Letters to the Editor
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Safety and efficacy concerns regarding elective coronary artery surgery in patients with prior coronary stents

Recent data have raised concerns about the safety of drug-eluting stents.1 Stents are used in over 70% of percutaneous coronary interventions (PCIs) because they reduce the risk of acute major complications of PCI and long-term restenosis. Despite the low procedural morbidity afforded by stenting, coronary artery bypass graft (CABG) surgery offers significantly greater freedom from angina, repeat revascularization, myocardial infarction, stroke, and death in the treatment of multivessel ischaemic heart disease.2 Stenting has, however, become first line treatment in increasing numbers of patients that are surgical candidates. Many of these patients eventually require coronary artery surgery: in the UK over 5% of patients undergoing CABG have a history of previous PCI.3

Surgeons suspect that prior stenting may have a negative impact on outcome after elective coronary surgery for several reasons. In-stent restenosis is associated with a higher risk of early venous graft failure.4 The presence of coronary stents means that grafts are anastomozed more distally, and endarterectomy may be required. Anti-platelet medication adds to increased morbidity and potential mortality, resulting from excess post-operative bleeding, and stopping this medication has been associated with stent thrombosis in off-pump CABG patients.

Pathophysiological processes associated with stenting may adversely affect surgical outcomes.5 Stenting causes prolonged endothelial dysfunction and local and systemic inflammatory syndromes, more profound than after balloon angioplasty, because of persistent radial mechanical strain, vessel wall rupture, and the presence of an intravascular foreign body. The local inflammatory response is stimulated by disruption of the coronary endothelium, and is characterized by a florid macrophage response that does not occur after balloon angioplasty. Six months after PCI endothelium-dependent vasomotor function has been shown to be more abnormal in stented coronaries compared with those undergoing balloon angioplasty. Two years after stenting a chronic inflammatory response surrounds the stent, characterized by non-occlusive mural thrombi. Drug-eluting stents induce this inflammatory response, most prominently at the edges of the stent, but also where endothelialization is delayed leaving the intima exposed to metal within the stent. Troponin release is higher in patients receiving stents compared with those undergoing angioplasty alone, and is associated with increased peri-procedural mortality, rates of myocardial infarction, and repeat revascularization.

UK data suggest that patients with previous PCI had higher mortality after CABG than patients without prior PCI, even when patients undergoing salvage CABG for failed PCI are excluded from the analysis.6 Unfortunately, the timing of PCI in 90% of the remaining patients, and the proportion of PCIs that involve stenting are unknown. We propose a prospective longitudinal multicentre study to evaluate the effect of previous stenting on clinical outcomes after elective coronary surgery, using peak oxygen consumption (VO2) as the primary outcome. For 80% power to detect a difference of 1.5 mL/kg/min in VO2 at the 5% two-tailed significance level, we would need 176 patients in each group. Secondary outcome measures include major adverse clinical events, quality-of-life adjusted years, and perfusion imaging with a 5-year follow-up.

References
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Addition of milk prevents vascular protective effects of tea

The paper of Lorenz et al.1 concludes that milk may counteract the favourable health effect of tea on vascular function. Impaired vasodilation is one of the mechanisms underlying hypertension. We agree with the authors in that all dietary components should be considered, and that potential interactions should be examined when interpreting effects.

We, however, would like to address two major aspects. That there was a significant positive effect of a large serving of tea on vasodilation is plausible considering what we know about the biological function of tea ingredients and phenolic compounds in other foods. It was, however, minor in absolute terms, just 3.5% above the control response. It is open how long the effect would last and what it could mean for the vasodilatory response throughout a whole day.

The authors then assumed that the inhibitory effect (or rather non-stimulatory) effect of tea if consumed together with milk might be caused by casein complexing the tea catechins. If such a complex is formed in the intestinal tract, would catechins remain complexed once casein is broken down to amino acids and peptides? We doubt this. Casein is a well-digestible protein. But casein forms a curd in the stomach. Release into the small intestine is delayed in comparison with other proteins, like whey proteins. When milk was fed to miniature pigs, whose gastrointestinal

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