alyses changes the QRS amplitude in the electrocardiogram. Int J Cardiol 1993; 41:141–147
16 Wagner GS. Marriott’s Practical electrocardiography. 9th ed. Baltimore, MD: Williams & Wilkins, 1994; 23–26
17 Constant J. Learning electrocardiography. 3rd ed. Little, Brown and Company, 1987; 103

Low Voltage With Pericardial Effusion

Complexity of Mechanisms

In this issue of CHEST (see page 2064), Japan’s leading investigator of pericardial disease, Tetsuro Sugiura, MD, and his colleagues contribute a unique report of patients free of heart disease with asymptomatic pericardial effusions, small to large, some of whom had PR segment and ST-T wave changes. The report raises many questions as it answers, and these will require further investigation. By standard definition, 32 of 121 patients had low voltage QRS. In the entire group, widespread ST-segment elevation was found in only 8 patients, and widespread PR-segment depression in 32 patients. PR-segment depressions were significantly more frequent in those with low QRS voltage than in those free of low voltage (more cases may have escaped detection when low voltage applied to more than the QRS complexes). The authors appropriately lumped moderate and large pericardial effusions, as it has been shown that the physiologic effect—increased ventricular interaction—of even asymptomatic effusions was similar in moderate-to-large effusions as opposed to small effusions.

Eight of 32 patients with PR-segment deviations had ST-segment deviations, while none with isoelectric PR segments had ST deviations, and significantly more patients with isoelectric PR segments had moderate-to-large effusions than small effusions. Small voltage and PR-segment deviations were rare in patients who had clinically silent pericardial effusions with an unsurprising trend to lower voltage in moderate-to-large effusions.

Certain findings are relatively remarkable: all patients with PR deviations had either a malignancy or a connective tissue disease. In contrast, it is not surprising that all patients with hypothyroidism and with renal disease had no ECG changes.

The authors conclude that there is subepicardial myocardial involvement by inflammation that they believe can cause low voltage despite small effusions and conclude “even a small but inflammatory effusion can cause low voltage in the presence of PR-segment depressions.” They thus assume the fluid to be inflammatory and, presumably because the patients were asymptomatic, they assumed that neither drainage nor biopsy was indicated. In this connection, no patients had tamponade, and Sugiura and colleagues did not describe any of the large effusions as massive. PR-segment deviations can be safely attributed to early appearance of the atrial T wave because they are always opposite to the direction of the P wave with an appropriate spatial vector. However, it is not certain that all PR-segment deviations must be inflammatory. Though rarely ubiquitous as in acute pericarditis, they appear frequently enough in “routine” ECG practice and are seen regularly in the tachycardia of exercise testing with no evidence of inflammation. In the resting ECG, truly widespread PR deviations are indeed characteristic of acute pericarditis (when depressed elsewhere, PR segments are often elevated in lead aVR and sometimes in lead V1). That all of the patients of Sugiura and colleagues with PR deviations had either malignancy or connective tissue disease is in itself remarkable and, at least for the latter, might suggest inflammation. These relationships would bear further clinical investigation.

That neither ST nor PR deviations were discovered in patients with hypothyroidism and renal failure is not surprising. Indeed, classic uremic pericarditis is unique. Despite even life-threatening inflammatory effusions and intrapericardial hemorrhage, the ECG does not change, since even a brisk uremic intrapericardial inflammation does not penetrate the myocardium; indeed, any such pericarditic
changes suggest intercurrent infection (classically pneumococcal and recently viral, particularly hepatitis viruses). The 10 patients with pulmonary malignancies must have included some patients with emphysema, a common cause of low voltage, particularly in the limb leads. There were also no statistics on the incidence of pleural effusions, another correlate, on its own, of low voltage. Moreover, in view of the pioneering work of Madias et al demonstrating fluid retention to be a potent and frequent cause of low voltage in hospital patients, we would have to know more about the patients’ fluid-electrolyte status and whether edema or ascites was present. We also do not know the effects of various therapies on PR and ST deviations. Finally, another matter of interest from the standpoint of inflammation would be the ratio of the height of the J point (ST takeoff) to the height of the T wave in lead V6, one of the most reliable signs of acute pericarditis when it exceeds 25%. A question also arises in younger patients, particularly men, of early repolarization in any patients with significant J-point deviations.

Unlike coronary disease, clinical pericardial disease is not a massive threat to our patients; therefore few, if any, grant funds have been available for intensive investigation. Sugiura and colleagues may wish to pursue some of their tantalizing findings by follow-up observations of their patients including postmortem investigation (unless autopsies have “died” in Japan as they have in the United States), which should eventually be possible in some of their patients and particularly those with malignancies.

David H. Spodick, MD, DSc, FCCP
Worcester, MA

Dr. Spodick is Professor of Medicine, University of Massachusetts Medical School Director, Cardiovascular Medicine Fellowship Program, Saint Vincent Hospital. Reproduction of this article is prohibited without written permission from the American College of Chest Physicians (e-mail: permissions@chestnet.org). Correspondence to: David H. Spodick, MD, DSc, FCCP, University of Massachusetts Medical School, Cardiovascular Medicine Fellowship Program, Saint Vincent Hospital, 20 Worcester Central Building, Worcester, MA 01608; e-mail: David.Spodick@tenethealth.com

REFERENCES

Drug Therapy for Pulmonary Arterial Hypertension

What’s on the Menu Today?

Pulmonary arterial hypertension (PAH) is a clinical hemodynamic syndrome characterized by elevation of pulmonary artery pressure (PAP) and pulmonary vascular resistance in the absence of left-sided heart disease, lung disease, or pulmonary thromboembolic disease. PAH can be idiopathic (primary pulmonary hypertension [PPH]) or familial, and can arise in association with connective tissue diseases (CTDs), infection with the HIV, portal hypertension, and congenital heart disease (CHD).

Patients with PAH have dyspnea, a progressive limitation of exertion tolerance, and an impairment of right ventricular (RV) function, frequently culminating in RV failure and death within 2 to 3 years. Until recently, there were few therapeutic options for patients with PAH. However, ongoing basic and clinical research has led to tremendous advances in our understanding of the pathobiology of PAH. Moreover, an increasing number of novel therapeutic agents that target these pathobiological features are being studied in randomized clinical trials (RCTs).

Over the past few years, many new therapeutic choices have been added to the menu for clinical use in patients with PAH. But how does one choose between them? What are the relative benefits and risks of each? Which patients with PAH are likely to benefit? What are the costs? The decision to initiate therapy in a patient with PAH depends on objective assessment of the severity of PAH, characterization of pulmonary circulation hemodynamics, and knowledge of some basic concepts of PAH pathobiology.

The pathobiology of PAH is complex. Until re-