Statins (HMG-CoA reductase inhibitors) have played the most significant role in lowering low-density lipoprotein (LDL)-cholesterol and, in the era of strong statins, the more LDL decreases the more coronary events are reduced. Stabilization of vulnerable coronary plaque is thought to be the main mechanism by which statins reduce coronary events, as well as their other pleiotropic effects. Further, animal studies have revealed that statins can decrease the lipid core and inflammatory cell infiltration, both of which are factors in vulnerable plaque, increase the thickness of the fibrous cap and reduce the size of atherosclerotic plaque.

In the ESTABLISH trial, coronary events in both primary and secondary prevention.

In addition, IVUS may be able to demonstrate structural change of coronary plaques induced by statin, the change that animal studies have demonstrated, as described above.

Angioscopy, which has especially evolved in Japan, can show detailed characteristics of the luminal surface, including color-coded information. The surface color of coronary plaque is yellow or white. Yellow plaque represents plaque with a thin fibrous cap through which lipid accumulation can be seen. Thus the color of coronary plaque can be graded according to its intensity and the grade may be negatively related to the thickness of the fibrous cap; for example, the thinner fibrous cap of atheromatous plaque has a higher intensity of yellow color, whereas white plaque may indicate a thick fibrous cap with a lipid core that cannot be seen, a fibrous plaque without a lipid core or simply intimal hyperplasia. One limitation of angioscopy is that information obtained from the images (eg, plaque color or geometric information) is difficult to objectively and quantitatively evaluate compared with IVUS. The intensity of yellow color has been visually categorized into several grades or scores. Takano et al. demonstrated using angioscopy that 12-month atorvastatin administration significantly decreased the yellow score of coronary plaque, with a final average LDL-cholesterol of 99 mg/dl, and that the change in the yellow score of the plaque was linearly correlated to the change in LDL.

In the study published in the current issue of the Circulation Journal, Hirayama et al. serially evaluated the effect of atorvastatin on coronary plaque, using both angioscopy and IVUS at 3 time points (baseline, 28 weeks and 80 weeks), whereas IVUS or angioscopy was performed at only 2 time points (baseline and follow-up) in the previous studies. They categorized coronary plaque color into 6 grades. Initial interobserver reproducibility for the interpretation of plaque color was quite low compared with the previous study by
Takano et al; however, 2 independent reviewers of plaque color trained for color grading on more than 800 coronary plaques until they reached the agreement under the blinded process. In their results, angioscopy demonstrated that atorvastatin might increase the thickness of the fibrous cap in the initial 28 weeks. After 28 weeks, angioscopy could not demonstrate intraplaque qualitative changes, probably because the fibrous cap was too thick. IVUS revealed that plaque volume was continuously reduced over 28 and 80 weeks, with preservation of lumen volume and decreased external elastic membrane volume, which may indicate reverse remodeling. Qualitative change in coronary plaque after 28 weeks may be evaluated by IVUS RF analysis, as previously reported, or by optical coherent tomography.

In summary, serial observation of coronary plaque using both angioscopy and IVUS provides valuable information about the mechanism of stabilization of vulnerable coronary plaque by a strong statin.

References