I am still delivering babies, doing hospital work and office surgical procedures, and coming in after hours to see patients, but I am looked upon as a museum piece.

The list of things FPs do just keeps getting shorter

In his editorial, Dr. Hickner posed an important question: Have family physicians abandoned acute care? (*J Fam Pract*. 2013;62:333). My answer is Yes, they have abandoned acute care—and a lot more. FPs no longer do hospital care, obstetrics, pediatrics, orthopedics, gynecology, procedures, or continuity care. FPs have been so dumbed down, there is nothing they do that a mid-level cannot do.

I have been practicing family medicine for more than 25 years. I'm still delivering babies, doing hospital work and office surgical procedures, and coming in after hours to see patients, but I am looked upon as a museum piece by other physicians in my area.

So I'll pose another question to my colleagues here: What, exactly, is the role of a family physician in today's brave new health care model?

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Additional insights on how best to utilize statins

I agree wholeheartedly with Dr. Wahawisan et al (Statin therapy: When to think twice. *J Fam Pract*. 2013;62:726-730,732) that one shouldn't use medications to ameliorate dyslipidemia unless those medications have been proven to stabilize or regress plaque and/or improve atherothrombotic disease (ATD) outcomes. However, I believe the article deserves further comment.

I have a fairly large (about 250 patients) lipid clinic that I established in 1974; approximately 20% of my patients are taking statin-fenofibrate therapy. I use statin-fibrate (fenofibrate, never gemfibrozil[1]) in patients with both low high-density lipoprotein and high triglycerides, although I usually do so after starting statins. When the lipid goal is achieved, fibrates can induce plaque stabilization/regression.[2]

I rarely use niacin because of the associated itching and flushing, and because no stand-alone trials have shown a positive effect of niacin on ATD events. The Coronary Drug Project failed to find immediate effects of niacin, but there seemed to be a survival benefit 15 years after the trial ended.[3] I use fish oil (but not krill oil, since there have been no population studies) combined with low-dose aspirin for these agents' antiplatelet and vasodilatory effects. The dosages I use are docosahexaenoic acid plus eicosapentaenoic acid 1 g/d and aspirin 81 mg/d.

I stopped using ezetimibe when the SEAS trial[4] found an increase in total cancers and fatal cancers in the simvastatin-ezetimibe cohort as compared with the simvastatin cohort.

Finally, one must be careful about the new American Heart Association/American College of Cardiology guidelines, which relied solely on randomized controlled trials (RCTs) that studied high-risk patients, many of whom already had ATD. It is not clear that the results of those RCTs are applicable to the general population. I suggest treating patients in the at-risk (for ATD) population and to treat them to low-density lipoprotein levels that are associated with maximum plaque stabilization/regression[5] or alternately so that their risk factors are in line with those of the low-risk population.

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1. Feeman WE. Statin-fibrate combination therapy. *Am J Cardiol*. 2008;101:1521.
2. Feeman Jr WE. Prediction of angiographic stabilization/regression of coronary atherosclerosis by a risk factor graph. *J Cardiovasc Risk*. 2000;7:415-423.
3. Canner PL, Berge KG, Wenger NK, et al. Fifteen year mortality in Coronary Drug Project patients: long-term benefit with niacin. *J Am Coll Cardiol*. 1986;8:1245-1255.
4. Rossebø AB, Pederson TR, Boman K, et al; SEAS Investigators. Intensive lipid lowering with simvastatin and ezetimibe in aortic stenosis. *N Engl J Med*. 2008;359:1343-1356.
5. Feeman Jr. WE. LDL lowering: Evidence for a plaque non-progression threshold. *Int J Cardiovasc Res*. 2013;2:5.