SPEAKER 1: Yeah, I mean a very interesting question concerned with how far we should go with a PCSK9 inhibitors is that, should one, should the cardiologist, should the interventionalist who just finished doing the procedures. They're on high intensity statins at the moment. And the LDL is 69.

So should he or she decide, OK that's just fine. You've hit the target. Off you go, we'll see you in a year. The answer is no, because clearly if this patient has a prior history of recurrent events, and progression of disease, that's a red flag. One has to say, what can we do to modify that progression. We've got to think broadly. We've got to think broadly.

PCSK9 inhibition is one of the options. Because we know from the trials, not yet from the guidance, that coming even lower, maybe down to 25, would be a reasonable target. But what about other options? They are-- there are around the corner different ways of interference RNA to modify PCSK9. Those trials are underway as well. So it may not just be the monoclonals.

Secondly, there's the whole issue do we alter inflammation? We know about CANTOS and canakinumab. That's quite an expensive strategy at the moment. And then in terms of these vascular patients, the end event are atherothrombotic.

And as a co-chair of Compass, I would have to raise the question about the combination of very low dose rivaroxaban and aspirin. Because of the impact not only on MACE, but on cardiovascular death, and all cause death, and the 42% reduction in stroke. So I think that we have a very interesting and challenging time. And that these are not alternatives.

I can see in the future, we may be wanting to target more than one strategy, just as we do now. We don't target hypertension or LDL lowering, we do both. With diabetes, we're not simply modifying HbA1c. So I think in the future, we're going to be looking at atherothrombotic risk and LDL lowering as ways of modifying future risk. It's got to be expensive, but that may be the option.