Letter to the Editor

What is the best method of cerebral protection?

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An editorial comment by Pacini [1], which I read with great interest, regarding our recent contribution to the topic of unilateral cerebral perfusion (UCP) appeared in the June 2010 issue of European Journal of Cardio-thoracic Surgery (EJCTS) [2]. I appreciate his kind comments acknowledging our results; however, I do not agree with his conclusion that UCP cannot be recommended for patients who require complex aortic repair. Admittedly, the mean time of circulatory arrest (CA) was only 17.2 min, but the 21% share of complete arch replacements in our study matches with this proportion in other studies reporting elective arch surgery. Moreover, the very good results reported are not exceptional and, as pointed out in our article, have been reported by others. All the series have in common that fact that UCP was performed in mild-to-moderate hypothermia. On the other hand, there is growing evidence (based on experimental and clinical experience) that cold cerebral perfusion can lead to neurological injury [3,4]. Strauch et al. have even postulated that cold perfusion time of more than 30 min should be avoided [3,4].

Milewski and Pacini et al. demonstrated recently that there were no differences between deep hypothermic circulatory arrest (DHCA) and bilateral cerebral perfusion (BCP) regarding operative mortality and neurological morbidity [5]. It is not surprising considering the perfusion temperature, and because of the fact that for establishment of BCP direct cannulation of arteries with atherosclerotic plaques, which otherwise were identified as a predictor of adverse neurological outcome, was performed. I am surprised, however, that a CA time of up to 45 min needed for elective hemiarch replacement was defined by authors as 'short arch reconstructive time'. Normally, it takes several minutes, for which DHCA is, for sure, not necessary. Why should the negative side effects of unphysiological cold perfusion, which are pertinent for all aspects of cardiac surgery necessitating extracorporeal circulation, not be considered for surgery of the aortic arch? We have postulated, therefore, the adaptation of body temperature to the expected time of CA [2]. This, in turn, can be limited by a proper surgical strategy (e.g., arterial cannulation site and cannulation of vascular prosthesis after completing distal anastomosis for resuming visceral perfusion). Because prolonged arch reconstruction time is associated with adverse outcome independent of technique, limiting this time plays a key role in arch surgery and is the best method of protection.

Since 2002, we have performed a total of 130 complete arch replacements using UCP, including different forms of complex arch procedures, for example, dissection or aneurysm of arch arteries, re-dos, etc. The mean time of UCP was 39 ± 16 min and the deepest temperature was 29.2 ± 2.0 °C. Thirty-day mortality and operative neurological morbidity were 3.0% and 1.5% for all patients, 2.9% and 2.9% for those (35) having acute aortic dissection and 0 and 0 in those cases with CA time longer than 60 min (11, range 60—105 min). This, together with data presented [2], demonstrates that UCP with all its advantages mentioned [2], plays an important role in simplifying arch surgery and shortening CA time, leading to excellent results even in complex forms of aortic diseases.

References


Letter to the Editor

Is interleukin-10 the only predictor for inflammation?

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We have read the study of Onorati et al. with great interest [1]. In this study, in the pulsatile cardiopulmonary bypass (CPB) group, the maximum level of interleukin (IL)-10

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was found to be around 600 pg ml\(^{-1}\), whereas, in the standard linear CPB group, it was 400 pg ml\(^{-1}\) (methylprednisolone was not used in this study). The difference was statistically significant. Our concerns are as follows:

Although there was a drop in IL-10 level, it was not so for IL-6 and IL-8 levels. We think that this condition should be clarified in terms of anti-inflammatory mechanisms. It has been previously shown that administration of 1 g methylprednisolone raised IL-10 levels up to 300 pg ml\(^{-1}\) and kept IL-6 at 80 pg ml\(^{-1}\) and IL-8 at 14 pg ml\(^{-1}\) [2]. How could IL-8 and IL-6 levels remain the same while IL-10 levels increase?

References


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Reply to the Letter to the Editor

Reply to Aksun et al.

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Keywords: Anti-inflammatory cytokines; Inflammatory response; Cardiopulmonary bypass

We thank Dr Aksun et al. for their appreciation and comments about our article investigating endothelial activation and inflammatory response after different types of perfusion [1,2].

The colleagues claimed for a reported absence of interleukin (IL)-6 and IL-8 leakage after cardiopulmonary bypass (CPB). However, we never reported that IL-6 and IL-8 levels remained the same. On the contrary, as demonstrated in the Fig. 2 of our article [2], there is a nearly 250-fold rise from baseline values of IL-6 and a nearly 300-fold rise from baseline values of IL-8 during CPB, with a peak time at the 12th hour after the ending of the surgical procedure. Accordingly, two-way analysis of variance (ANOVA) clearly showed a statistically significant rise of both cytokines in both groups (within-group \(p = 0.001\) for IL-6 and IL-8). What proved to be similar was the overall leakage of IL-6 and IL-8 during the postoperative course between the two groups (between-groups \(p = 0.448\) for IL-6 and \(p = 0.111\) for IL-8), showing a quite similar pro-inflammatory activation with the two different CPB strategies. The pro-inflammatory leakage, however, paralleled with a different IL-10 release, which was statistically higher in patients undergoing intra-aortic balloon pump-induced pulsatile CPB (between-groups \(p = 0.001\) for IL-10).

Furthermore, as stated in the study, we did not pre-treat patients with steroids to avoid any potential bias on perioperative cytokine release. Therefore, given the absence of any administration of anti-inflammatory drugs, a significantly higher pro-inflammatory cytokine release compared with the values reported by Giomarelli et al., who treated patients with steroids, can be expected [3]. It has been clearly shown that the higher the pro-inflammatory release, the higher the anti-inflammatory release, so that any conclusion on the inflammatory cascade should be based on the pro-inflammatory/anti-inflammatory ratio [4,5]. Another possible explanation for the different values of IL-6, IL-8 and IL-10 levels observed by us, compared with those reported by Giomarelli et al., can rely on the different cardioplegia, CPB conductance, cardioplegic temperature, CPB temperature, aortic cross-clamp/CPB time, anaesthesia, etc., employed in the two studies [2,3,5]. Therefore, for any study investigating biochemical and cytokine release, the conclusion can be extrapolated only to similar clinical environments. Finally, at the time of our study, we investigated cytokine release with the only available cytokine assay approved for clinical practice (Randox, UK). The assay kit of the cited study [3] was not reported, and, possibly, it could be not approved for the clinical practice, given the date of publication (2003).

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