murmurs: emphasis on the billowing mitral leaflet syndrome, AM. HEART J. **90**:636, 1975.
4. Shappell, D. S., Marshall, C. E., Brown, R. E., and Bruce, T. A.: Sudden death and the familial occurrence of midsystolic click–late systolic murmur syndrome, Circulation **48**:1128, 1973.
5. Chesler, E., Matison, R. E., Lakier, J. B., Pocock, W. A., Obel, I. W. P., and Barlow, J. B.: Acute myocardial infarction with normal coronary arteries, Circulation **54**:203, 1976.
6. Rakowski, H., Waxman, M. B., Wald, R. W., and Wigle, E. D.: Mitral valve prolapse and ventricular fibrillation (Abstract), Circulation **51** and **52** (Suppl.II):II-93, 1975.
7. Chahine, R. A., Raizner, A. E., Ishimori, T., Luchi, R. J., and McIntosh, H. D.: The incidence and clinical implications of coronary artery spasm. Circulation **52**:972, 1975.
8. Awdeh, M., and Ghohston, D. E.: Mitral valve prolapse and coronary artery spasm (Letter to the Editor), Circulation **56**:329, 1977.
9. Sabom, M. B., Curry, R. C., Pepine, C. J., and Conti, C. R.: Effect of ergonovine maleate in patients with chest pain and mitral valve prolapse (Abstract), Clin. Res. **25**:251 A, 1977.

PR segment depression, pericardial knock, and pericardial rub in pericarditis

To The Editor:

The paper by Wheatley[1] on acute pericarditis in calcific aortic stenosis is an interesting and well-presented discussion of the interaction of two pathologic conditions in the same patient. I should like, however, to add a few remarks to some of the data and the discussion.

Dr. Wheatley's Fig. 1 shows an electrocardiogram of left ventricular hypertrophy. With the T-P interval representing the appropriate baseline, there are, indeed, ST (J) elevations. These occur in only four leads, and there are as many ST depressions as elevations plus an equal number of isoelectric J-points. The ST elevations, notably in Leads V_1 to V_3, are of an order frequently seen with deep S waves and sometimes considered "reciprocal" to the ST depressions of LVH in the lateral precordial leads. What is more striking about this electrocardiogram is the *PR segment depressions* in Leads 1, 2, 3, aV_F and V_1 to V_6. These are equally characteristic of acute pericarditis as are ST elevations[2, 3] and, indeed, in this ECG are the principal sign of pericarditis.

Amid the interesting clinical information and its correlation with the operative and pathologic findings is the presence of a "pericardial knock." An abnormal third heart sound of this kind would virtually never be found in pure cardiac tamponade,[4] although it is characteristic of constriction, and can be heard and recorded in combined effusive-constrictive pericarditis. To some extent that combined lesion may have been present, since the pericardium was thickened and, on the photomicrograph (Fig. 4), the epicardium may actually be both thickened and neovascularized. If not, the patient's "pericardial knock" requires further discussion as possibly a strong third heart sound associated with the diminished ventricular compliance of LVH and not suppressed by tamponade.

Finally, the presence of the pericardial rub is also of interest and is consistent with the observation that pericardial rubs are the rule (rather than, as traditionally taught, the exception) in the presence of effusions which can be quite large, with or without tamponade.[5]

These remarks are meant to supplement and not to detract from a nice presentation of an interesting subject.

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REFERENCES

1. Wheatley, C. E.: Acute idiopathic pericarditis and calcific aortic stenosis, AM. HEART J. **96**:87, 1978.
2. Spodick, D. H.: The electrocardiogram in acute pericarditis: Distributions of morphologic and axial changes by stages, Am. J. Cardiol. **33**:470, 1974.
3. Spodick, D. H.: Pathogenesis and clinical correlations of the electrocardiographic abnormalities of pericardial disease, *in:* Rios, G. ed.: Clinico-Electrocardiographic Correlations, Philadelphia, 1977, F.A. Davis Company.
4. Lange, R. L.: Compressive cardiac and circulation disorders, AM. HEART J. **74**:419, 1967.
5. Spodick, D. H.: Acute cardiac tamponade: Pathophysiology, diagnosis and management, Progr. Cardiovasc. Dis. **10**:64, 1967.

Reply

To The Editor:

I am very grateful to Dr. Spodick for his additions to my presentation. His enlargement of the electrocardiographic changes are particularly appropriate. The TP segment must be considered the baseline when acute pericarditis is suspected since it is the only segment which is isoelectric. This allows detection of PR and ST segment shifts. His references 2 and 3 are excellent discussions of this subject.

The sound designated as a "pericardial knock" was at the appropriate time interval after the second sound in early diastole and was either a knock or a third sound, but the frequency of this sound was higher than a third heart sound. Since the first and second sounds were greatly reduced at the apex and this sound was heard easily, it seemed unlikely to be a third sound. Surface recordings were not made.

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Misdiagnosis of pericardial effusion in presence of MAC

To The Editor:

Dashkoff and associates[1] in their article, "Echocardiographic features of mitral annulus calcification" (MAC), state that the echoes from the calcified annulus are contiguous with the echoes from the posterior aortic wall.

Their figures 5, 6, and 8 clearly demonstrate that it is the