4. ECHO-DOPPLER CARDIOGRAPHY DURING PERCUTANEOUS TRANSLUMINAL CORONARY ANGIOPLASTY

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Tennant and Wiggers, using an optical myograph, demonstrated in a classic study that ligation of a coronary artery in dogs resulted in paradoxical systolic motion of the affected myocardium within 60 s [1]. Since then, the function of ischemic and nonischemic myocardium has been studied extensively in animal models by a variety of techniques [2–11], including M-mode and two-dimensional echocardiography. Similar data in humans on the effect of transient ischemia on left ventricular function have been limited to observations during spontaneous or provoked attacks of angina pectoris [12–15]. The advent of percutaneous transluminal coronary angioplasty (PTCA), however, provides the opportunity to assess the time course of changes in myocardial wall motion during transient, total interruption of coronary blood flow in humans, and its relation to clinical and electrocardiographic signs of ischemia.

Hauser et al. [16] studied 18 patients undergoing PTCA of 22 coronary stenoses. At baseline (i.e., after introduction of the balloon catheter through the coronary stenotic lesion, but prior to balloon inflation), 14 patients had normal wall motion in the territories of 18 stenotic vessels. During PTCA, left ventricular wall motion abnormalities developed in 16 territories; the remaining two patients had no wall motion abnormalities induced during PTCA. One of these patients had a "highly collateralized" lesion of the anterior descending artery. In four patients (with four lesions) with at least some degree of wall motion abnormality at baseline, there was an increase in
left ventricular dysfunction in two, whereas no changes occurred in the other two, one of these being the only postinfarct patient with a large area of anterioapical akinesis at baseline. If wall motion abnormalities did develop, they usually progressed rapidly from hypokinesis to dyskinesis. The first signs of hypokinesis developed 19 ± 8 s after balloon inflation. Balloons were kept inflated for 30–60 s. After deflation, it took 20 ± 8 s before normalization of wall motion began, and restoration to baseline was always complete 2 min after reperfusion. There were no differences between the first and last inflations (a total of 52 inflations were studied in these 18 patients) as far as time to start of asynergy (19 ± 7 vs 20 ± 8 s) and time to start of normalization (20 ± 8 vs 15 ± 8 s) are concerned. Quantitative measurements of left ventricular function were obtained in only one patient with left anterior descending stenosis whose ejection fraction dropped from 55% to 25%.

We studied the effects of PTCA in 15 patients undergoing PTCA of 15 lesions (3.3 inflations per patient) [17]. All patients developed new areas of asynergy 8 ± 3 s after balloon inflation (which was continued for 38 ± 8 s). Again all degrees of asynergy were encountered from hypokinesis to dyskinesis, but dyskinesis was found only in patients with stenosis of the left anterior descending artery (8 of 11). Balloon deflation was followed by complete normalization of wall motion as early as 19 ± 8 s and was followed in 12 patients by regional hyperkinesis at 25 ± 7 s after deflation (figure 4–1). A semi-quantitative left ventricular wall motion score (13 segments, graded 0 for normokinesis, +1 for hypokinesis, +2 for akinesis, +3 for dyskinesis, and −1 for hyperkinesis) showed an increase from 0.5 ± 1.4 at baseline (after positioning of the balloon catheter, but before inflation) to 6.9 ± 2.2 during inflation, and a decrease to −1.9 ± 1.6 after deflation. In eight patients, left ventricular size (the average of the lengths of diastolic endocardial outlines in the apical two- and four-chamber and long-axis views) was calculated: it increased from 170 ± 9 to 196 ± 10 during inflation. Finally, the total number of segments showing any degree of asynergy during balloon inflation was divided by the total number of left ventricular segments (13) as an estimate of the amount of myocardium being supplied by the stenotic vessel: for the whole group, it was 30 ± 8%, for the patients with anterior descending lesions 34 ± 2%, and, for the patients with stenosis of the right coronary, 17 ± 4%.

Pandian et al. [18] studied 12 patients during PTCA. During inflation, all 12 developed regional dysfunction involving 29 ± 11% of left ventricular myocardium. Regional dysfunction took 1–2 min after balloon deflation to resolve.

Interestingly, both our study [17] and that by Hauser et al. [16] demonstrated that regional dyssynergy is the first sign of ischemia to appear, followed by ischemic electrocardiographic changes in four of 15 and eight of 18 patients, respectively. The last ischemic symptom to appear was angina, which occurred in five of 15 patients in our study. After deflation, ischemic