The Similarity of Changes in Segmental Contraction Patterns Induced by Postextrasystolic Potentiation and Nitroglycerin

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SUMMARY  Despite a fundamental difference in their underlying mechanisms, both postextrasystolic potentiation (PESP) and administration of nitroglycerin (TNG) have been utilized to predict reversibility of abnormal segmental wall motion in patients with ischemic heart disease. To determine whether these interventions induce the same changes in segmental contraction pattern, we analyzed biplane ventriculograms of 14 patients who had an adequately visualized PESP beat on a basal ventriculogram as well as a post-TNG ventriculogram. Four segments in each plane were defined and the area ejection fraction of each segment was calculated for a basal sinus, PESP, and post-TNG beat. To correct for global differences in the response to PESP and TNG, we normalized each segmental ejection fraction (NSEF) by the ventricular ejection fraction for that beat and then compared the differences in NSEF from the basal value after PESP and TNG. Eleven patients demonstrated similar responses to both interventions. The three patients whose responses were discordant had elevated or unchanged left ventricular systolic or end-diastolic pressures at the time of the TNG ventriculogram. Our data suggest that, provided these pressures are lower than basal values at the time of the TNG ventriculogram, PESP and TNG will induce similar changes in segmental contraction patterns. Seven patients with similar responses had a PESP beat on their post-TNG ventriculogram. Changes in NSEF after PESP + TNG were identical to those after either intervention. This implies that the combination of interventions does not induce further changes in segmental contraction pattern beyond that produced by either intervention alone.

IN PATIENTS WITH ISCHEMIC HEART DISEASE, localized segments of the ventricle which display decreased systolic wall motion may be composed of compromised but viable muscle fibers rather than scar tissue. With the development of coronary artery bypass surgery, the concept of restoring contractile function by improving blood supply to an ischemic area has received increased consideration and has been supported by documentation of improvement in left ventricular function in some patients after such surgery. To preoperatively identify ischemic regions of the ventricle with the potential to improve their contractile function after adequate revascularization, several groups of investigators have evaluated the response of abnormally contracting segments to postextrasystolic potentiation (PESP), which enhances myocardial contractility, or to administration of nitroglycerin (TNG), which usually decreases the workload of the heart. Both of these interventions have been reported to improve the systolic motion of some hypokinetic and akinetic segments, and limited postoperative studies appear to confirm the predictive value of these induced changes.

The use of PESP requires a ventriculogram with an early extrasystole followed by an adequately visualized sinus beat, whereas the administration of TNG requires the performance of a second ventriculogram. In an individual patient, the choice of which intervention to use may be limited. Therefore, we performed this study to ascertain whether the comparative effects of PESP and TNG are the same for all segments of a given ventricle regardless of the basal function of any segment and despite the difference in the underlying mechanisms of the two interventions.

Methods

In patients with chest pain suggestive of angina, we performed retrograde left heart catheterization with the patient in the postabsorptive state and following premedication with diazepam, 10 mg i.m. A #7F NIH catheter was passed from the right brachial artery or a #8F pigtail catheter was passed from the right femoral artery. Left ventricular pressures were monitored using a Statham P23Db
strain gauge and recorded at 50 mm/sec paper speed with an Electronics for Medicine DR12 recorder. After recording basal pressures, we obtained a biplane (posteroanterior and lateral) left ventriculogram with a General Electric Fluoricon I system alternatively filming in each projection at 60 frames/sec as 45 cc of a 66% meglumine diatrizoate, 10% sodium diatrizoate solution (renografin-76) was injected at a rate of 15 cc/sec. Fifteen to 20 min following the basal ventriculogram, left ventricular pressures were recorded and the patient was given 0.4 mg sublingual TNG. Four to 5 min later, we recorded the pressures again and obtained a second biplane ventriculogram in an identical manner. Then, we performed selective coronary angiography using either the Sones or Judkins technique.

For this study, 14 patients were selected whose ventriculograms were of good quality both before and after TNG, and with at least one extrasystole identified on their basal angiograms. On the basal ventriculogram the first sinus beat following the extrasystole and the next sinus beat were chosen for analysis. On the post-TNG ventriculogram, we chose sinus beats occurring at least two beats after an extrasystole. To determine the beat-to-beat variability in our measurements, 12 pairs of consecutive sinus beats on the basal and/or post-TNG ventriculograms of nine of the 14 patients were also analyzed.

In addition, ten patients had well-visualized postextrasystolic beats on the post-TNG ventriculogram. We analyzed these beats to compare the effects of PESP + TNG with those of both PESP and TNG alone in the same patient.

In both projections, we chose the end-diastolic frame as that with the largest area of the ventricular silhouette immediately prior to complete mitral valve closure and the end-systolic frame as that demonstrating the smallest area of the ventricular silhouette with no further inward motion of any segment of the perimeter. For all the beats chosen, the ventricular silhouette was completely outlined with contrast material at both end-diastole and end-systole. By frame count, the systolic ejection times in each projection agreed within 1 frame (16.7 msec).

To eliminate interobserver error for volume and ejection fraction calculations,11,12 the same observer traced the appropriate silhouettes for all the beats. Biplane left ventricular volumes were obtained by the modified Simpson's Rule method of Goerke and Carlsson.13 The ventricular ejection fraction was calculated as the ratio of angiographically determined stroke volume to end-diastolic volume. Using the method of Hamilton et al.,14 we quadrisected each silhouette by constructing the longest axis from the midpoint of the aortic valve plane to the region of the apex and drawing a perpendicular at the midpoint of that axis. These angiographic segments were designated A through H as shown in figure 1. On the end-diastolic and end-systolic silhouettes of each beat, we measured the area of each segment by planimetry, and calculated the segmental ejection fraction as the percent change in segment area.10,12,15 To compare the differences in a segment's relative contribution to ventricular performance independent of differences in global function, we normalized each segmental ejection fraction by dividing it by the biplane ventricular ejection fraction for that beat.

Since all the patients were in sinus rhythm, we determined heart rates from the measurement of the mean of at least three R-R intervals of a monitor electrocardiogram recorded on the pressure tracings. For each pressure, we took the mean of the measurements of three to five beats. Left ventricular end-diastolic pressure was measured at the incisura following the "a" wave on the pressure tracing. The heart rate, peak systolic, and end-diastolic pressures recorded immediately prior to the basal and post-TNG ventriculograms were chosen for comparison with the angiographic findings.

Results

Random Variation in Normalized Segmental Ejection Fraction

For each segment, we determined the difference in normalized segmental ejection fraction (NSEF) between the beats in the 12 pairs of consecutive sinus beats. The mean of the actual values for the 96 segments was 0.5 ± 1.9% (mean ± SEM). To obtain 95% tolerance limits for random variation, we multiplied the standard deviation by 2.06 and added it to the mean,16 which yielded a value of 39%. The mean of the absolute values of the differences in NSEF was 15 ± 1.2% (mean ± SEM), and the calculated 95% tolerance limit for the absolute value was also 39%. For convenience, we chose ±40% about zero as the limits of random variation in NSEF.

Comparison of Changes in NSEF Following PESP and TNG

For each of the 14 patients, the differences in NSEF were computed between the values for a basal sinus beat and

![Diagram](position)
those following PESP and TNG. Of the 112 segments, 14 demonstrated an increase in NSEF after PESP which was greater than 40%. The highest basal segmental ejection fraction among these segments was 34%, and only two segments had a basal segment ejection fraction greater than 12%. Of the 83 segments whose PESP-induced change in NSEF fell within the random tolerance limits, four had basal segmental ejection fractions less than 12%, and among the 15 segments whose NSEF was significantly decreased after PESP, the lowest basal segmental ejection fraction was 21%. Among the 16 segments with a significant increase in NSEF after TNG, the highest basal segmental ejection fraction was 33%, and four had basal segmental ejection fractions greater than or equal to 15%. Eighty-three segments did not demonstrate a significant change in NSEF after TNG and only five of these had basal ejection fractions less than or equal to 15%. Of the 13 segments whose NSEF decreased more than 40% after TNG, one had a basal ejection fraction less than 36%. In figure 2, the mean values of basal segmental ejection fraction for each group of segments is also provided. When tested by analysis of variance, the mean basal segmental ejection fractions of those segments with an improved NSEF after each intervention were both significantly lower ($P < 0.001$) than those of the segments whose NSEF was decreased or unchanged. The mean basal segmental ejection fraction of the segments with a decreased NSEF after PESP was higher than that of the segments which did not change ($P < 0.05$), but a similar difference was not found for the mean basal segmental ejection fraction of the segments whose NSEF decreased after TNG.

In order to compare the changes caused by each intervention in an individual patient, we constructed a grid on which we plotted the differences in NSEF from basal values about zero on the y-axis for the PESP beat and on the x-axis for the TNG beat. The tolerance limits for random changes were superimposed on each patient’s grid plot.

The plots of three patients (group A) demonstrated the pattern shown in figure 3; all the points fell within the tolerance limits of random variation for both interventions. This pattern would result if there was no significant change in ejection fraction for any segment; or there were significant changes in all segments, but the ratio of segmental to global ejection fraction was not different from the basal state for any segment. In either case, neither PESP nor TNG induced alterations in the segmental contraction patterns of these patients, and in this sense, the response to each intervention was the same.

The plots of eight patients (group B) demonstrated the pattern shown in figure 4; some of the points were outside the tolerance limits of random change for both interventions and distributed near an identity line. This pattern would be present only if both interventions induced significant changes in the ejection fractions of the segments; for some segments, the ratio of segmental to global ejection fraction following both interventions was different from that of the basal state; and for each of these segments, the change in the segmental to global ratio was the same after each intervention. Of the 64 segments in these patients, the plotted differences of only four segments fell in the upper left or lower right quadrants of the PESP vs TNG grids and outside the limits of random variation for one of the interventions. Hence, PESP and TNG induced similar alterations in the segmental contraction patterns of these patients.

The plots of the three remaining patients (group C) are shown in figure 5; some of the points were outside the tolerance limits for one intervention but not for both. These patterns would result if at least one of the interventions caused significant changes in the ejection fractions of the
segments; and for some of the segments, the ratio of segmental to global ejection fraction after the intervention was different from that of the basal state. However, for each of these segments the change in the segmental to global ratio was different after each intervention. Therefore, in these patients, the change in segmental contraction pattern after PESP was different from the change after TNG.

Since the 11 patients in groups A and B represented those cases in which the alterations of segmental contraction pattern were similar after PESP and TNG, their data points were pooled and are plotted in figure 6. The line obtained by the least-squares method with PESP-induced changes as the dependent variable is shown with its slope and intercept. The 95% confidence limits for the slope and 95% confidence limits for the data points are also indicated. The line obtained with TNG-induced changes as the dependent variable had a slope of 0.79 (95% confidence interval 0.67 to 0.91) and intercept of 1. For both lines $r = 0.81$.

**Figure 4.** PESP vs TNG grid plot for a representative patient in group B. The data points indicate the difference in NSEF from the basal value following PESP and TNG for each segment. The 95% tolerance limits for random change are shown as in figure 2.

**Figure 5.** PESP vs TNG grid plots for the three patients in group C. The data points indicate the difference in NSEF from the basal value following PESP and TNG for each segment with the 95% tolerance limits for random change superimposed as in figures 2 and 3.

**Relationships Between Segmental and Global Responses to PESP and Nitroglycerin**

The individual hemodynamic and global angiographic data are shown in table 1 with the patients grouped according to the patterns of segmental response to the interventions. In all patients, the left ventricular peak systolic and end-diastolic pressures both decreased after administration of TNG. However, at the time of the TNG ventriculogram, one of these pressures was not lower than its basal value for patients A. H., W. F., J. G., and W. G. Despite individual exceptions, the calculated means from all the patients for end-diastolic volume index, ejection fraction, systolic pressure, end-diastolic pressure, and heart rate demonstrated changes with PESP and TNG which were similar to those reported by other investigators.5-7,9,17,18 Using analysis of variance to look for differences between the three groups of patients, the only statistically significant difference we found was in basal ejection fraction.

The ventricular ejection fractions after PESP and TNG correlated well ($r = 0.84$) for the 11 patients whose segmental response was similar following both interventions. However, when the patients with discordant responses were added to the data base, the overall correlation was poor ($r = 0.62$). The changes in ejection fraction with PESP and TNG correlated poorly for groups A and B patients ($r = 0.69$), and this relationship was even weaker ($r = 0.37$) when data from group C patients were included. Between the groups, we found no statistically significant differences in the changes in ejection fraction caused by either intervention.

When the changes from basal values were calculated for all the other parameters listed in table 1, we did not find any significant differences between groups. In addition, there was no clear evidence of a relationship between the magnitude of the relative compensatory pause and the changes in end-diastolic volume associated with the PESP beats.

**Comparison of Changes in NSEF with PESP+TNG and with PESP or TNG Alone**

For the ten patients who had a postextrasystolic beat on their TNG ventriculograms, we computed the difference from basal value for each NSEF on the PESP+TNG beat. These data were pooled and plotted against the changes in
NSEL measured in the same segments after PESP alone. The changes correlated ($r = 0.75$) and the slope of the least-squares line was 0.79 with an intercept of 6. When the same data were plotted against changes in NSEL after TNG, $r$ was 0.81 and the slope of the regression line was 0.87 with an intercept of 3. However, three of these patients were those with discordant segmental responses to PESP and TNG alone. When we analyzed only the data from the seven group A and group B patients, changes with PESP+TNG correlated with those following PESP alone, with $r = 0.85$ and the slope of the regression line was 0.94 with an intercept of 3 (fig. 7, left). Compared to changes after TNG, those with PESP+TNG correlated, $r = 0.88$, and the slope of the least-squares line was 1.00 with an intercept of 1 (fig. 7, right).

**Discussion**

Because they act in continuity with each other, the function of individual segments of a ventricle cannot be appropriately evaluated without consideration of their interaction. Thus, to determine whether an intervention has induced an alteration in the segmental contraction pattern, the changes measured in the systolic motion of each segment should be examined in relation to the changes observed in the others. If all the segments demonstrated the same degree of improvement, one would conclude that the intervention produced an increase in global function but did not cause an additional localized change in segmental contraction pattern; if all the segments improved but the degree of change was different for each segment, the most reasonable interpretation would be that the intervention produced both an increase in global function and an alteration of segmental contraction pattern; and if some segments improved while others worsened, the logical conclusion would be that regardless of its effect on global function, the intervention induced a change in segmental contraction pattern. In our study, we wanted to compare the changes in segmental contraction pattern induced by different interventions. We thought it appropriate to presume that each intervention might cause a different change in global function. There-
### Table 1. Hemodynamic and Global Findings Associated with Basal Sinus, Postextrasystolic and Postnitroglycerin Beats

<table>
<thead>
<tr>
<th>Group</th>
<th>Patient</th>
<th>EDVI (cc/m²)</th>
<th>EF (%)</th>
<th>LVSP (mm Hg)</th>
<th>LVEDP (mm Hg)</th>
<th>Heart Rate</th>
<th>RCP</th>
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<tr>
<td></td>
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<td>TNG</td>
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<td>PESP</td>
<td>TNG</td>
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<td>A</td>
<td>J.D.</td>
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<td>70</td>
<td>60</td>
<td>52</td>
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<td>57</td>
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<tr>
<td>Mean ± SEM</td>
<td>49 ± 11</td>
<td>54 ± 9</td>
<td>49 ± 6</td>
<td>62 ± 6†</td>
<td>67 ± 7</td>
<td>64 ± 4</td>
<td>143 ± 7</td>
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<td>60</td>
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<tr>
<td>Mean ± SEM</td>
<td>75 ± 14</td>
<td>78 ± 13</td>
<td>63 ± 10</td>
<td>37 ± 3†</td>
<td>52 ± 4</td>
<td>44 ± 4</td>
<td>136 ± 5</td>
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<td>58</td>
<td>50</td>
<td>47</td>
<td>94</td>
<td>49</td>
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<tr>
<td>Mean ± SEM</td>
<td>67 ± 14</td>
<td>71 ± 14</td>
<td>63 ± 15</td>
<td>42 ± 3†</td>
<td>67 ± 13</td>
<td>43 ± 5</td>
<td>140 ± 5</td>
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<td>All pts:</td>
<td>Mean ± SEM</td>
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<td>71 ± 8*</td>
<td>60 ± 6*</td>
<td>44 ± 3</td>
<td>59 ± 4*</td>
<td>48 ± 4</td>
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*Significantly different from basal by $P < 0.05$ or less (paired Student's t-test).
†Group A different from group B at $P < 0.005$ and group C at $P < 0.005$; no significant difference between group B and group C (analysis of variance).

Abbreviations: EDVI = end-diastolic volume index; EF = angiographic left ventricular ejection fraction; LVSP = left ventricular peak systolic pressure; LVEDP = left ventricular end-diastolic pressure; RCP = relative compensatory pause with postextrasystolic potentiation; Basal = values associated with normal sinus beat on the basal ventriculogram; PESP = values associated with postextrasystolic beat on the basal ventriculogram; TNG = values associated with normal sinus beat on the post-nitroglycerin ventriculogram.
fore, we reasoned that the measurements we made for each segment should be normalized by a factor which would provide a common baseline, independent of changes in global function, from which the relative changes in different segments could be compared.

If a given parameter of segmental performance is normalized by a similar global index, the normalized value is a function of the ratio of segmental to global performance. Furthermore, because the normalized parameter is a function of a segment to global ratio, it is independent of the actual level of global function. For each segment, it reflects the relative contribution to the ventricle as a whole. If the relative contribution of a segment does not change despite an alteration in global performance, the normalized value will remain constant. Changes in the normalized value must represent an alteration of the segmental to global ratio and thus a primary change in the extent to which that segment contributes to overall ventricular performance. It logically follows that if the relative contributions of the segments has been changed, the segmental contraction pattern of the ventricle has been altered.

The area ejection fraction of angiographic segments represents the contributions of the contraction of myocardial fibers along the perimeter of the ventriculosgraphic silhouette, as well as in adjacent regions which are not seen “end-on” by contrast techniques. Therefore, the determination of segmental ejection fraction does not require the assumption that the endocardial fibers forming the perimeter are the same throughout the cardiac cycle. Because we were comparing differences between ventriculosgrams obtained in an identical manner and in the same patient, we assumed that any measurement errors due to rotation of the heart or the use of arbitrary axes to define the segments would be minimal. Previous studies have indicated that the ventricular ejection fraction is the most reproducible parameter of ventricular function obtained from contrast ventriculography,11,12,24,25 with the practical advantage that calculations of actual ventricular volumes are not required. For these reasons, we chose NSEF as the parameter to be evaluated in this study.

Prior to evaluating changes in NSEF caused by PESP and TNG, it was necessary to define limits above which a change could be considered significant. Because random changes in NSEF for a group of normal ventricles would not describe limits relevant to this study, we calculated this variation from 12 consecutive beats in nine of the ventricles subsequently used to compare changes induced by PESP and TNG. If no change in segmental contraction occurred from beat to beat, the difference in NSEF would be zero and the average of the values we measured in consecutive beats was 0.5 ± 1.5%. Since changes in opposite directions would tend to cancel each other, we also calculated the mean of the absolute values for the differences which was 15 ± 1.2%. Although this value seems relatively large, it must be recalled that the ventricular ejection fraction is always less than 100% and its position in the denominator causes the NSEF to be greater than the non-normalized value.

After establishing the magnitude of change in NSEF to be considered significant, we examined the basal segmental ejection fractions of the segments demonstrating an increase, no change, or decrease in NSEF after each of the interventions. By definition, a significant increase in NSEF for a segment implies a significant increase in that segment’s relative contribution to global function regardless of the actual changes measured in either segmental or ventricular performance. Quantitative studies by others have shown that some hypokinetic and akinetic segments demonstrate improved systolic wall motion after PESP and TNG, but dyskinetic segments do not.1,4,6 We did not define limits of basal segmental ejection fraction to classify segments as normal, hypokinetic, akinetic or dyskinetic because the ventriculographic projections in our study were different from those in which such standards have been established.1,4,12 Nevertheless, we found the segments with an increased NSEF following PESP or TNG had a significantly lower basal ejection fraction than those with no change or a decrease. Furthermore, there were relatively few segments with basal ejection fractions within the overlap of the ranges of values for the groups. We felt it was reasonable to assume that the lower basal ejection fractions of these segments implied that their basal function was depressed, and their improvement in NSEF after PESP or TNG suggests they correspond to hypokinetic or akinetic segments with reversible asynergy.

Following each intervention, 83 segments had insignificant changes in NSEF. The range of basal ejection fraction for these segments was broad, but relatively few had low or negative values. Based on the work of others4,6,8,9 we suspect that the vast majority of these segments had normal basal function while those with lower ejection fractions had asynery which was not reversed by either PESP or TNG. Finally, the overwhelming majority of segments the NSEF of which decreased after PESP and TNG had basal ejection fractions equal to or greater than those of the segments in which NSEF did not change. Thus, a decrease in NSEF did not imply that the basal function of a segment was depressed; rather, it suggested that in most cases, the initial function of such a segment was normal, and following the intervention, its relative contribution to ventricular performance was diminished because those of other, initially depressed segments had increased.

The interpretation of the data for group A patients was relatively straightforward. Following PESP and TNG, the differences for all the segments fell within the random limits. Thus, no significant changes in segmental contraction pattern were induced by either intervention. Hence, this group does represent one of the situations in which the segmental response to PESP was similar to that for TNG. Group B patients also had a similar qualitative response to both interventions, but in contrast to group A patients, the interventions did produce alterations of segmental contraction pattern.

When the data points of group A and B patients were combined, they yielded best-fit lines with slopes reasonably close to identity and intercepts close to the origin regardless of the choice of dependent variable. This fact lends considerable support to our conclusion that, in these patients, the responses to each intervention were also quantitatively similar. This data confirms a recent report which described a good correlation between PESP and TNG in unmasking the
residual contractile ability of asynergic zones. In addition, use of NSEF as the measurement of segmental performance has enabled us to demonstrate that the induced changes are similar for all segments in a given ventricle regardless of the basal function of that segment and despite any differences in the magnitude or direction of the global response to each of the interventions.

As noted in table 1, we examined the hemodynamic data to identify any factors which might determine the pattern of segmental response. The only statistical difference between groups was in basal ejection fraction. Because group A patients had normal basal ejection fractions, it seems logical that neither intervention would cause a significant alteration in contraction. Patients in group B had abnormally low basal ejection fractions, thereby providing a margin for change. These results are similar to those of our earlier study of the ventriculographic response to coronary artery bypass surgery; patients with normal preoperative ejection fractions demonstrated no significant postoperative changes whereas patients with depressed preoperative ejection fractions demonstrated a significant improvement after surgery.

Although other investigators have noted that the timing of the postextrasystolic beat does affect the augmentation of contraction seen on the PESP beat, we could not detect any differences in the relative compensatory pause between the groups. In addition, we did not find a relationship between the relative compensatory pause and the change in end-diastolic volume or ejection fraction with PESP. These findings suggest that in the patients we studied, the length of the compensatory pause probably was not a significant factor in determining the similarity of induced changes by PESP and TNG. In this study we analyzed potentiated beats following spontaneous extrasystoles originating from unknown sites in the ventricles. Therefore we could not determine the possible effects of different sites of extrasystolic depolarization upon the changes seen with PESP in a particular segment. In preliminary experiments using open-chest dogs, we measured the area of the stress-strain loop as an index of segmental performance, and noted a relatively small (coefficient of variation = 10%) variability in the work performed by an anterior-apical segment of the LV when the preceding extrasystole was initiated at the same interval following the normal beat but from locations throughout both ventricles (Tyberg JV and Mathey D, unpublished observations). Given the intrinsic variability of the angiographic measurements, we suspect that such effects were not a significant factor in the changes in NSEF observed with PESP.

For the patients in group C, the changes in segmental contraction patterns following PESP and TNG were not similar. The grid plot of patient W. F. (fig. 5A) is consistent with a significant alteration following TNG but no change from the basal pattern with PESP. For patient J. G. (fig. 5B) the plot demonstrates significant alterations in contraction pattern after both interventions, but the segments which changed significantly were not the same. Patient W. G. (fig. 5C) demonstrated a significant alteration of contraction pattern with PESP and none with TNG. The individual hemodynamic data reveals that at the time of the TNG ventriculogram, the end-diastolic pressure was greater than its basal value for patient W. F. and unchanged for patient J. G. All patients in groups A and B had a lower end-diastolic pressure at the time of the TNG ventriculogram. Patient W. G. had a higher systolic pressure at the time of his TNG ventriculogram, a finding which was noted in only one group B patient (A. H.). Therefore, we suspect that in these patients the discrepancy between the induced changes may have been related to the functional state of the myocardium at the time of the TNG ventriculogram. Based on the results of our study, we would recommend that changes appearing after TNG should be interpreted with caution if both systolic and end-diastolic pressures are not decreased from basal levels at the time of the TNG ventriculogram.

Ten patients had a PESP beat on their post-TNG ventriculogram which enabled us to compare the changes in NSEF induced by PESP+TNG with those caused by PESP or TNG alone. When we analyzed the combined data from all ten patients, the slope of the best-fit line of the changes with PESP+TNG had a slope of 0.79 against changes with PESP and a slope of 0.87 against TNG. Since three of these patients were those in group C, we felt it was appropriate to also analyze the findings with their data points excluded. As noted in figure 7, this analysis yielded regression lines with slopes much closer to identity than the previous comparisons. This finding strongly suggested that in patients in whom the changes in segmental contraction pattern with PESP are similar to those with TNG, the changes with PESP+TNG will be identical to those seen with either intervention. Thus, our data supports the previously reported agreement between changes in asynergic segments after TNG alone and PESP+TNG. Furthermore, we have demonstrated the changes to be the same for all segments of the ventricle regardless of basal function and have documented the similarities of response to PESP, TNG and PESP+TNG in the same patients.

Since we performed our study with biplane ventriculography, we were able to examine the responses of segments seen in orthogonal views. Previous reports of the response to PESP, TNG, or both, have been based on single plane ventriculography and may not have detected changes which occurred in orthogonal projections. The importance of using biplane techniques in patients with coronary artery disease has been recently emphasized and confirmed in studies relating the extent of abnormally contracting segments to global ventricular function.

In summary, our study has demonstrated that in most patients, the use of either PESP or administration of TNG will induce the same change in segmental contraction pattern regardless of the initial function of any segment and despite any difference in the magnitude or direction of the global response to the intervention. Because the combination of PESP+TNG induces the same change as that produced by either intervention alone, the satisfactory application of one of these techniques should provide the clinician with sufficient data to assess the presence or absence of reversible asynergy. The choice of which intervention to be utilized may depend on the capabilities of the catheterization facility and the clinical status of the patient.

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