WINTER

NEWSLETTER

ISCVPR

February 2026

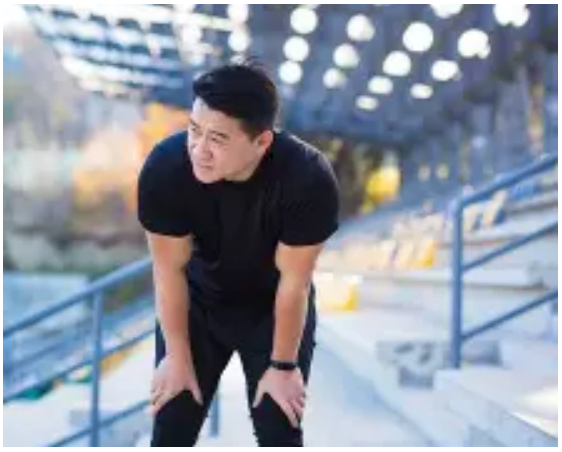
Welcome to the Winter Edition of ISCVPR Newsletter

In this issue, we reflect on the history of the organization and spotlight our meaningful contributions to advancements in cardiac and pulmonary patient care across the state. We feature stories from a founding member, helpful guidance for patient care, and a glimpse into ongoing efforts. The newsletter wraps up with ways to stay connected and engaged with the community!

ISCVPR's History

A founding member of ISCVPR, Rose Mary Wasielewski, shares her account on the establishment and growth of ISCVPR. She highlights how ISCVPR monthly meetings helped build a strong foundation for cardiac rehab programs across Indiana. If you are interested in joining the ISCVPR board of directors, you can find a service application on page 4!

Jane Miller, Cindy McCoy, Peg Martin, Susie Carter, & Rose Mary Wasielewski with Gov. Evan Bayh. 1987.



Breathlessness in CVD Patients

A recent review of studies has highlighted the effect impairments in lung function have on patients with cardiovascular disease. By expanding the understanding of pulmonary dysfunction beyond traditional lung diseases, we can better understand how breathlessness can impact performance in cardiac patients. The article discusses the need of further research into heart-lung integration during exercise in order to improve clinical management in those patients.

Evaluating Pulmonary Patients

Debbie Koehl, RT shares tips and tricks to assessing PR patients that were shared at the recent AASCVPR pre-conference. Read how you can better tailor your PR programs to each patient's disease, symptoms, and functional needs in order to improve both performance and long-term disease management.

2026

ISCVPR ANNUAL CONFERENCE

APRIL 23RD, 2026

755 E MAIN ST. GREENWOOD, INDIANA 46143

Indiana Society of Cardiovascular & Pulmonary Rehabilitatio

https://www.iscvpr.org/

Our Awesome Speakers:

SUSAN BAUMAN, BSN, CCRP
Reimbursement

LISA DUNNIVANT, BSN, RN, CCRN
LVAD education and considerations

CEMAL OZEMEK, PHD
New Guideline Review / ACSM guideline update

DEBBIE KOEHL, MS, RRT-NPS, AE-C, FAARC
Quality Programming and Reimbursement

MATT THOMAS, MBA, MS, ACSM-CEP
Frailty Talk

CONNIE WILSON, MSN, RN, CCRP, CCEP, PR CERT
Quality Programming

SHIRLEY ELLIS, PMP, CEO
Motivational Speaker & Leadership Coach

Memories of ISCVPR

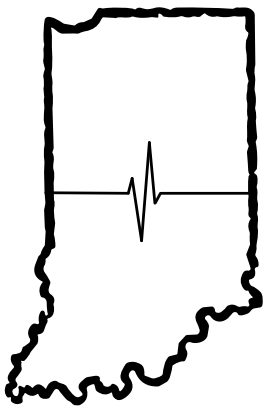
Being a part of the development and growth of ISCVPR has been a great honor to me. The camaraderie and support of my colleagues on the board throughout the years has been a gift that I cherish, even now in my retirement.

ISCVPR began as an outreach from an educational conference in Indianapolis. At the conference, the idea of forming a state-wide organization was presented. There was a request for volunteers to establish the organization, and I felt compelled to raise my hand. As a result, I became part of the dynamic group that established ISCVPR and I remained on the board for most of my career years in cardiac rehab.

Soon after the conference, meetings were set up by the core group of volunteers, and we began to meet on a monthly basis. We became the first board of ISCVPR. Even though it was an almost 3 hour drive each way for me, I always looked forward to meeting with my colleagues. As we worked on establishing an organization, we shared ideas about our programs and our goals. Being a part of this group was nourishing to me as a professional in the field of cardiac rehab.

Through it, I grew both personally and professionally.
Now in my retirement, I am proud to say that I had a part in establishing an organization that has grown and thrived for many years. I know that the efforts of ISCVPR have played a great part in the continuing professionalism and progressiveness of the cardiac and pulmonary rehab programs in Indiana and in the professionals that work in those programs.

Carry on, my dear colleagues!
Rose Mary Wasielewski, RN, MSN, FAACVP
ISCVPR Founding Board member



STATE OF INDIANA

EXECUTIVE DEPARTMENT
INDIANAPOLIS

Executive Order

PROCLAMATION

TO ALL TO WHOM THESE PRESENTS MAY COME, GREETING:

WHEREAS, this year, diseases of the heart and blood vessels will kill almost 1,000,000 Americans which nearly equals the total number of deaths from all other diseases; and

WHEREAS, of the patients hospitalized with coronary heart disease, nearly 6,000 are seen yearly in cardiac rehabilitation settings in Indiana; and

WHEREAS, the objective of the Indiana Society of Cardiovascular Rehabilitation is to restore these individuals to their previous level of functioning and to facilitate lifestyle changes; and

WHEREAS, the Indiana Society of Cardiovascular Rehabilitation's goal is to provide guidelines throughout the state of Indiana to insure quality care and to provide research programs and information as well as health care education in the community through professional and public education and community service programs;

NOW, THEREFORE, I, EVAN BAYH, Governor of the State of Indiana, do hereby proclaim February 10 - 15, 1991 as

CARDIAC REHAB WEEK

in the state of Indiana.

IN TESTIMONY WHEREOF, I have hereunto set my hand and caused to be affixed the Great Seal of the State of Indiana at the Capitol in Indianapolis on this 16th day of January, 1991.

BY THE GOVERNOR: Evan Bayh
Governor of Indiana

Joseth H. Hewitt

STATE

Indiana Society of Cardiovascular & Pulmonary Rehabilitation

Indiana Society of Cardiopulmonary Rehabilitation
Board of Directors – Service Application

The Indiana Society of Cardiopulmonary Rehabilitation is pleased to announce upcoming elections for members of the Board of Directors. Qualification for the Board of Directors consists of:

- Current ISCVPR member for at least one consecutive year.
- Must be committed to the ISCVPR mission and vision.
- Must be willing to make a time commitment to work on behalf of ISCVPR.
- Attend Board of Directors meetings quarterly

All applications will be reviewed by the ISCVPR nominating committee. After approval from the nominating committee, elections by ISCVPR membership will take place 30 days before the annual meeting

Name:_____Credentials_____

Email:_____

Address:_____City:_____State:_____Zip:_____

Phone:_____

Current Job Responsibilities:

_____

_____

Years of ISCVPR Membership_____ AACVPR Member Yes No

Involvement in ISCVPR:

_____

_____

Why do you want to become a member of ISCVPR Board of Directors?

_____

_____

What skills, abilities, or attributes do you feel you can contribute to ISCVPR?

_____

_____

_____

What do you view are the two greatest challenges for ISCVPR and Cardiopulmonary Rehabilitation?

_____

_____

What do you recommend as a solution to one of these issues?

_____

_____

What is your vision of ISCVPR?

_____

_____

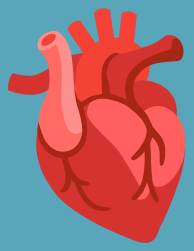
I hereby attest to meeting the above stated qualifications for ISCVPR Board of Directors. I verify that all information contained within this application is accurate.

Signature: _____(esignature accepted)

Please return to: Aaron Wright at (awrigh11@iuhealth.org)

Cardiac Rehabilitation:

A review on how pulmonary function can affect patients with CVD.



Inspiratory Capacity and Dynamic Hyperinflation During Exercise in Patients with Cardiovascular Disease: A SYSTEMATIC REVIEW.
https://journals.lww.com/jcrjournal/abstract/2026/01000/inspiratory_capacity_and_dynamic_hyperinflation.2.aspx

This article explores how expiratory flow limitation (EFL) and dynamic hyperinflation (DH) may significantly impact exercise capacity in patients with heart disease. Although commonly linked to lung diseases, recent evidence highlights their role in cardiovascular disease (CVD), contributing to exercise intolerance. This systematic review examines studies from the past decade on pulmonary function during exercise, measured using inspiratory capacity (IC) maneuvers, in patients with CVD, emphasizing prevalence and clinical significance. The findings of this review underscore the need for further research into heart-lung integration during exercise to develop more effective, personalized treatment strategies for patients with CVD experiencing ventilatory constraints.

Congratulations to IU - Health Ball Memorial Cardiac Rehabilitation for celebrating their 50th Anniversary as a program!

Pulmonary Rehabilitation:
Evaluating Your Pulmonary Patients

Debbie Koehl, MS, RRT-NPS, AE-C, FAARC

Ever wonder if you are evaluating your pulmonary rehab to your fullest potential? Are you discussing your patients' goals in PR? Do they understand what goals they could accomplish in PR?
Well, I know I often think of that when we evaluate patients in our program. This past AACVPR meeting there was a great PR pre-conference entitled “Hands-on Bootcamp: Master your Pulmonary Rehabilitation Assessment/Intervention Skills”. Let me tell you, I have been doing PR for over 20 years, and this program really made me do some self-reflection of our program and what we could do to enhance it. Now, this was a 4-hour course. So, I am only really going to do a quick summary of things you should think about and discuss with your PR team.
Let’s look at the areas discussed during that conference.

#1 Dyspnea Assessment and Breathing Retraining

- Purpose of assessing
  - Establish the patient’s baseline dyspnea
  - Create a personalized treatment program
  - Measure clinical outcomes
- How to assess
  - Use open ended questions, not just yes and no
    - For examples: Can you describe your shortness of breath to me?
  - Using a validated tool
    - UCSD Shortness of Breath Questionnaire
- Are you listening to breath sounds?
  - Do you know what a patient sounds like at baseline to compare to when they are ill?
- What kind of breathing retraining are you doing?
  - Pursed lip breathing, diaphragmatic breathing, paced breathing, box breathing, mindful breathing, recovery positions

#2 Inhaler Techniques and Other Classes of Respiratory Medications

- Do you assess your patient inhaler techniques?
  - Do you use an In-Check Dial to see what device might work best for your patient?
  - Do you know all at the different inhaler devices that they patient may be using?
  - Can you teach all the different devices?

Pulmonary Rehabilitation:
Evaluating Your Pulmonary Patients

#3 Supplemental Oxygen

- Do you know what type of oxygen system the patient has at home?
- Is it working for the patient?
- Who is providing that service for your patient?
- After you complete their oxygen assessment, will their prescription change and if so, will their device need to change as well?

#4 Exercise Assessment

- How are you evaluating your patient for exercise?
  - Six-minute walk test? Are you converting the test to a treadmill walking speed for your exercise prescription?
  - Are you doing a sit to stand test to look at muscle strength and weakness?
  - Are you evaluating balance?
  - Are you looking at frailty?
  - Are you looking at flexibility?

#5 Pulmonary Devices for Patient Use

- Does your asthma patient have or use a peak flow meter?
- Does the asthma patient have an asthma action plan?
- Airway clearance techniques and devices for you patient with secretion issues
  - Bronchodilator therapy with the use of hypertonic saline?
  - What device do they use to clear secretions?
    - PEP therapy such as an Aerobika or Acapella
    - Therapy vest
  - Do you teach controlled or huff coughing techniques?
- Do you use Inspiratory Muscle Training devices?
  - New research shows these work with a select group of patients.
  - All devices are not created equal!!!

This is truly a summary of the details provided by the speakers. This was a hands-on program where we all got to play and take home some of the devices. The goal is to make sure we are looking at our pulmonary patients based on their symptoms, diseases and what we can do in pulmonary rehab to not only improve their strength and endurance but also their disease management to make them as healthy as possible.

Cardiopulmonary Rehabilitation Competency Series

With Joni B.

Curated by:
S. Joni Bertsch MSN, RN, CCRP, Certificate in Pulmonary Rehabilitation AARC/AACVPR
IU Health Ball Memorial Cardiopulmonary Rehabilitation

This series of educational tools and articles has been thoughtfully put together to support continued growth and excellence in cardiopulmonary rehabilitation. These materials can be used to strengthen patient education efforts, enhance clinical knowledge, and promote best practices across the state.

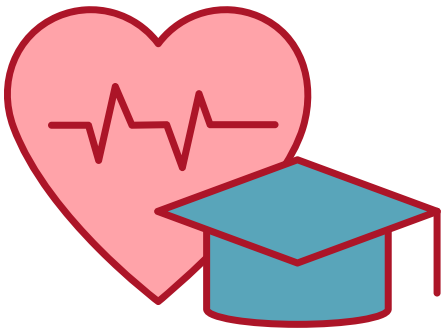
Please feel free to explore and implement these materials to further empower your staff and elevate the care you provide.

Summer 2025

Tobacco Cessation

Fall 2025

Patient Assessment



Winter 2026

Blood Lipid Management

Spring 2026

Non-COPD Respiratory Diseases

CHAPTER 5

BLOOD LIPID MANAGEMENT

TODD M. BROWN, MD, MSPH, FACC, FAACVPR

INTRODUCTION

High cholesterol is recognized as a major risk factor for the development of coronary heart disease (CHD) and is associated with an increased number of CHD-related events in individuals with CHD. A major focus of risk reduction in individuals with CHD is the lowering of blood cholesterol. As such, health professionals working in cardiac and pulmonary rehabilitation need to be knowledgeable about the adverse cardiovascular effects of high cholesterol as well as current lifestyle and pharmacologic approaches to the treatment of high cholesterol. **In this chapter, we will review the following:**

1. The basics of lipoprotein metabolism and the components of a standard lipid profile,
2. The data on hypercholesterolemia as a cardiovascular risk factor and the role that hypercholesterolemia plays in the atherosclerotic disease process,
3. Lifestyle recommendations for the treatment of high cholesterol, and
4. Pharmacologic approaches to the treatment of high cholesterol, with particular emphasis on guidelines for the treatment of elevated blood cholesterol published by the American Heart Association (AHA) and American College of Cardiology (ACC).

OVERVIEW OF LIPOPROTEINS

A detailed description of lipoprotein metabolism and function was provided by the National Cholesterol Education Program (NCEP) Adult Treatment Panel III (ATP III) and will be reviewed briefly here.1 Lipids, including triglycerides and cholesterol, are relatively insoluble in water and, therefore, carried in the bloodstream on lipoproteins. The term lipoprotein comes from the concept that each of these particles contains both a lipid component and a protein component. The proteins found on these lipoprotein particles are termed apolipoproteins (apo). The density of a lipoprotein particle is related to its lipid content. Those with less lipid content and therefore more protein are more dense. Similarly, those that contain higher concentrations of lipid are less dense. As such, lipoprotein particles can be classified based on density (Table 1). Major bloodstream lipoproteins include chylomicrons, very low density lipoproteins (VLDL), low density lipoproteins (LDL), and high density lipoproteins (HDL).1

TABLE 1. MAJOR BLOODSTREAM LIPOPROTEINS1

PARTICLE	DENSITY	LIPID CONTENT	APOLIPO-PROTEINS
Chylomicron	Least	TG	B-48, C, E
VLDL		TG, cholesterol (10-15%*)	B-100, C, E
LDL		cholesterol (60-70%*)	B-100
HDL	Most	cholesterol (20-30%*)	A

*percent of total cholesterol, HDL=high density lipoprotein, LDL= low density lipoprotein, TG=triglyceride, VLDL=very low density lipoprotein

Chylomicrons are the least dense of the lipoproteins and are triglyceride rich particles formed in the intestines. They transport dietary fat and can be found in the bloodstream at highest concentration soon after a meal. They contain apo B-48, apo C (C-I, C-II, and C-III), and apo E. Similar to chylomicrons, VLDL particles are also triglyceride rich, but they do contain some cholesterol. About 10-15% of the total serum cholesterol is found in VLDL particles. Also, similar to chylomicrons, VLDL particles contain apo C and apo E, but contain apo B-100 instead of apo B-48. LDL, the most atherogenic of all the lipoproteins, carries the majority (60-70%) of the cholesterol in the blood and contains only apo B-100. HDL particles carry 20-30% of the cholesterol in the blood and have apo A-1 and A-II.1

STANDARD BLOOD LIPID PROFILE

A standard fasting lipid profile and associated normal ranges as established by ATP III are shown in Table 2.1 Lipid profiles obtained for clinical purposes will usually report the amount of total cholesterol, the amount of cholesterol found on high density lipoprotein particles (the high density lipoprotein cholesterol [HDL-C]), and the amount of triglycerides.

TABLE 2. COMPONENTS OF A STANDARD LIPID PROFILE WITH NORMAL RANGES (MG/DL) AS DEFINED BY THE NCEP ADULT TREATMENT PANEL III1

TC	<200	desirable	**HDL-C**	>40	optimal in men
	200-240	borderline high		>50	optimal in women
	>240	high			
TG	<150	optimal	**LDL-C**	<100	optimal
	150-199	borderline high		100-129	near or above optimal
	200-499	high		130-159	borderline high
				160-189	high
	>500	very high		>190	very high

HDL-C=high density lipoprotein cholesterol, NCEP=National Cholesterol Education Panel, TC=total cholesterol, TG=triglyceride, LDL-C=low density lipoprotein cholesterol

Some laboratories will also report the amount of cholesterol found on low density lipoprotein particles (the low density lipoprotein cholesterol [LDL-C]) from direct measurement of LDL-C. However, not all laboratories will directly measure LDL-C. As shown in Table 3, if not directly measured, the amount of LDL-C can be calculated based on the Friedewald equation.2 This equation uses the triglyceride concentration to estimate the amount of cholesterol on the very low density lipoprotein particles (VLDL-cholesterol [VLDL-C]). The VLDL-C is estimated to be one-fifth of the triglyceride concentration. Thus, since the amount of total cholesterol in the bloodstream is the sum of the cholesterol present on LDL, VLDL, and HDL particles, the LDL-C can be estimated by subtracting the sum of the HDL-C and estimated VLDL-C from the total cholesterol. It is well known that this method of estimating the LDL-C becomes increasingly more inaccurate as the TG levels exceed 400 mg/dL, so it should only be used in individuals with a TG level of <400 mg/dL.2

TABLE 3. FRIEDEWALD EQUATION2

$$\text{LDL-C} = \text{TC} - \text{HDL-C} - \frac{\text{TG}}{5}$$

HDL-C=high density lipoprotein cholesterol, LDL-C=low density lipoprotein cholesterol, TC=total cholesterol, TG=triglyceride

In addition to the components of a standard lipid profile (Table 2), there are additional lipid subfractions and biomarkers which are clinically available such as LDL and HDL subfractions, apolipoprotein-B, LDL particle concentration, lipoprotein(a), lipoprotein-associated phospholipase A2, and c-reactive protein.3 The clinical utility of these and other biomarkers have recently been reviewed by an expert panel of lipid specialists and cardiovascular epidemiologists.3, 4 Other than the use of c-reactive protein in individuals at intermediate risk for coronary heart disease, none were recommended for routine clinical use.3 In addition, these additional lipid subfractions and biomarkers were not routinely recommended in the 2013 ACC/AHA Guideline on the Assessment of Cardiovascular Risk and are not recommended for routine use in the 2013 ACC/AHA Guideline on the Treatment of Blood Cholesterol to Reduce Atherosclerotic Cardiovascular Risk in Adults, and will, therefore, not be covered in detail here.4, 5

HYPERCHOLESTEROLEMIA AND CARDIOVASCULAR DISEASE RISK

Numerous epidemiologic studies have demonstrated that abnormal lipid parameters are associated with increased cardiovascular disease risk. The majority of these data suggest that high levels of LDL-C are atherogenic, and that elevated LDL-C is the most atherogenic of the dyslipidemias. High levels of LDL-C contribute to both the development of CHD and the frequency of clinical events among individuals with CHD.6, 14 As a result of these data, treatments aimed at reducing LDL-C have been the primary focus of therapy for hypercholesterolemia in CHD prevention.

Although most of the focus has been directed toward LDL-C, other abnormalities in lipid parameters are considered atherogenic. Hypertriglyceridemia is associated with high levels of VLDL-C and is now felt to be an independent

risk factor for CHD.15, 16 Furthermore, high levels of HDL-C are associated with decreased cardiovascular disease risk.6, 17-18 Therefore, a clinically useful method of estimating the total amount of cholesterol present on any of the atherogenic lipoproteins is to calculate the non- HDL-cholesterol (non-HDL-C) by subtracting the HDL-C from the total cholesterol. As a result, clinical guidelines aimed at reducing cardiovascular disease risk tend to focus on therapies that lower LDL-C and non-HDL-C and strategies to increase HDL-C.

Abnormalities in lipid parameters frequently do not occur in isolation. Recently, there has been increased recognition of the concept of the "atherogenic dyslipidemia" which consists of the simultaneous presence of elevated levels of triglycerides, low HDL-C, and only modest elevations in LDL-C.1 Although LDL-C levels are not markedly elevated, the LDL particles which carry the LDL-C are smaller and more dense in this condition and are generally felt to be more atherogenic.1

This "atherogenic dyslipidemia" is common in individuals with insulin resistance and diabetes.1 Frequently individuals may have a constellation of risk factors which includes this "atherogenic dyslipidemia" as well as obesity, hypertension, and hyperglycemia termed the metabolic syndrome (Table 4).

TABLE 4. CRITERIA FOR THE METABOLIC SYNDROME [19]

Abdominal Obesity	WC >102 cm (40 in.) in men or >88 cm (35 in.) in women
Hyper triglyceridemia	>150 mg/dL or drug treatment
Low HDL-cholesterol	<40 mg/dL in men or <50 mg/dL in women or drug treatment
Elevated Blood Pressure	Systolic BP >130 mmHg, Diastolic BP >80 mmHg, or drug treatment
Hyperglycemia	Fasting glucose >100 mmHg or drug treatment

BP=blood pressure, HDL=high density lipoprotein, WC=waist circumference

The current harmonized definition of metabolic syndrome requires the presence of any three of the following conditions as detailed in Table 4: abdominal obesity, elevated triglycerides, low HDL-C cholesterol, elevated blood pressure, and hyperglycemia.19 Individuals with metabolic syndrome are considered to be at high risk for CHD events, and those without diabetes are at very high risk of developing diabetes.20, 21

THE PATHOPHYSIOLOGY OF ATHEROSCLEROTIC CORONARY HEART DISEASE

Atherosclerosis results from a combination of traditional risk factors such as hypercholesterolemia, hypertension, diabetes, tobacco abuse, obesity, and physical inactivity which interact with inflammatory cells to produce the clinical manifestations of CHD.22 Our current understanding of atherosclerosis is that traditional risk factors are a source of inflammatory changes in the blood vessel wall which in turn attract lipid laden macrophages and other inflammatory cells to enter the blood vessel wall where they proliferate and develop atherosclerotic plaques, which are the source of the clinical manifestations of CHD.22

The atherosclerotic process begins in early adulthood as a fatty streak.23 Fatty streaks consist of lipid deposition (mainly lipid laden macrophages) in the arterial wall. Over many years, if risk factors, particularly hypercholesterolemia, remain uncontrolled, these plaques grow in size and, in some cases, ultimately enlarge enough that they narrow the lumen (or opening inside of the blood vessel), resulting in obstruction to blood flow.23 These larger and more obstructive lesions are termed fibrous plaques as they consist of an outer fibrous cap and an inner lipid core. Reduced coronary blood flow (myocardial ischemia) resulting from these fibrous plaques contributes to the symptoms of exertional angina and dyspnea that individuals with CHD experience.23

In addition, inflammatory changes inside of the atherosclerotic plaque can promote the breakdown and subsequent rupture of the fibrous plaque.22,23 In some cases, this follows the development of exertional symptoms, but in many cases, plaque rupture occurs prior to the development of any cardiovascular symptoms. This is because atherosclerotic plaques that are prone to rupture tend to be younger, more immature plaques that usually do not produce the degree of luminal narrowing required to develop exertional symptoms.24

This plaque rupture is then followed by clot formation within the lumen of the coronary artery, potentially resulting in an acute coronary syndrome from the sudden development of severe obstruction to coronary blood flow (acute myocardial ischemia).22, 23 The term

acute coronary syndrome describes a spectrum of clinical presentations of acute myocardial ischemia that ranges from unstable angina, to acute myocardial infarction, to even sudden cardiac death that occurs as a consequence of ischemia induced fatal ventricular arrhythmias. Coronary plaques that have high cholesterol content and a large lipid core (so-called "soft plaques") have been identified as plaques that are more inflammatory and are particularly susceptible to acute rupture, producing the substrate for acute myocardial infarction.23

TREATMENT OF HYPERCHOLESTEROLEMIA

The treatment of hypercholesterolemia involves a multifaceted approach of diet and lifestyle changes followed by pharmacologic therapy in those who are at higher risk and/or are unable to achieve recommended lipid levels with diet and lifestyle changes. Given that cholesterol, particularly LDL-C, is a major component of atherosclerotic plaque and that "soft plaques," which consist of a larger lipid inner core, are thought to be more prone to rupture and result in presentations of acute coronary syndrome, much of the focus on the prevention of CHD and the prevention of CHD-related events among individuals with CHD has been on the treatment of high LDL-C.

Dietary Modification as a Treatment for Hypercholesterolemia

In terms of macronutrients, diets high in cholesterol and fat, especially saturated and trans-fats, are associated with elevations in plasma LDL-C.25-30 Likewise, diets that replace saturated and trans-fat intake with monounsaturated and polyunsaturated fats are associated with reductions in LDL-C.31 Table 5 summarizes some of the common foods in the American diet based on their macronutrient concentration based on data collected from the AHA website, recognizing that most foods contain multiple types of fats.32 Dietary cholesterol is found in animal products with eggs, dairy, and meat being the major source of dietary cholesterol in the American diet. There is no dietary cholesterol contained in foods from plants.32

TABLE 5. CHOLESTEROL AND FATTY ACID COMPOSITION OF COMMON FOODS

MACRONUTRIENT	EXAMPLES
Dietary cholesterol	Animal products (meat, poultry, fish, eggs, butter, cheese, whole and 2% milk)
Saturated fatty acids	High fat meats (fatty beef, lamb, pork, poultry with skin, beef fat, lard), dairy products (cream, butter, cheese, whole and 2% milk), and tropical oils (palm oil, palm kernel oil, and coconut oil)
Trans-fatty acids	Fried foods (French fries, doughnuts), baked goods (pastries, pie crusts, biscuits, pizza dough, cookies, crackers), stick margarines, and shortenings
Monounsaturated fatty acids	Vegetable oils (olive oil, canola oil, peanut oil, sunflower oil, sesame oil), avocados, peanut butter, and many nuts and seeds
Polyunsaturated fatty acids	Vegetable oils (soybean oil, corn oil, safflower oil), fatty fish (salmon, mackerel, herring, trout), nuts (walnuts), and seeds (sunflower seeds)

Data shown obtained from the American Heart Association website.[32]

Saturated fats are also found in many foods that are also high in dietary cholesterol such as fatty meats and high fat dairy products. In addition, many tropical oils contain a high percentage of saturated fat but do not contain dietary cholesterol such as palm oil, palm kernel oil, and coconut oil. More recently, increased focus has been placed on trans-fats which are found in foods containing partially hydrogenated oils including fried foods, baked goods, stick margarines, and shortenings.32

Current recommendations on dietary patterns to reduce LDL-C, have been discussed in great detail in the 2013 ACC/AHA Guideline on Lifestyle Management to Reduce Cardiovascular Risk.31 These guidelines made four primary dietary recommendations for LDL-C lowering which are summarized in Table 6, and which focused on reductions in the consumption of saturated and trans-fats. No recommendation was made regarding the benefits of reducing dietary cholesterol.31

The committee did review two meta-analyses but found insufficient evidence to recommend reducing dietary intake of cholesterol due to a lack of adequate clinical data demonstrating that reductions in dietary cholesterol favorably impact the lipid profile.31, 33, 34

TABLE 6. 2013 ACC/AHA LIFESTYLE MANAGEMENT GUIDELINE DIETARY RECOMMENDATIONS FOR LOWERING LDL-C31

1. Consume a dietary pattern that emphasizes:
a) the intake of vegetables, fruits, and whole grains,
b) includes low-fat dairy products, fish, legumes, non-tropical vegetable oils, and nuts, and
c) limits the intake of sweets, sugar-sweetened beverages, and red meats
2. Aim for a dietary pattern that achieves 5-6% of calories from saturated fat
3. Reduce percent of calories from saturated fat
4. Reduce percent of calories from trans-fat

ACC=American College of Cardiology, AHA=American Heart Association, LDL-C=low density lipoprotein cholesterol

The committee emphasized that reductions in the dietary consumption of saturated and trans-fat are associated with improvements in the lipid profile.31 Lower saturated fat intake is associated with reductions in both LDL-C and HDL-C, but the reductions in LDL-C are larger than those of HDL-C resulting in an overall favorable impact on the lipid profile.35, 36 There is a variable response in the lipid profile based on whether the saturated fat is replaced by carbohydrates, monounsaturated fatty acids, or polyunsaturated fatty acids. The committee favored the use of polyunsaturated fatty acids, followed by monounsaturated fatty acids, and then carbohydrates to replace saturated and trans-fats as higher intakes of carbohydrates raise triglyceride levels and lower HDL-C.31 Among carbohydrates, complex carbohydrates are preferred to simple carbohydrates and whole grains are preferred to more refined carbohydrates.31

In addition to reviewing evidence on the macronutrient content of diets, the 2013 ACC/AHA Guideline on Lifestyle Management to Reduce Cardiovascular Risk also emphasized that recent research has focused on dietary patterns as opposed to simply the macronutrient content of foods as foods are typically consumed in combinations rather than individually.31

Two dietary patterns that were evaluated in detail in the guideline were the Mediterranean and Dietary Approaches to Stop Hypertension (DASH) dietary patterns.31 The basic components of each of these dietary patterns are summarized in Table 7. The committee felt that the strongest evidence supported adopting a DASH-like dietary pattern.31 Further details about the composition of the DASH dietary pattern can be found online at: http://www.nhlbi.nih.gov/health/public/heart/hbp/dash/new_dash.pdf.

TABLE 7. DIETARY PATTERNS FOR LOWERING LDL-C EXAMINED IN THE 2013 ACC/AHA LIFESTYLE MANAGEMENT GUIDELINE31

Mediterranean Pattern	higher in fruits (particularly fresh fruits), vegetables (roots and green varieties), whole grains (cereals, breads, rice, pasta), fatty fish (high levels of omega-3 fatty acids); lower in red meat; substituted lower-fat or fat-free dairy products for higher-fat dairy foods; used oils (olive or canola), nuts (walnuts, almonds, hazelnuts) or margarines with rapeseed or flaxseed oils instead of butter or other fats
DASH Pattern	higher in vegetables, fruits, low-fat dairy products, whole grains, poultry, fish, and nuts; lower in sweets, sugar-sweetened beverages, red meats

ACC=American College of Cardiology, AHA=American Heart Association, DASH=Dietary Approaches to Stop Hypertension, LDL-C=low density lipoprotein cholesterol

The DASH dietary pattern is also similar to those recommended by the 2006 AHA Scientific Statement on Diet and Lifestyle Recommendations and the 2010 US Department of Health and Human Services Dietary Guidelines.37, 38 The committee emphasizes that patients should be educated and provided resources to assist them in selecting foods including the use of food labels to identify the macronutrient content of foods and that referral to dietary and nutritional counseling is often necessary to assist patients in dietary changes.31

Physical Activity as a Treatment for Hypercholesterolemia

In addition to examining dietary patterns as a mechanism to lower LDL-C, the 2013 ACC/AHA Guideline on Lifestyle Management to Reduce Cardiovascular Risk examined data on physical activity as a mechanism to lower LDL-C and non-HDL-C.31 Based on their evaluation of the available evidence, the committee concluded that aerobic physical activity alone is associated with improvements in LDL-C and non-HDL-C but does not have significant effects on triglycerides or HDL-C.31 In contrast, resistance training was associated with improvements in LDL-C, HDL-C, and triglycerides, but not HDL-C.31

The committee's final recommendation is detailed in Table 8. This recommendation is based on the 2008 Physical Activity Guidelines Advisory Committee Report that a minimum of 150 minutes of moderate intensity physical activity (such as brisk walking) per week is necessary for most health benefits.31 The committee also recognizes that higher levels of physical activity are associated with more health benefits.31

TABLE 8. 2013 ACC/AHA LIFESTYLE MANAGEMENT GUIDELINE PHYSICAL ACTIVITY RECOMMENDATION FOR LOWERING LDL-C AND NON-HDL-C31

3-4 sessions per week of aerobic physical activity of moderate to vigorous intensity lasting on average 40 minutes per session

ACC=American College of Cardiology, AHA=American Heart Association, LDL-C=low density lipoprotein cholesterol, HDL-C= high density lipoprotein cholesterol

Additional Lifestyle Recommendations to Promote Cardiovascular Health

The 2006 AHA Scientific Statement on Diet and Lifestyle Recommendations provides additional guidance on lifestyle choices beyond diet and physical activity.37 These guidelines emphasize the importance of smoking cessation, maintaining a healthy body weight (defined as a body mass index of 18.5 to 24.9 kg/m2), and moderate alcohol consumption.37 Although the use of alcohol for the purposes of lowering cholesterol is not advocated, women and men who choose to consume alcohol should consume no more than one and two alcoholic drinks daily, respectively.37

Pharmacologic Approaches for the Treatment of Hypercholesterolemia

The 2013 ACC/AHA Guideline on the Treatment of Blood Cholesterol to Reduce Atherosclerotic Cardiovascular Risk in Adults has reviewed the scientific data that supports the use of medications to decrease cardiovascular risk in those at highest risk of cardiovascular events.5 This guideline replaces the ATP III guideline that had previously set treatment goals for individuals with CHD and those at high risk of CHD.1 In this review, we will provide a summary of the recommendations made by the committee.

Statins reduce cholesterol production in the liver by inhibiting the enzyme HMG CoA reductase. As discussed in more detail in the 2013 ACC/AHA Guideline on the Treatment of Blood Cholesterol to Reduce Atherosclerotic Cardiovascular Risk in Adults, the preponderance of the clinical trial evidence supports the use of statins to lower LDL-C for both primary and events and secondary prevention of cardiovascular events.

Thus, statin therapy is the mainstay of pharmacologic treatment of hypercholesterolemia.

Prior to or in addition to initiating drug therapy for elevated LDL-C, clinicians should first advise patients to follow the diet and lifestyle recommendations as described in the previous section. In addition, in some individuals, particularly those with LDL-C >190 mg/dL and triglycerides >500 mg/dL, investigations into possible secondary causes of hyperlipidemia may be warranted. Common etiologies of secondary causes of hyperlipidemia specifically mentioned in the 2013 ACC/AHA Guideline on the Treatment of Blood Cholesterol to Reduce Atherosclerotic Cardiovascular Risk in Adults are listed in Table 9.5

TABLE 9. COMMON SECONDARY CAUSES OF DYSLIPIDEMIA AS OUTLINED BY THE 2013 ACC/AHA GUIDELINE ON THE TREATMENT OF BLOOD CHOLESTEROL TO REDUCE ATHEROSCLEROTIC CARDIOVASCULAR RISK IN ADULTS5

Elevated LDL-C	diets high in saturated or trans-fat, weight gain, anorexia, diuretics, cyclosporine, glucocorticoids, amiodarone, biliary obstruction, nephrotic syndrome, hypothyroidism, obesity, pregnancy
Elevated Triglycerides	diets high in refined carbohydrates, weight gain, very low-fat diets, excessive alcohol intake, estrogens, glucocorticoids, bile acid sequestrants, protease inhibitors, retinoic acid, anabolic steroids, sirolimus, raloxifene, tamoxifen, beta blockers, thiazide diuretics, chronic renal failure, nephrotic syndrome, lipodystrophies, diabetes, hypothyroidism, obesity, pregnancy

ACC=American College of Cardiology, AHA=American Heart Association, LDL-C=low density lipoprotein cholesterol

Because the degree of LDL-C lowering is highly variable between different statins and different doses of statin therapy, the 2013 ACC/AHA Guideline on the Treatment of Blood Cholesterol to Reduce Atherosclerotic Cardiovascular Risk in Adults classified statin therapy based on the degree of expected LDL-C lowering according to the agent and dose chosen

(Table 10).5 As will be discussed in more detail below, individuals at highest risk who require more aggressive therapy will usually be treated with high intensity regimens, with low to moderate intensity regimens reserved for those at lower risk or who do not tolerate high intensity therapy. Statin therapy classified as low intensity lowers LDL-C on average by <30%, moderate intensity on average by 30-<50%, and high intensity on average by >50%.5

TABLE 10. CLASSIFICATION OF STATIN THERAPY

BASED ON INTENSITY OF LDL-C LOWERING BY THE 2013 ACC/AHA GUIDELINE ON THE TREATMENT OF BLOOD CHOLESTEROL TO REDUCE ATHEROSCLEROTIC CARDIOVASCULAR RISK IN ADULTS5

	LOW INTENSITY	MODERATE INTENSITY	HIGH INTENSITY
Rosuvastatin		5, 10 mg	20, 40 mg
Ator vastatin		10, 20 mg	40, 80 mg
Simvastatin	10 mg	20, 40 mg	
Pravastatin	10, 20 mg	40, 80 mg	
Lovastatin	20 mg	40 mg	
Fluvastatin XL		80 mg	
Fluvastatin	20, 40 mg	40 mg twice daily	
Pitavastatin	1 mg	2, 4 mg	

All doses given once daily except as noted.

ACC=American College of Cardiology, AHA=American Heart Association, LDL-C=low density lipoprotein cholesterol

One major change in this guideline as compared to ATP III was that the decision to initiate statin therapy and, once initiated, the intensity of statin therapy prescribed are no longer recommended based on LDL-C and treatment thresholds. Rather, the new guideline has established four clinical groups (Table 11) in which the committee feels, based on a review of the evidence, that the benefits of statin therapy outweigh any risks for adverse events.5 In this way, the decision about whether to initiate statin therapy and at what dose is based on an individual's global cardiovascular risk, rather than simply a LDL-C level. Those at higher risk get more intensive therapy; whereas, those at lower risk do not require drug treatment or are treated with less intensive therapy.

TABLE 11. FOUR CLINICAL GROUPS THAT BENEFIT FROM STATIN THERAPY AND RECOMMENDED INTENSITY OF STATIN THERAPY FOR ELEVATED LDL-C BASED ON THE 2013 ACC/AHA GUIDELINE ON THE TREATMENT OF BLOOD CHOLESTEROL TO REDUCE ATHEROSCLEROTIC CARDIOVASCULAR RISK IN ADULTS5

1. Clinical ASCVD*
   - high intensity statin therapy for those <75 years of age†
   - moderate intensity statin therapy for those >75 years of age
2. LDL-C >190 mg/dL
   - high intensity statin therapy†
3. Diabetes, aged 40-75 years, LDL-C 70-189 mg/dL, and no clinical ASCVD*
   - moderate intensity statin therapy
   - if 10 year risk of ASCVD >7.5%, ‡ optional use of high intensity statin therapy (based on expert opinion)
   - consideration of statin in those <40 and >75 years (based on expert opinion)
4. Aged 45-70, LDL-C 70-189 mg/dL, no diabetes or clinical ASCVD,* and 10 year risk for ASCVD >7.5%‡
   - moderate to high intensity statin therapy

*Clinical ASCVD defined as acute coronary syndrome, myocardial infarction, stable or unstable angina, coronary or other arterial revascularization, stroke, transient ischemic attack, or peripheral arterial disease.

†Use moderate intensity statin in those who cannot tolerate or are not candidates for high intensity statin.

‡Based on Pooled Cohort Equations.4 An online calculator is available at http://www.cvriskcalculator.com/.39

ACC=American College of Cardiology, AHA=American Heart Association, ASCVD=atherosclerotic cardiovascular disease, LDL-C=low density lipoprotein cholesterol

There are a number of additional sub-groups that are not covered in Table 11 that are worth mentioning. First, the efficacy of statin therapy in those with heart failure and end-stage renal disease has been questioned as the result of clinical trials which failed to demonstrate a benefit of statin therapy in reducing cardiovascular events in these two groups.40-43 As a result, the committee chose to make no recommendation regarding statin therapy in these

two groups. The committee stopped short of advising against statin therapy in those with heart failure and end-stage renal disease, but advised an individualized approach based on individual patient's own preferences and likelihood of benefit, risks for adverse events, and potential for drug-drug interactions.5 Second, some groups of patients might be at excess cardiovascular risk but do not fall into one of the four clinical groups outlined in Table 11. These factors include: 1) LDL-C >160 mg/dL, family history of premature atherosclerotic cardiovascular disease, elevated high-sensitivity-C-reactive protein (>2 mg/L), elevated coronary artery calcium score (>300 Agatston units or >75th percentile for age, sex, and ethnicity), ankle-brachial index <0.9, or elevated lifetime risk.5 Although the committee does not make specific recommendations for these individuals, similar to recommendations for those with heart failure or end-stage renal disease, they advised an individualized approach based on individual patient's own preferences and likelihood of benefit, risks for adverse events, and potential for drug-drug interactions.5

Although current treatment recommendations do not use LDL-C levels to drive therapy, the committee does still recommend repeating lipid profiles 4-12 weeks after initiation of statin therapy and 3-12 months periodically thereafter to assess for the expected response to therapy.5 The committee cites two reasons for a less than expected response to therapy. First, measuring LDL-C is an effective method of assessing patient adherence. Practitioners should question patients about reasons for non-adherence and educate them on the importance of regularly taking their medications. In patients who poorly adhere to statin therapy because of adverse effects or intolerances, alternative statin medications or lower intensity statin therapy may be better tolerated.5 Secondly, if patients adhere to therapy yet still have a less than expected reduction in LDL-C, investigations for biologic reasons underlying this variability may be warranted, particularly aimed at investigating for the presence of secondary causes of dyslipidemia (Table 9), which may reduce the effectiveness of statins.5 Further difficulty adequately lowering LDL-C due to adverse effects from or intolerances to medical therapy or failure to respond to therapy as expected should prompt referral to a lipid specialist for assistance with further management.

The two most common reasons that result in non-adherence to statin therapy are muscle side effects (which range in severity from mild myalgias to rhabdomyolysis) and transaminitis. The routine measurement of creatine kinase (CK) levels prior to initiating statin therapy or for surveillance of myopathic side effects in asymptomatic patients is not recommended.5 Rather, CK levels are only recommended to investigate muscle related symptoms.5 In terms of monitoring for liver toxicity, liver transaminases should be measured prior to the initiation of statin therapy in everyone, and then only re-measured if there is clinical suspicion for hepatotoxicity such as unusual fatigue, weakness, loss of appetite, abdominal pain, dark colored urine, or jaundice.5 Recently, there has been increased attention to the observation that individuals on statins are at increased risk of developing diabetes.44 However, because of the clear benefit of statins in reducing cardiovascular risk in individuals with diabetes, no treatment modification has been recommended on the basis of this risk.5 Individuals at risk for diabetes, on or off of statin therapy, should be screened per current guidelines.5

Non-statin Therapy

Non-statin pharmacologic therapy can be considered in patients as add on therapy in addition to statin therapy or as an alternative to statin therapy in those individuals who are intolerant of statins. The ACC/AHA Guideline on the Treatment of Blood Cholesterol to Reduce Atherosclerotic Cardiovascular Risk in Adults reviewed the available clinical trial data for these two indications and concluded that there were insufficient data to support the routine use of non-statin pharmacologic therapy for these two clinical scenarios.5 The committee stopped short of advising against non- statin therapy, but advised an individualized approach based on individual patients' own preferences and likelihood of benefit, risks for adverse events, and potential for drug-drug interactions.5 Subsequent to this, in 2016, an expert consensus panel, convened by the ACC, provided additional recommendations regarding non-statin therapies.45 This panel recommended against the use of niacin for LDL-C reduction given the lack of efficacy and potential harms associated with its use. A patient centered approach to non-statin therapy was recommended that took the patient's global cardiovascular risk and desired LDL-C level into consideration. The addition of non-statin medications

must also balance the possible cardiovascular benefit with the potential for side effects and drug-drug interactions. As such, no single strategy will be equally effective in all individuals. A summary of lipid effects and potential side effects to non-statin lipid lowering medications is provided in Table 12.

TABLE 12. LIPID EFFECTS AND POTENTIAL SIDE EFFECTS OF COMMONLY USED NON-STATIN LIPID LOWERING THERAPIES

AGENT	LIPID EFFECTS	POTENTIAL SIDE EFFECTS
Niacin*	↓ LDL-C 5-25%, ↑ HDL-C 15-35%, ↓ TG 20-50%	flushing, transaminitis, hyperglycemia, hyperuricemia
Bile acid sequestrants	↓ LDL-C 15-30%, ↑ HDL-C 3-5%, little effect on TG	Hypertriglyceridemia, GI disturbances, transaminitis
Ezetemibe	↓ LDL-C 15-20%, little effect on HDL-C, ↓ TG 5-10%	Transaminitis, GI disturbances, URI symptoms
Fibrates	↓ LDL-C 5-20%, ↑ HDL-C 10-35%, ↓ TG 20-50%	myopathy (especially gemfibrozil), †renal dysfunction (especially fenofibrate)
Omega 3 fatty acids	↑ LDL-C 10-45%,‡ ↑ HDL-C 10%, ↓ TG 25-45%	GI disturbances, skin changes, bleeding
PCSK9 inhibitors	↓ LDL-C 45-60%	Injection site reactions, nasopharyngitis, influenza

Data compiled from references 1, 5, 45, and 46.

*Niacin is not recommended for LDL-C lowering by ACC consensus panel.45

†Gemfibrozil should not be used concomitantly with statin therapy.5

‡LDL-C may increase in patients on omega 3 fatty acids especially if the TG is elevated at the start of therapy.46

GI=gastrointestinal, HDL-C=high density lipoprotein cholesterol, LDL-C=low density lipoprotein cholesterol, PCSK9=proprotein convertase subtilisin/kexin 9, TG=triglycerides, URI=upper respiratory infection

SUMMARY

High cholesterol, particularly elevated LDL-C, is a major risk factor for the development of CHD and is associated with an increased number of CHD-related events in individuals with CHD. Current approaches to lower cholesterol levels include lifestyle interventions, such as dietary modifications and physical activity, as well as drug therapy, primarily with statins. Health care professionals working in cardiac rehabilitation have a unique opportunity to educate patients with cardiovascular disease on lifestyle changes and promote adherence to secondary prevention medications.

BLOOD LIPID MANAGEMENT

McKool, K RN, MSN, CTTS-M. (Ed.). (2018). Blood Lipid Management *CR Best practice resource: A CCRP study guide*. (pp. 94-102). The American Association of Cardiovascular and Pulmonary Rehabilitation.

NAME_____DATE_____

1. High cholesterol is recognized as a _____ risk factor for the development of coronary heart disease.
   a. Possible
   b. Mild
   c. Moderate
   d. Major

2. The density of a lipoprotein particle is related to its lipid content. Those with less lipid content and more protein are more dense (HDL). Those with higher concentration of lipid are less dense (LDL). Treatment of hypercholesterolemia aims at reducing_____.
   a. LDL-C low density lipoprotein cholesterol
   b. HDL- C high density lipoprotein cholesterol

3. Individuals with Metabolic Syndrome are considered to be at high risk for Coronary Heart Disease (CHD) events. List the 5 conditions that are identified as criteria for Metabolic Syndrome.
   a._____
   b._____
   c._____
   d. _____
   e. _____

4. Treatment of hypercholesterolemia involves a multifaceted approach. Choose all the correct answers.

   a. Dietary changes that emphasize a high intake of vegetables, fruits, and whole grains
   b. Dietary changes that increase the intake of saturated fat.
   c. Dietary changes that reduce calories from trans-fat
   d. Minimum of 150 minutes of moderate intensity physical activity per week.
   e. The use of medications to decrease cardiovascular risk in those at highest risk of cardiovascular events
   f. All the above

Use TABLE 10 and TABLE 11 on page 100 to answer questions 5-7

5. John is a 40-year-old patient who experienced a recent MI and continues to have stable angina.
   His LDL-C is 120mg/dL. The recommended intensity of statin therapy is:
   a. Low Intensity
   b. Moderate Intensity
   c. High Intensity

6. Mary is 80 years old and recently diagnosed with peripheral arterial disease. Her LDL-C is 100mg/dL. Her physician has told her that she needs to start taking Atorvastatin. The recommended intensity of statin therapy will be _____ with an Atorvastatin dose of _____.
   a. High Intensity at 40mg
   b. Moderate Intensity at 20mg
   c. Moderate Intensity at 40mg

7. Claudia is a 41-year-old diabetic patient who was recently diagnosed with COPD and will be participating in outpatient pulmonary rehab. She says that her primary doctor recently started her on statin therapy. In her chart you read that her LDL-C is 90mg/dL. The recommended intensity of statin therapy is:
   a. No statin therapy is needed
   b. Low Intensity
   c. Moderate Intensity
   d. High Intensity

8. The two most common reasons that persons do not adhere to statin therapy are:
   a. Cost and nausea
   b. Concern for the development of diabetes and preferring alternative therapies.
   c. Belief that is not needed or effective
   d. Muscle side effects and transaminitis

9. For patients with poor adherence because of adverse effects to statin therapy:
   a. Stop all statin therapy
   b. Continue despite adverse effects
   c. Offer alternative statin medications
   d. Lower intensity statin therapy
   e. Both c. and d.

10. It is important for Cardiopulmonary Rehab staff to know and understand the recommendations for Lipid Management because:
   a. Cardiopulmonary Rehab staff have a unique opportunity to educate patients
   b. Cardiopulmonary Rehab staff have the ability to promote needed lifestyle changes
   c. High cholesterol is a major risk factor for the development of coronary artery disease
   d. All the above

BLOOD LIPID MANAGEMENT - ANSWER KEY

McKool, K RN, MSN, CTTS-M. (Ed.). (2018). Blood Lipid Management *CR Best practice resource: A CCRP study guide*. (pp. 94-102). The American Association of Cardiovascular and Pulmonary Rehabilitation.

NAME_____DATE_____

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   a. LDL-C low density lipoprotein cholesterol
   b. HDL- C high density lipoprotein cholesterol

3. Individuals with Metabolic Syndrome are considered to be at high risk for Coronary Heart Disease (CHD) events. List the 5 conditions that are identified as criteria for Metabolic Syndrome.
   a. Abdominal Obesity
   b. Hypertriglyceridemia
   c. Low HDL-cholesterol
   d. Elevated Blood Pressure
   e. Hyperglycemia

4. Treatment of hypercholesterolemia involves a multifaceted approach. Choose all the correct answers.
   a. Dietary changes that emphasize a high intake of vegetables, fruits, and whole grains
   b. Dietary changes that increase the intake of saturated fat.
   c. Dietary changes that reduce calories from trans-fat
   d. Minimum of 150 minutes of moderate intensity physical activity per week.
   e. The use of medications to decrease cardiovascular risk in those at highest risk of cardiovascular events
   f. All the above

Use TABLE 10 and TABLE 11 on page 100 to answer questions 5-7

5. John is a 40-year-old patient who experienced a recent MI and continues to have stable angina.
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   c. High cholesterol is a major risk factor for the development of coronary artery disease
   d. All the above

Sugar-Free Gluten-Free Maple Orange Glazed Chicken

Ingredients

- 8 chicken thighs
- ⅓ c. sugar-free maple syrup
- ¼ c. fresh orange juice
- 2 tbs. coconut aminos
- 2 tbs. olive oil
- 1 tbs. Dijon mustard
- 2 garlic cloves, minced
- ½ tsp. crushed red chili flakes
- Thyme for seasoning
- Salt and Pepper to taste
- 2 boxes natural Heaven brown rice hearts of palm
- 1 medium onion chopped
- 2 small carrots, chopped and any other veggie you would like

Directions

Preheat oven to 400˚. Spray a 9x13 baking dish. In the bottom of the dish place the Veggie base (brown rice, onion, carrots) mix well and spread in the bottom of the pan. Salt and pepper both sides of the chicken and place on top of the veggie base. Mix together maple syrup, orange juice, coconut aminos, olive oil, Dijon mustard, garlic, chili flakes. Pour mixture over chicken and sprinkle with thyme. Cover with foil and bake at 400˚ for 30 minutes, then remove the foil. Bake for another 30 minutes or until chicken reaches 165˚ internal.

RECIPE BY: COOKING WITH CONNIE

Recently Released:

AACVPR
American Association of Cardiovascular & Pulmonary Rehabilitation

An AACVPR Update on Virtual Cardiac and Pulmonary Rehab Legislation.

February 3, 2026

Earlier today, Congress advanced the Consolidated Appropriations Act, 2026 (H.R. 7148), a funding package that includes provisions to extend Medicare telehealth flexibilities and in-home cardiopulmonary rehabilitation flexibilities through December 31, 2027. Next, the legislation will be sent to the president to be signed into law.

This milestone represents a major advocacy achievement for our field and a clear reflection of what we can accomplish together. Through persistent outreach, Day on the Hill participation, and tireless grassroots engagement, you helped drive this win forward. AACVPR members have consistently shown what it means to champion equitable, high quality, evidence-based rehabilitation for patients across the country – and today, that work pays off.

What this Means for Programs

Cardiac, intensive cardiac, and pulmonary rehabilitation (CR/ICR/PR) programs in both physician office and hospital outpatient settings can now deliver services virtually, via synchronous two-way audiovisual telecommunications technology, through December 31, 2027. This two-year extension creates a critical window for programs to implement and scale virtual and hybrid care – a powerful opportunity to improve participation rates and reach patients who have historically been unable to enroll or complete rehabilitation.

It allows programs to:

- Launch or expand hybrid CR/ICR/PR services
- Explore innovative care models that break down barriers to participation and improve access and adherence
- Plan with greater confidence while longer-term policy solutions are pursued

Hybrid delivery is a powerful tool to meet patients where they are and close long-standing gaps in access and participation.

Recently Released:

An AACVPR Update on Virtual Cardiac and Pulmonary Rehab Legislation.

February 3, 2026

What's Next: Education to Support Implementation

Join AACVPR for an upcoming webinar, Launching & Expanding Hybrid CR/ICR/PR: What Programs Need to Know Now, featuring national experts and program leaders who will break down the policy change and share strategies for successful implementation.

Launching & Expanding Hybrid CR/ICR/PR: What Programs Need to Know Now

Thursday, February 19

Noon - 1 p.m. CT

This timely session will cover:

- What the policy change means for CR/ICR/PR programs
- Key clinical, operational, and safety considerations
- Real-world examples from programs already delivering hybrid care

This webcast will serve as the first step in a broader AACVPR education effort designed to help programs take full advantage of this two-year extension and translate this policy victory into meaningful improvements for patients and programs alike.

ISCVPR Program Highlights

Regional Meetings -

Central

February 12th, 2026 @5-6pm, hosted by Aaron Wright.
We are meeting virtually with Dr. Fulkerson to discuss pulmonary hypertension.
Microsoft Teams link to join:
https://teams.microsoft.com/meet/28428382468299?p=ZLicRPcKXGaaj6yc4p

www.iscvpr.org

Is your program ready to celebrate for Cardiac and Pulmonary Rehabilitation week?

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February 8-14, 2026

PR Pulmonary Rehabilitation
Inhale Confidence, Exhale Limits

March 8-14, 2026

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