SPEAKER: Sure. That's interesting. We are entering clinical practice. And what I see is-- let's say a patient that has survived a myocardial infarction, has undergone success for PCI, and is stable with the classical secondary prevention after discharge.

And when we see such a patient at some later stage-- let's say clinical control-- we see many patients who are actually very motivated to adhere to therapy, very interested in optimizing both therapies and lifestyle, and many patients who come back asking, doctor, what else can I do? What can I do to optimize my situation?

And then we, of course, enter immediately the discussion about cholesterol levels. Let's say such a patient actually has achieved the goals. Target of LDL is just around 1.8 millimoles per liter. And let's say that patient is on the high-dose statin.

It is almost obligatory to discuss with the patient an extra-- an additional reduction of LDL. And there [INAUDIBLE] comes in the discussion and PCSK9 inhibition comes in. Let's say that patient has already [INAUDIBLE], which is not unusual. We combine high-dose statin and [INAUDIBLE].

I feel that I owe the patient and his and her relatives also to pinpoint the utility of PCSK9. Perhaps portraying an additional, let's say, 60% LDL reduction and also even mentioning the scientific evidence-- because many people are actually aware of that, and scientific evidence is now available to the public-- to pinpoint that additional benefits can be derived from this.

So that last patient I put on PCSK9 fully understood the concept, adhered to, let's say, triple cholesterol therapy, and at the next control no disadvantage from that.