Sequence and Timing of Ventricular Wall Motion in Patients with Bundle Branch Block Assessment by Radionuclide Cineangiography

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SUMMARY We determined the sequence and timing of inward ventricular wall motion by least-square phase analysis of radionuclide cineangiograms in 10 patients with left bundle branch block (LBBB), five patients with right bundle branch block (RBBB) and 11 patients with normal conduction. All LBBB and RBBB patients had normal coronary arteries and no segmental wall motion abnormalities. The left ventricle (LV) was divided into eight segments and the right ventricle (RV) into three; sequence and timing were scored by three observers.

In normal subjects, wall motion begins in either or both ventricles and ends in the LV or both ventricles. In patients with LBBB it begins in the RV and ends in the LV; in patients with RBBB it begins in the LV and ends in the RV or both ventricles. The intraventricular wall motion is altered in the ventricle ipsilateral to a bundle branch block. In LBBB, the mean time of onset of LV wall motion is delayed 1.9 frames (38 msec), whereas RV wall motion is normal. In RBBB, the onset of RV wall motion is delayed 1.3 frames (26 msec), whereas LV wall motion is not delayed.

The sequence and timing of ventricular wall motion in normal conduction and bundle branch block, as represented by least-square phase analysis of radionuclide cineangiograms, conform with previous angiographic and electrophysiologic studies. Thus, this method may be useful in analyzing other conditions characterized by abnormal ventricular activation.

MUCH INFORMATION is available concerning the pathogenesis and electrophysiology of both right and left bundle branch block (RBBB and LBBB). Little information has been obtained on the mechanical correlates of these electrical abnormalities and even less concerning their effects on the sequence and timing of ventricular wall motion. We have used least-square phase analysis (LSPA) of equilibrium radionuclide cineangiograms to identify abnormal initial sites of ventricular activation. We now report the extension of this technique to an analysis of the sequence and timing of segmental ventricular wall motion in patients with bundle branch block.

Methods

Patient Selection Criteria

All patients with conduction abnormalities in the present study had a complete bundle branch block on a standard 12-lead ECG. Patients with isolated left hemiblocks or incomplete bundle branch block were excluded. RBBB was defined as QRS prolongation to 0.12 second or greater in conjunction with an S wave in leads I, V₅ and V₆ and an R' in leads V₁ and V₂. The criteria for LBBB included QRS prolongation to 0.12 second or greater with either a Q wave nor an S wave in leads I, aV₁, V₅ and V₆ and an RR' complex in leads V₅ and V₆. All patients with bundle branch block...
were required to have angiographically normal coronary arteries and no segmental wall motion asynergy on single-plane left ventriculography.

Eleven normal subjects served as controls. These subjects were required to have no clinical evidence of heart disease by history, physical examination, ECG and chest radiograph, and a normal conduction pattern. All normal subjects were also required to have subjectively normal left ventricular segmental wall motion and ejection fraction (LVEF) on radionuclide cineangiograms (RNCAs).

The study protocol was approved by the institutional Human Investigation Committee. All participants gave informed consent.

Radionuclide Cineangiography

Red blood cells were labeled in vivo with 25–35 mCi of technetium-99-m.® Equilibrium RNCAs were collected in the 30° right anterior oblique (RAO) and 45° modified (30° caudal tilt) left anterior oblique (LAO) projections with an Anger scintillation camera (LEM, Siemens Gammasonics, Inc.) interfaced to a dedicated minicomputer (Gamma-11, Digital Equipment Corporation).

The camera was fitted with a high-sensitivity, parallel-hole collimator with holes slanted at 30°. The acquisition software supplied by the manufacturer of the minicomputer was used to collect data in 21–37 20-msec, 64 × 64 frames at 3.4 × 10^6 counts/frame. All subjects were studied at rest during stable normal sinus rhythm.

LVEFs were determined from the LAO views of the RNCAs by a modification of the method originally described by Parker et al.® This modification has been previously validated in our laboratory against LVEFs determined by radiographic ventriculography. Regions of interest (ROIs) were drawn around the left ventricle (LV) at end-diastole (ED) and end-systole (ES) with a manually controlled cursor. The operator was aided by guide marks drawn around the stroke volume (ED – ES) image with the cursor. A background ROI was drawn adjacent to the inferior lateral quadrant of the LV ED ROI. The LVEF was then computed from the counts within the LV ED ROI and the counts within the LV ES ROI in the customary manner after subtraction of background counts normalized to the area of each ROI. The LVEF for each study was taken as the mean of three determinations by the same operator.

Least-square Phase Analysis

LSPA has been described.® Briefly, a cosine function was fitted to the ejection and rapid filling portions of the time-activity histogram of each pixel by a least-square technique. The parameters of the fit were the amplitude, phase, and average value of the cosine function. The period was taken as 500 msec if the heart rate during collection of data was 120 beats/min or less and was set equal to the period of the average cardiac cycle for faster heart rates. The curve was fitted from frame 1 of the raw data to an arbitrarily selected frame during rapid ventricular filling two to three frames after the nadir of the global LV time-activity curve. Thus, the diastasis portion of the cardiac cycle was not included in the determination of the cosine function. The phase of each fitted cosine function was taken to represent the time of onset of inward movement of that portion of the myocardium located within the associated pixel at ED.

Phase determined in this manner is largely independent of diastolic events, but does depend on the characteristics of the entire systolic interval. For example, variations in the systolic ejection interval or the isovolumic contraction interval will give rise to phase changes. However, we shall take these phase values to represent the time of onset of inward myocardial motion.

The phase information was displayed as a series of “isophase” frames in a manner similar to that described by Verba et al.® Each cosine function was divided into equal segments to correspond to the 20-msec intervals represented by the frames of the LSPA display. Each frame was displayed as the ED image of the heart with a pixel shown in black if the cosine function corresponding to the pixel reached its maximal value during the interval represented by that frame. The result is a series of functional images in which the onset of mechanical systole is represented in black as a wave of onset of inward movement sweeping over the atria and ventricles.

The statistical error in the determination of the phase of a time-activity histogram by LSPA is a function of the average value and amplitude. Thus, calculated phases are subject to large statistical errors in regions of the image in which average values or amplitudes are relatively low, such as extracardiac structures and akinetic myocardial segments. Display of the erroneous phase data in these regions is distracting and misleading. To suppress such data, ROIs were drawn by hand around the ventricles and data outside these regions were not displayed. Furthermore, the phase of a pixel was not displayed unless four or more adjacent pixels were in the same frame.

Interpretation of Phase Displays

The ventricles were divided into segments (fig. 1). The LV was divided into eight segments: anterobasal, anterolateral and apical in the RAO projection; superior lateral, inferior lateral, posterolateral, apical septal and basal septal in the LAO projection. The posterobasal and diaphragmatic segments of the LV were not included in the present analysis because of the overlapping position of the RV in the RAO projection during equilibrium-gated cardiac blood pool imaging. The RV was arbitrarily divided into three segments (inflow, outflow and apex). The RV inflow segment was seen only in the RAO projection because of the overlapping position of the RV apex and outflow segments in the LAO projection.

Throughout this report, “sequence” refers to the order of the onset of motion for a given segment in relation to other segments (i.e., first, second, . . . fi-
nal). "Timing" refers to the number of frames that have elapsed since the first detectable motion in any ventricular segment. Thus, between groups (normal, LBBB and RBBB), a segment could vary in sequence but not in timing, vary in timing but not in sequence, or vary in both sequence and timing.

The sequence and timing of all segments of both the RV and LV were determined by three independent observers. The first frame in which a given segment showed black pixels was considered to be the frame in which motion first began for that segment. The frame number of initial motion for each segment was recorded by the observers, and disagreements were settled by majority vote. Of the 286 total ventricular segments to be analyzed, all three observers agreed on the frame of onset for 130 segments and two of three observers agreed in an additional 134 segments. In 22 segments, there was no agreement between observers; these segments were excluded from the analysis. The interobserver agreement was highly significant (kappa 0.51, p < 0.001).12

Since the trigger for the ECG gating was the peak of the R wave, the patients with conduction defects could have an onset of wall motion not coincident with the first frame of the display. The data were therefore normalized such that the frame demonstrating the initial wall motion (regardless of which ventricle it was located in) was designated frame 1, and all subsequent frames were numbered consecutively. From this segment-by-segment analysis, the sequence of onset of ventricular wall motion for each subject was determined, including both the initial and final segments overall as well as the initial and final segments within each ventricle.

Statistical Analysis

Values are mean ± SEM. The sequences of ventricular wall motion were compared by chi-square test13 or Fisher's exact test.14 Concerning the timing of motion (mean frame of onset), a two-tailed t test for unpaired data was used for intergroup comparisons and a two-tailed t test for paired data was used for intragroup comparisons.

**Results**

Subjects

There were 11 normal volunteers, mean age 34 years (range 24–50 years). Ten patients with LBBB were studied; their mean age was 57 years (range 46–64 years). Five had completely normal hemodynamics and LVEF and five had cardiomyopathies, globally reduced LV wall motion and LVEFs of 12–44%. Five patients with RBBB were studied, mean age 59 years (range 45–73 years). All five had normal LV function.

Least-square Phase Analysis Display

An LSPA display from a subject with normal conduction is shown in figure 2. Wall motion begins in the RV (RAO, frame 1; LAO projection, frame 2) and then spreads to the RV apex (RAO and LAO, frame 4); the latest onset occurs in the RV outflow tract. The LV in this example begins to move slightly later than the RV, in the anterolateral and anterobasal segments (RAO, frame 2); wall motion quickly spreads to involve the remainder of the LV. In the RAO view, the final area of onset of LV wall motion appears at the LV apex, while in the LAO view the onset of wall motion is seen slightly later in the paraseptal region.

Figure 3 shows the LSPA display of a patient with RBBB. Wall motion begins in the superior lateral segment of the LV in frame 1 of the LAO projection and spreads as a wave around the posterolateral surface of the LV (LAO view) and down the anterior wall toward the apex (RAO view). The earliest movement in the RV, however, does not appear until frame 2 (near the RV apex, LAO view), and most of the RV wall does not begin to move until the latter part of systole in both RAO and LAO projections.

The LSPA display of an LBBB patient with normal LVEF is shown in figure 4. In the LAO view, wall motion starts in the paraseptal and apical areas of the RV and progresses normally toward the RV outflow tract. The onset of LV wall motion can most clearly be seen in the third frame of the LAO and RAO views and appears to spread across the septum from the RV.
proceeding across the base and apex to end in the posterolateral LV.

Sequence of Wall Motion

Overall Sequence of Motion

Initial overall ventricular wall motion, when both ventricles were considered together, included segments of the LV in normal subjects (N) and RBBB patients, but in only two LBBB patients (N eight of 11, RBBB five of five, LBBB two of 10; \( \chi^2 = 10.51, \text{df} = 2; p < 0.005 \)); initial overall wall motion included segments of the RV in most normal subjects and LBBB patients but in only one RBBB patient (N nine of 11, RBBB one of five, LBBB nine of 10; \( \chi^2 = 9.04, \text{df} = 2; p < 0.01 \)). Thus, in most normal patients, initial wall motion included segments from both ventricles; wall motion begins in the LV in patients with RBBB and in the RV in patients with LBBB.

The final segments to begin moving were located in the LV in most subjects from all groups (N 11 of 11, RBBB four of 5, LBBB 10 of 10; NS). In contrast, the final segments to begin moving included the RV in five patients with RBBB, in four of 11 normal subjects and in none of the patients with LBBB (\( \chi^2 = 15.55, \text{df} = 2; p < 0.001 \)).

Sequence in the Left Ventricle

Both ventricles were also analyzed separately for intraventricular variation in the sequence of onset of wall motion in the three groups. The anterobasal, basal septal, and superior lateral LV segments began to move first in most subjects from all groups (N 11 of 11, RBBB five of five, LBBB seven of 10; NS). The apical and apical septal segment were the last to begin moving in most normal and RBBB subjects. However, the apical segments were rarely among the last to begin moving in patients with LBBB (N 10 of 11, RBBB four of five, LBBB two of nine; \( \chi^2 = 10.83, \text{df} = 2; p < 0.005 \)). (The apical segments could not be evaluated in one patient with LBBB.) In LBBB patients, the final segments included the posterolateral and inferolateral segments (N four of 11, RBBB two of five, LBBB nine of 10; \( \chi^2 = 6.96, \text{df} = 2; p < 0.03 \)). Thus, the sequence of wall motion in the LV was similar in normal subjects and patients with RBBB, beginning in the base and ending in the apex. In contrast, the sequence within the LV was markedly different in patients with LBBB: initial wall motion included the basal areas as it did in the other groups, but the apical areas began to move in the early or intermediate frames, and the latest areas to begin moving were the posterior and inferior lateral segments.

Sequence in the Right Ventricle

The initial segments of the RV to move most often included the lower portion of the RV outflow tract (fig. 1) in normal subjects and LBBB patients, but less often in RBBB patients (N nine of 11, RBBB two of five, LBBB seven of 10; NS); in RBBB patients, the RV apex was among the first RV segments to move (N three of 11, RBBB five of five, LBBB four of 10; \( \chi^2 = 7.56, \text{df} = 2; p < 0.01 \)). The final segments in the RV to begin moving were not significantly different among the three groups.

Timing of Ventricular Wall Motion

The mean frame during which onset of motion of each ventricular segment occurred in the two groups of patients with bundle branch block was compared with that of the normal subjects (figs. 5 and 6). In the 10 LBBB patients, there was no significant delay in the time of onset of RV wall motion for any of the three RV segments. There was, however, a significant delay (\( p < 0.05 \)) in six of eight LV segments; the exceptions were the apical and apical septal segments. For the LBBB patients, the mean delay for all LV segments compared with the respective segment of the normal group was 1.9 frames (38 msec). Five of the 10 patients with LBBB had normal global function (LVEF > 0.55) and five had a depressed LVEF. When compared with controls, the movement of LV segments significantly delayed for the entire LBBB group was also significantly delayed in each of the subgroups. However, the magnitude of the delay was greater in the group with depressed LVEF (2.4 frames, or 48 msec, vs 1.5 frames, or 30 msec; \( p < 0.025 \)).

A similar analysis of the subjects with RBBB (fig. 6) revealed no significant difference from the normal subjects in the time of onset of wall motion for seven of eight LV segments. The superior lateral LV segment

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**Figure 3.** Least-square phase analysis display of radionuclide cineangiograms from a patient with right bundle branch block. RAO = right anterior oblique; LAO = left anterior oblique.

**Figure 4.** Least-square phase analysis display of radionuclide cineangiograms from a patient with left bundle branch block. RAO = right anterior oblique; LAO = left anterior oblique.
two independent methods of analysis, concluded that the middle inferior LV wall began to move 25 msec before the apex and the middle anterior LV wall 18 msec before the apex. Because of the overlying position of the RV in the RAO projection of the equilibrium-gated RNCAs, we could not visualize the middle inferior wall. The anterolateral segment (middle anterior wall) began to move an average of 0.9 frame earlier than the apex, which would correspond to a delay of 18 msec for the apex, identical to that found angiographically (figs. 5 and 6). Similar results were also reported by Leighton et al.,17 who used single-plane RAO left ventriculography in normal patients.

Very little information is available concerning the sequence and timing of the onset of RV wall motion in either normal persons or patients with conduction abnormalities. Testelli20 observed a delay of 20–30 msec in the onset of the pressure rise in the RV in four patients with transient RBBB during cardiac catheterization. Although the delayed pressure rise is not identical to a delay in wall motion, it would be reasonable to assume that they would be related, since the pressure increase is a function of wall motion. The delay of 20–30 msec in pressure corresponds well to the mean delay of 26 msec in onset of motion found in the present study.

Haft, Herman, and Gorlin1 examined the timing of LV contraction in patients with LBBB and various underlying cardiac diseases. They found a mean delay of 35 msec in the onset of LV wall motion in patients with LBBB, which is similar to the mean delay of 1.9 frames (38 msec) in our patients with LBBB. The LV contraction patterns in their patients without coronary artery disease or cardiomyopathy were considered normal, in contrast to the results of the present study. However, these authors were primarily concerned with asymmetry and asynergy rather than ventriculographic

Discussion

Using a least-squares method of curve fitting to the time-activity histograms of individual pixels of equilibrium-gated radionuclide cineangiograms, we found significant differences in the sequence and timing of ventricular wall motion in patients with bundle branch block compared with normal subjects. Since all of the subjects with bundle branch block had normal coronary arteries on coronary angiography and no segmental wall motion asynery on left ventriculography, we believe that the differences observed are due to altered sequences of activation rather than to primary myocardial disease. Our results are consistent with those from angiographic1, 15–17 and electrophysiologic mapping studies.2–4, 18, 19

Comparison with Angiographic and Hemodynamic Studies

Using cineangiography and cineradiography of epicardial markers in normal humans, McDonald15 demonstrated that initial LV movement occurred during the prejection phase of systole and involved a descent of the basal areas toward the apex that continued during the ejection phase. This finding is consistent with ours in that LV wall motion in patients with normal conduction began in the anterobasal, basal septal and superior lateral segments.

In a single-plane RAO angiographic study of subjects with normal left ventricles, Clayton et al.,16 using

Figure 5. Mean frame of onset of wall motion for each ventricular segment in subjects with normal conduction and patients with left bundle branch block (LBBB). IN = inflow; OUT = outflow; other abbreviations as in figure 1.

Figure 6. Mean frame of onset of wall motion for each ventricular segment in subjects with normal conduction and patients with right bundle branch block (RBBB). IN = inflow; OUT = outflow; other abbreviations as in figure 1.
evidence of a difference in the sequence of segmental wall motion.

Thus, the results of this noninvasive study are consistent with those of angiographic studies. Further, our study extends these studies to include segmental RV wall motion.

Comparison with Electrophysiologic Studies

Our hypothesis was that the sequence and timing of ventricular wall motion should be altered predictably by abnormal patterns of ventricular activation. For this to be true, the pattern and timing of wall motion as depicted by LSPA should conform to the known patterns of ventricular activation during both normal and abnormal conduction.

The most extensive analysis of the activation of the normal human heart has been the study of seven isolated hearts by Durrer et al. These investigators found that there was considerable variability in sequence between hearts, a phenomenon also noted in the present study. The earliest RV activity occurred in the area pretrabecularis, near the insertion of the anterior papillary muscle, which corresponds to our finding of earliest RV wall motion in the lower portion of the RV outflow segment. Durrer et al. found that the earliest LV activity occurred in three locations in the anterior basal and the anterior and posterior paraseptal areas. These observations correspond to our finding of the earliest LV wall motion in the anterobasal, basal septal, anterolateral, and superior lateral segments (figs. 5 and 6) during normal conduction. Wyndham et al. examined epicardial activation in 11 patients with normal conduction during cardiopulmonary bypass. Despite interpatient variability, patterns emerged consistent with the data of Durrer et al. All patients had early epicardial breakthrough in the RV paraseptal region, which corresponds to our observation of earliest wall motion in the lower RV outflow segment in nine of 11 normal subjects. Early breakthrough sites in the LV were found in the anterolateral wall, which correspond to the early LV wall motion in the anterobasal segment in our patients. An additional site of early breakthrough was noted in the inferior RV in 10 of 11 of their patients an average of 16 msec after the anterior RV breakthrough. This difference would not be detectable in the present study, given the time resolution of our technique, and we could not confirm a mechanical correlate to this finding.

Thus, for the areas visible during equilibrium-gated RNCA and within the time resolution achieved in the present study, the sequence of wall motion determined by the LSPA technique corresponds to the sequence of activation determined electrophysiologically for normal conduction for the sites of earliest activation. However, the sites of latest electrical activation within the LV were most commonly seen in the posterobasal and posterolateral areas during normal conduction. In our study the latest segments to begin moving were in the apical areas; although this finding is consistent with the angiographic studies mentioned previously, it differs from the electrophysiologic data. This difference may be due to the inability of the equilibrium-gated technique to evaluate the posterobasal segment in the views obtained because of the overlying position of the RV, as well as the small numbers of patients examined and interpatient variability observed with all three techniques. In addition, all patients in the study of Wyndham et al. had significant coronary artery disease, which may have led to some difference from the normal pattern.

Kastor et al. used electrode catheters to determine the sequence of endocardial activation in patients with RBBB and in normal subjects. In the patients with RBBB, activation of the three RV areas (inflow, outflow and apex) was significantly delayed. The timing of activation of the LV apex was normal in patients with RBBB, and activation of the LV outflow tract occurred earlier than normal. In the present study, the onset of RV wall motion was delayed in the RV inflow and outflow segments, but did not appear to be delayed in the RV apex. In the study of Kastor et al., the mean delay was 26 msec in the RV inflow tract and 38 msec in the RV outflow tract; these values correspond well to the mean delays we observed (1.4 frames, or 28 msec, for the RV inflow and 2.1 frames, or 42 msec, for the RV outflow segment) (fig. 6). The early activation of the LV outflow tract with RBBB may well correspond to the early wall motion seen in the superior or lateral LV segment of the present study. Wyndham et al. described the pattern of epicardial activation in three patients with RBBB and noted normal LV activation, which is consistent with the results of the present study. Absence of the normal right paraseptal breakthrough seen in their mapping corresponds to the delayed wall motion of the RV outflow tract in the present study.

Wyndham et al. also described epicardial activation in five patients with LBBB who underwent coronary artery bypass grafting. In these patients, the sequence of RV activation was comparable to that of normal subjects; in the present study the sequence of RV wall motion in the LBBB patients did not differ from that in the normal subjects. Mapping demonstrated that the LV was activated by a wave of depolarization that spread across the septum and that the latest LV activation occurred in the posterobasal and posterolateral areas. In the present study, the latest areas to begin moving were in the superior, posterior, and inferior lateral LV segments (figs. 4 and 5). Electrophysiologically, terminal LV activation in LBBB was delayed by 54 msec compared with normal controls; as depicted by LSPA, terminal LV wall motion in LBBB (inferolateral segment, fig. 5) was comparably delayed for the entire group (3.2 frames, or 64 msec).

Thus, LSPA of equilibrium-gated RNCA demonstrates a sequence and timing of wall motion in patients with bundle branch block that is quite similar to the pattern of activation demonstrated by electrophysiologic techniques.

Limitations of the Technique

The time resolution of LSPA is limited by statistical error and is a function of the number of counts collected for the RNCA and the amplitude of regional wall
motion. In the current study, display of the results of LSPA in frames shorter than 20 msec was not justified. This limitation of time resolution made it impossible to explore the mechanical correlates of the more subtle electrophysiologic observations. However, major features of the electrophysiologic studies of both normal and abnormal conduction were found to have correlates in the sequence and timing of wall motion, and the angiographic data were closely reproduced. The major differences between the timing of wall motion as depicted by this study and activation as shown by the electrophysiologic data were observed in the apical and apical septal segments. The overlying position of the RV and LV in the RAO projection and the difficulty in orienting the LAO projection precisely parallel to the septum may have led to some difficulty in correctly separating RV and LV motion in this region. Inability to visualize the inferior wall of the LV in the RAO projection is a limitation of the equilibrium-gated technique that might be overcome by obtaining a left posterior oblique view or using a first-transit technique.

Theoretically, phase shifts are not uniquely associated with the onset of ejection, for the phase of the fitted cosine is affected by data from all portions of the cardiac cycle included in the analysis. Diastolic phenomena may have a large effect on the calculated phase if diastasis is included in the data analysed. LSPA does not require analysis of the full cycle; it can be applied to any segment of a time-activity curve if the period of the fit is specified. (The only limitation in the number of frames excluded is that statistical errors become larger as the number of frames fitted becomes smaller.) In our study, all data after the first 60–100 msec of rapid ventricular filling were excluded from the fit. Thus, diastolic phenomena could have had little effect on our results.

Prolongation of the systolic ejection period causes a global phase delay if the period is not included as a parameter of the fit. While this would affect the overall timing of the onset of wall motion, we would not expect prolongation of the ejection period to affect the underlying sequence of wall motion in the absence of an abnormality of electrical conduction. Because our results compare favorably with the results of electrophysiologic mapping, the differences in sequence and timing of wall motion described by the present technique reflect the underlying differences in electrical activation in patients with bundle branch block.

Conclusions

Our data suggest that LSPA of gated-equilibrium RNCAs provides a reliable representation of the sequence and timing of wall motion in patients with normal conduction or bundle branch block. Phase analysis of RNCAs yields data on ventricular movement that conform with angiographic data. Furthermore, in both sequence and timing, the changes in wall motion due to bundle branch block parallel changes in activation shown by electrophysiologic studies. We reported the use of this method to identify the site of ventricular activation during pacing and ventricular tachycardia. The least-squares technique may prove useful in studying other disorders of ventricular activation such as atrioventricular nodal bypass tracts. It may also be applied to gated rest and exercise equilibrium RNCAs to localize coronary obstruction based on a change in sequence or timing of movement of ventricular segments.

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