

Furthermore, in their experience, reperfusion catheter usage resulted in a longer delay in the initiation of CPB and subsequently, worse outcomes. They state that the use of reperfusion catheters may lead to a false sense of security, potentially resulting in higher rates of postoperative myocardial infarction and early mortality. Non-use of perfusion catheters was also preferred by us.

In contrast to most of the reports listed in Table 6, none of our patients were admitted with acute ST-segment elevation infarction. This is likely to be one cause of the higher operative mortality in the other series.

Twelve (12%) of the patients in our study suffered postoperative myocardial infarction when compared with 1% for the rest of 10 192 patients. However, 11 of them had preoperative ST-segment elevation. Nevertheless, it denotes that there is potential for improvement. There are no reliable data on the time elapsed from the start of trouble in the catheterization laboratory until the decision to operate was taken. We do suspect that the time in many of these cases had been so long that irreversible myocardial damage had occurred. The risk of myocardial infarction is proportional to the duration of myocardial ischaemia. The dilemma for the invasive cardiologist is the understandable struggle over potentially successful salvage with further percutaneous techniques vs the time limit for inducing irreparable myocardial injury beyond the possibility of complete recovery. Yet, and without doubt, to obtain the best surgical outcome, the limit of irreversibility must not be crossed. The timing of the operative intervention is evidently crucial to minimize ischaemic time and thereby the reduction of infarctions in number or at least in size.

We used antegrade crystalloid cardioplegia, and it remains open whether blood cardioplegia and/or retrograde delivery might have reduced the myocardial infarction rate. Many would consider these measures as mandatory in acute coronary occlusion situations. The answer is that blood cardioplegia was not available in our institution, and catheterization of the coronary sinus was not integrated in our routine during the study time.

It is noteworthy that no patient needed revision surgery for postoperative mediastinitis despite the fact that many of them were in a critical state, and hence the start of the operations was done under less-than-optimal disinfection conditions. After circulatory and cardiopulmonary stabilization was achieved, disinfection was handled with rewashing, irrigation and additional antibiotics, leading to complete success in all.

In the literature, there are few reports on iatrogenic Type A aortic dissection caused by cardiac catheterization procedures. The current study includes 4 patients, of whom all survived. Recently, a 50% operative mortality in 12 patients with catheterization-associated Type A aortic dissection has been reported [9]. The patients were collected from 135 262 who underwent cardiac catheterization from 1995 to 2010. The authors point out that the high mortality rate is discouraging when considering that the indication for the diagnostic procedure was elective in the majority of patients. We were fortunate to have no deaths, but the number of patients with iatrogenic aortic dissection is so low in our study that comparison is not really justified.

However, diagnostic cardiac catheterization resulted in 11 more patients with life-threatening complications. As summarized in Table 2, their haemodynamic situation was at least as grave as for the post-PCI patients. Two of them were massaged onto CPB after wire-induced left main artery dissection with occlusion. It is as expected that intima tear with vessel lumen occlusion located in the left or right coronary ostium induced severe haemodynamic instability.

Three of our patients in the diagnostic group had perforations of the left ventricle. It occurs infrequently without a myocardial infarction. Echocardiographically guided pericardiocentesis is the primary measure [10]. If haemodynamic stability is not restored, surgery is necessary as was the case with our patients.

One other difficult complication in the diagnostic catheterization group was that of the patient with fractional flow reserve measurement of stenotic LAD. A fractional flow reserve-specific wire became stuck, and the manipulations resulted in a localized dissection with threatened anterior wall infarction, which was why emergency surgery was undertaken. The case demonstrates that even this type of invasive investigation may result in a major complication.

In conclusion, life-threatening complications associated with cardiac catheterization procedures requiring emergency operative action are infrequent. When they do occur, it is possible to obtain a low mortality (1%) rate by immediate operative intervention, avoiding any delays in care delivery.

Conflict of interest: none declared.

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eComment. Management of percutaneous coronary intervention complications

Authors: Georgios Dimitrakakis, Ulrich O. von Oppell and Ahmed A. Azzu
Department of Cardiothoracic Surgery, University Hospital of Wales, Cardiff, UK
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We read with great interest the article by Almdahl *et al.*, regarding the cardiac surgical outcome of patients with complications of percutaneous coronary intervention

(PCI) [1]. Complications of PCI include perforation (of the coronary artery or ventricle), retained wires, dissection, bleeding (subepicardial or not), tamponade and myocardial ischaemia or infarction [1, 2].

There is an angiographic classification of three types of coronary artery perforation post PCI according to Ellis *et al.* [3]. In Type I, the angiographic findings are consistent with extraluminal crater with no contrast extravasation. In Type II the contrast extravasation is limited to 'blushing' in the myocardial or epicardial fat. In Type III there is a contrast extravasation through frank (>1 mm) perforations or Type III 'cavity spilling' extravasation into either the left ventricle, the coronary sinus, any of the cardiac chambers, or the pericardium [3].

This classification is quite important as a useful tool for the diagnosis, management and prognosis of this potentially severe complication. Type I perforations are associated with the lowest incidence of tamponade (8%) with no reported incidence of myocardial infarction or mortality. As a result, the vast majority (85%) of Type I are treated conservatively. Type II perforations show a higher incidence of tamponade and myocardial infarction (13% and 14%, respectively) with no reported mortality, and conservative treatment is successful in 90% of cases. In contrast, Type III perforations have a high morbidity with a much higher incidence of tamponade and myocardial infarction (63% and 50%, respectively) and conservative treatment has been reported to be successful in only 44% of cases, with a mortality independent of treatment in 19% of cases [2, 3].

Shimony *et al.*, in their recent systematic review and meta-analysis (16 studies, 197 061 PCIs), reported that the incidence of PCI complications was 0.43%. Furthermore, the tamponade rates were 0.4%, 3.3% and 45.7% for patients with Ellis classes I to III coronary artery perforations, respectively [4].

It is important to take into consideration the fact that currently (to the best of our knowledge), no established protocol guidelines exist regarding management strategies for PCI complications [2-4]. According to the suggested algorithm of Shimony *et al.*, which is consistent with our department's practice as well, all these patients should be under continuous monitoring and assessment (sequential echocardiography studies). In the case of haemodynamic instability (mainly Ellis class III), the patient is either initially treated conservatively under the care of the cardiologists [pericardiocentesis (definitive or bridging treatment for open surgical intervention),

heparin reversal, discontinuation of IIb/IIIa inhibitors/bivalirudin, prolonged balloon inflation for 5 to 15 minutes, embolization, polytetrafluoroethylene-covered stent etc], or surgically by the cardiac surgeons [4].

Surgical management is not standardized and depends on the surgical anatomy, patient co-morbidities and clinical condition. Surgical reports include simple suturing of the perforation, ligation of bleeding vessels, pericardial patch application, surgical glues, CABG (on or off CBP) with and without endarterectomy, stent removal, or vein patch [1-5].

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