ANNE C. GOLDBERG: So where we are now is that in patients who are still having high LDL levels on maximally tolerated statin, often plus Ezetimibe, you want to take the very high risk patients and treat them further. So for example, a patient with severe coronary disease, high risk patient who has, say, had an event relatively recently-- acute coronary syndrome, MI, bypass surgery-- they're on a high dose statin. They're on Ezetimibe. The LDL is above 70.

That would be somebody that I would consider for a PCSK9 monoclonal antibody, because that person is still at high risk. We know that lowering LDL is better. In the clinical trials [INAUDIBLE], and in Odyssey outcomes, people were treated down into the ranges of LDLs 20 to 30. And there were clear outcomes measures in terms of major coronary events that were better.

So that is the kind of person I would consider treating based on the current clinical trial data. And in fact, when you start looking at the intravascular ultrasound studies, you start seeing some improvement in atheromas when you get down into that very low level, 20 to 30. I think that most of these people definitely need to be below 50 as a minimum in terms of trying to decrease progression of their coronary disease.

The other persons who are very high risk would be people with genetic hypolipidemias. And so for example, there are a lot of patients with heterozygous FH. And you put them on high dose statin-- 80 [INAUDIBLE], 40 [INAUDIBLE]. You add Ezetimibe. Their starting LDLs are somewhere in the 250 to 400 range. They're at very high risk. And when you get them down to an LDL of, say, 150, they're still at high risk, especially those who have coronary disease already. Those are people who are clear candidates for PCSK9 monoclonal antibody. There's no question, the number needed to treat in a patient with an LDL of 160 familial hypocholesterolemia, known coronary disease-- the number needed to treat is less than 10. And so clearly there is a significant benefit to that population of patients.