Keywords: Clopidogrel; Antiplatelet therapy; Coronary artery bypass grafting; Blood loss; Aprotinin

As Shrivastava and Akowuah note, in our observational study [1] we only recorded clopidogrel use within three days prior to surgery indiscriminately if it was stopped 1, 2 or 3 days prior to surgery. However, onset of action can be expected within hours (especially if a loading dose is administered) and once established in therapy effects are present for the lifespan of platelets. We therefore appreciate the point if the mean time clopidogrel has been stopped before surgery in the no clopidogrel group was less than 7 days. Although this was not recorded for study purpose (which is a limitation) clinical practice allows to suggest that the majority of patients in the no clopidogrel group have had no clopidogrel at all. In both groups a comparable number of patients were on aspirin (37.3% in the clopidogrel group vs. 41.5% in the no clopidogrel group, P=ns).

A recent meta-analysis of randomized clinical trials [2] points out clearly, that concerns about perioperative myocardial infarction (early graft closure) linked to aprotinin use are limited in evidence. This is in accordance to our clinical experience. It has been recognized early that aprotinin is also efficient for the reduction of blood loss after CPB in a low-dose protocol [3]. We use it routinely in the pump-prime dose (two million KIU) also in patients who are not at an increased risk of bleeding supplemented by the high dose protocol in patients are at risk.

As a next point Shrivastava and Akowuah presume a possible bias due to different practice among the surgeons in our unit. However, this can be ruled out by the fact that in the respect of interest (stopping of clopidogrel before surgery) our surgical group is very homogenous. Additionally, the continental system of a surgical unit in a university hospital differs from the consultant-system practiced in UK.

Finally we agree that further clinical trials are welcomed in this field, especially including specific measurements of platelet function. In addition, factors reducing perioperative bleeding complications (e.g. aprotinin and other antifibrinolytics) have to be evaluated for patients operated under ongoing clopidogrel therapy.

References


Letter to the Editor

Coronary graft flow and cardiac pacing modalities: the importance of the cardiac resynchronisation trials

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D’Ancona et al. present important information on coronary graft flow and optimal pacing techniques [1]. Optimal cardiovascular haemodynamics in the marginal postoperative patient may enhance graft patency. This has previously been shown in patients who require intra-aortic balloon pump support [2].

A number of important points, however, need clarification.

First, no mention of pre or postoperative intra-ventricular conduction defects are mentioned. Patients with left bundle branch block are significantly better cardiovascularly after the specific pacing of the lateral aspect of the left ventricle [3]. This has been shown in the cardiac resynchronisation trials [4,5]. However D’Ancona et al. just mention ‘two ventricular pacing wires were placed in each patient’, making no mention of the anatomical placement of the electrodes.

Second, recommending an optimal A-V delay of 175 ms is potentially non safe practice. Personnel experience of altering A-V delay in A-V sequential paced patients, while monitoring cardiac indices via Swan-Ganz catheter readings shows that inter patient variability in optimal A-V pacing delay is quite high. This fact is demonstrated by their figure 1, which shows as the A-V delay is increased from 100 to 175 ms, at least six of the ten patients had
decreased mean graft flow! This fact has already been reported in the literature [3].

References


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Reply to the Letter to the Editor

Reply to Poullis and Shackcloth
Intraoperative graft flow measurement and pacing modalities

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We have read with interest Dr Poullis letter and we thank him for his timely comments. We are well aware that, as specified by Dr Poullis, and demonstrated in previous literature, optimal cardiovascular hemodynamics in the marginal postoperative patient may enhance graft patency or better improve myocardial perfusion. In reality our study, differently from any other published in the literature, including the resynchronization trials mentioned by Dr Poullis, did not focus on the mere hemodynamic parameter changes resulting from different pacing modalities but did center on the immediate changes in intraoperative coronary graft flows as measured by transit time technique and as resulting from different pacing techniques.

Ventricular pacing wires were routinely placed in the right ventricle and right atrium and graft flows were measured during DDD and VVI pacing and at different A-V delays. Patients with preoperative atrio-ventricular or intraventricular conduction anomalies were specifically excluded from the study.

Although we do believe that DDD pacing can significantly improve coronary grafts flow when compared to VVI pacing, we do agree that it is difficult to indicate the best A-V delay. The inter patient high variability in optimal A-V pacing delay is not a secret and can be easily deducted in the second part of our study where statistical significance was not achieved. In this regard we are extending our analysis to a larger group of patients with the aim to better define a specific range of A-V delays that may significantly allow for optimal flows in newly constructed coronary grafts.

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