levels increase the kidney's absorption of sodium and water, which in turns increases blood volume and elevates the BP.¹⁹ Obese patients require a larger-than-normal cardiac output because of increased blood volume. In addition, sympathetic system in obese patients is overactive, leading to constriction of peripheral arteries, sodium retention and vascular resistance. The presence of any of these actions, alone or synergistically, can lead to hypertension.

In 2009, a group of organizations released a set of diagnostic criteria on which they agreed (*Table 2*).²⁰

The organizations involved were the International Diabetes Federation (IDF) Task Force on Epidemiology and Prevention; the National Heart, Lung, and Blood Institute; the AHA; the World Heart Federation; International Atherosclerosis Society; and the International Association for the Study of Obesity.

Consequences of metabolic syndrome

In many individuals with insulin resistance, hormonal imbalance and obesity are interrelated, resulting in even more devastating consequences of the metabolic syndrome. Santos and Fonseca found that metabolic syndrome was more prevalent in patients with inflammatory disorders, such as systemic lupus erythematosus (SLE) and rheumatoid arthritis.²¹

The researchers also discovered increased incidents of atherosclerosis in this same subset of patients. In a post hoc analysis of the Multiethnic Study of Atherosclerosis, Afonso et al discovered that of the four groups studied (no metabolic syndrome and no microalbuminuria, microalbuminuria only, metabolic syndrome only and metabolic syndrome and microalbuminuria), the group with both metabolic syndrome and microalbuminuria had higher levels of inflammatory markers and more subclinical atherosclerosis than the other groups.²²

Further studies have shown that patients with metabolic syndrome demonstrate increased stiffness in precapillary vessels, which impedes subcutaneous microcirculation and makes cardiac events more likely.⁸ Investigators for the Strong Heart Study proved that patients with metabolic syndrome had a higher in-hospital death rate after acute ST-elevation MI.²³ Zhao et al determined that in patients with metabolic syndrome, progression of coronary stenosis was increased by 50% and frequency of cardiovascular events was increased by 64% compared with those without the syndrome.²⁴

In addition to increased cardiovascular risk, metabolic syndrome has been linked to polycystic ovary syndrome (PCOS), sleep apnea, dementia and fatty liver.¹⁰ Moreover, recent studies have linked the metabolic factors of obesity, elevated glucose and increased triglycerides to the development of macrosomia, obesity and metabolic syndrome in the unborn child.^{25,26} Clinicians will have to rethink the care of the pregnant woman, insisting on lifestyle interventions to prevent the passage of unhealthy metabolic conditions to her child.

Tenenbaum and Fisman reported that patients with metabolic syndrome and hyperglycemia had higher mortality rates than patients with the same level of hyperglycemia and no metabolic syndrome; they also reported a 30-day mortality rate of 8.3% vs 2.5% ($P <.05$), respectively.²⁷

Towfighi, Zheng and Ovbiagele reported that the incidence of stroke has more than tripled for women in the past 20 years, and they attribute this increase to the rising incidence of obesity.²⁸ In a meta-analysis that included 951,083 patients and 87 studies, Mottillo and colleagues found that metabolic syndrome was associated with a twofold increase in cardiovascular outcomes and a 1.5-fold increase in all-cause mortality.²⁹

After analyzing the data from 22,719 individuals in the Reasons for Geographic and Racial Differences in Stroke (REGARDS) study, Brown, Voeks, Bittner and Safford found that 47% of the participants had metabolic syndrome.⁶ With such large numbers of patients falling into the metabolic syndrome category, the need for standardized diagnostic criteria and treatment protocols persists even in the midst of the controversy over the syndrome's characteristics and utility.

Screening guidelines

A practical guide to metabolic syndrome

The assessment of patients suspected of having metabolic syndrome should include a physical examination, a dietary history and laboratory workup.

Physical examination. During the initial visit, measure the patient's waist and calculate the BMI. Measurement of waist circumference is important because adiposity is related to cardiovascular risk.³⁰ Obesity is defined as a BMI >30, and overweight is defined as a BMI of 25 to 29; a BMI of 18.5 to 24.9 indicates normal weight.³¹

However, a study of 32,024 participants revealed that the method used to define obesity—whether by body-fat percentage, truncal obesity measurements or BMI—did not change the prevalence of metabolic syndrome, indicating that all measurements were equal.³² Coutinho et al analyzed 16,000 patients with coronary disease and found that increased waist circumference was associated with increased risk of death (hazard ratio [HR], 1.70; 95% confidence interval, 1.58-1.83).³³ The study showed that central obesity was associated with an increased risk of death regardless of BMI. Thirty percent of all deaths in the study were attributed to central obesity independently,³³ thereby confirming the need to measure both BMI and waist circumference to more adequately determine risk category and tailor treatment to the individual.

BMI can easily be calculated in adults once the height and current weight are plugged into the formula: weight (lb)xC3height (in)2x703. The NIH classifications of obesity should be used only as a general guideline, as there are ethnic-specific considerations of obesity that should be applied to each patient.²⁰ Rahman and Berenson found that increased percentage of body fat and obesity among white, black and Hispanic patients corresponded more with a BMI >=25.5, 28.7 and 26.2, respectively.³⁴ Therefore, counseling on ways to prevent obesity should begin long before the patient reaches a BMI of 30.

After the BMI has been determined, a screening waist circumference should be obtained with the provider standing on the patient's right side. The patient's right iliac crest is palpitated and marked, and then a vertical line is drawn from the mid-axillary line. The measuring tape is then placed in a horizontal plane at the level of the mark. The tape should be parallel to the floor and the measurement should be taken during normal respiration.³¹

According to guidelines in *The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure* (JNC 7), a screening BP measurement should be taken in the office while the patient is seated, with the arm at the level of the heart.³⁵ Two separate readings are obtained, and elevated readings are confirmed in the contralateral arm. Every office visit should include BP measurement and assessment of risk factors or other comorbidities.

A systolic BP of 120 to 139 mm Hg and diastolic BP of 80 to 89 mm Hg is considered prehypertension. BP readings >130/80 mm Hg in patients with diabetes or kidney disease are a risk factor for metabolic syndrome, and readings >130/85 mm Hg are a risk factor for those without chronic disease. BP readings >140/90 mm Hg require treatment, often with more than one antihypertensive. BP screenings are recommended every two years in patients aged 20 years and older.¹¹

Diet and exercise history. The next step in the assessment for metabolic syndrome is to document a diet and exercise history. Overeating and inactivity are known risk factors for obesity.¹¹ Discussion with patients should address barriers to exercise, support systems and their readiness to change their current regimen.

Open-ended, nonjudgmental questions, such as "How do you feel about exercise?" rather than "Do you exercise?" will provide the clinician with more useful information to help determine the patient's needs; unbiased comments will also help establish trust with the patient.³⁶ Establishing a trusting relationship with the patient can help both patient and provider meet short- and long-term goals for weight loss.

A careful food-consumption history should be taken on the first visit as well. Review of the diet history will give clues as to what dietary habits need improving and provide an estimate of the number of calories consumed.³⁷ That information will be important when determining an appropriate caloric goal.³¹

Laboratory studies. Assessment for metabolic syndrome includes screening for lipids during normal stress levels, as acute illness, increased stress and eating within nine hours of testing can produce falsely elevated results.³⁰ A

A practical guide to metabolic syndrome

complete lipid profile should be obtained, including LDL (normal, <100 mg/dL), HDL (normal, >40 mg/dL), triglyceride (normal, <150 mg/dL) and total cholesterol (normal, <200 mg/dL) levels. Additional tests to rule out other differential diagnoses of elevated lipid levels include serum thyroid-stimulating hormone, blood urea nitrogen, creatinine, liver function tests and urinalysis.

Once the lipid level is obtained, assess the risk of coronary heart disease. The Framingham risk assessment can be used to determine the patient's risk factors for coronary heart disease or a cardiac event in the next 10 years.³⁸ Diabetes, peripheral vascular disease, abdominal aortic aneurysm and symptomatic carotid disease are considered risk equivalents for CVD in the Framingham risk calculations.¹²

Other Framingham risk predictors are age, total cholesterol, HDL, smoking, antihypertensive treatment and BP. Points are assigned to each risk factor, and the total number of points determines LDL levels at which to begin therapeutic lifestyle changes and drug therapy.³⁰ The risk factors are stratified separately for men and women.

Note that Framingham risk calculations underestimate cardiovascular risk in patients with T2DM, so be cautious about using them in that subset of patients.³⁸

In addition, Sumner and colleagues have challenged the validity of the previously stated lipid classifications for metabolic syndrome as they relate to blacks.³⁹ The Triglyceride and Cardiovascular Risk in African Americans (TARA) study showed that even obese, insulin-resistant black patients often have low triglyceride levels; 30% of the study participants were insulin-resistant, but only 2% had high triglyceride levels.³⁹ The researchers stated that in comparative studies, 60% of whites with insulin resistance also had elevated triglycerides. Moreover, the researchers believe that both the IDF and the AHA criteria miss many blacks who are at risk for metabolic syndrome.³⁹

The oral glucose tolerance test (OGTT) is the most sensitive assay for detecting overt T2DM in patients with prediabetes; however, performing this test in the office setting is time-consuming. Therefore, a fasting glucose determination is used more often. Patients with a history of impaired fasting glucose (IFG) or impaired glucose tolerance (IGT) should be screened every one to two years for T2DM.⁴⁰ Balkau et al proved that serum glucose levels were more predictive of diabetes than was an HbA_{1c} determination.⁴¹ In 2010, however, the ADA added an HbA_{1c} determination to the tests for diabetes; levels >6.5% are diagnostic. The levels of serum glucose and OGTT results diagnostic of diabetes were unchanged at >=126 mg/dL and >=200 mg/dL, respectively.

A random blood glucose >200 mg/dL that is accompanied by complaints of increased thirst, urination, hunger and fatigue is considered a positive indicator of diabetes. IFG, which is a risk factor for metabolic syndrome, is defined as a blood glucose reading of 100 to 126 mg/dL.⁴⁰

Treatment of metabolic syndrome

The goal of metabolic syndrome treatment is to delay or prevent CVD and diabetes. Treatment of metabolic syndrome and its risk factors focuses on healthy lifestyle interventions, such as exercise and a nutritious diet. Patients whose metabolic syndrome persists may try behavior modification or pharmacotherapy.

Exercise. Vigorous activity for 45 to 60 minutes at least five times a week, but preferably daily, is recommended to aid in achieving and maintaining a healthy weight.⁴² The Diabetes Prevention Program study showed that for at-risk persons, a loss of approximately 10% of body weight could prevent or delay development of diabetes or other metabolic disorders.¹²

Resistance training in particular has received a great deal of attention in the treatment of metabolic syndrome. It was found to have a null effect on triglycerides, HDL, LDL and diastolic BP, but resistance training has been shown to decrease systolic BP, HbA_{1c} and obesity.⁴³ The AHA recommends resistance training two days a week in addition to behavioral changes in a physical-activity regimen.¹¹ Using a pedometer to track exercise, taking the stairs, and reducing the amount of time spent in such sedentary activities as watching television can all be effective in reducing body weight.

A practical guide to metabolic syndrome

Weight reduction results in improvement of all metabolic risk factors. Therefore, providers are encouraged to tailor an exercise regimen based on the patient's individual characteristics and specific risk factors that predispose him or her to metabolic syndrome. Patients with metabolic syndrome who are at high risk for cardiovascular events should be medically supervised in their physical activity; some may require an exercise stress test to detect life-threatening abnormalities before initiating an exercise program.⁴²

The Oslo Diet and Exercise Study revealed that diet and exercise together produced the greatest reduction in the incidence of metabolic syndrome. After one year, metabolic syndrome affected 32.6% of the diet-and-exercise group, 64.7% in the diet-only group, and 76.5% of the exercise-only group.⁴⁴ Similarly, Yassine et al found that exercise and caloric restriction vs. exercise alone produced greater weight loss (6.8 kg \times 2.7 kg vs. 3.4 kg).⁴⁵ After controlling for diet, increased physical activity was most associated with a decreased likelihood of developing metabolic syndrome in the Finnish Diabetes Prevention Study.⁴⁶

Healthy diet. Treatment of metabolic syndrome also includes eating a proper diet. The Mediterranean diet consists of a lot of green leafy vegetables, fiber, fish, olive oil and nuts; low intake of saturated fats, *trans* fats and cholesterol has proven to be effective in aiding weight loss in patients with metabolic syndrome.^{40,47} Reducing daily caloric intake by 500 to 1,000 calories along with exercise will help place the body in a calorie-deficient state so that weight loss can occur.

A reduction in consumption of refined sugars, sodium and high-glycemic food can aid in weight loss as well.⁴⁸ Much attention has been given to studies showing that consumption of diet drinks leads to metabolic syndrome and increases weight gain.⁴⁹ Scientists hypothesize that the artificial sugar increases cravings for other refined sugars, leading to weight gain and disruption of glucose metabolism. Also, patients with elevated lipid levels should keep fat intake in the range of 25% to 35% of calories. Fat intake >35% has the potential to increase LDL, whereas levels <25% will cause HDL to decrease and triglycerides to increase.³⁰ A consultation with a dietitian can help the patient reach preset goals. Patients with a history of kidney disease should refrain from high-protein diets because reduced renal function can ultimately lead to insulin resistance.⁴⁸

Behavior modification. Another important approach to maintaining weight loss is behavior modification. Providers should encourage patients to read all food labels, set goals for weight loss, keep food diaries or journals, and be active in their journey to a healthy weight.⁴⁰

For patients who are unable to achieve weight loss after a reasonable time, such as six to 12 months of aggressive lifestyle interventions, a trial of oral weight-loss medications may be beneficial. Orlistat (Xenical) is the only drug approved by the FDA for weight-loss maintenance. Bariatric surgery is another option for obese patients. Weight-loss surgery can be recommended for patients with a BMI >40 or a BMI >35 with comorbid conditions.⁵⁰

Medications. Treatment of dyslipidemia in metabolic syndrome centers around the goal of reducing LDL with lipid-lowering medications.²⁸ The revised ATP III recommends an LDL of <70 mg/dL for high-risk patients and <100 mg/dL for those at moderate risk.³⁰ The LDL for patients at low risk is <160 mg/dL, which is unchanged from the original 2001 guidelines. Once the primary goal of LDL reduction is reached, the secondary goal is to reduce elevated triglycerides. The tertiary goal is to increase HDL if levels are <40 mg/dL for men and <50 mg/dL for women.⁵¹

The 10-year risk of a first cardiovascular event averages 16% to 18% in metabolic syndrome patients.² Recent studies have evaluated the positive effect of statins, which include reduction of LDL and the inflammatory marker CRP, in metabolic syndrome patients.⁵² Statins combined with fibrates have the additive effect of reducing triglycerides and increasing HDL, but this combination has the potential to cause myopathy. Therefore, patients will have to be monitored closely.

Other combinations, such as fenofibrate and nicotinic acid, can be considered.⁵³ Great concern over the use of nicotinic acid in patients with metabolic syndrome continues because one of the side effects is possible elevation of blood glucose.⁵² Thus, providers are cautioned to use the smallest effective dose of nicotinic acid in patients with dyslipidemia and prediabetes.

A practical guide to metabolic syndrome

More recently, the presumed positive effect of niacin combined with statins has been scarred by the discontinuation of the AIM-HIGH trial.⁵⁴ The high-dose niacin-and-statin combination reduced triglyceride levels and raised HDL levels, but there was no reduction in heart attacks, strokes or hospitalizations for acute coronary syndrome. There was a small increase in ischemic stroke with niacin use. The usefulness of raising HDL has been questioned with the discontinuation of this study, leaving many researchers searching for another marker of lipid control beyond LDL.

Controlling blood sugar. IFG and IGT are also risk factors for metabolic syndrome that should be treated initially with lifestyle interventions. As previously stated, a weight loss of 7% to 10% of total body weight greatly reduces the patient's potential to develop diabetes.¹¹ Exercise improves insulin sensitivity and aids in weight loss. Reducing caloric intake by 500 calories, avoiding refined sugars and following a healthy diet will also be beneficial to the patient with IFG or IGT.⁴² The drug metformin has also been proven to slow down and impede the development of diabetes in patients with metabolic syndrome.⁵⁵

The ADA does not routinely recommend treatment of insulin resistance in the absence of diabetes. Instead, the organization states that treatment can be "considered" in patients who have a BMI >35, are younger than age 60 years, or have IFG and IGT plus other risk factors.⁴⁰ However, once T2DM develops, the recommendation is to use a combination of therapies, including lifestyle modifications and medications, to achieve an HbA_{1c} <7% to reduce risk of CVD.⁴⁰

Addressing BP. Lowering BP to <130/85 mm Hg is important in reducing the chance of cardiovascular events in patients at risk for metabolic syndrome. Lifestyle modifications are the starting point in any treatment related to hypertension control.

The findings in the JNC 7 support use of the Dietary Approaches to Stop Hypertension (DASH) diet, weight loss and moderate alcohol intake to reduce cardiovascular risk.³⁵ Also, according to the guidelines, patients with a history of diabetes should aim for a BP <=130/80 mm Hg.

Several drug combinations for the treatment of hypertension and metabolic syndrome have been studied, but no one combination has supremacy over another. Researchers have supported the use of angiotensin-converting enzyme (ACE) inhibitors in patients with metabolic syndrome, especially those who also have IFG or IGT.³⁰ Diuretics have been proven to reduce cardiovascular risk, but there is debate about progression to frank diabetes in those who have IFG or IGT. Hypertensive blacks with metabolic syndrome respond better to thiazide diuretics than to ACE inhibitors as first-line therapy, based upon findings of the ALLHAT trial.⁵⁶ Thus, clinical judgment is required to produce results that will bring the patient's BP to goal.

Other considerations. The prothrombotic state and the proinflammatory state in metabolic syndrome are also recognized as major risk factors for CVD.¹² The proinflammatory state involves elevation of cytokines and CRP. Weight loss improves the inflammatory response. Coagulation factors increase in patients with metabolic syndrome. Aspirin is a low-cost medication that can help prevent the likelihood of stroke in women and MI in men.

If there are no contraindications, aspirin should be considered for women aged 55 to 79 years and men aged 45 to 79 for primary prevention of CVD. According to the US Preventive Services Task Force, the daily use of aspirin will reduce the relative risk of stroke in women by 17% and the relative risk of MI in men by 32%.⁵⁷ There is no consensus on the aspirin dosage - either 81 mg or 325 mg can be considered; however, the higher dose is linked to GI bleeding.

Implications for practice

Health-care providers must suspect that patients with any one of the five metabolic risk factors can have other hidden risk factors that are silently working together to exacerbate the situation. This suspicion should lead to further inquiry into family and personal medical history.

Consideration should be given to prescriptions for lifestyle modifications, investigative diagnostic procedures and therapeutic medications, as deemed appropriate. The components that make up metabolic syndrome are at

A practical guide to metabolic syndrome

epidemic levels in the United States, and as waistlines are continuing to increase, solutions to this global problem are urgently needed.

This paper has provided a review of the metabolic syndrome and reported guidelines that can be utilized in the office setting. With continuing research, modifications in the recommendations for metabolic syndrome can be expected. The turbulent atmosphere surrounding the syndrome may continue, but as long as the goal is improvement in patient outcome, the medical community will have to put aside differences and treat patient as a whole and not as a set of debatable numbers.

Jacinta Thomas, APRN-C, practices internal and family medicine in Atlanta, and is a student in the DNP Program at the University of Alabama, Birmingham, where Deborah K. Walker, DNP, CRNP, AOCN, is an assistant professor. Neither author has any relationships to disclose relating to the content of this article.

HOW TO TAKE THE POST-TEST: To obtain CME/CE credit, please [click here](#) after reading the article to take the post-test on [myCME.com](#).

References

1. [National Cholesterol Education Program \(NCEP\) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults \(Adult Treatment Panel III\). "Third Report of the National Cholesterol Education Program \(NCEP\) Expert Panel on Detection, Evaluation and Treatment of High Blood Cholesterol in Adults \(Adult Treatment Panel III\) final report." Circulation. 2002;106:3143-3421.](#)
2. Ervin B. "Prevalence of metabolic syndrome among adults 20 years of age and over, by sex, age, race, and ethnicity, and body mass index: United States, 2003-2006." *Natl Health Stat Report.* 2009;13:1-7.
3. Wu SH, Liu Z, Ho SC. "Metabolic syndrome and all-cause mortality:a meta-analysis of prospective cohort studies." *Eur J Epidemiol.* 2010;25:375-384.
4. Sullivan PW, Ghushchyan V, Wyatt HR et al. "Productivity costs associated with cardiometabolic risk factor clusters in the United States." *Value Health.* 2007;10:443-450.
5. Boudreau DM, Malone DC, Raebel MA et al. "Health care utilization and costs by metabolic syndrome risk factors." *Metab Syndr Relat Disord.* 2009;7:305-314.
6. Brown TM, Voeks JH, Bittner V et al. "Variations in prevalent cardiovascular disease and future risk by metabolic syndrome classification in the REasons for Geographic and Racial Differences in Stroke (REGARDS) study." *Am Heart J.* 2010;159:385-391.
7. [de Simone G, Devereux RB, Chinali M et al. "Prognostic impact of metabolic syndrome by different definitions in a population with high prevalence of obesity and diabetes." Diabetes Care. 2007;30:1851-1856.](#)
8. [Bayturan O, Tuzcu EM, Lavoie A et al. "The metabolic syndrome, its component risk factors, and progression of coronary atherosclerosis." Arch Intern Med. 2010;170:478-484.](#)
9. Reaven GM. "The metabolic syndrome: time to get off the merry-go-round?" *J Intern Med.* 2011;269:127-136.
10. [National Heart, Lung and Blood Institute. What is metabolic syndrome? Website.](#)
11. [American Heart Association. What is metabolic syndrome? Website.](#)
12. Rosenzweig JL, Ferrannini E, Grundy SM, et al. Primary prevention of cardiovascular disease and type 2 diabetes in patients at metabolic risk; an Endocrine Society Clinical Practice Guideline. *J Clin Endocrinol Metab.* 2009;93:3671-3689.

A practical guide to metabolic syndrome

13. Gade W, Schmit J, Collins M, Gade J. Beyond obesity: the diagnosis and pathophysiology of the metabolic syndrome. *Clin Lab Sci.* 2010;23:51-61.
14. Whitmore C. Type 2 diabetes and obesity in adults. *Br J Nurs.* 2010;19:880-882.
15. Carlson JJ, Turpin AA, Wiebke G et al. "Pre- and post-prandial appetite hormone levels in normal weight and severely obese women." *Nutr Metab (Lond).* 2009;6:32.
16. [Antuna-Puente B, Feve B, Fellahi S, Bastard JP. "Adipokines: the missing link between insulin resistance and obesity."](#) *Diabetes Metab.* 2008;34:2-11.
17. [Després JP, Lemieux I, Bergeron J et al. "Abdominal obesity and the metabolic syndrome: contribution to global cardiometabolic risk."](#) *Arterioscler Thromb Vasc Biol.* 2008;28:1039-1049.
18. Fernández-Sánchez A, Madrigal-Santillán E, Bautista M et al. "Inflammation, oxidative stress and obesity." *Int J Mol Sci.* 2011;12:3117-3132.
19. Reaven GM. "Insulin resistance/compensatory hyperinsulinemia, essential hypertension and cardiovascular disease." *J Clin Endocrinol Metab.* 2003;88:2399-2403.
20. [Alberti KG, Eckel RH, Grundy SM et al. "Harmonizing the metabolic syndrome: a joint interim statement of the International Federation Task Force on Epidemiology and Prevention; National Heart, Lung and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity."](#) *Circulation.* 2009;120:1640-1645.
21. Santos MJ, Fonseca JE. "Metabolic syndrome, inflammation and atherosclerosis - the role of adipokines in health and in systemic inflammatory rheumatic diseases." *Acta Reumatologica Portuguesa.* 2009;34:590-598.
22. Afonso L, Hari P, Kondur A et al. "Usefulness of microalbuminuria in patients with the metabolic syndrome to predict subclinical atherosclerosis and cardiovascular disease outcomes." *Am J Cardiol.* 2010;106:976-983.
23. [Lee MG, Jeong MH, Ahn Y et al. "Impact of the metabolic syndrome on the clinical outcome of patients with acute ST-elevation myocardial infarction."](#) *J Korean Med Sci.* 2010;25:1456-1461.
24. Zhao XQ, Krasuski RA, Baer J et al. "Effects of combination lipid therapy on coronary stenosis progression and clinical cardiovascular events in coronary disease patients with metabolic syndrome: a combined analysis of the Familial Atherosclerosis Treatment Study (FATS), the HDL-Atherosclerosis Treatment Study (HATS), and the Armed Forces Regression Study (AFREGS)." *Am J Cardiol.* 2009;104:1457-1464.
25. [Pal L. "A look at metabolic syndrome through the lens of an ob/gyn."](#) *Contemp Ob Gyn.* 2009;54:50-53.
26. [Gunderson EP, Quesenberry CP Jr, Jacobs DR Jr et al. "Longitudinal study of prepregnancy cardiometabolic risk factors and subsequent risk of gestational diabetes mellitus: The CARDIA study."](#) *Am J Epidemiol.* 2010;172:1131-1143.
27. [Tenenbaum A, Fisman EZ. "The metabolic syndrome...is dead: These reports are an exaggeration."](#) *Cardiovasc Diabetol.* 2011;10:11.
28. [Towfighi A, Zheng L, Ovbiagele B. "Weight of the obesity epidemic: rising stroke rates among middle-aged women in the United States."](#) *Stroke.* 2010;41:1371-1375.
29. [Mottillo S, Filion KB, Genest J et al. "The metabolic syndrome and cardiovascular risk: a systematic review and meta-analysis."](#) *J Am Coll Cardiol.* 2010;56:1113-1132.

A practical guide to metabolic syndrome

30. [Grundy SM, Cleeman JL, Merz CN et al. "Implications of recent clinical trials for the National Cholesterol Education Program Adult Treatment Panel III guidelines." Circulation. 2004;110:227-239.](#)
31. [National Heart, Lung and Blood Institute. "Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults: The Evidence Report." National Institutes of Health. Bethesda, MD: National Institutes of Health; 1998. NIH Publication No. 98-4083.](#)
32. Vega GL, Barlow CE, Grundy SM. "Prevalence of the metabolic syndrome as influenced by the measure of obesity employed." *Am J Cardiol.* 2010;105:1306-1312.
33. Coutinho T, Goel K, Corrêa de Sá D et al. "Central obesity and survival in subjects with coronary artery disease: a systematic review of the literature and collaborative analysis with individual subject data." *J Am Coll Cardiol.* 2011;57:1877-1886.
34. [Rahman M, Berenson AB. "Accuracy of current body mass index classification for white, black and Hispanic reproductive-age women." Obstet Gynecol. 2010;115:982-988.](#)
35. [Chobanian AV, Bakris GL, Black HR et al. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure.](#)
36. [Obesity Society position statement. Weight bias and discrimination.](#)
37. [Bray GA. "Lifestyle and pharmacological approaches to weight loss: efficacy and safety." J Clin Endocrinol Metab. 2008;93\(11 Suppl 1\):S81-88.](#)
38. [Greenland P, Alpert JS, Beller GA et al. "2010 ACCF/AHA guideline for assessment of cardiovascular risk in asymptomatic adults: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines." Circulation. 2010;122:584-636.](#)
39. [Sumner AE, Finley KB, Genovese DJ et al. "Fasting triglyceride and the triglyceride-HDL cholesterol ratio are not markers of insulin resistance in African Americans." Arch Intern Med. 2005;165:1395-1400.](#)
40. American Diabetes Association. "Standards of medical care in diabetes - 2011." *Diabetes Care.* 2011;34 Suppl 1:S11-S61.
41. [Balkau B, Soulimane S, Lange C et al. "Are the same clinical risk factors relevant for incident diabetes defined by treatment, fasting plasma glucose, and HbA1c?" Diabetes Care. 2011;34:957-959.](#)
42. [Colberg SR, Sigal RJ, Fernhall B et al. "Exercise and type 2 diabetes: the American College of Sports Medicine and the American Diabetes Association: joint position statement." Diabetes Care. 2010;33:e147-e167.](#)
43. Strasser B, Siebert U, Schobersberger W. "Resistance training in the treatment of the metabolic syndrome: a systematic review and meta-analysis of the effect of resistance training on metabolic clustering in patients with abnormal glucose metabolism." *Sports Med.* 2010;40:397-415.
44. Anderssen SA, Carroll S, Urdal P, Holme I. "Combined diet and exercise intervention reverses the metabolic syndrome in middle-aged males: results from the Oslo Diet and Exercise Study." *Scand J Med Sci Sports.* 2007;17:687-695.
45. [Yassine HN, Marchetti CM, Krishnan RK et al. "Effects of exercise and calorie restriction on insulin resistance and cardiometabolic risk factors in older obese adults - a randomized clinical trial." J Gerontol A Biol Sci Med Sci. 2009;64:90-95.](#)
46. [Ilanne-Parikka P, Laaksonen D, Eriksson JG et al. "Leisure-time physical activity and the metabolic syndrome in the Finnish Diabetes Prevention Study." Diabetes Care. 2010;33:1610-1617.](#)

A practical guide to metabolic syndrome

47. [Bland J. "Metabolic syndrome: the complex relationship of diet to conditions of disturbed metabolism."](#) [*Functional Foods Health Dis.* 2011;2:1-12.](#)
48. Falentin F. "The metabolic syndrome, an overview; new scientific evidence suggests that a healthy lifestyle might be the best treatment alternative and prevention strategy." [*Nutr Perspect.* 2010;33:13-21.](#)
49. [Malik VS, Popkin BM, Bray GA et al. "Sugar-sweetened beverages and risk of metabolic syndrome and type 2 diabetes: a meta-analysis."](#) [*Diabetes Care.* 2010;33:2477-2483.](#)
50. Sampalis JS, Sampalis F, Christou N. Impact of bariatric surgery on cardiovascular and musculoskeletal morbidity. [*Surg Obes Relat Dis.* 2006;2:587-591.](#)
51. [Wilson PW, Grundy SM. "The metabolic syndrome: a practical guide to origin and treatment: part II."](#) [*Circulation.* 2003;108:1537-1540.](#)
52. [Devaraj S, Siegel D, Jialal I. "Statin therapy in metabolic syndrome and hypertension post-JUPITER: what is the value of CRP?"](#) [*Curr Atheroscler Rep.* 2011;13:31-42.](#)
53. Robinson JG. "LDL reduction: how low should we go and is it safe?" [*Curr Cardiol Rep.* 2008;10:481-487.](#)
54. [National Heart, Lung, and Blood Institute. Press release: NIH stops clinical trial on combination cholesterol treatment.](#)
55. [Ratner R, Goldberg R, Haffner S et al. "Impact of intensive lifestyle and metformin therapy on cardiovascular disease risk factors in the diabetes prevention program."](#) [*Diabetes Care.* 2005;28:888-894.](#)
56. [Black HR, Davis B, Barzilay J et al. "Metabolic and clinical outcomes in nondiabetic individuals with the metabolic syndrome assigned to chlorthalidone, amlodipine or lisinopril as initial treatment for hypertension: a report from the Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial \(ALLHAT\)."](#) [*Diabetes Care.* 2008;31:353-360.](#)
57. [U.S. Preventive Services Task Force. "Aspirin for the Prevention of Cardiovascular Disease: Recommendation Statement."](#)
58. Alberti KG, Zimmet PZ. "Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: diagnosis and classification of diabetes mellitus provisional report of a WHO consultation." [*Diabet Med.* 1998;15:539-553.](#)
59. Balkau B, Charles MA. "Comment on the provisional report from the WHO consultation. European Group for the Study of Insulin Resistance (EGIR)." [*Diabet Med.* 1999;16:442-443.](#)
60. Einhorn D, Reaven GM, Cobin RH et al. "American College of Endocrinology position statement on the insulin resistance syndrome." [*Endocr Pract.* 2003;9:237-252.](#)
61. [International Diabetes Federation. The IDF consensus worldwide definition of the metabolic syndrome.](#)

All electronic documents accessed February 8, 2012.

HOW TO TAKE THE POST-TEST: To obtain CME/CE credit, please [click here](#) after reading the article to take the post-test on [myCME.com](#).

Classification

Language: ENGLISH

Publication-Type: Magazine

Journal Code: The Clinical Advisor

Subject: CHOLESTEROL (99%); DIABETES (92%); TYPE 2 DIABETES (90%); DISEASES & DISORDERS (90%); OBESITY (90%); CARDIOVASCULAR DISEASE (89%); HYPERTENSION (89%); ASSOCIATIONS & ORGANIZATIONS (87%); PUBLIC HEALTH (78%); ASTRONOMY & SPACE (75%); HEART DISEASE (74%); HEALTH CARE PROFESSIONALS (72%)

Industry: HEALTH CARE (74%); HEALTH CARE PROFESSIONALS (72%)

Geographic: UNITED STATES (94%)

Load-Date: February 27, 2012

End of Document