

Is re-modification of Ross operation necessary?[†]

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This is an interesting article [1]. We enthusiastically support the use of Dacron graft in Ross operation to prevent aortic dilation in long term. The diameter of sinus of Valsalva increases by 16% during systole in native aorta [2] (although thicker than native pulmonary artery). As the Dacron graft could not change its diameter during systole, we think that this may cause an increase in incidence of aneurysm/dissection formation. To overcome this problem, would it be more convenient to insert Dacron graft between coronary buttons?

[†]The corresponding author of the original article [1] was invited to reply but did not respond.

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Tackling reperfusion injury after cardiopulmonary bypass with tetrahydrobiopterin: new therapeutic potentials for this phenylketonuria drug?[†]

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Szabo et al. [1] demonstrated that administration of tetrahydrobiopterin (BH4) during early reperfusion improves cardiopulmonary recovery after cardioplegic arrest and extracorporeal circulation in dogs. Application of BH4 restored myocardial, endothelial, and pulmonary function and increases plasma cyclic guanosine monophosphate (cGMP) levels.

Szabo et al. concluded that the beneficial effect of BH4 on reperfusion injury after cardiopulmonary bypass can be explained by recoupling of endothelial nitric oxide synthase (eNOS), as supported by increased cGMP levels measured by radioimmunoassay. However, there are many factors that can cause induction of cGMP other than NO, such as carbon

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monoxide and natriuretic peptides [2], which are also released in ischemia/reperfusion damage [3]. In addition, plasma arterial cGMP is correlated with atrial natriuretic peptide levels, and its production is secondary to an enhancement of the local tissue cGMP of the vascular bed [4]. It is required to measure natriuretic peptide to exclude this as another factor of cGMP production. Moreover, there are several more direct ways to evaluate eNOS coupling, such as eNOS monomer/dimer analysis, eNOS activity, eNOS-dependent superoxide generation, and quantification of BH4 and BH2 levels [5].

In addition, it is not clear whether the dose of BH4 that is used in the study of Szabo et al. increases myocardial and endothelial levels of BH4. Subsequently, it is unclear whether the improved left-ventricular function is due to improved myocyte function or is

secondary to improved endothelial function. Furthermore, administration of BH4 results in increased BH2 levels, which have been described to have an inhibitory role on eNOS function. Therefore, measurement of myocardial and endothelial levels of both BH4 and BH2 is recommended from both a safety and a mechanistic point.

Szabo et al. have paved the way to further reduce reperfusion injury after cardioplegic arrest and extracorporeal circulation. However, more focused molecular analysis is needed to further unravel the mechanism of action and optimize the use of this eNOS-modulator (BH4), which is already approved as phenylketonuria (PKU) drug, in this clinically relevant model of reperfusion injury.

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The philosophy of a nerve-sparing thoracotomy closure[†]

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We read with interest Bayram and colleagues' recent article [1] on the randomized trial evaluating a modified thoracotomy closure technique to reduce compression of the intercostal nerve and thereby reduce post-thoracotomy pain. The authors' report a statistically significant reduction in postoperative pain using this technique. The described procedure is a modification of a technique proposed by Cerfolio and colleagues [2, 3], who harvest the intercostal muscle flap at the beginning of surgery, retract the flap outside the thoracotomy field using a soft Penrose drain, and, at closure, drill holes in the lower rib to avoid compressing the nerve. This approach is theoretically a more attractive concept as it avoids nerve entrapment both by the chest retractor during surgery and the pericostal sutures after closure. Bayram's technique, on the other hand, does not avoid nerve compression intraoperatively by the chest retractor, and nerve compression at this stage could still cause postoperative neuropathic pain. We have seen Cerfolio's group perform their procedure with impressive results and are halfway through a randomized trial to evaluate the technique in our unit. Similar to Cerfolio's group, we retract the harvested flap with a soft drain and this gives unrestricted intrathoracic access.

We agree with the authors' results that postoperative pain appears to be less in patients where the modified thoracotomy closure technique was adopted; however, we are unclear

regarding the primary outcome measure on which the sample size was calculated – was it the mean analgesic drug consumption or the actual pain scores? If they were pain scores, at what time interval after surgery? This is especially important considering the multiplicity of variables compared in the study. Calculating mean drug consumption, though technically possible by statistical methods, may not be a very relevant clinical endpoint; pain scores, though necessarily subjective, would be a more important outcome. We also disagree with the authors' conclusion that the reduced pain resulted in earlier discharge from hospital, especially as the authors' own results (Table 1) show otherwise! Nevertheless, we congratulate Bayram and colleagues on their important randomized trial which adds to the growing evidence supporting the hypothesis that avoidance of nerve entrapment at thoracotomy helps reduce postoperative pain.

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