The Effects of Pericardial Effusion on Respiratory Variations in Hemodynamics and Ventricular Function*

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The present study. In this issue of the Journal, Gonzales et al. (1) employed Doppler measurements of transmural blood flow velocity, pressures and cardiac output to assess the effects of inspiration on the pattern of left ventricular filling, left ventricular isovolumetric relaxation and hemodynamics. This study makes an important contribution to the general topic of the effects of respiration on cardiac function, a topic that has long intrigued clinicians and physiologists alike (2). The study sheds new light on the pathophysiology of pericardial effusion in general and cardiac tamponade in particular. The authors have demonstrated pronounced abnormal respiratory variation in the velocity and pattern of ventricular filling when intrapericardial pressure has been increased a mere 1 or 2 mm Hg by instilling saline solution into the pericardial sac of conscious, sedated instrumented dogs with closed chest breathing spontaneously. Contrary to expectations, this respiratory variation, which began early, did not progress with increasing severity of cardiac tamponade beyond the level associated with the advent of right atrial compression and did not correlate with pulsus paradoxus.

The principal findings of the study were that pericardial effusion is accompanied by an increase in early diastolic mitral blood flow velocity and left ventricular isovolumetric relaxation. As expected, changes in the pattern of left ventricular inflow, as assessed by Doppler echocardiography, were accurately reflected by simultaneous recordings of the instantaneous left atrial to left ventricular diastolic pressure gradient. Perhaps somewhat less expected was the observation that these changes begin at very modest elevations of pericardial pressure, before equilibration of ventricular diastolic or right atrial pressure. Some of these observations could be partly resolved by further experiments, such as adapting the model to study dogs by using nonventricular pacing at the same heart rate during baseline and the various stages of tamponade.

The pericardial fluid pressures reported in the early stages of cardiac tamponade in this investigation are well within the reported range of normal pericardial contact pressure measured with use of a flat unstressed balloon inserted between the pericardium and myocardium (3). This observation means that pericardial fluid pressure elevated to a level equal to or even less than normal pericardial contact pressure adversely affects ventricular function and induces abnormal respiratory variation in left ventricular filling. It is important to distinguish clearly between these two means of measuring pericardial pressure.

The concept of pericardial contact pressure. This concept has been generally accepted by physiologists, who usually employ a balloon to measure pericardial pressure. Clinicians, who lack the means to use these devices in their patients, except in the operating room, and who are accustomed to measuring pericardial fluid pressure before and after pericardiocentesis, have been much more reluctant to embrace the idea of pericardial contact pressure, with all its implications including regional variation and minimal right heart transmural diastolic pressure. In the instance of pericardial effusion, this difficulty is not an issue, but it is necessary to resolve the paradox of a fluid pericardial pressure of 2 mm Hg initiating abnormal hemodynamic respiratory variation while pericardial contact pressure of 5 to 8 mm Hg does not.

Pericardial tamponade. Gonzales et al. (1) very properly caution the reader that evaluation of the severity of cardiac tamponade depends on the proper integration of clinical and echocardiographic findings. Appleton (a coauthor of the
report) and Hatle have made major contributions to the application of Doppler echocardiography in pericardial and myocardial disease. It may be, as suggested by Spodick et al. (4), that a truly lax pericardial effusion seldom if ever exists, although in a previous study, Appleton et al. (5) found abnormal respiratory variation in only about half of the patients with pericardial effusion but no clinical evidence of cardiac tamponade whom they studied.

Important differences exist between clinical cardiac tamponade and experimental cardiac tamponade induced in dogs. These include the rate of pericardial fluid accumulation and differences in pericardial and mediastinal structure in the two species. These differences account to some extent for the relatively low pericardial pressure elevations reported by Gonzales et al. (1). Nevertheless, their study makes a most important contribution to our knowledge of pericardial effusion and cardiac tamponade and strengthens the concept first proposed by Reddy et al. (6) that cardiac tamponade is a continuum and not, as many clinicians continue to believe, a phenomenon that is either present or absent. Moderate and severe cardiac tamponade must be treated by closed or open drainage, but mild tamponade may under many circumstances be safely managed expectantly.

References