# Cholesterol:

The good, the bad, and the ugly

High cholesterol, especially elevated low-density lipoprotein levels, is a major cause of atherosclerosis. Nurses play a leading role in helping patients manage their cholesterol levels. We show you how.

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The author has disclosed that she has no significant relationships with or financial interest in any commercial companies that pertain to this educational activity.



Coronary artery disease (CAD) is a narrowing of the arteries supplying blood and oxygen to the heart. Almost every year since 1900, cardiovascular disease has caused more

deaths than any other disease in the United States, according to the CDC. In fact, each day, a person dies every 38 seconds from cardiovascular disease; more than cancer, chronic respiratory disease, and accidents combined.

A main cause of CAD is high cholesterol. When cholesterol is high, it blocks the blood supply to the heart. As it builds up, the coronary vessels constrict and atherosclerotic plaques or lesions are formed (see *Understanding atherosclerosis*). These cholesterol plaques then initiate clot formation and inflammation, which further block the vessels. This means that any increase in cholesterol causes a subsequent increase in cardiovascular disease risk.

#### What's cholesterol?

Cholesterol is a fatlike substance produced by the liver. Some of its functions include:

- building and maintaining cell membranes
- assisting in the manufacture of androgens, estrogens, and aldosterone
- aiding in the production of bile
- assisting the synthesis of vitamin D
- helping metabolize fat-soluble vitamins
- insulating nerve fibers.

Cholesterol can't dissolve in blood or water, so it's packaged in protein-covered molecules called lipoproteins. This allows it to easily mix and flow with blood. Some of these molecules are large and light and others are small and heavy. The most important lipoproteins are low-density lipoprotein (LDL), high-density lipoprotein (HDL), very low-density lipoprotein (VLDL), and triglycerides. The total cholesterol level is the sum of all these lipoproteins.

*LDL* carries cholesterol from the liver to the rest of the body. Cells capture LDL and



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extract the cholesterol. When there's too much LDL in the bloodstream, it's deposited on the artery wall, causing the formation of a cholesterol plaque. Over time, the plaque increases, hampering the blood flow in the arteries. This is known as atherosclerosis. LDL is about 60% to 70% of the total serum cholesterol. The optimal level for LDL is under 100 mg/dL. Research has shown that reducing LDL significantly decreases the risk of cardiovascular disease.

*HDL* extracts cholesterol from the artery walls and bloodstream, bringing it back to the liver for excretion. This decreases the LDL level. HDL is inversely associated with the risk of cardiovascular disease, meaning a higher HDL level decreases risk. HDL is about 20% to 30% of the total serum cholesterol. The optimal level for HDL is greater than 40 mg/dL.

Triglycerides, composed of fatty acids, are manufactured in the liver or come from the diet. The liver removes triglycerides from the blood by packing them into VLDL. These particles are harder to move through the

blood vessels and can contribute to the formation of atherosclerosis. High triglyceride levels are also a risk factor for cardiovascular disease. The optimal level for triglycerides is under  $150 \, \text{mg/dL}$ .

## Get to know dyslipidemia

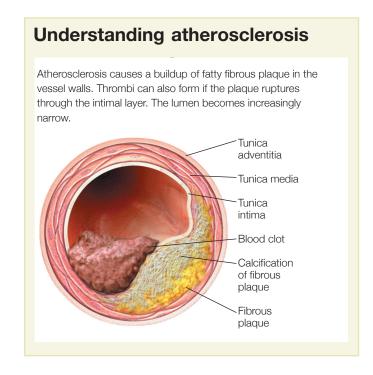
Both heredity and lifestyle factors can influence a person's cholesterol level. Total cholesterol, LDL, HDL, and triglycerides are all part of the equation. Alterations in any of these lipoproteins increase the risk of significant cardiovascular disease. When a person has an abnormal lipoprotein metabolism, it's known as dyslipidemia. It can be classified as either primary or secondary.

*Primary dyslipidemia* includes disorders that arise from genetic alterations in lipoprotein metabolism. Some of these include monogenic familial hypercholesterolemia, familial defective apolipoprotein B-100, and polygenic hypercholesterolemia. In all of the genetic dyslipidemias, there's a faulty mechanism in either the manufacture or balance of cholesterol. Family testing can be done to identify affected individuals.

Secondary dyslipidemia refers to acquired defects in lipoprotein metabolism. Common causes of secondary dyslipidemia are hypothyroidism, liver disease, renal disease, obesity, diabetes, excessive alcohol intake, and complications from medications such as some antihypertensives, antipsychotics, and estrogens. The liver is crucial in the manufacture of cholesterol and its secretion into the bloodstream. It can also remove cholesterol from the blood through active receptors on the surface of its cells. If a person's liver is damaged or overtaxed by a chronic illness or medications, there are less cell receptors for removal of the cholesterol. This can cause high serum cholesterol levels.

# The lipid profile

A lipid profile measures HDL, LDL, triglycerides, and total cholesterol—the sum of all the blood cholesterol content (see *Evaluating lipid test results*). HDL is sometimes called



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"good" cholesterol because it helps remove LDL, keeping arteries open and blood moving freely. LDL is often called "bad" cholesterol because elevated levels result in the buildup of fatty deposits, or plaques, in the artery, reducing blood flow. The rupture of these plaques can cause a myocardial infarction or stroke. Triglycerides are a type of fat in the blood. Any calories not utilized by the body are converted to triglycerides for storage in fat cells. A high triglyceride level means a person is regularly consuming more calories than are burned.

The cholesterol/HDL ratio is used to determine a person's risk of heart disease because high HDL levels can protect against the detrimental effects of high LDL levels. This means that if both the LDL and HDL levels are high, the ratio may come within the desired range. The goal is to keep the cholesterol/HDL ratio below 5.1, with an optimum ratio of 3.5. This ratio is obtained by dividing the total cholesterol number by the HDL number.

# Lipid management guidelines

Starting in 1988, the National Heart, Lung and Blood Institute (NHLBI) formed an expert Adult Treatment Panel (ATP) to survey and report on the detection, evalua-

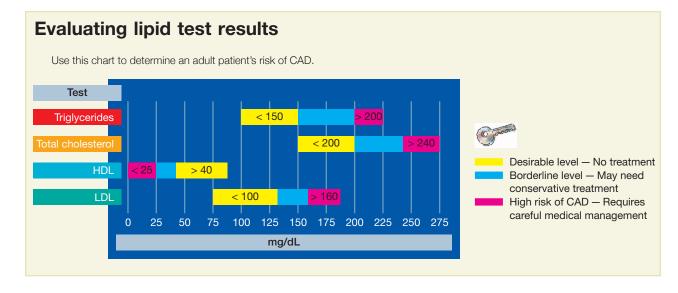
tion, and treatment of high cholesterol. The ATP focused on clinical interventions to prevent heart disease. Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (ATP I), the first such panel, directed a strategy for prevention of atherosclerosis in people with an elevated LDL level without any atherosclerosis. ATP II in 1998 reinforced primary prevention of cardiovascular disease and added treatment guidelines for people already diagnosed with atherosclerosis who have a high LDL level. ATP II also recommended a new goal of a lower LDL level (under 100 mg/dL).

ATP III, the most recent panel in 2004, addresses the prevention of cardiovascular disease in people with other risk factors such as diabetes or cigarette smoking. In addition, the panel recommends an increase in the HDL level to 40 mg/dL as a more accurate measure.

The ATP III guidelines recommend the identification of major risks for cardiovascular disease as a reason to start treatment. Some of these major risk factors include cigarette smoking, hypertension, age, family history of early heart disease, and diabetes. The recommended LDL level differs based on the number of risk factors the person has.

What's this I see? Test results aplenty!





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In other words, individuals with multiple risk factors are advised to initiate treatment for hyperlipidemia at a lower level.

ATP III recommends the following steps in a lipid management program. First, a fasting lipid profile is drawn. The patient's risk factors are then evaluated, including atherosclerotic disease, diabetes, smoking, age, and weight. If a person has two or more risk factors, in addition to a high LDL level, his or her 10-year risk of a major coronary event is evaluated. At this point, the patient's risk category and the associated recommended LDL level at which treatment should be started can be identified.

The NHLBI plans to release ATP IV in the spring of 2012.

# Goals of lipid management

The first goal of lipid management is to keep the total cholesterol level under 200 mg/dL. Lifestyle modifications through dietary changes, weight loss, and exercise are started. If these changes don't decrease total cholesterol to under 200 mg/dL, then medications are started. Statins are the first line of pharmacologic treatment. However, if a patient also has a low HDL level and high triglycerides, fibric acid derivatives or niacin can be used. Combination therapy may be useful in targeting high LDL and triglyceride levels and low HDL.

# The skinny on fats

Fats provide energy and are essential to life. In our diet, there are unsaturated fats, saturated fats, and trans fats.

**Unsaturated fats,** found in plant-based products such as nuts, seeds, and vegetable oil, can decrease serum cholesterol levels and inflammation.

On the other hand, *saturated fats* can increase the risk of cardiovascular disease because they increase LDL levels. The body makes all the saturated fat it needs, so there's no need to eat any saturated fat in our diet. Major sources of saturated fats include meat, seafood, skinned chicken, whole milk dairy products, and coconut or palm oil.

*Trans fats* are made by hydrogenation. This is a process of heating liquid vegetable oils in the presence of hydrogen gas to make vegetable oil less likely to spoil and solidify it for ease of use. Trans fats are worse for cholesterol levels than other fats because they increase the LDL level and decease the HDL level. Trans fats also increase inflammation.

Keeping the triglycerides under 150 mg/dL is the second goal of lipid management. Again, lifestyle modifications are the first step of treatment. When the triglyceride level is above 150 mg/dL, the patient requires conservative treatment; if triglyceride levels are above 200 mg/dL, the patient may be started on a fibric acid or niacin to target this lipoprotein alteration. The omega-3 fatty acids are also beneficial in lowering triglycerides levels by stopping the synthesis of VLDL in the liver.

Let's take a closer look at therapeutic lifestyle changes and pharmacologic management.

# Therapeutic lifestyle changes

Therapeutic lifestyle changes can improve cholesterol levels. Changing the diet by decreasing fats is the first step (see *The skinny on fats*). It's important to keep the intake of saturated fats low and eliminate trans fats all together. In fact, eliminating trans fats from the diet can decrease the risk of cardiovascular disease up to 19%.

Eating lean cuts of meats and trimming visible fat and skin from meat before cooking can decrease saturated fat intake. When consuming dairy products, choosing low-fat cheeses, yogurts, and milk can help decrease fat intake. Avoiding margarines, salad dressings, and mayonnaise—all high in saturated and trans fats—is important. Canola oil or olive oil can be substituted for cooking and dressings. Educate your patients on reducing saturated fats by reading food labels and avoiding fast food eateries.

Fiber can also help lower cholesterol levels. Soluble fiber binds to the cholesterol we eat and helps the body eliminate it. There's also some evidence that soluble fiber can decrease the manufacture of cholesterol in the liver and make LDL particles less dense or easier to flow through the blood vessels.

The next step in therapeutic lifestyle changes is increasing physical activity. Help your patients accomplish this goal by providing instruction to start with aerobic exercise,

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such as walking every other day for at least 30 minutes as an initial step. Then, as endurance increases, the exercise routine can be changed to engaging in aerobic exercise for 40 minutes, 5 days each week. Because long-term compliance with an exercise program may be a problem, you can help your patients pick a plan that will be easy to follow, with favorite exercises and realistic workout times. You can also assist your patients to identify ways of incorporating more exercise in daily routines, such as using the stairs rather than an elevator.

# Pharmacologic management

The major classifications of medications to treat dyslipidemia are statins, bile acid se-

questrants, cholesterol absorption inhibitors, nicotinic acid derivatives, and fibric acid derivatives (see *Common medications used to treat dyslipidemia*). Each has its benefits and adverse reactions.

Statins are most effective in lowering LDL cholesterol. They block the enzyme in the liver that controls the production of LDL. By reducing LDL levels, statins decrease the development of atherosclerotic plaque formations, decrease inflammation, and curtail the clotting response. Statins are only mildly effective in lowering triglycerides or increasing HDL levels. Some of the medications in this class are pravastatin, lovastatin, atorvastatin, simvastatin, fluvastatin, and rosuvastatin.

Drug	Туре	Adverse reactions	Nursing pearls
Lovastatin	Statin	<ul><li>GI symptoms</li><li>Muscle and liver damage</li></ul>	<ul> <li>Baseline/periodic liver enzymes</li> <li>CPK level to evaluate muscle damage</li> <li>Take at bedtime</li> <li>Take with food</li> </ul>
Pravastatin	Statin	GI symptoms     Muscle and liver damage	<ul><li>Baseline/periodic liver enzymes</li><li>CPK level to evaluate muscle damage</li><li>Take at bedtime</li></ul>
Simvastatin	Statin	GI symptoms     Muscle and liver damage	<ul> <li>Baseline/periodic liver enzymes</li> <li>CPK level to evaluate muscle damage</li> <li>Take at bedtime</li> <li>Decreases triglycerides, increases HDL</li> </ul>
Fluvastatin	Statin	GI symptoms     Muscle and liver damage	<ul><li>Baseline/periodic liver enzymes</li><li>CPK level to evaluate muscle damage</li><li>Take at bedtime</li></ul>
Atorvastatin	Statin	GI symptoms     Muscle and liver damage	<ul> <li>Baseline/periodic liver enzymes</li> <li>CPK level to evaluate muscle damage</li> <li>Take at bedtime</li> <li>Effect is dose related</li> <li>Long half-life</li> </ul>
Rosuvastatin	Statin	GI symptoms     Muscle and liver damage	<ul> <li>Baseline/periodic liver enzymes</li> <li>CPK level to evaluate muscle damage</li> <li>Take at bedtime</li> <li>Decreases triglycerides, increases HDL</li> </ul>
Cholestyramine	Bile acid sequestrant (powder)	GI symptoms	<ul><li> Take with meals</li><li> Don't take with other medications</li><li> Avoid taking with carbonated beverage</li></ul>

In general, statins are well tolerated, with minor adverse reactions such as gastrointestinal (GI) upset and headache. A more bothersome adverse reaction is muscle pain and weakness, which can be a sign of rhabdomyolysis (muscle breakdown). It's important to obtain a baseline phosphokinase (CPK) level and then repeat the test for any complaint of muscle pain and weakness. An elevated CPK would indicate that rhabdomyolysis is occurring. Also, if statins are combined with erythromycin, niacin, or gemfibrozil, the risk of myopathy may increase. Advise your patients to talk to their healthcare provider if they experience severe muscle pain or leg cramps.

Liver toxicity is another serious adverse reaction of statins. At the start of treatment, a

cheat

#### **Diet changes**

**Therapeutic** 

• Trim visible fat and skin from meat and poultry before cooking.

lifestyle changes

- Eat fish high in omega-3 fatty acids, such as salmon and trout.
- Select low-fat cheeses, milk, and yogurts.
- · Cook with canola or olive oils.
- Decrease use of mayonnaise and salad dressings.
- Increase intake of fruits, vegetables, and legumes.
- Learn to read food labels and pick those with fresh ingredients.
- Switch from white rice or bread to whole grain products.
- In restaurants, watch the serving size; eat half and take home the rest.
- In fast food eateries, try lower-fat choices by avoiding sauces and fries.

#### Physical activity changes

- Pick a fitness plan with realistic goals, favorite exercises, and realistic workout times.
- Take the stairs instead of the elevator.
- Park the car far from the front door of stores.
- · Walk during a lunch break.
- Increase aerobic exercise to 40 minutes, 5 days a week

baseline liver panel should be drawn, and then every 3 months to monitor liver function. If the patient has liver damage or drinks large amounts of alcohol, statins may not be the best drug treatment. In addition, statins should be used with caution in patients with compromised renal function. Proteinuria can occur, especially with rosuvastatin. If using high doses of these medications, renal function tests may be necessary.

There are minor differences among the statins. Atorvastatin is dose-related; increasing the dosage will reduce cholesterol more. Rosuvastatin and simvastatin can also decrease triglyceride levels and increase HDL levels, an advantage if your patient has changes on all three levels. Medications used to treat hypertension or arrhythmia can interfere with the metabolism of statins, resulting in increased plasma levels of the medications. It's important to educate your patients about possible drug interactions.

Most statins should be taken at bedtime because that's when a larger amount of cholesterol is produced. Exceptions are lovastatin, which works better when taken with food, and atorvastatin, which has a long half-life (peak levels of the medication will still occur during sleep).

*Bile acid sequestrants* bind the bile acids in the intestine and prevent reabsorption in the ileum. This disruption in the circulation of bile acids causes an increase in the conversion of cholesterol into bile acids, resulting in lower LDL levels. These medications are also effective at lowering triglyceride levels. Some of the medications in this class include cholestyramine, colestipol, and colesevelam.

Bile acids aren't absorbed, so adverse reactions are minimal. The most common adverse reactions are GI upset, including diarrhea, gas, nausea, and weight loss. Bile acid sequestrants should be taken with meals because they block cholesterol in the GI tract.

Bile acid sequestrants often come as powders or granules to be mixed with water,

juice, or applesauce for better absorption. These medications have the potential to bind with other medications, such as thyroid hormones, fat-soluble vitamins, or antibiotics, decreasing their bioavailability. Advise your patients to take these medications either 1 hour before or 4 hours after taking a bile acid sequestrant.

Cholesterol absorption inhibitors, such as ezetimibe, cause mild lowering of LDL but aren't effective at raising HDL or lowering triglyceride levels. They decrease absorption of cholesterol in the small intestine, leading to a decrease of delivery to the liver. Adverse reactions include headache, diarrhea, and abdominal pain. Like the statins, any complaints of muscle pain or weakness should be taken seriously because rhabdomyolysis may occur.

Nicotinic acid derivatives inhibit the mobilization of free fatty acids from the peripheral tissues, reducing the hepatic synthesis of triglycerides. This decreases both the triglyceride and LDL levels and increases HDL levels. Patients taking niacin often experience flushing and itching for a short period after taking it. The flushing is caused by increased prostaglandins, and can be treated with 325 mg of aspirin half an hour before taking the niacin. Niacin can increase liver enzymes, uric acid, and glucose. A baseline fasting blood glucose level and liver panel should be obtained before starting the medications and every 6 months while on the medication. Also, niacin may not be the first choice for patients with diabetes or gout.

Fibric acid derivatives, such as fenofibrate and gemfibrozil, stop the production of VLDL and speed up the removal of triglycerides. Fibric acid derivatives are effective medications to help lower triglyceride levels. Gemfibrozil is also modestly effective in increasing HDL levels. The most common adverse reactions include GI upset, such as flatulence, dyspepsia, nausea, vomiting, and diarrhea. As with other cholesterol-lowering medications, liver toxicity can occur. Liver function tests should be obtained before the

# did you know?

The American Heart Association has updated its heart disease prevention guidelines for women. Effectiveness-Based Guidelines for the Prevention of Cardiovascular Disease in Women—2011 Update focuses on understanding risks and applying measures likely to work in the "real world." Visit http://www.circ.ahajournals.org/cgi/reprint/ CIR.0b013e31820faaf8v1 to download the new guidelines.

medication is started and every 3 months afterward.

# Your role in successful outcomes

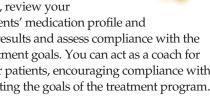
Nurses play a crucial role in lipid management programs by taking responsibility for getting patients involved in the treatment plan. Many studies demonstrate improvements in clinical outcomes and reduction of risk factors when a nurse is involved in care. Your role includes education, assessment and monitoring, consultation, and referral. Teach your patients about cholesterol and how abnormal levels can cause cardiovascular dis-

ease. Also teach about the lifestyle modifications that assist in improving the lipid profile.

Lipid-lowering agents are an integral part of management, and education about these drugs is a vital component of an effective lipid management program. At each visit, review your

patients' medication profile and lab results and assess compliance with the treatment goals. You can act as a coach for your patients, encouraging compliance with meeting the goals of the treatment program. You can help your patients set realistic

Do you think you're ready to pass the test now?



lifestyle modification goals and provide positive reinforcement when the steps to those goals are met.

In all these ways, nurses are important in securing successful outcomes for patients with dyslipidemia.

#### Learn more about it

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#### Cholesterol: The good, the bad, and the ugly

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