Interobserver variability in the interpretation of lesions in other coronary vessels might be similarly translated into different decisions about the necessity for coronary artery bypass surgery, or if coronary artery bypass surgery is to be performed, which vessels are bypassable. Interobserver variability is a significant limitation of coronary angiography and clearly requires further study.

References


Intervention Ventriculography
Comparative Value of Nitroglycerin, Post-extrasystolic Potentiation and Nitroglycerin Plus Post-extrasystolic Potentiation

VIDYA S. BANKA, M.D., MONTY M. BODENHEIMER, M.D., RAJNIKANT SHAH, M.D.,
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SUMMARY The comparative value of nitroglycerin (TNG), post-extrasystolic potentiation (PESP) and their combination (TNG + PESP) to unmask asynergic residual contraction was examined, each patient serving as his own control. Twelve of 13 hypokinetic zones improved both with TNG and PESP. One remained unchanged with either. Of 15 akinetic zones, four improved with both TNG and PESP, while ten remained unchanged. One akinetic zone, although improved with TNG, remained unchanged with PESP. Four dyskinetic zones did not change with either. Six asynergic zones responding to TNG alone demonstrated further augmentation with TNG + PESP. However, none of 13 TNG unresponsive zones improved with TNG + PESP. Thus, TNG, PESP, and TNG + PESP are each equally capable of unmasking asynergic residual contractile ability.

RECENT STUDIES have shown that the residual contractile ability of asynergic zones can be assessed ventriculographically using nitroglycerin,1,2 post-extrasystolic potentiation,4,5 or catecholamine (epinephrine) infusion.6 These interventions involve very different mechanisms for unmasking contractile reserve. Nitroglycerin acts presumably by improving the balance between oxygen demand and supply to the chronically ischemic zone1 either due to its unloading effect and/or to increased regional coronary blood flow. Post-extrasystolic potentiation has been found superior to epinephrine as an intervention which increases contractility.6

A comparison of the value of nitroglycerin and post-extrasystolic potentiation in assessing contractile reserve has not been previously examined. In addition, the potential utility of a combined intervention which would both decrease ischemia (i.e., nitroglycerin) and increase contractility (i.e., post-extrasystolic potentiation) in unmasking reversible asynergy in a zone unresponsive to a single intervention is unknown. The present study was, therefore, undertaken to compare the ventriculographic changes induced by both nitroglycerin and post-extrasystolic potentiation in patients with coronary artery disease and asynergy. These responses were also compared to a nitroglycerin plus post-extrasystolic potentiation intervention.

Material and Methods

Thirty-six patients undergoing cardiac catheterization for evaluation of coronary heart disease were selected for study based on the following criteria: 1) asynergy on ventriculography (defined as a localized abnormality of left ventricular contraction); 2) appearance of one to three premature ventricular beats during injection of contrast material into the left ventricle during the initial ventriculogram and/or nitroglycerin ventriculogram; 3) significant (≥75% decrease in diameter) obstruction of one or more of the three major
coronary arteries (left anterior descending, right, and circumflex arteries); 4) absence of angiographic evidence of other etiologic heart disease. All patients were in the postabsorptive state and were premedicated with 50 mg Nembutal, 50 mg Demerol, and 0.4 Atropine. An informed consent had been obtained from each patient regarding the use of nitroglycerin during ventriculography.

Right heart catheterization was performed via an antecubital vein cutdown and left heart catheterization either percutaneously through a femoral artery or via a right brachial arteriotomy. Following recording of left ventricular pressure (using Statham P23 D6 transducers) and cardiac output (dye dilution method using indocyanine green), left ventriculography (initial and post-extrasystolic potentiation ventriculogram) was performed in 30° right anterior oblique projection using 30–40 cc of meglumine diatrizoate (Renografin-76) injected into the left ventricle. When asynnergy was observed, nitroglycerin (grs. 1/150 sublingual) was administered 15–20 min following the initial ventriculogram. When the characteristic hemodynamic effect of nitroglycerin was observed (i.e., fall in systolic and end-diastolic pressure and increase in heart rate), the ventriculogram (nitroglycerin and nitroglycerin + post-extrasystolic potentiation ventriculogram) was repeated in the same degree of obliquity using the same amount of contrast material and tube-to-tabletop distance. Selective cine coronary arteriography was then performed in multiple views using either the Judkins or Sones technique. Cines were taken on a 10 × 6 inch dual field image intensifier (Siemens) at 64 frames/sec using 35 mm Kodak Shellburst film. Hemodynamics were monitored and recorded on an Electronics for Medicine oscillographic recorder.

Ventriculograms were analyzed with respect to location and severity of asynnergy. Location was determined according to the anatomic areas of the left ventricle perfused by each of the three major coronary arteries. The anterior wall and apical zone was defined as the "left anterior descending segment"; the portion of the inferior wall between the mitral valve and posterior papillary muscle was considered the "right coronary segment"; and the inferior wall between the posterior papillary muscle and the apex was taken to be a representative portion of the "circumflex segment." The severity of the contraction abnormality of each segment was defined as follows: hypokinesis indicated diminished contraction; akinesis referred to absence of contraction; and dyskinesis to paradoxical systolic expansion.

A quantitative analysis was performed by superimposing tracings of end-diastolic and end-systolic frames using the cardiac apex and mid-aortic valve as fixed points. The position of the diaphragm was kept constant during cineventriculography, and the outline of the diaphragm was used as an internal marker to allow correct superimposition of the end-diastolic and end-systolic frames. Hemiaxes were drawn which quadrisection the long axis at right angles to it. Each hemiaxis was measured and recorded as a percentage change from end-diastole to ascertain the amount of regional contraction. Apical motion was calculated on the basis of percent change of the apex to base axis. Qualitatively, an asynergenic zone was assigned to the hypokinetic group by the consensus of three experienced observers (VSB, MMB, and RHH). Quantitatively, a hypokinetic zone was defined as that zone which demonstrated <25% hemiaxis shortening in the initial ventriculogram. An asynergenic segment was considered to have responded following post-extrasystolic potentiation, nitroglycerin or nitroglycerin + post-extrasystolic potentiation when it either normalized or changed to a lesser degree of severity, e.g., a dyskinetic segment becoming akinetic, an akinetic segment becoming hypokinetic, etc. Quantitatively, an asynergenic segment was considered to have improved when it showed a ≥10% increase in the corresponding hemiaxis shortening. Left ventricular volumes were determined using the single plane method of Sandler and Dodge.

The study was divided into two parts. In part I (composed of 23 patients) ventricular ectopic beats were present in the initial ventriculogram but not during the nitroglycerin ventriculogram. In this study group, a comparison of control, post-extrasystolic potentiation and nitroglycerin was made. In part II (consisting of 13 patients) the nitroglycerin ventriculogram was accompanied by ventricular premature beats thus allowing a comparison to be made of control, nitroglycerin, and nitroglycerin + post-extrasystolic potentiation.

The following criteria were applied to select the various ventriculographic beats for comparison: 1) "control beat" was defined as a sinus beat in the initial ventriculogram which preceded a ventricular premature beat; 2) a "post-extrasystolic beat" (PESP beat) was defined as that beat in the initial ventriculogram which followed one or more ventricular premature beats allowing for a compensatory pause of at least one and one half times the R-R interval of subsequent sinus intervals. Using these criteria, the PESP beat was characterized by the presence of a larger end-diastolic volume than the control beat; 3) a "nitroglycerin beat" (TNG beat) selected from the nitroglycerin ventriculogram satisfied the same criteria as mentioned in 1 for the control beat; 4) a "nitroglycerin plus post-extrasystolic potentiation beat" (TNG + PESP beat) was selected from the nitroglycerin ventriculogram using the same criteria as 2. Statistical analysis was performed using the Students t-test for paired values and all values are given as mean ± standard error of the mean (SEM).

Results

Comparison of Ventriculographic Changes Induced by Nitroglycerin and Post-extrasystolic Potentiation

Correlation of Changes in Asynnergy

Table 1 shows the qualitative and quantitative changes in each asynergenic zone with nitroglycerin and post-extrasystolic potentiation. Of 32 asynergenic zones, 13 were hypokinetic, 15 akinetic, and four dyskinetic. Twelve of the 13 hypokinetic zones qualitatively improved with both nitroglycerin and post-extrasystolic potentiation. Quantitative responses were also similar. Nitroglycerin resulted in an increase in hemiaxes shortening from 16.3 ± 2.3 to 36.8 ± 2.3% (P < 0.001) and improvement with post-extrasystolic potentiation from 16.3 ± 2.3 to 36.4 ± 3.1% (P < 0.001). One hypokinetic zone remained unchanged both with nitroglycerin and post-extrasystolic potentiation. Of the 15 akinetic zones, four improved qualitatively with
Table 1. Qualitative and Quantitative Responsiveness of Asynergic Zones with Nitroglycerin and Post-extrasystolic Potentiation

<table>
<thead>
<tr>
<th>Patient</th>
<th>Asyn seg</th>
<th>Qualitative assessment (% hemiaxes shortening)</th>
<th>Quantitative assessment: Control TNG PESP</th>
</tr>
</thead>
<tbody>
<tr>
<td>J.L.</td>
<td>RCA Akin</td>
<td>2.7 WNL 29.3 WNL 25.5</td>
<td></td>
</tr>
<tr>
<td>J.B.</td>
<td>RCA Akin</td>
<td>7.0 Akin Akin 11.3 11.3</td>
<td></td>
</tr>
<tr>
<td>P.P.</td>
<td>RCA Akin</td>
<td>7.4 Akin Akin 13.6 34.5 35.0</td>
<td></td>
</tr>
<tr>
<td>M.G.</td>
<td>RCA Akin</td>
<td>0 Akin Hypo 13.2 0</td>
<td></td>
</tr>
<tr>
<td>F.H.</td>
<td>RCA Akin</td>
<td>-2.3 Dysk Dysk -13.5 0</td>
<td></td>
</tr>
<tr>
<td>F.R.</td>
<td>RCA Akin</td>
<td>3.5 Akin Dysk 18.8 17.3</td>
<td></td>
</tr>
<tr>
<td>J.S.</td>
<td>RCA Akin</td>
<td>13.6 Akin Akin 29.0</td>
<td></td>
</tr>
<tr>
<td>J.R.</td>
<td>RCA Akin</td>
<td>4.6 Akin Akin 11.1 7.0</td>
<td></td>
</tr>
<tr>
<td>J.L.</td>
<td>RCA Akin</td>
<td>7.6 Hypo Akin 19.6 23.7</td>
<td></td>
</tr>
<tr>
<td>J.S.</td>
<td>RCA Akin</td>
<td>14.6 Hypo WNL 46.0 34.2</td>
<td></td>
</tr>
<tr>
<td>J.S.</td>
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<td></td>
</tr>
<tr>
<td>E.B.</td>
<td>RCA Akin</td>
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<tr>
<td>G.S.</td>
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<td></td>
</tr>
<tr>
<td>J.L.</td>
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<td>2.9 Dysk Akin 5.9 2.2</td>
<td></td>
</tr>
<tr>
<td>J.B.</td>
<td>RCAler</td>
<td>13.9 Dysk Akin 23.2 29.3</td>
<td></td>
</tr>
<tr>
<td>J.B.</td>
<td>RCA Akin</td>
<td>-3.2 Dysk Dysk -4.3 -3.5</td>
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<tr>
<td>A.S.</td>
<td>RCA Akin</td>
<td>35.8 Dysk Akin 13.3 16.5 13.0</td>
<td></td>
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<tr>
<td>J.S.</td>
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<tr>
<td>T.C.</td>
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<td>J.H.</td>
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<tr>
<td>J.H.</td>
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<tr>
<td>L.G.</td>
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<td>J.H.</td>
<td>RCA Akin</td>
<td>13.0 Hypo Hypo 67.0 31.0</td>
<td></td>
</tr>
<tr>
<td>J.H.</td>
<td>RCA Akin</td>
<td>22.0 Hypo WNL 51.0 43.0</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: Asyn seg = asynergic segment; Hypo = hypokinetic; Akin = akinetic; Dysk = dyskinetic; WNL = normal contraction; RCA = right coronary segment; LAD = left anterior descending segment; LCF = circumflex segment; TNG = nitroglycerin; PESP = postextrasystolic potentiation.

Both nitroglycerin and post-extrasystolic potentiation. Quantitatively, with nitroglycerin mean hemiaxes shortening increased from 6.7 ± 1.3 to 33.7 ± 11.4% (P < 0.05) while with post-extrasystolic potentiation mean hemiaxes shortening increased from 6.7 ± 1.3 to 24.8 ± 11.6% (P < 0.05). Ten akinetic zones remained unchanged following both nitroglycerin (hemiaxis shortening changed from 6.1 ± 0.9 to 7.5 ± 0.8%) and post-extrasystolic potentiation (from 6.1 ± 0.9 to 7.6 ± 0.9%). One akinetic zone, although improved with nitroglycerin (hemiaxis shortening from zero to 15.2%) remained unchanged with post-extrasystolic potentiation. The four dyskinetic zones did not change either with nitroglycerin (mean hemiaxis changed from −4.1 ± 0.8 to −2.4 ± 0.9%) or post-extrasystolic potentiation (hemiaxis changed from −4.1 ± 0.9 to −3.45 ± 1.2%). As illustrated in figure 1, the correlation of responsiveness in asynergic zones with nitroglycerin and post-extrasystolic potentiation was strong (r = 0.866). A typical study is illustrated in figure 2.

Correlation of Changes in Normal Zones

The 27 zones which showed a normal control contraction pattern with a mean hemiaxis shortening of 37.4 ± 4.3% did not change in the nitroglycerin ventriculogram (hemiaxis

![Figure 1](http://circ.ahajournals.org/)

**Figure 1.** Correlation of hemiaxis shortening with nitroglycerin (TNG) and post-extrasystolic potentiation (PESP).

![Figure 2](http://circ.ahajournals.org/)

**Figure 2.** A representative ventriculographic study (Pt. 1, table 1) demonstrating the effect of nitroglycerin and post-extrasystolic potentiation on asynergy. The control ventriculogram shows hypokinesis of the anterior wall ("left anterior descending segment") and akinetic of the inferior wall ("right coronary segment"). Both the asynergic zones show a normal contraction pattern after both nitroglycerin (TNG) and post-extrasystolic potentiation (PESP).
shortening 37.1 ± 4.8%). However, with post-extrasystolic potentiation, the normal zones demonstrated a significant further increase in hemiaxis shortening to 52.2 ± 5.5% (P < 0.02).

Correlation of Changes in Ventricular Performance

**Ventricular Volumes.** With nitroglycerin, end-diastolic volume decreased from 206.0 ± 23.6 to 187.5 ± 22.1 cc while the end-systolic volume decreased from 130.8 ± 24.9 to 109.0 ± 25.3 cc. In contrast, the post-extrasystolic beat exhibited an increased end-diastolic volume compared to the control beat from 206.0 ± 23.6 to 219.0 ± 22.0 cc (P < 0.05) while the end-systolic volume decreased from 130.8 ± 24.9 to 125.9 ± 24.5 cc.

**Ejection Fraction.** The ejection fraction increased significantly with both nitroglycerin and post-extrasystolic potentiation. With nitroglycerin it increased from 0.43 ± 0.04 to 0.51 ± 0.07 (P < 0.05) while with post-extrasystolic potentiation the increase was from 0.43 ± 0.04 to 0.56 ± 0.06 (P < 0.001). Figure 3 illustrates the strong correlation of changes in ejection fraction during the two interventions (r = 0.965).

**Stroke Index.** The stroke index showed no significant change with nitroglycerin (from 41.9 ± 3.1 to 43.8 ± 3.9 cc/beat/m²) but increased significantly from 41.9 ± 3.1 to 59.9 ± 3.9 cc/beat/m² (P < 0.01) with post-extrasystolic potentiation.

**Comparison of Ventriculographic Changes Induced by Nitroglycerin and Nitroglycerin Plus Post-extrasystolic Potentiation (TNG + PESP)**

**Correlation of Changes in Asynergy**

Of the 19 asyneric zones in this group, six responded to nitroglycerin alone while 13 were unresponsive. The six responsive zones increased hemiaxis shortening from 15.1 ± 5.9% to 38.2 ± 6.6% (P < 0.001) with nitroglycerin alone and demonstrated a small but statistically significant further augmentation in hemiaxis shortening to 43.0 ± 6.5% (P < 0.05) with TNG + PESP (fig. 4). However, both qualitatively and quantitatively none of the 13 asyneric zones which were unresponsive to nitroglycerin alone showed improved contraction with TNG + PESP. Hemiaxis shortening was 13.6 ± 3.4% during control, 10.4 ± 4.2% with nitroglycerin, and 9.9 ± 4.9% with TNG + PESP (fig. 4).

**Correlation of Changes in Normal Zones**

The normal zones showed no significant change in hemiaxis shortening with nitroglycerin (from 37.4 ± 4.3 to 37.2 ± 4.9%) but demonstrated a marked increase in hemiaxis shortening to 53.5 ± 4.0% (P < 0.01) with TNG + PESP.

**Correlation of Changes in Ventricular Performance**

**Ventricular Volumes.** Nitroglycerin resulted in a lower end-diastolic volume compared to control (table 2). However, TNG + PESP caused a higher end-diastolic volume than nitroglycerin alone (from 262.5 ± 32.9 to 295.7 ± 35.9 cc) and a lower end-systolic volume (from 174.9 ± 36.8 to 143.8 ± 34.9 cc).

**Ejection Fraction.** Although in this group of patients ejection fraction with nitroglycerin remained unchanged from control (from 0.42 ± 0.05 to 0.39 ± 0.07), it demonstrated a significant increase to 0.58 ± 0.06 with TNG + PESP (P < 0.001).

**Stroke Index.** The stroke index decreased insignificantly

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**Figure 3.** Correlation of ejection fraction with nitroglycerin (TNG) and post-extrasystolic potentiation (PESP).

**Figure 4.** Comparative effect of nitroglycerin (TNG) and nitroglycerin plus post-extrasystolic potentiation (TNG + PESP) on asyneric zones. The further change in responsive zones when TNG + PESP was applied was significant (P < 0.001). None of the changes in the unresponsive zones were statistically significant.
from 62.1 ± 6.9 cc/beat/m² to 45.8 ± 7.1 cc/beat/m² with nitroglycerin but showed significant increase to 80.3 ± 4.7 cc/beat/m² (P < 0.001) with TNG + PESP (table 2).

**Discussion**

In the present study a close correlation was found between improvement in asynery with nitroglycerin and post-extrasystolic potentiation when each patient served as his own control (fig. 1). Only one of the 32 asynergic zones differed in its response to the two interventions. As shown in a previous study, the primary determinant of responsiveness appeared to be the severity of asynery. Qualitatively, 12 of the 13 hypokinetic zones improved with nitroglycerin or post-extrasystolic potentiation in contrast to only four of 15 responsive akinetic zones and none of four dyskinetic zones. In addition, the quantitative changes in hemiaxis shortening were quite close with the two methods (fig. 1).

Both nitroglycerin and post-extrasystolic potentiation affected global left ventricular function as reflected by ejection fraction similarly (fig. 3). These findings are consistent with the previous studies of McNulty et al. and Shah and Helfant as well as Hamby et al. Utilizing nitroglycerin, it was found that the increase in ejection fraction was dependent upon the responsiveness of asynery, i.e., an increase in ejection fraction was seen when the asynergic zone had demonstrated improvement in contraction while if there was no change in asynery, ejection fraction did not change.

Hamby and co-workers showed a consistent increase in ejection fraction of the post-extrasystolic beat compared to the control beat in patients with coronary artery disease with or without asynery.

Several hemodynamic changes occurring in the ventricle with nitroglycerin or post-extrasystolic potentiation may be responsible for bringing about the enhanced contraction in the asynergic zones. In the case of the post-extrasystolic pause, there is prolonged filling resulting in elevation of the end-diastolic pressure and there is also a fall in the aortic pressure resulting in decreased initial outflow resistance or afterload. Both these mechanisms, by increasing myocardial contractility, would result in enhanced contraction of an asynergic zone possessing residual contractile ability. The action of nitroglycerin, on the other hand, is based on both a decrease in afterload and preload (as well as a mild increase in heart rate). In addition, although the precise mechanisms are still controversial, nitroglycerin improves the balance between myocardial oxygen supply and demand. This would allow enhanced contraction to occur in a zone which is asynergic because of ischemia.

It appears that despite the fact that considerably different mechanisms are involved, the responses of both asynergic zones and the left ventricle as a whole to these two interventions are remarkably similar. However, the combined effect of nitroglycerin and post-extrasystolic potentiation could possibly unmask residual contractile ability in asynergic zones not responsive to either intervention alone. This revealing of further zones of reversible asynery was not found in the present study. None of 13 zones which were unresponsive to nitroglycerin alone exhibited improved contraction with the combined effect of nitroglycerin and post-extrasystolic potentiation, although the combination of the two interventions produced a small further augmentation of hemiaxis shortening in the responsive zones (fig. 4). The higher ejection fraction seen with post-extrasystolic potentiation alone or in combination with nitroglycerin can be explained on the basis of the increased contractile response of the normal zones produced by extrasystolic potentiation.

Therefore, nitroglycerin, post-extrasystolic potentiation, and their combination are each equally capable of qualitatively unmasking the residual contractile ability of asynergic zones. The responsiveness to each of these individual interventions is relatively similar quantitatively as well. These findings strongly imply that the responsiveness of asynergic zones to nitroglycerin, post-extrasystolic potentiation, or their combination is dependent primarily on the amount of viable myocardium which compromises the asynergic zone. This is consistent with recent observations of the electrophysiological and histopathological characteristics of asynergic zones which were responsive or unresponsive to nitroglycerin. Studies from our laboratory at the time of open heart surgery have shown that asynergic zones which respond to nitroglycerin are generally characterized by local R waves on epicardial electrograms and histopathologically intact myocardium. In contrast, unresponsive zones were associated with significant Q waves on the epicardial electrogram and replacement of myocardium with fibrous or necrotic tissue. These data, in addition to the present study, confirm the likelihood that asynergic zones unresponsive to nitroglycerin would not be capable of exhibiting enhanced contraction to these different or additional interventions.

Quantitative analysis of ventriculograms has several inherent limitations since this involves superimposition of images during end-diastole and end-systole, and no consensus has been reached for selection of fixed points for superimposition of these images. Chairman and co-workers and Leighton et al. have proposed improved methods for selection of fixed points to avoid errors which may be induced by movement of the patient or diaphragm, the change in the long axis within the ventricle, systolic movement in an apical direction of noncontracting basal structures and systolic rotation of the left ventricle usually evident in the 30° RAO projection as a lifting up of the apex. Although such factors may not affect the analysis of gross contraction abnormalities, lesser degrees of asynery, i.e., hypokinesis, can be overestimated or underestimated quantitatively due to limitations of the technique. These objective methods of quantitative analysis, however, have been found superior to the subjective visual analysis of the ventriculogram for diagnosis of hypokinesis, particularly regarding the reproducibility of analysis.
ACKNOWLEDGMENTS

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REFERENCES


Myocardial LDH Isozyme Distribution in the Ischemic and Hypoxic Heart

GRAEME L. HAMMOND, M.D., BERNARDO NADAL-GINARD, M.D., NORMAN S. TALNER, M.D., AND CLEMENT L. MARKERT, PH.D.

SUMMARY Small myocardial specimens were obtained from 12 patients undergoing coronary reconstructive surgery and from 12 patients undergoing surgical correction for cyanotic congenital heart defects. The specimens were analyzed for LDH isozyme distribution. A control analysis was performed on myocardial specimens obtained at the time of surgical correction for acyanotic congenital heart defects in seven patients with normal coronary arteries. There was a 42% increase in the proportion of A subunits in the hearts of coronary patients as compared to controls. This represented a shift toward an anaerobic isozyme distribution. There was no change in the percentage of A units from the hearts of cyanotic patients as compared to acyanotic hearts of the same age.

Cardiac muscle from patients with coronary vascular disease had an altered LDH subunit composition. Such an alteration was not present with chronic systemic hypoxia. These deficiencies may or may not be related to differing local metabolic responses to the two conditions. However, in the clinical situations, ischemic heart muscle may be oxygen deprived to the point of lactate acid production while hypoxic heart muscle usually is not. Consequently, these findings may represent a compensatory cellular mechanism which provides for continued energy production during chronic ischemia by enhancing glycolysis.

NORMALLY, ENERGY DELIVERY IN HEART MUSCLE proceeds by aerobic metabolism. However, when the heart is required to work under clinical conditions of chronic oxygen deprivation, we have observed that satisfactory or even excellent cardiac contractions are often main-

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