

EDITORIAL

**THE CARDIAC LYMPHATIC SYSTEM
AND CORONARY ARTERY DISEASE**

In this issue of *Lymphology*, Solti et al present some of their recent studies and thinking on coronary artery disease based on studies in dogs with cardiac lymphatic obstruction, and have drawn certain conclusions about cardiac lymphatic impairment in man. The Budapest group has studied various aspects of the lymphatic system for many years, and the reports of Jellinek et al on coronary artery changes in dogs with cardiac lymphatic obstruction go back to at least the mid-1960s. While it is appropriate to publish the present paper that describes studies that are most difficult to perform, the experimental techniques and conclusions must be viewed with considerable skepticism.

That the coronary artery wall is an active metabolic organ is without question, but the study of its metabolic processes is far from simple. Certainly the intentions of the authors are meritorious; indeed, it is my prejudice to agree with the thesis that interference with cardiac lymph flow predisposes to significant biochemical and pathologic alterations in the coronary arteries. Solti et al and others have embraced this thesis, and certain of the work their group has done in the past supports it. But in this paper the authors have presented a complex, difficult to control series of experiments that make it dubious that they have seriously contributed to the issue at hand. For example, measuring coronary artery blood flow using an electromagnetic flow meter with findings of 15% reduction (15-20 ml/min) with "lymph stasis" is of questionable validity, particularly after serial thoracotomy and use of sodium pentobarbital with its known cardiac depressant effects for general anesthesia. It is highly improbable that the authors have been able to maintain uniformity among the groups they have studied. Another example of a problem in the paper is the duration of coronary arterial

wall metabolic and pathologic changes. Solti et al have described these changes as occurring over several weeks after lymphatic ligation, but these abnormalities may be only short-term manifestations. With lymphatic regeneration and "normalization" of lymph flow, will these acute changes subside? Longer term observations might provide better insight into whether lymph stasis and the abnormalities in the coronary arteries persist.

Thoughtful investigators have speculated for years that impaired cardiac lymph flow may contribute to coronary atherosclerosis, and recently our group hypothesized a possible role for impaired cardiac lymph flow in the accelerated atherosclerosis after cardiac transplantation in humans. But speculation, though honorable, is not proof. Thus one must interpret the Solti et al paper as providing limited evidence that impaired cardiac lymph flow leads to coronary atherosclerosis. Some of the leaps to conclusions in man are also dubious. Thus, for example, x-radiation of the mediastinum may injure coronary arteries directly, rather than, as the authors invoke, producing changes via damage to the lymphatic drainage system.

Does impaired cardiac lymph flow lead to changes in the coronary artery wall? There is good evidence that this occurs, and certainly the previous careful histologic work of the Budapest group has contributed to this concept. I, too, believe that interference with cardiac lymph flow predisposes to coronary atherosclerosis, but we still have a long way to go to prove such a relationship.

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