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Reply to the Letter to the Editor

Reply to Lunkenheimer

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It was a real pleasure to see your Letter to the Editor, because of many reasons. First, it is always good to know that our work [1] is attracting your interest so strongly. We thank you. Then, it is very interesting to follow your evolution [2], switching from the valuable participation in a development of the ventricular myocardial band concept, to the position of its passionate critic. Finally, it is again pleasure to find out, that your calls for denial of such concept, do not always find a 'desirable' echo in a scientific world [3].

Examining elctrophysiological and functional data, in order to test the hypothesis of activation sequence in the ventricular myocardium, James Cox (coauthor) and his associates, calculated the delivery of the impulse throughout the ventricular band. They have very carefully plotted out how the electrical impulse is delivered to the myocardium, by the specialized conduction system, and correlated those findings with the velocities of conduction in thin and thick areas of the heart. It came out that impulse delivery precisely mimicked predicted sequence of activation. The same was true for other researchers applying different investigational methods, mentioned in our article (i.e. Fourier analyses of ventricular MUGA scans, MRI-based mathematical modeling) [1]. The results obtained from those studies, initiated by the evidence of the ventricular myocardial band, not only provided a solid experimental ground for present publication [1], but also for many other researches derived from this new morpho-functional concept [3].

Careful readers may also find a precise concordance between the context and references cited in our article [1]. Thus, they can notice, for example, that some conclusions given by Henein and Gibson are not interpreted, but transferred from the their valuable original work on ventricular long axis [4]. It is not our misunderstanding, but our disagreement with proposed influence of the atrial muscle on generating ventricular suction force, by abrupt upward movement of the entire ventricular base. The same is true for the excellent Brutsaert's review [5]. There is no such place in our article [1] arguing with Brutsaert's explanations of different loading conditions influence on isolated myocardial fiber activation. On the contrary, we have emphasized this well-known fact several times, explaining dynamic changes of the 'hemoskeleton' and their significant influences on the ventricular performance. Therefore, the sources of your conclusion about "delayed shortening activity as a typical behaviour of the ascending limb" would remain a secret for us, but definitively, they do not belong to our article [1].

Beyond your personal, undoubtedly benevolent, concerns, the readers may also be aware of the some other opinions, declaring that: "This model is an example of an emphasis that relates fiber architecture to chamber shape and mechanics and has implications for improved understanding of electrical, electromechanical and mechanical determinants of cardiac function" [3].

We are quite confident that you had carefully read our article [1], but if you could, in addition, participate on the NIH-NHLBI 2002 Workshop: 'Form and Function: New Views on Development, Diseases and Therapies for the Heart', you probably wouldn't miss some important facts.

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Letter to the Editor

Biological effects of coronary surgery: role of surgical trauma and CPB

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We did read with interest the Letter to the Editor from Dr Raja in the May issue of the EJCTS [1]. He commented the prospective randomised study, comparing off-pump and on-pump CABG, by Wehlin recently published in the EJCTS [2]. Moreover, he quoted as reference a review paper about the same topic from our group [3]. Here we would make some comments about the contents expressed by Dr Raja. First, quoting the review paper from our group, he stated that excessive shear stress during CPB may cause damage to blood constituents, activation of the inflammatory response, platelet activation and it may contribute to endothelial injury. We recognize that the shear stress may have a potential role in the pathophysiology of the CPB. Nevertheless, we have to point out that our literature review did not investigate the evidences about the relationships between shear stress and CPB [3]. Moreover, we also recognize that the CPB may elicit the inflammatory response by means of several pathways. However, the evidences emerged from the available literature comparing on-pump surgery to off-pump surgery, including even major thoracic and abdominal surgery, suggest that CPB may have a limited role in inflammatory and hemostatic derangements during the perioperative period. Moreover, such role seems to be limited to the final steps of the operation and the very early postoperative hours [3]. On the other side, it is likely that the trauma to the tissue, imposed by the surgical procedure, may be the more consistent source of pro-inflammatory and pro-coagulant mediators during the entire perioperative period. In our opinion, the observations from Wehlin and colleagues are a further confirmation of this hypothesis [2].

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Reply to the Letter to the Editor

Reply to Cannata et al.
Trauma or no trauma, cardiopulmonary bypass is the major contributor to inflammatory response after cardiac surgery

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Keywords: Cardiopulmonary bypass; Systemic inflammation; Coronary surgery

In an era of evidence based medicine, Cannata and colleagues' views that cardiopulmonary bypass (CPB) may have a limited role in inflammatory and hemostatic derangements during the perioperative period and that the trauma to the tissue, imposed by the surgical procedure, may be the more consistent source of pro-inflammatory and pro-coagulant mediators during the entire perioperative period seem a bit naive and contrary to available robust evidence from a large number of randomized controlled trials (RCTs).