We thank Dr Mehlhorn and his colleagues for their valuable input on the subject.

With this opportunity, we would like to clarify some issues in our previous letter [1].

Actually, our statement “The knowledge on cardiac lymphatics is very limited and their importance is almost always neglected in cardiac surgery practice” did not refer to knowledge on the literature. Rather, we wanted to point out that the clinicians (i.e. cardiac surgeons) have limited knowledge and they usually ignore the lymphatic system of the heart while performing surgery on the heart. In other words, the surgeon almost never considers the factor of the lymphatic system in the surgery. He/she acts as if there are not any lymphatic vessels or they mean nothing to the heart functions. The only things the surgeon focuses on are the arterial, venous and connection system. Again, it is not usual to find some tips and pitfalls for avoiding obstructing or damaging the lymphatic circulation in the classical text-books.

In our letter, we wanted to emphasize the drawbacks of this underestimation or negligence and point out the importance of developing some surgical strategies to protect the lymphatic circulation.

The second issue was one of our questions to Dr Vazquez-Jimenez in the first letter. We believe the question was misunderstood by Dr Mehlhorn and his co-workers. We are aware that high central venous pressure impedes fluid removal from the cardiac interstitium via myocardial lymphatics and causes myocardial edema. However, our question was not related with intraluminal superior vena caval (SVC) pressure.

After placing venous cannulas into SVC and inferior vena cava, sometimes we encircle and secure caval veins by using a snare or vena cava clamp as some procedures require total cardiopulmonary bypass (CPB) and total circulatory arrest. Thus, we iatrogenically create great pressure to the caval vein itself (between the external and internal wall) rather than the lumen. We assumed [1] that the lymphatic circulation would be impaired especially in the ‘right drainage types’ [2]. And we asked their opinion about using cuffed (with self-inflating balloon) venous cannulas instead of regular ones. In their reply [3], Dr Vazquez-Jimenez pointed out that because of the complete network of lymph collaterals, the lymph drainage of the heart would be able to compensate.

We would like to learn Dr Mehlhorn and his colleagues’ input on the same issue.

We wish to express our appreciation to Dr Mehlhorn and his team for their valuable and intense research on cardiac lymphatics and myocardial fluid balance. We read their previous studies with great interest.

As these researchers had shown, plegic myocardium is prone to edema formation because of both relatively enhanced fluid filtration and lymph flow cessation [4]. They also concluded that increasing the colloid osmotic pressure of normothermic blood cardioplegia (BC) minimizes myocardial edema, thus preventing post CPB cardiac dysfunction [5].

Actually, these data are supported by our recent study [6]. We had compared intermittent antegrade cold BC with crystalloid cardioplegia (CC). Interestingly, we found that in the BC group, aerobic metabolism was not used more than in the CC group. In other words, we found that most of the consumed oxygen could not be used in energy production (oxidative phosphorylation). Thus we speculate that the
superiority of BC over CC does not depend on oxygen content, but on other factors such as buffering, free radical scavengers and higher osmotic pressure.

After reading Dr Mehlhorn’s studies, we believe the importance of colloidal osmotic pressure should be more intense than we previously thought. Of course, there would be a significant amount of accumulation of interstitial myocardial fluid and that would be decreased by improved lymphatic flow. Thus, if we consider the lymphatic system factor, ‘beating heart’ technique or ‘cardiac surgical conditions’ [7] may be good alternatives to cardioplegic arrest as these techniques produce minimal myocardial contraction during CPB (suppressing myocardial chronotropy and inotropy).

In conclusion, we believe that as clinicians we should give more attention to the edema factor in myocardial protection.

References