# EDITORIAL

## Some Observations Concerning Pericardial Effusions and their Relationship to the Venous and Lymphatic Circulation of the Heart

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The many etiologies for pericardial effusion have been extensively documented, but the *mechanisms* involved in the formation and persistence of the pericardial fluid have received little attention. Investigators interested in the etiologies of pericardial effusion have concerned themselves primarily with changes in the parietal pericardium. This is understandable, as the parietal pericardium can anatomically be approached as a distinct structure and is needled and biopsied with relative ease. On the other hand, the visceral pericardium has been neglected, since it is much less accessible for study.

Just as the parietal pericardium has been assumed to be the source of pericardial effusion, so "inflammation" has been generally considered to be the insult which causes the effusion. However, understanding the mechanism of pericardial effusion requires a more thorough examination of the problem. Though "inflammation" can explain pericardial effusion in certain clinical settings, substantial non-inflammatory effusions do occur with, for example, congestive heart failure.

### *The Vasculature of the Structures Surrounding the Pericardial Space*

The pericardial space is formed by the visceral and parietal pericardium and normally contains a small amount of clear, straw-colored fluid containing protein. It will be worthwhile to examine certain aspects of the vasculature adjacent to the pericardial space before describing certain of our experimental findings.

The serous inner lining of the parietal pericardium is continuous with the epicardium (the visceral pericardium) of the heart muscle. The subepicardial area contains an elaborate venous circulation, the major components of which drain into the coronary sinus and the anterior cardiac veins. There are many anastomoses between these two venous systems (1), both of which drain into the right atrium. In addition to the venous system, the mammalian heart has an extensive lymphatic system which drains the muscle wall from subendocardium to subepicardium. Collecting lymphatics in the sub-

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epicardium eventually join to form a large channel which leaves the heart at its base (2–6). It is apparent that the epicardial region underlying the visceral pericardium has an extensive venous and lymphatic drainage system.

Though the normal mammalian parietal pericardium does have small arteries and veins on its outer surface, it is not a very vascular organ. In addition, the lymphatics of the parietal pericardium, which tend to be located mainly in the basal fat, are relatively scant (2). Thus, in marked contrast to the visceral pericardium, the venous and lymphatic drainage system of the parietal pericardium is relatively poorly developed.

### *The Movement of Substances into and out of the Pericardial Space*

The routes by which various substances enter and leave the pericardial sac are unknown. We have found in the dog that micropulverized barium sulfate leaves the pericardial space as well as the heart muscle itself by the same basic lymphatic pathway to the mediastinal collecting system (7, 8). These studies suggested that this material left the pericardial sac via the subepicardial lymphatics. Unfortunately, there is little data on the removal of other types of substances, including water and electrolytes, from the pericardial sac. Nor do the few limited studies (2, 7, 9) give any clear answers about the exact routes of removal of these materials. Though cardiac lymph flow undoubtedly in part represents drainage from the pericardial space, the extent to which the latter site contributes to the lymph composition and volume is unknown (10, 11).

### *The Experimental Production of Acute Pericardial Effusion*

In view of the possibility that the pericardial space drains mainly to subepicardial veins and lymphatics, as was suggested by the anatomical and barium sulfate studies, it seemed reasonable to hypothesize that significant interference with both the venous and lymphatic outflow from the heart muscle might result in the acute formation of a pericardial effusion. However, we were unable to produce such effusions in dogs when we obstructed the mediastinal lymphatic system draining the heart and at the same time ligated the coronary sinus. When we made the venous obstruction more complete by also ligating the anterior cardiac veins, an outpouring of fluid occurred *from the epicardial surface of the heart.* Beads of fluid formed, coalesced and ran down the surface of the heart in small rivulets; the surface of the heart became "wet" in appearance. This "beading" could be demonstrated repeatedly after wiping the surface of the ventricles. Following these initial observations it has been possible to regularly produce acute pericardial effusions in dogs by this method (12), confirming that significant obstruction to venous and lymphatic drainage from the heart muscle leads to an acute pericardial effusion which arises from the surface of the heart. Undoubtedly this fluid comes from the interstitial space of the heart muscle and "overflows" into the pericardial space when the normal venous capillary and lymphatic routes available to return it to the circulation are obstructed. Because the pressure development at the endocardium is greater than at the epicardium, the transmural pressure gradient would also tend to facilitate the movement of interstitial fluid towards the epicardial sur-

face. Local inflammation would serve to enhance effusion by impairing capillary permeability and increasing protein, water and electrolyte loss into the extra-vascular space.

### *The Significance of the Experimental Observations*

We believe that these early observations are pertinent to understanding pericardial effusions in man, and briefly cite two clinical states to illustrate the possibilities: 1. Myocardial lymph and venous blood both drain to the right side of the heart. It is likely, in the light of our experimental findings, that the pericardial effusion seen in severe congestive heart failure is due to an elevation in central venous pressure which interferes with both the venous blood and lymph drainage from the heart muscle itself. 2. In such conditions as radiation pericarditis with effusion, the critical pathologic process may be considered damage to the *visceral* surface of the pericardium and the underlying myocardium, causing interference with epicardial venous blood and lymph flow. The resultant pericardial effusion due to the loss of interstitial fluid from the myocardium to the pericardial space may be further augmented by radiation injury to the mediastinal lymphatic system draining the heart muscle. If damage to the visceral pericardium and its underlying tissue is the major pathology seen in radiation pericarditis (13), the relative infrequency of obtaining meaningful information from biopsies of the parietal pericardium becomes understandable.

It is apparent from the foregoing discussion that more attention should be directed to the visceral pericardium and the subepicardial region of the myocardium to seek critical pathologic changes when pericardial effusion is present. Furthermore, a continued careful assessment of the mechanisms of formation of pericardial effusion is warranted.

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