cholesterol <2.59 mmol/l and non-HDL cholesterol <3.37 mmol/l. Before statin treatment, relative concentrations of atherogenic particles matched those of ACC-recommended targets well. After 16 weeks of statin therapy, however, 0.9 g/l apolipoprotein B corresponded to 2.14 mmol/l LDL cholesterol in patients with triglyceride levels <2.26 mmol/l, and to 1.84 mmol/l LDL cholesterol and 2.70 mmol/l non-HDL cholesterol in patients with elevated triglyceride levels. These concentrations were closer to the targets identified by the ACC and AHA as 'reasonable' for all patients with coronary heart disease and other atherosclerotic vascular disease (i.e. LDL cholesterol <1.81 mmol/l and non-HDL cholesterol <2.59 mmol/l), than to ACC-recommended targets.

To attain an apolipoprotein B concentration of 0.9 g/l in patients treated with statins, therefore, intensive therapy might be required to achieve 1.81–2.07 mmol/l LDL-cholesterol and 2.59 mmol/l non-HDL-cholesterol levels.

Original article Ballantyne CM *et al.* (2008) Statin therapy alters the relationship between apolipoprotein B and low-density lipoprotein cholesterol and non-high-density lipoprotein cholesterol targets in high-risk patients: the MERCURY II (measuring effective reductions in cholesterol using rosuvastatin therapy II) trial. *J Am Coll Cardiol* **52:** 626–632

Elevated thromboxane A_2 level independently predicts 'no-reflow' after PCI

Platelet aggregation is thought to be involved in the pathogenesis of the 'no-reflow' phenomenon, in which tissue perfusion is not improved after restoration of arterial patency. Niccoli *et al*. hypothesized that a high plasma level of thromboxane A_2—a mediator of platelet activation—could be associated with no-reflow after primary percutaneous coronary intervention (PCI).

Their single-center study included 47 patients with ST-segment elevation myocardial infarction who were scheduled to undergo primary PCI. After arterial recanaliation, angiographic no-reflow (defined as Thrombosis In Myocardial Infarction flow grade ≤2, or flow grade 3 with myocardial blush grade of <2) occurred in 46.8% of patients. Electrocardiographic no-reflow was defined as a reduction in the ST-segment elevation value of 50% or less from baseline, and occurred in 44.7% of patients. Plasma levels of thromboxane A_2 were higher, on average, among patients with angiographic no-reflow, and electrocardiographic no-reflow, than in those with normal perfusion (17.74 pg/ml versus 3.91 pg/ml, $P = 0.005$, and 19.58 pg/ml versus 3.99 pg/ml, $P = 0.001$, respectively). Multivariate analysis revealed that plasma level of thromboxane A_2 was an independent predictor of both angiographic no-reflow ($P = 0.04$) and electrocardiographic no-reflow ($P = 0.013$). The incidence of no-reflow under both criteria increased with rising tertiles of thromboxane A_2 level ($P < 0.01$ for trend). The authors concluded that thromboxane A_2 could represent a novel therapeutic target in patients who undergo primary PCI.

Original article Niccoli G *et al.* (2008) Plasma levels of thromboxane A2 on admission are associated with no-reflow after primary percutaneous coronary intervention. *Eur Heart J* **29:** 1843–1850