Does prior PCI increase the risk of subsequent CABG?

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This editorial refers to ‘Impact of previous percutaneous transluminal coronary angioplasty and/or stenting revascularization on outcomes after surgical revascularization: insights from the imagine study’ by S. Chocron et al.,† on page 673

Chocron and colleagues have reported that prior percutaneous coronary intervention (PCI) leads to a worse outcome in patients subsequently undergoing coronary artery bypass graft (CABG).¹ In view of the fact that around one-third of patients with multivessel disease treated with bare metal stents will require re-intervention within a few years,² this conclusion is potentially worrying and raises several questions. (i) Is the conclusion justifiable? (ii) Are the findings consistent with other studies in the literature? (iii) If real, what are the likely pathophysiological mechanisms? (iv) Will the findings be different drug-eluting stents? (v) What are the clinical implications for patients and the economic implications for health services?

Is the conclusion justifiable?

The obvious weakness of the study of Chocron et al.¹ is that it is a re-analysis of the primary end-point of the IMAGINE trial according to whether the patients had undergone PCI prior to CABG, a question which the IMAGINE trial was not designed to answer. Rather, IMAGINE was a randomized trial of the angiotensin-converting enzyme (ACE) inhibitor quinapril in 2553 subjects with preserved left ventricular function after CABG, which concluded that there was no difference in time to occurrence of the primary end-point (a composite of cardiovascular death, revascularization on account of disease treated with bare metal stents will require re-intervention within a few years,² this conclusion is potentially worrying and raises several questions. (i) Is the conclusion justifiable? (ii) Are the findings consistent with other studies in the literature? (iii) If real, what are the likely pathophysiological mechanisms? (iv) Will the findings be different drug-eluting stents? (v) What are the clinical implications for patients and the economic implications for health services?

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events [HR = 2.28; (95% CI 1.38–3.59); \( P < 0.001 \)]. In a sub-
sequent propensity-matched group based on 13 pre-operative 
risk factors, logistic regression analysis again confirmed multiple 
prior PCIs to be associated with increased in-hospital mortality 
(HR = 3.01; \( P < 0.01 \)) and major adverse cardiovascular events 
(HR = 2.31; \( P < 0.01 \)). In a subset of 621 of the same group of 
patients with diabetes there were almost identical findings.7

If real, what are the likely 
pathophysiological mechanisms?

An explanation for why prior PCI should increase the risk of sub-
sequent CABG is not immediately apparent. Obvious explanations 
such as differences in age, gender, ventricular function, extent of 
coronary artery or other vascular disease (often less severe in 
the prior PCI group), or completeness of appropriate revasculari-
zation are not supported by the data.

While the prior PCI group often present with more unstable 
symptoms, as in the study of Chocron and colleagues,1 it is not 
clear from the literature whether the ‘new culprit’ lesion is 
related to the previous PCI or de novo remote disease. Other 
possibilities might be that prior stents encourage more distal 
bypass grafting with less favourable graft run off, or may compro-
mise collateral blood flow.8 While it is well recognized that 
drug-eluting stents (DES) cause dysfunction of the endothelium 
both overlaying the stent and further downstream,9 is it possible 
that bare metal stents (BMS) also compromise endothelial func-
tion overlaying the stent which is exaggerated by changes in the 
inflammatory and coagulation status precipitated by cardiac 
operations?

Will the findings be different 
with drug-eluting stents?

A potential criticism of the studies by Chocron, Hassan, and 
Thielmann is that PCI may have been suboptimal as there was a 
relatively low use of DES in comparison with BMS. However, as 
several meta-analyses have consistently demonstrated that while 
DES reduce the risk of restenosis in low-risk coronary lesions 
they do not reduce the risk of mortality or subsequent myocardial 
infarction,10 it is counter-intuitive to believe that they will improve 
results post-CABG.

There is a further concern with DES: the FDA have warned that 
their use is ‘associated with increased risks of both early and late 
stent thrombosis, as well as death and myocardial infarction’,11 
DES impair endothelialization, leaving a potentially prothrombotic 
substrate within the vessel,12 and leave a further conundrum for 
the surgeon in terms of control of antiplatelet medication and 
whether to perform bypass grafts to a coronary vessel with a 
DES without critical restenosis in patients who have multivessel 
disease. These clinical concerns are compounded by cost impli-
cations; not only are DES significantly more expensive than BMS, 
but new recommendations that patients remain on clopidogrel 
for at least a year, and possibly indefinitely, add significantly to 
overall costs.

What are the clinical implications for patients and the economic 
implications for health services?

In economic terms there is already strong evidence that stenting in 
multivessel coronary artery disease is not a cost-effective treat-
ment,13 and the studies of Chocron and others will add to these 
concerns. However, the major implication of the finding that 
prior PCI increases the risk of subsequent CABG is to add ammu-
nition against the spurious belief that CABG can always be safely 
defered in favour of an initial strategy of PCI in multivessel 
disease, where several large registries already show a consistent 
survival advantage for CABG over PCI in propensity-matched 
patients.2,10 These dual observations should be carefully con-
sidered in patients with multivessel disease who are likely even-
tually to require CABG, and underline the importance of the 
proposed interventions being discussed by a multidisciplinary 
team including a surgeon rather than by the individual 
cardiologist.10,14

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**Post-traumatic ventricular septal defect**

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A 20-year-old male was admitted to the emergency department with a stab wound in his chest in the cardiac region. Given the haemodynamic instability and suspected cardiac tamponade, urgent thoracotomy was performed with drainage of haemopericardium and cardiac surgery was performed immediately, during which right ventricular free wall laceration was sutured under extracorporeal circulation (Panel A). Before discharge, the patient underwent routine transthoracic echocardiography that revealed defect in the distal third of the interventricular septum, with sharply demarcated edges, 4–5 mm wide, and left to right shunt flow (Panel B). These findings were confirmed by magnetic resonance imaging (MRI) (Panel C and D). Given the asymptomatic course of the patient and size of the shunt, which has been assessed as non-significant ($Q_p/Q_s \approx 1.4/1$, according to MRI), a conservative approach was proposed with the possibility of a future catheter-based treatment. At the 3-month follow-up the patient was asymptomatic.

Panel A. Suture of right ventricular free wall.
Panel B. Transthoracic echocardiography. Apical four-chamber view. Left to right shunt in the distal third of the interventricular septum (white arrow).
Panel C. Magnetic resonance imaging in the short axis showing the ventricular septal defect (white arrow).
Panel D. Magnetic resonance imaging in the long axis showing the ventricular septal defect with shunt flow (white arrow).