large rise of arterial pressure, heart rate and left ventricular contractility. The effect was reflexly produced, since it could be prevented by infiltration of the aorta by xylocaine. The efferent limb of the reflex involved both α and β sympathetic effects, since the rise of pressure could be blocked by phenoxbenzamine, and that of heart rate by propranolol. This reflex is thus unusual in that it has a positive feedback loop, which would tend to lead to a progressively greater rise of arterial pressure. This situation of a generalized sympathetic overactivity is thus similar to the paradoxical postoperative hypertension; since renin release is also under sympathetic control, the increased levels of plasma renin activity reported by Rocchini might also reflect sympathetic overactivity.

THOMAS PICKERING, M.D.
The New York Hospital-Cornell Medical Center
New York, New York

References


The authors reply:

We would like to thank Dr. Pickering for his interesting comments. We agree that the mechanism underlying the activation of the sympathetic nervous system and the renin angiotensin system following coarctation resection is at least in part mediated through the autonomic nervous system. However, we did not cite all of the possible mechanisms underlying paradoxical hypertension since they are numerous and many are speculative with little or no experimental data to substantiate them.

Our data do not eliminate baroreceptor activation as an initial and transient mechanism leading to postoperative hypertension. While we have not observed an immediate reduction in arterial pressure following coarctation repair, such a decrease in pressure could have occurred in the operating room during anesthesia immediately upon release of the aortic cross clamps.

One of the most dramatic hemodynamic consequences of coarctation repair is the sudden increase in arterial pressure distal to the coarctation site. However, the spinal sympathetic reflex suggested by Dr. Pickering is probably not the major mechanism involved in paradoxical hypertension. The study of Liy et al. indicates that the cardiovascular response to mechanical stretch of the thoracic aorta in the anesthetized cat is significant only if both carotid arteries are occluded. A more plausible consequence of the increased pressure in distal aorta following coarctation resection is its effect on distal vascular responsiveness. Broglio et al. have demonstrated a progressive disappearance of the vascular α-adrenergic response in the hindlimbs of dogs with chronic aortic coarctation. The decrease in responsiveness of the α-receptor could result in increased activity of the sympathetic system in the postoperative period. In any event, the basic mechanism underlying paradoxical hypertension is still unclear and requires further investigation.

ALBERT P. ROCCHINI, M.D.
Children's Hospital Medical Center
Boston, Massachusetts

Type A WPW and Mitral Valve Prolapse

To the Editor:

In the article entitled “Wolff-Parkinson-White Syndrome (WPW)” by Gallagher et al. (Circulation 51: 767, 1975) an association between WPW and mitral valve prolapse was noticed for the first time. Out of 68 patients with WPW seven had a midystolic click with or without an apical murmur, and ballooning of the posterior mitral leaflet was documented by echocardiogram, by left ventriculogram or both.

Chandra et al. (Circulation 53: 943, 1976) utilized echocardiography to determine the motion of the interventricular septum in 26 patients with WPW, and two of their patients had mitral valve prolapse.

In these nine patients published so far with the combination of WPW and mitral valve prolapse the echocardiogram has shown type A WPW in all of them.

Is this relationship between type A WPW and mitral valve prolapse a coincidence or could there possibly exist a causal relationship between them?

The findings in one of our patients with mitral valve prolapse would seem to favor a causal relationship. An 11-year-old girl was catheterized because of occasional episodes of chest discomfort and a grade 1-2/6 systolic murmur along the left sternal border. At one occasion a midystolic click was noticed with maximum between the apex and the left sternal border. The routine electrocardiogram varied between normal and type A WPW. Right heart catheterization revealed no detectable shunt and the pressures were within normal limits. A cineangiogram was obtained injecting into the pulmonary artery in the RAO projection focusing especially on the left ventricle, and a prolapse of the posterior mitral leaflet was demonstrated. The systolic contraction pattern of the left ventricle was compatible with the descriptive term: “ballerina foot.”

When the patient was seen six months later in the outpatients’ clinic, the midystolic click was heard only intermittently and the electrocardiogram showed alternately normal conduction pattern and type A WPW. When phonocardiography was performed (fig. 1) it was then demonstrated that the midystolic click was only and always present when the electrocardiogram showed WPW.

Our patient has then demonstrated that the clinical manifestation of mitral valve prolapse in terms of a midystolic click can be influenced by type A WPW. We therefore suggest that the absolute dominance of type A electrocardiograms in patients with WPW and mitral valve prolapse published so far is due to a causal relationship between these to distinct abnormalities and not merely a coincidence.

How could one possibly explain such a relationship?

In type A WPW the early activation is thought to occur in the posterior area of the left ventricular wall unlike type B where the lateral right ventricular myocardium is believed to undergo initial activation. This will in patients with type A WPW create a 2 to 3 mm discrete early systolic anterior movement of the posterior left ventricular wall as recently demonstrated by DeMaria et al. (Circulation 53: 249, 1976) using echocardiography. Since this abnormal left ventricular contraction pattern may interfere with the function of the mitral valve apparatus possibly leading to mitral valve prolapse (Scamporidin et al., Circulation 48: 287, 1973), patients with type A WPW could develop such a prolapse, or as in

References

Type A WPW and mitral valve prolapse.
P Bjerregaard and E L Peterson

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