

13. The patient requests your advice on whether he should start taking coenzyme Q10 (CoQ10) supplement. Which *one* of the following statements is CORRECT regarding CoQ10?
- (A) CoQ10 functions as an electron carrier in the mitochondrial respiratory chain and as an intracellular antioxidant.
 - (B) Statin-associated myopathy can be attributed to a decrease in serum CoQ10 levels.
 - (C) CoQ10 supplementation is necessary when statins are administered.
 - (D) Muscle tissue levels of CoQ10 have been shown to be lower in statin-treated patients and CoQ10 supplementation corrects the defect.
14. The patient agrees to start another trial of statin therapy with rosuvastatin 5 mg. However, his myalgia symptoms recur, although to a milder degree, and rosuvastatin is discontinued but he remains on ezetimibe 10 mg. Which *one* of the following therapies would be LEAST likely to achieve further significant reduction in LDL-C?
- (A) Fluvastatin.
 - (B) Pravastatin.
 - (C) Colesevelam.
 - (D) Fenofibrate.

■ Items 15-17

A 39-year-old woman who is a non-smoker is referred for management of resistant hypertriglyceridemia. She had one episode of acute pancreatitis 2 years previously associated with milky-appearing plasma and she recalls her triglyceride level being “in the thousands”. Her medical history is significant for type 2 diabetes mellitus (DM), which was diagnosed at the time of the pancreatitis episode, and hypertension. She believes her glycemic control is fair and has not been monitoring her glucose. She tries to follow a diet low in fat/cholesterol and concentrated carbohydrates but she does not have a structured exercise program. Her medical records indicate that she has gained about 10 pounds over the last 6 months. Her current medications include metformin, glipizide, lisinopril/hydrochlorothiazide, atorvastatin 80 mg and gemfibrozil 1200 mg daily. Her BMI is 31 kg/m² and her waist circumference is 39”. Her laboratory values are as follows:

Total cholesterol	255 mg/dL
Triglycerides	950 mg/dL
HDL-C	42 mg/dL
LDL-C	NA
Glucose	205 mg/dL
HbA1c	9.5%
Creatinine	1.2 mg/dL
Urinalysis	Normal
Electrolytes	Normal
ALT	Normal
TSH	Normal

15. Which *one* of the following mechanisms BEST explains this patient's lipid profile?
- (A) Increased release of free fatty acids from adipose tissue and overproduction and impaired catabolism of apoB-containing lipoproteins.
 - (B) Complete lipoprotein lipase deficiency.
 - (C) ApoCII deficiency.
 - (D) ApoE2 homozygosity.
16. Which *one* of the following interventions will MOST rapidly decrease the triglyceride level in this patient?
- (A) Titration of both metformin and glipizide to maximal doses.
 - (B) Addition of pioglitazone.
 - (C) Addition of intermediate- or long-acting s.c. insulin at bedtime.
 - (D) Initiation of TLC-based diet focusing on restricting refined carbohydrates.
17. The patient is instructed on an intensive lifestyle modification program with the aim of losing 5–7% of body weight in the following 3 months. She refuses to start insulin despite your recommendations. Which *one* of the following additional therapies is contraindicated for managing her hypertriglyceridemia?
- (A) Addition of a thiazolidinedione with combined dietary and structured exercise prescription.
 - (B) Addition of omega-3 polyunsaturated fatty acids.
 - (C) Addition of niacin.
 - (D) Substitution of fenofibrate for gemfibrozil.

Bibliography

1. Executive Summary of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III): *JAMA* 2001;285:2486-97.

Item XX Answer X

The HATS trial (Figure 6) demonstrated that simvastatin and niacin resulted in a net regression of angiographic atherosclerosis compared to placebo. This net regression was also associated with a significant reduction in clinical events. This study supports the concept that lowering LDL and raising HDL results in a significant reduction in atherosclerotic development.

In patients with hypertriglyceridemia, statins have a greater effect in raising HDL. In one study with fluvastatin XL 80 mg, in patients with hypertriglyceridemia, HDL increased by 20%. In a study with fluvastatin plus niacin, the rate of liver toxicity did not increase significantly greater than with each agent used repeatedly especially at doses of niacin extended release of 1,000 mg/day. The risk of myopathy is also not significantly increased when combining a statin such as fluvastatin with a niacin extended release dose of 1,000 mg. Myopathy with combining a statin with niacin appears to occur less frequently than with a fibrate and a statin. The myopathy with the statin-niacin combination appears to be associated with niacin-induced hepatotoxicity initially, and the liver impairment affects the pharmacokinetic clearance of statins, increasing the blood levels inducing the myopathy. Niacin does lower Lp(a) modestly, but at doses of 1,000/day, the reduction in Lp(a) is approximately 10% to 20%

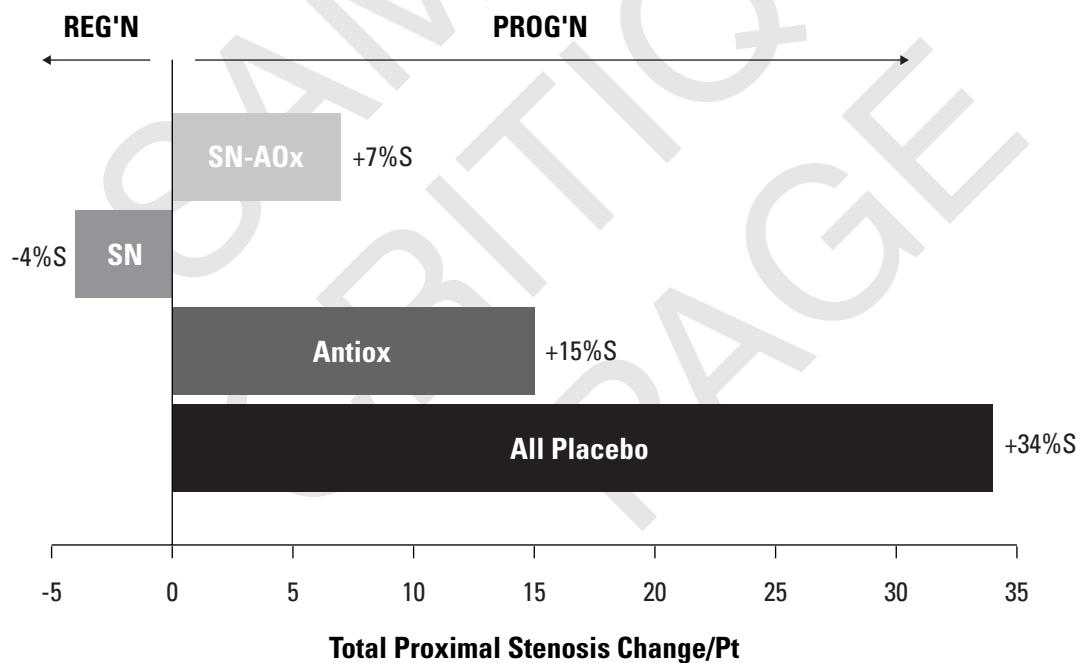


Figure 6. HATS Results: QCA

SN=Simvastatin=Niacin; Aox=Antioxidant
As presented at the 2000 AHA; Abstract #2461