the findings reported by Gentzler et al. despite our having examined a much larger cohort of patients with mitral valve prolapse. This discrepancy exemplifies the danger of conclusions based on experience with relatively small groups of patients (e.g., nineteen) despite statistical significance. We conclude that there is no congenital anomaly, absence, or abnormality in the distribution of the left circumflex coronary artery in patients with prolapsing mitral valves, when compared to those who do not have this entity.

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Reference


The author replies:

To the Editor:

Thank you for forwarding the summary of Dr. Peter and associates concerning coronary artery anatomy in their substantial experience with the systolic click syndrome (SCS). These investigators describe a significantly lower incidence of absent left circumflex coronary artery (LCCA) in their patients with SCS, as well as a significantly higher incidence of this finding in the group they consider to represent the normal incidence of coronary artery branching patterns. In our view a number of factors may account for a varying incidence of absent LCCA in SCS. Perhaps the most important of these is patient selection. All patients with the SCS included in our study were disabled by symptoms, particularly chest pain and arrhythmia; none was studied because of the auscultatory or minor electrocardiographic findings observed in many asymptomatic patients with SCS. There is no doubt, then, that our patient group was highly selected, and may not be representative of the broad spectrum of the SCS. Also, we chose to exclude from our control group those patients with chest pain and normal coronary arteries, primarily because they often exhibited atypical patterns of chest pain similar to those described by our patients with SCS. Were we to include these patients in our control population, the incidence of absent LCCA would be comparable to the “normal” group described by Dr. Peter and coworkers.

Previously we reported that our patients with SCS exhibited a characteristic regional disorder of left ventricular contraction, with reduced shortening of the proximal left ventricular inflow tract and mitral valve ring. Perhaps the most compelling argument in support of a regional contraction disorder as a potential basis for the peculiar mitral valve abnormality in SCS was the additional observation in our study that each of seven patients with isolated arteriosclerotic occlusion of the LCCA exhibited not only a similar ventricular contraction disorder to SCS, but also a systolic click and late systolic murmur, physical findings indistinguishable from SCS.

It remains a moot point whether a congenital variation in coronary anatomy may be responsible for a regional myocardial abnormality in some patients with SCS. We have recently studied two patients by means of Thallium radionuclide myocardial imaging in whom clear-cut underperfusion of the left ventricular inflow tract was demonstrated. In both of these patients, intractable arrhythmias and chest pain were present, and no LCCA could be demonstrated angiographically.

We remain convinced that a segmental myocardial contraction disorder underlies the mitral valve abnormality in some patients with SCS. The questions of whether a vascular abnormality is responsible for this disorder of contraction, and the frequency with which this variety of the click syndrome occurs, remain at issue.

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Right Ventricular Dysfunction in Acute Myocardial Infarction

To the Editor:

In their interesting article, Rigo and colleagues (Circulation 52: 268, 1975) detect a relatively high incidence of right ventricular dysfunction in patients with acute inferior myocardial infarction, a statement with which we heartily agree.

In our judgment, the reason why this right ventricular dysfunction in acute myocardial infarction (AMI) is so seldom mentioned in the literature lies in the fact that the clinical manifestations and the hemodynamic findings characteristic of right-sided heart failure are often lacking, and “may only be manifest after plasma volume expansion.”

In these cases, the same results can be obtained and repeated at will and more safely by studying the well known changes of venous pressure induced by the abdominal compression, whose functional mechanism is easily understandable.

In fact, at variance to what happens in normal people, in patients with right-sided heart failure (even subclinical), the abdominal compression, performed while the patient breathes normally, raises the central venous pressure (CVP) more than 2 cm H2O. This easily explains the genesis of the so-called “hepatojugular reflux.”

The variations of the CVP registered before and during abdominal compression are independent of the zero reference level used and, in the absence of other factors, they depend only upon the functional condition of the right heart chambers.

In around 70% of our CCU patients with AMI we have observed that the CVP, even normal at rest, rises abnormally during abdominal compression, especially in the course of the first seven days of evolution.

Even in the absence of necrosis of its wall (which has not been a very frequent finding in our autopsies), this right ventricular dysfunction depends mainly on the involvement of its myocardial fibers entailed by the myocardial infarction localized in the left ventricle, and not exclusively on the hemodynamic alterations caused by the left-sided heart failure.

In patients with AMI, the simple and safe maneuver of abdominal compression is very useful not only to reveal the right ventricular dysfunction, but also to follow its evolution, and for studying the variations of the left ventricular pressure originated by the changes of the right ventricular filling pressure.

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References


Apexcardiography 1956–1976

To the Editor:

We read with interest Dr. Howard Wayne’s letter to the Editor (Circulation 52: 521, 1975) which treats us somewhat urgently by
Letter: Right ventricular dysfunction in acute myocardial infarction.

M Blas

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