

was often not necessary and a stent could be directly implanted ("direct stenting"). In the conventional group, most patients needed predilatation.

Patients randomised to aspiration had improved myocardial perfusion and clinical outcome compared with the conventional group.^{1,2} However, is there evidence to suggest that the higher rate of direct-stenting in the aspiration group has augmented these results?

As shown by Loubeyre and colleagues,³ direct stenting can be safe and effective in selected patients undergoing primary percutaneous coronary intervention (PCI). However, in unselected patients with ST-elevation myocardial infarction (STEMI), direct stenting has never been shown to improve myocardial perfusion or clinical outcome compared with predilatation.⁴ Direct stenting in patients without adequate flow leads to the risk of undersized stenting, which is the most important predictor of restenosis. This problem is illustrated by a trial comparing direct stenting with predilatation in unselected STEMI patients: in-stent restenosis rate at 1-year angiographic follow-up was significantly higher in the direct stenting group.⁴

Finally, the use of predilatation in TAPAS was not limited to patients randomised to conventional PCI. Predilatation was done in 207 of 502 (41.2%) of the patients randomly assigned aspiration. There were no differences between patients with and without predilatation in rates of cardiac death (7/207 [3.4%] vs 8/295 [2.7%], respectively; $p=0.67$), nor in cardiac death or non-fatal reinfarction (10/207 [4.8%] vs 16/295 [5.4%], respectively; $p=0.77$). This finding indicates that predilatation is not associated with impaired clinical outcome.

Jaewon Oh and colleagues raise the important issue of the value of platelet inhibitors during primary PCI. In TAPAS, all patients were pretreated with clopidogrel, aspirin, and heparin. During the procedure, abciximab was given routinely to 469 of 502 (93.4%) in the thrombus-aspiration

group and 452 of 503 (89.9%) in the conventional PCI group ($p=0.12$).¹

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Secondary prevention of stroke

Geoffrey Donnan and colleagues, in their Seminar on stroke (May 10, p 1612),¹ mention cholesterol reduction with statins among the proven secondary prevention strategies. However, although statin treatment might be effective in the prevention of stroke in patients with known coronary-artery disease but without a history of cerebrovascular disease, its role in the secondary prevention of stroke raises some serious concerns.

Indeed, the SPARCL study² found that, compared with placebo, the use of 80 mg atorvastatin per day in patients who had a stroke or transient ischaemic attack was associated with a non-significant 13% relative risk reduction of non-fatal stroke during a 5-year follow-up, without improving

survival, and with a 66% increase in the relative risk of haemorrhagic stroke among patients receiving high-dose statin. Furthermore, a review³ found that, in the secondary prevention of cerebrovascular events, statin therapy decreases the occurrence of ischaemic stroke but that this effect is counterbalanced by an increase in the occurrence of haemorrhagic stroke.

Lower LDL cholesterol concentrations with or without statin treatment have also been shown to be strongly and independently related to a higher risk of symptomatic haemorrhagic transformation after ischaemic stroke thrombolysis.⁴

Finally, an inverse relation has been shown between stroke severity and cholesterol concentrations.⁵ Higher total serum cholesterol favours the development of minor stroke, and, owing to selection, major strokes are more often seen in patients with lower total serum cholesterol concentrations.⁵ Therefore, it is also plausible that statin therapy might decrease only milder stroke subtypes.

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