Reviewer’s report

Title: Coronary bypass grafting using Crossclamp Fibrillation does not result in reliable reperfusion of the Myocardium when the crossclamp is intermittently released: A prospective cohort study.

Version: Date: 1 October 2006

Reviewer: Brian Fabri

Reviewer’s report:

General
This could have been a potentially highly interesting study that unfortunately falls short of expectations and essentially ends up as an observational report.

The authors report on intra-myocardial pH monitoring in the setting of CABG using intermittent X-clamp fibrillation. The authors use the (recently ‘re-packaged’) Khuri Myocardial pH monitor, which has in fact been around since the early 1980’s. Terumo re-launched the product and made it much more user-friendly but unfortunately due to poor adoption of the system this product has now been withdrawn.

The Myocardial pH Monitor is an early warning system for intra-operative myocardial acidosis and allows for pH guided myocardial management. In the introduction the authors state that they had two hypotheses. Firstly they hoped to define ‘a safe ischaemic time for cross clamping’ and secondly ‘to measure ischaemic preconditioning in clinical practice’. Unfortunately they fail to achieve either of these aims.

The study comprises 16 patients who have been arbitrarily divided into 2 groups depending upon whether there is perceived to be pH recovery. They define recovery as a pH ? 6.8. Is this an arbitrary value or is it based on previous reports? Why not define it based on the pre-clamp pH seeing that the initial pH range is wide (6.974 to 7.239). In figure 1 the ‘pH recordings (of all the patients) from application of crossclamp, through release, until application of second cross clamp, allowing for maximal time for recovery’ is shown. How is ‘maximal time for recovery’ defined? Is it when the proximal anastomosis was completed? Is it after a ‘pre-determined’ interval of graft perfusion?

The authors report the pH findings by describing average pH values of the anterior and posterior probes. Are there differences between the anterior and posterior probes? Is it valid to combine the two values and assume a global pH measurement when in fact each probe measures pH within its vicinity and is only ‘representative’ of myocardial pH? Would one not expect differences between the two probes especially as grafting (and therefore revascularisation) continues sequentially?

The final clinical outcome (Table 3) appears to have been the same on both groups but this is only in regard to ‘crude’ outcome measures such as length of stay and inotrope usage. Surprisingly the authors do not report on any measures on myonecrosis. Post-operative measurement of enzymes (CKMB or Troponin) would have been ideal in the context of apparent differences in intra-operative pH. There is ample evidence in the PCI and cardiac surgical literature that relatively minor increases in post-operative enzymes (3-5 times the upper limit of normal) is associated with a worse prognosis. Could the patients with apparent lack of pH recovery have higher cardiac enzyme release in the post-operative period? That correlation would have had significant importance. Indeed no mention is made about whether there were any ECG changes peri-operatively.

The authors comment of the rate of pH descent in the recovery group and assume that the ‘reduced rate of descent’ is indicative of preconditioning. This is too big a step to take especially without further evidence, such post-operative enzyme release. A reduced rate of descent is also seen in the non-recovery group (Table 2) but no comment/mention is made of this in the text. Also, how would you explain ischaemic preconditioning happening in one group but not the other? Incidentally Table 2 shows a drop of 0.152 after the third X-clamp and the text reports a drop of 0.0152. Also why is the ‘rate of descent’ reported over the first 5 minutes of X-clamp? What happens during the rest of the X-clamp period? Why not report the rate of descent over the whole X-clamp interval (per minute).

In the discussion the authors ‘hypothesize’ on why some patients did not show recovery in pH after X-clamp release. They consider this is due to lack of adequate perfusion of the native coronaries either because of the non-pulsatile, ‘lower’ aortic pressure during cardiopulmonary bypass (BUT wouldn’t this be the same for
both groups?), or the cardioplegia pressure (BUT CARDIOPLEGIA WAS NOT USED IN THESE
PATIENTS!!) or because of increased resistance from ‘twisted or compressed’ vessels!! Why in that case
was no attempt made to improve the perfusion pressure and see whether this resulted in an improvement in
pH?

In conclusion, this paper is just a description of pH changes in a small group of patients undergoing CABG
using X-clamp fibrillation. It is quite remarkable that exactly 50% of the group showed ‘pH recovery’ after
X-clamp release. I do not think one can reach any conclusions from this report. There are too many gaps in
the design, execution and interpretation, which unfortunately renders this paper almost meaningless.

-----------------------------------------------------------------------------------------------
Major Compulsory Revisions (that the author must respond to before a decision on publication can be
reached)

-----------------------------------------------------------------------------------------------
Minor Essential Revisions (such as missing labels on figures, or the wrong use of a term, which the author
can be trusted to correct)

-----------------------------------------------------------------------------------------------
Discretionary Revisions (which the author can choose to ignore)

What next?: Reject as not sufficiently sound

Level of interest: An article of limited interest

Quality of written English: Acceptable

Statistical review: No, the manuscript does not need to be seen by a statistician.