Concepts and Controversies: The 2013 American College of Cardiology/American Heart Association Risk Assessment and Cholesterol Treatment Guidelines

Seth S. Martin, MD, and Roger S. Blumenthal, MD

On 12 November 2013, the American College of Cardiology and American Heart Association (ACC/AHA) issued clinical guidelines on cardiovascular disease risk assessment (1) and cholesterol treatment (2). The recommendations are intended to guide decision making but do not replace clinical judgment. We highlight the core concepts of the guidelines that rest on widespread consensus, discuss controversial aspects, and suggest a potential middle ground for clinicians and patients struggling with how to proceed in the midst of the controversy.

**Core Concepts**

**Value of the Art of Medicine**

The new guidelines value the art of medicine in that they allow room for individualizing primary prevention on the basis of shared decision making between the patient and clinician. When one is considering whether to initiate a statin for primary prevention in adults with a low-density lipoprotein cholesterol (LDL-C) level of 70 to 189 mg/dL and an estimated 10-year risk for myocardial infarction or stroke of 5% or greater, the guidelines advise that patients and clinicians engage in a “risk discussion.” Such a discussion can address potential benefits and harms of statin therapy, drug–drug interactions, and patient preferences. Along these lines, we previously suggested consideration of the “5 Ps” (3): preference, precision, participation, potency, and price (Table).

Encouraging greater patient–provider dialogue is a virtue of the new guidelines, but finding time for these risk discussions will be challenging. We suspect that nearly an entire visit, or perhaps several visits, will need to be dedicated to this purpose. Careful discussion will be critical to clinical decision making, particularly in patients with prior statin intolerance and elderly patients with greater propensity for drug–drug interactions and competing risks. Even when high-quality evidence is available, the art of medicine still matters. In fact, the more we learn, the finer an art it becomes.

**Statins as First-Line Pharmacologic Therapy**

Several classes of drugs decrease atherogenic cholesterol levels and cardiovascular risk (4). In 2001, the Adult Treatment Panel III guideline recommended a statin, bile acid sequestrant, or nicotinic acid (4). Since then, efficacy and safety data have accumulated for statins, with trial data from more than 170 000 patients representing various populations (5). Generic high-intensity statins have also become available. The new guidelines strongly recommend statins as first-line pharmacologic therapy. Furthermore, they advise clinicians to prioritize efforts to maximize statin dose and to try alternative statins or dosing regimens in patients with intolerance.

**Expanding the Scope of Prevention**

The new guidelines expand the scope of prevention from coronary heart disease to atherosclerotic cardiovascular disease. Adding stroke to the risk calculator created a challenge because stroke is not uniformly atherosclerotic in origin; however, the guidelines succeeded in incorporating it. This expansion should help unite primary care providers, cardiologists, neurologists, and gynecologists in addressing the shared risk factors of cerebrovascular and coronary disease and their common pathophysiologic pathway: atherosclerosis.

**Areas of Controversy**

**Accuracy of Risk Assessment**

The most controversial aspect of the new guidelines is the inclusion of a new calculator for 10-year risk for myocardial infarction or stroke based on 4 cohort studies by the National Heart, Lung, and Blood Institute (1). During the development of the guideline, calculator validation in 2 external cohorts yielded c-statistics ranging from 0.56 to 0.77, with systematic overestimation of risk. After its re-
lease, some clinicians began testing the calculator and questioning its clinical accuracy. Compounding concern, examination of the calculator’s performance in 3 additional cohorts showed a 75% to 150% overestimation of risk (6). Future studies should clarify reasons for overestimation and evaluate refinements to the calculator.

To manage potential limitations of the risk calculator, a reasonable middle ground might be to expand the definition of intermediate risk from a range of 5.0% to 7.5% to a range of 5% to 15%. Patients falling in this range who desire greater certainty could then consider their family history or coronary artery calcium score to refine risk assessment. For example, the Canadian guidelines double the estimated risk if a family history of premature cardiovascular disease is present (first-degree relative aged <60 years) (7). A coronary artery calcium score of 0 portends a favorable 10-year prognosis, and a score of 100 or greater signifies a risk level that approximates that in persons who have already had an event (8).

Abandonment of Lipid Goals

Unlike Adult Treatment Panel III and recent European and Canadian guidelines, the new recommendations do not target fixed LDL-C and non–high-density lipoprotein cholesterol (HDL-C) goals. Rather, they recommend lipid measurement at baseline, 1 to 3 months after statin initiation, and yearly thereafter to check for the expected percentage decrease of LDL-C levels (30% to 45% with a moderate-intensity statin and ≥50% with a high-intensity statin). The guideline panel considered only selected randomized, controlled trials and concluded that treatment targets are not “evidence-based.” However, true evidence-based medicine is “the conscientious, explicit, and judicious use of current best evidence in making decisions about the care of individual patients” (9). Therefore, a truly evidence-based approach may be to synthesize randomized evidence with other lines of evidence, which supports selective use of LDL-C or non–HDL-C targets in high-risk adults.

Strict devotion to lipid targets might inadvertently lead to withholding treatment in high-risk patients with favorable baseline lipid levels or unnecessary addition of nonstatin drugs. Still, some experts maintain that lipid targets can also enhance care when applied during follow-up in high-risk patients. Indeed, LDL-C goals were part of the expressed strategy in landmark secondary prevention trials (TNT [Treating to New Targets], COURAGE [Clinical Outcomes Utilizing Revascularization and Aggressive Drug Evaluation], and AIM-HIGH [Atherothrombosis Intervention in Metabolic Syndrome with Low HDL/High Triglycerides: Impact on Global Health Outcomes]).

In addition, some studies, such as AIM-HIGH and ACCORD (Action to Control Cardiovascular Risk in Diabetes) (10), suggest possible benefits of add-on therapy for patients with high triglyceride levels and low LDL-C levels, which are markers of remnant lipoprotein cholesterol. A follow-up non–HDL-C goal could guide the treatment of these patients. In fact, add-on therapy to optimize atherogenic cholesterol levels in high-risk patients is compatible with the guideline advice for familial hyperlipidemia that “nonstatin cholesterol-lowering medications are often needed to lower LDL-C to acceptable levels” (2).

Our view is that risk- and lipid-based paradigms are not mutually exclusive and could be complementary. At baseline, obtaining the most accurate assessment of risk is crucial in deciding whom to treat, whereas in follow-up, lipid measurements can serve as a marker of therapeutic response, promote adherence, motivate lifestyle improvements, and guide discussions about add-on pharmacologic therapy for patients who are clearly established as high-risk.

CONCLUSION

The 2013 ACC/AHA risk assessment and cholesterol treatment guidelines emphasize important core concepts to rally behind. We intend our perspective to be a springboard for more detailed discussions on how best to implement and enhance the optimum care of individual patients. A patient’s risk estimate is now the number to know rather than his or her LDL-C level, unless the LDL-C level is 190 mg/dL or greater. We hope that the results of ongoing studies will help us determine whether aiming for a non–HDL-C target is more “evidence-based” than simply giving a statin once risk exceeds a threshold level.

From Johns Hopkins Ciccarone Preventive Cardiology Center, Baltimore, Maryland.

Financial Support: Dr. Martin is supported by the Pollin Cardiovascular Prevention Fellowship and the Marie-Josée and Henry R. Kravis endowed fellowship. Dr. Blumenthal is supported by the Kenneth Jay Pollin Professorship in Cardiology.

Potential Conflicts of Interest: Disclosures can be viewed at www.acponline.org/authors/icmje/ConflictOfInterestForms.do?msNum=M13-2805.

Requests for Single Reprints: Roger S. Blumenthal, MD, Johns Hopkins Hospital, 1800 Orleans Street, Blalock 524C, Baltimore, MD 21287; e-mail, rblument@jhmi.edu.

Current author addresses and author contributions are available at www.annals.org.


References


Current Author Addresses: Drs. Martin and Blumenthal: Johns Hopkins Hospital, 1800 Orleans Street, Blalock 524C, Baltimore, MD 21287.

Author Contributions: Conception and design: S.S. Martin, R.S. Blumenthal.
Analysis and interpretation of the data: R.S. Blumenthal.
Drafting of the article: S.S. Martin.
Critical revision of the article for important intellectual content: S.S. Martin, R.S. Blumenthal.
Final approval of the article: S.S. Martin, R.S. Blumenthal.