Glagovian remodelling, plaque composition, and stenosis generation

Necropsy and intravascular ultrasound (IVUS) studies in vivo show that while the angiogram is good at detecting high grade stenosis it is very insensitive for demonstrating the actual extent of atherosclerosis. Segments of an angiographically normal coronary artery can harbour many occult plaques. There are two reasons for this insensitivity. The first is that behind the plaque itself the media may vanish allowing the plaque to bulge into the adventitia rather than toward the lumen. In extreme cases the internal elastic lamina breaks allowing the plaque to be extruded from the artery wall. The second process is that of arterial remodelling (compensatory dilatation) described by Glagov. This process and associated compensatory dilatation varies widely from plaque to plaque. Within one coronary artery there may be a plaque with no remodelling, while in another segment of the same artery there may be a 100% increase in the artery cross sectional area. Remodelling is plaque specific rather than patient specific.

**Figure 1** A plaque five days after thrombolysis. There is a cavity containing some residual thrombus which communicates with the arterial lumen via a hole in the plaque cap. The lipid in the core has been washed out in vivo to create the cavity. Reproduced from Davies MJ. Atlas of coronary artery disease, with permission of the publishers Lippincott-Raven.

**Plaque disruption**

The study in this issue of Heart by von Birgelen and colleagues casts some light on what plaque factors are associated with positive compensatory remodelling. It is now soundly established that culprit plaques which undergo an acute episode of disruption have certain key features. These include a large lipid core, a high macrophage content, a low smooth muscle content, and a thin cap. The lipid core is a space in the collagenous tissue of the intima containing cholesterol rich lipid debris. Core size can be determined by IVUS, particularly if the lipid within the core has been displaced by the entry of blood.
from the lumen via a tear in the cap. The core cavity is particularly striking after lysis has removed thrombus (figs 1 and 2).

Mechanisms of remodelling
The study reported in this issue shows that core size as defined by IVUS of culprit lesions for acute coronary syndromes is positively associated with previous positive remodelling (compensatory dilatation) of the artery at the site of the plaque. Of the 51 lesions studied 36 had compensatory enlargement (71%) while 15 (29%) did not. The core size was significantly larger in the group where compensatory enlargement had occurred. These findings have the important implication that many vulnerable plaques at risk of future acute thrombotic events will have undergone sufficient remodelling to be angiographically invisible. This fits well with the angiographic data showing that the thrombi which cause infarction often do not develop at sites of previous high grade stenosis. More important, however, is the suggestion that core size and positive remodelling are linked. The link may be causal. Circumferential wall stress across the plaque with a large lipid core is altered. The core, being soft and deformable, cannot carry this stress which has to be redistributed to adjacent areas of the vessel wall.

Previous work on stress distribution using finite element analysis concentrated on the increased forces exerted on the plaque cap, but behind the plaque force is also increased which may potentiate stretching of the tissue and therefore remodelling. The association of a large lipid core and positive remodelling may be indirect. Remodelling of the arterial media depends on the complex interaction of growth factors and metalloproteinases concerned both with smooth muscle cell movement and degradation of connective tissue matrix. Large lipid cores are associated with high macrophage densities and these cells are major stimulators and producers of metalloproteinases. It may therefore be that core size is simply a surrogate for plaques with high macrophage activity, and it is this factor which enhances remodelling.

M J DAVIES
St George’s Hospital Medical School, BHF Cardiovascular Pathology, Cranmer Terrace, London SW17 ORE, UK


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M K DAVIES
A HOLLMAN
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M J DAVIES

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