I want to thank Dr. Konuralp and colleagues for his kind comments. With regard to the preparation of the efferent cardiac lymph vessel in swine, we first identified the lymph vessel after injection of Evans blue and dissected it from the tissue under the superior vena cava (SVC), pulling the SVC to the right with a forceps. Once the dissection was completed we placed a silk tape around the SVC, avoiding an injury of the lymph vessel by keeping the tape directly around the vena cava. Total cardiopulmonary bypass does not produce lymph edema as blood of the upper body is drained by the SVC cannula and even after occlusion of the main lymphatic efferent vessel there is a complete network of lymph collaterals in pericardium, trachea and lungs which is able to compensate for lymph drainage of the heart. A compromised lymph drainage does exist in some children after the Fontan operation. With mobilization of the pulmonary arteries and the SVC many of the efferent lymph vessels and collaterals are destroyed and due to the high venous pressure in the upper part of the body (in contrast to the arterial switch operation where almost the same anatomical preparation is made) the lymph drainage of the heart is severely compromised. The result is a high incidence of pericardial and thoracic effusions and chronic impairment of the ventricular function.

With regard to the flow rate of the 'right drainage type' this group did indeed show a higher flow than the other types (these data will be published in a paper about cardiac lymph and myocardial protection in very near future).

The lymphatic drainage of the transplanted heart is a very interesting issue since global lymphatic interruption occurs with the cardiac transplantation. As hypothesized by Albert J. Miller [1] interrupted cardiac lymphatics do not regenerate and this fact predisposes the heart to coronary vasculopathy and chronic rejection after cardiac transplantation. Miller et al. [1] published an experimental study in 10 dogs showing no regeneration of the transected principal cardiac lymphatic vessel, although small lymphatic collaterals from the distal side were seen in two animals. However, dogs were used for his experiment, which to my opinion is not the ideal lymphatic model. Furthermore he did not perform a complete explantation and reimplantation of the heart, thus keeping many collaterals open and therefore suppressing the stimulus for lymphatic regeneration. I think that depending on the individual regeneration capacity of the cardiac lymphatics, the transplanted heart will be more or less predisposed to coronary vasculopathy and chronic rejection after cardiac transplantation.

In conclusion I believe that investigation of the cardiac lymphatic system is mandatory for the understanding of the pathophysiology of many cardiac diseases (coronary atherosclerosis [2], pericarditis, myocardialpathy [3], etc.) and I hope that our work will stimulate other cardiac surgeons to investigate this fascinating issue.

References