Body Surface Distributions of Repolarization Forces During Acute Myocardial Infarction

I. Isopotential and Isoarea Mapping

DAVID M. MIRVIS, M.D.

SUMMARY Although ST-segment abnormalities during acute myocardial infarction are clinically important, the total thoracic distribution of these repolarization potentials has not been reported. To provide this information, 24 patients with acute myocardial infarction were studied. Isopotential body surface maps were constructed from potentials sensed by 150 anterior and posterior electrodes. Patterns from 12 patients with anterior lesions demonstrated the appearance of repolarization potentials 21.3 ± 4.6 msec before the end of the QRS complex. During the ST segment, potential distributions were characterized by a single anterior maximum that remained fixed in location but increased in intensity as repolarization progressed. Distributions in the remaining subjects with inferior lesions were analogously characterized by (1) the onset of repolarization 34.6 ± 12.4 msec before termination of the QRS complex and (2) a single anterior minimum located on the left anterior superior thorax, with positive potentials distributed around the lower thoracic margins. These data suggest that electrocardiographic changes after acute myocardial infarction include (1) marked overlap between activation and recovery patterns and (2) isopotential surface patterns with relatively simple topographic configurations, such as expected of a single-dipole equivalent cardiac generator.

THE ELECTROCARDIOGRAPHIC ST segment after myocardial infarction is significant for three reasons. First, it is the pattern during this period that may identify the acute phase of the disorder. Second, the distribution of abnormalities in the various ECG leads serves to approximate the cardiac locus of the ischemic damage. Third, the magnitude of the ST-segment deviation has been proposed as a measure of the extent of the ensuing myocardial infarction.

Methods of investigating these abnormalities include standard electrocardiography, vectorcardiography and, most recently, multielectrode grids placed upon the left precordial body surface. Each approach assumes that all relevant electrocardiographic information is sampled by the recording system. In each case, however, deficiencies may be identified. Both standard and multilead precordial systems depend on topographic proximity to the myocardial lesion and to the bulk of the generated electrical field. All lesion loci could not be expected to underlie such fixed location systems. Vectorcardiography, though relatively location-independent, is primarily sensitive to forces generated by a single-current dipole; it has been documented that nondipolar forces, such as generated by greater than one

From the Section of Medical Physics, Division of Circulatory Diseases, Department of Medicine, University of Tennessee Center for the Health Sciences, Memphis, Tennessee.

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Address for correspondence: David M. Mirvis, M.D., Section of Medical Physics, University of Tennessee Center for the Health Sciences, 956 Court Avenue, Room 2F18, Memphis, Tennessee 38163.

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cardiac wave front, may exist during much of the cardiac cycle.11,12

A fourth approach is to record ECG potentials from the entire thoracic surface and to display the sensed information as isopotential distributions.13-18 Thus, myocardial patterns projected to all parts of the thoracic torso may be detected. Additionally, such methods are capable, within limits, of evaluating multiple, simultaneously active cardiac effects and may thereby define the characteristics of the operative cardiac generator.14-18 Application of surface mapping techniques to the study of repolarization forces generated by acute ischemic lesions in man may therefore be advantageous. In this report, we describe our initial results using this process.

Methods

Study Population

Twenty-four men, ages 46–63 years, were studied during the first 48 hours after the onset of acute myocardial infarction. All were hospitalized in the Circulatory Care Center of the City of Memphis Hospital, where the diagnosis of acute infarction was confirmed by standard historical, electrocardiographic and enzymatic methods.

All patients were clinically stable at the time of study, had no more than mild congestive heart failure and were in sinus rhythm without bundle branch block. None were receiving digitalis glycosides or antiarrhythmic agents other than i.v. lidocaine, which was given routinely at this institution. Voluntary informed consent was granted before study.

Electrode System

Electrocardiographic signals were recorded from 150 chloridized silver disc electrodes placed on the anterior (100 electrodes) and posterior (50 electrodes) chest from the level of the clavicles to the inferior rib margin. Additional electrodes were located on the distal extremities to record the standard bipolar and unipolar limb leads, and to derive the Wilson central terminal potential.

Data Acquisition

ECG signals were amplified by a set of 33 dual-stage, low-noise (4 μV, peak-to-peak), differential, capacitor-coupled amplifiers.19 The first stage, including a differential input (grid electrode potential vs Wilson central terminal voltage) and a fixed gain of 1000, was located at the patient’s bedside. Thus, critically ill patients could be studied without transporting them to the research laboratory. The outputs of these amplifiers were transmitted via a 600-foot, shielded, multiconductor cable to the central electrocardiologic laboratory. There, signals were amplified by a bank of differential (output of first-stage amplifier vs remote patient room ground), variable gain and offset amplifiers. The gain of each amplifier was set at 1-16 and the offset at −4.5 to +4.5 V under computer control so that its output filled the input range of the analog-to-digital converter (−5.0 to +5.0 V). Thus, final gains varied from 1000–16,000.

ECG signals were acquired in five sets. Each set consisted of 30 electrode potentials plus standard leads I, II and III. These three standard leads, recorded with each of the five data sets, served to verify the stability of the recordings during acquisition of data and to document the correct merging of the data sets.

A 20-second sample of each of the five electrode groups was digitized on line at a sampling rate of 500 samples per channel per second. Baseline stability was carefully determined during data acquisition by oscillographic observation of ECG records. The nine-bit samples were recorded on digital magnetic tape for later off-line processing.

Data Processing

The five data sets were merged and individual grid electrode wave forms were averaged to form one set of 150 unipolar thoracic ECGs. Details of the methods used have been published elsewhere.20 Briefly, data sets were time aligned using a hardwired QRS trigger whose output was recorded along with the ECG data.

Beats to be averaged were chosen by an automated numerical routine comparing one PQRST wave form with another, and yielding a value or “wave form index” of unity if the two were identical; cycles with indexes of 0.8–1.2 were selected for averaging. Data sets with significant baseline wander were excluded. An example of the raw and averaged wave forms from three left precordial leads is presented in figure 1, showing the low noise content of the transmitted signal and the effect of the averaging upon it.

Isopotential maps were constructed from the averaged complexes. Onsets and offsets of the QRS complex, ST-T interval and TP segment were manually determined from plots of root-mean-square (RMS) potential, and maps were drawn at 2-msec intervals throughout the QRS period. Data points in a terminal 50-msec period of the TP segment were averaged and served as a baseline. A combined linear-bilinear interpolation routine was used to draw contour lines at predetermined potential levels (10, 20, 40, 60, 100, 200, 400, 600, 1000, 2000, 4000 and 6000 μV), which permitted adequate visualization of patterns during periods of low as well as high sensed voltages.21 Finally, isopotential maps were displayed in video form and photographed by a computer-controlled microfilm assembly.

Data Analysis

Isopotential patterns were studied both visually and quantitatively. Potentials were identified as emanating from ventricular excitation or recovery using the criteria described by Taccardi22 and used by Spach et al.22 Accordingly, surface forces beginning during the QRS complex but persisting into the ST segment or first appearing during the ST-T interval were classified
as being related to ventricular repolarization. Those evolving during the QRS complex but vanishing before or just at the onset of the ST segment were considered manifestations of ventricular depolarization.

**Results**

Subjects were divided into two major groups, based upon the localization of injury currents in the standard ECG. Twelve subjects had inferoposterior lesions (ST-segment elevation in standard leads II, III and aVF, with or without ST-segment depression in right precordial leads). Twelve others had anterior abnormalities, including patterns typically described as anterior, anteroseptal and anterolateral (ST-segment elevation in precordial leads V₁ to V₆, with or without ST-segment elevation in leads I and aVL). QRS complexes measured 84.2 ± 4.6 msec.

**Figure 1.** Examples of single PQRST wave forms from three right precordial electrodes as transmitted from the bedside to the ECG laboratory. Raw wave forms (left) and the complex resulting from averaging 18 complexes (center) show the low noise of the transmitted signals. The wave form index (WFI) (right) was calculated by comparing the first recorded, unaveraged cycle with each subsequent one in the lead; the constancy of the index near unity documents the reproducibility of the signal.

**Figure 2.** Isopotential body surface maps from one subject with an acute anterior myocardial infarction constructed from potentials sensed 30 hours after the onset of symptoms. Plus and minus signs corresponding to the polarity of the sensed voltage locate the electrode grid locations. The sternal notch is indicated by V and the six standard precordial electrode sites are marked by solid circles; the left and right edges of the maps are the midvertebral line. Contour intervals are drawn at plus and/or minus 10, 20, 40, 60, 100, 200, 400, 600 μV, etc. The zero isopotential line is overdrawn. Timing of each map is indicated by the fiducial marking on standard lead II in the upper right corner of each panel. (A) Pattern 24 msec before the end of the QRS, with peak positive and peak negative potentials of 1050 and -1801 μV, respectively. (B) Pattern 18 msec before the J point, with peak voltages of 543 and -923 μV. (C) Distribution 12 msec before end of QRS. The peak positive potential is 343 μV and the peak negative voltage is -492 μV. (D) Pattern 6 msec before the J point; maximum and minimum voltages are 168 μV and -277 μV, respectively.
Figure 3. Isopotential patterns for the same subject as in figure 2 and using the same conventions. (A) At the J point, the peak positive potential is 124 μV, and the peak negative potential is −102 μV. (B) At 10 msec into the ST-T interval, the maximum voltage is 166 μV and the minimum potential is −78 μV. (C) At 40 msec the maximum voltage is 263 μV and the minimum voltage is −74 μV. (D) At 80 msec, the maximum potential is 349 μV, and the minimum potential is −86 μV.

Anterior Myocardial Infarction

Isopotential maps depicting the patterns of repolarization potentials in one subject with an anterior myocardial infarction are presented in figures 2-4. All maps are timed relative to the J point, with negative values indicating instants during the QRS complex and positive values indicating points during the ST-T interval.

As all subjects displayed similar patterns, data from

Figure 4. Isopotential distributions at points 140 msec (A), 200 msec (B), 220 msec (C) and 260 msec (D) into the ST-T interval from the same subject and using the same markings as in figures 2 and 3. Maximal positive and negative voltages are: (A) 461 and −92 μV; (B) 389 and −158 μV; (C) 228 and −234 μV; (D) 44 and −219 μV.
one representative study will be presented. Data were recorded 30 hours after the onset of symptoms. The earliest evidence of repolarization forces was detected 24 msec before the end of the QRS complex, which lasted 82 msec (fig. 2A). Positive potentials, previously confined to the left axillary and paravertebral areas, migrated rightward, with the zero level line overlying the $V_2$ electrode site. Subsequently, these positive forces continued to expand over the left anterior chest as negative potentials, due to continuing ventricular excitation, were “pushed” down to lower portions of the thorax (figs. 2B-D). Thus, by 6 msec before the J point (fig. 2D), a maximum existed near the $V_2$ and $V_3$ electrode sites.

During the ST segment (fig. 3), this anterior maximum remained stationary in location but progressively increased in magnitude. At 0, 10, 20, 40, 60 and 80 msec into the ST segment, peak positive potentials were 124, 166, 212, 263, 310 and 349 $\mu$V, respectively. Little change, however, occurred in the locations of these peak positive potentials, all being recorded from an electrode near the $V_2$ site.

Later in the ST-T interval (fig. 4), changes in negative potentials dominated. A midprecordial maximum surrounded by inferoposterior negative potentials was observed 140 msec after the J point (fig. 4A). The negative potentials over the left lateral precordium subsequently increased in strength (fig. 4B) and moved rightward and superiorly during the T wave. Thus, by 220 msec (fig. 4C), the anterior minimum equaled the strength of the maximum (~234 $\mu$V vs 228 $\mu$V). Coincident with the development of an anterior minimum, the previously described maximum migrated rightward and decreased in strength. Thus, by the descent of the T wave, the anterior minimum dominated the precordial isopotential patterns.

All 12 patients with anterior infarction had similar patterns, although the locations of the anterior maximum varied from $V_1$ to $V_4$ in accordance with the laterality of the injury currents recorded on the standard ECG. The overlap between excitation and recovery forces was 16–28 msec (21.3 ± 4.6 msec, mean ± sd). In all cases, only a single maximum and/or minimum was observed at any single instant during the ST-T interval.

Potentials from grid electrodes making up the 35-electrode subset described by Maroko et al. were analyzed. The sums of all positive potentials in the 150-electrode set and in the 35-electrode set were calculated and the ratio of the latter to the former was computed. The left anterior precordial grid sensed 51.1 ± 20.6% and 51.5 ± 18.5% of total thoracic positive voltages 40 msec and 80 msec into the ST segment, respectively. The percentages detected varied from 15.6–69.9% and from 18.7–62.2% at these two instants.

**Inferior Myocardial Infarction**

Isopotential patterns at selected instants during the QRS complex and the ST-T interval from one subject with an acute inferior myocardial infarction are presented in figures 5–8. These data were registered 28 hours after the onset of symptoms. Sixty milliseconds before the end of the QRS complex, which lasted 92 msec, the pattern was dominated by an anterior minimum and a left posterior maximum (fig. 5A). Subsequently, the minimum migrated superiorly, as positive potentials surrounded the lower aspects of both the anterior and posterior thorax (figs. 5B and 5C). By 20 msec before the J point, the anterior minimum moved superiorly and to the left; positive potentials dominated the inferior thoracic surfaces, and negative potentials dominated the superior thoracic surfaces (fig. 5D).

This pattern continued throughout the first 80 msec of the ST segment (figs. 6A-C). The center of the anterior minimum was near the left clavicle, and positive potentials were spread along the inferior thoracic border. The location and strength of the minimum changed only slightly during the initial portions of the ST segment. For example, in the subject whose patterns are presented, peak negative voltages 10, 20, 40 and 60 msec into the ST segment were -319, -260, -287 and -278 $\mu$V, respectively.

All subjects in this group had similar ST-segment patterns. The presence or absence of ST depression in the anterior precordial leads correlated directly with the location of the ST-segment anterior minimum. In all cases, only one maximum and/or minimum was observed at a given instant. The overlap between activation and recovery forces was 28–54 msec (34.6 ± 12.4 msec), and was not significantly greater than that in the anterior infarction group ($p > 0.1$, unpaired $t$ test). Only 23.2 ± 14.1% of total thoracic positive potentials were registered by the left anterior precordial grid system described above, which was significantly less than that sensed in anterior infarction ($p < 0.01$, unpaired $t$ test).

Two patterns were observed later in the ST segment and during the T wave. Eight subjects had the pattern depicted in figures 6D and 7. By 100 msec into the ST-T interval, low anterior positive forces had begun to migrate superiorly (fig. 6D). At 140 msec, a discrete anterior midline maximum evolved alongside the lateralized anterior minimum (fig. 7A). This minimum subsequently moved inferiorly (fig. 7B) and later spread to engulf the anterior and posterior lower thorax with negative potentials (fig. 7C). The superior thorax was now positive in polarity — a pattern opposite to that observed earlier in the ST segment (figs. 6B-D). This two-layered distribution persisted until the end of the T-wave. Standard ECGs in these patients showed inverted T waves — the coved plane pattern described by Rothschild et al. — in the inferior leads.

The remaining four patients, all with upright T waves in the inferior leads, had the pattern shown in figure 8. These data were recorded 20 hours after onset of symptoms. An anterior maximum formed late in the ST segment (fig. 8A), as previously portrayed (figs. 6D and 7A). However, this maximum failed to spread superiorly during the T wave, and the left posterior minimum did not migrate inferiorly (figs. 8B
ISOPOTENTIAL MAPPING AFTER MI/Mirvis

FIGURE 5. Patterns from a subject who had an acute inferior myocardial infarction. Symptoms began 28 hours before study. (A) Distribution 60 msec before the J point, with peak positive and negative voltages of 333 and -1103 μV, respectively. (B) Distribution 54 msec before the J point, with peak voltages of 237 and -679 μV. (C) Distribution 40 msec before the end of the QRS: peak positive potential is 273 μV and peak negative potential is -412 μV. (D) Distribution 20 msec before the J point, with maximum and minimum voltages of 184 μV and -319 μV, respectively.

FIGURE 6. Isopotential distributions from the same subject as in figure 5. (A) At the J point, the peak positive potential is 234 μV and the peak negative potential is -266 μV. (B) At 10 msec into the ST-T interval, the maximal positive voltage is 184 μV and the peak negative voltage is -319 μV. (C) At 20 msec, the peak positive potential is 194 μV and the peak negative potential is -260 μV. (D) At 100 msec, the peak positive voltage is 194 μV and the peak negative voltage is -147 μV.
Distributions from the same patient as in figures 5 and 6, at instants 140–220 msec into the ST-T interval. Peak positive and negative voltages, respectively, are: (A) 382 and −132 μV; (B) 538 and −152 μV; (C) 607 and −177 μV; and (D) 316 and −160 μV.

Isoarea Distributions

Areas under the QRST interval in each of the 150 grid electrode wave forms were computed using Simpson's method for numerical integration. Results, expressed as millivolt-milliseconds, were processed in a manner analogous to that described for potentials and displayed as isoarea maps. Eighteen subjects had isoarea patterns characterized by a single site of peak positive and peak negative area. One example, from a patient with an inferior infarction, is presented in figure 9A. Locations of the maxima and minima varied, as expected, with the relative polarities and magnitudes of the QRS and C) as it did in the prior group (figs. 7B–D). Thus, the anterior chest remained engulfed with positive potentials during the T wave as in the late ST segment, with negative potentials persisting over the superior posterior chest.

Isopotential patterns from a second subject with onset of acute inferior myocardial infarction 20 hours before study. (A) Distribution 80 msec into the ST-T interval, with peak positive and negative voltages of 217 and −26 μV, respectively. (B) Distribution 120 msec into the ST-T interval, with a peak positive voltage of 270 μV and a peak negative potential of −48 μV. (C) Pattern 160 msec after J point; the peak positive potential is 245 μV and the peak negative potential is −74 μV.
ST-T abnormalities. In the remaining six patients (four with anterior and two with inferior lesions), isopolar distributions revealed dual maxima and/or minima. An example of a nondipolar pattern from a second patient with an inferior infarction is shown in figure 9B.

Discussion

Four significant concepts emerge from the presented data. First, repolarization forces were detectable as far as 54 msec back into the QRS complex. Overlaps between the end of cardiac activation and the onset of repolarization have been documented in dogs during the terminal portions of the QRS complex, and surface mapping techniques have demonstrated similar findings in man.

The mean values for the overlap between depolarization and repolarization potentials for both anterior and inferior lesions exceed the normal values published by Spach et al.; average values of less than 5 msec would have been anticipated for subjects over age 40 years rather than the measured quantities of 21.3 and 34.6 msec for anterior and inferior lesions, respectively. Although formal statistical comparison with age-matched controls was not possible from the reported data, the early onset of repolarization potentials may be characteristic of isopolar patterns after acute myocardial infarction. A similar overlap of injury currents and activation forces has been reported by Taccardi in an isolated canine model.

Experimental studies have provided a basis for this clinical observation. Transmembrane action potentials from ischemic tissues are characterized by reduced amplitude and duration relative to those recorded from normal tissues. Thus, soon after activation, ischemic tissue is more negative than surrounding tissue due to reduced amplitude of phase 0 and to increased slope of recovery phases. Current flows toward the ischemic zone, generating true ST-segment elevation; fibers not depolarized also contribute, as they remain at their resting membrane potential. In the absence of ischemia, significant intercellular gradients develop later during the plateau phase of the action potential. Thus, repolarization potentials would be expected to appear earlier in ischemic than in nonischemic tissue. However, the problems engendered by the lack of epicardial recordings from human subjects with acute infarction must be considered; although data from dogs and pigs are available, possible phylogenetic differences must not be discounted.

Second, a characteristic sequence of potential distributions was observed during the ST segment for each lesion location. During the terminal portions of the QRS complex, the anterior maximum in subjects with an anterior infarction migrated to the thoracic position where it remained throughout the ST segment. The intensity of the anterior maximum increased steadily during the translocation and after achieving a stable position on the torso. At all times, only a single maximum surrounded by a negative field was observed. A qualitatively similar sequence was found in patients with inferior myocardial lesions; presumably, the border of positive potentials surrounding the inferior thorax represents the margins of an intense maximum existing on the diaphragmatic surface.

The singularity of the extrema is consistent with a single-dipole cardiac equivalent generator. Such patterns must be considered consistent with and not diagnostic of a dipolar source because (1) multiple epicardial wave fronts, whether naturally occurring or experimentally induced, may generate surface patterns with single extrema; and (2) much quantitated nondipolarity may exist without the emergence of multiple discrete pairs of surface extrema. In contrast, the emergence of more than one maximum or minimum is generally considered to indicate a nondipolar source. Effects of the volume conductor may cause modest irregularities in contour lines, such as recorded here, but probably cannot cause sufficient fractionation to generate multiple extrema.

These patterns may be interpreted in light of recently reported experimental studies in rabbits. Electrical field studies of experimentally induced infarctions in isolated rabbit hearts have documented a highly dipolar effect appearing during the mid-to-late QRS and located near the ischemic region. This dipole then migrated progressively to a location overlying the injured area. During the ST segment, the injury-current dipole remained fixed in location, while it progressively increased in moment to reach peak force toward the end of the ST segment. The patterns of dipolarity, translocation during the QRS and stability of location with increasing extrema strength during the ST segment reported here correspond directly to
these experimentally quantitated characteristics of the ischemic lesion. Most authors have described the magnitudes but not the distribution of ST-segment voltages. Those that have addressed the pattern of injury currents have done so only for limited zones of the left precordium. Reid et al., Pelides et al.39 and Krotkiewski et al.30 reported left anterior precordial patterns of ST elevation similar to those described here for anterior infarctions. Selwyn et al.31 reported anterior thoracic distributions of ST-segment elevation and depression in subjects with inferior lesions similar to those detailed in this report. Murray et al.92 reported the multiple maxima and/or minima in left precordial isopotential maps in six of 27 patients (22%) with acute anterior infarction. The causes of difference between that study and the present study as well as those cited above,37, 29-31 which all demonstrated patterns consistent with a dipolar source, are not apparent.

The patterns presented relate to the use of left precordial electrocardiographic grids for calculating the magnitude of ST-segment deviations.6 First, the area covered by positive potentials after anterior infarction, as noted by Murray et al.,92 is wider than that covered by limited electrode grids. In this study, only 51.1% of total ST-segment positivity in subjects with anterior infarction was recorded by one limited electrode system. Whether or not this affects the accuracy of infarct size estimation is unknown; little can be said without the availability of an independent measure of lesion volume. However, the overlap and the relatively high potential values recorded at the edges of an electrode grid suggest that small deviations in electrical orientation of a lesion or small differences in grid placement may cause significant alterations in the computed sums of ST-segment elevations. Similarly, the localization of anterior minima and maxima in subjects with inferior lesions suggests that standard left precordial grid methods would be unable to sense the major components of this field.

A third significant feature of the isopotential distributions was the pattern observed during ending phases of the ST-T interval. In patients with anterior infarction, a minimum evolved over the left lateral thorax during the T wave as the ST-segment maximum shifted rightward. Midprecordial electrodes, previously under the dominance of the strong ST-segment maximum, came under the influence of this new, intense minimum, resulting in a conversion from marked ST-segment elevation to deep T-wave inversion — the coved plane pattern described by Rothschild et al.3 This pattern was then consistent with the T-wave patterns predicted to occur if refractory periods of ischemic lesions lengthened relative to the normal zones90 or if the observed ST-segment elevation resulted, in large measure, from systolic injury currents.34

Reorientation of surface patterns was at least as apparent during the T wave of one group of patients with inferior lesions (fig. 7). In a second group, however, the locations of the maximum and minimum remained relatively stable throughout the ST segment and the T wave (fig. 8). This concordance between ST-segment elevation and T-wave dipole orientation would be predicted if refractory periods of ischemic zones were abnormally shortened,32 or if ST elevation were due to significant diastolic currents of injury.34 Explanations for the differences among subjects are unknown.

A final effect studied was the distribution of QRST areas on the thoracic surface after myocardial infarction. Wilson et al.34 showed that the areas under the QRS and ST-T intervals represent the mean forces operative during those intervals, and that the area under the entire QRST wave form is "a measure of the effects produced by local variations in the excitation process and the mean electrical axis of QRST gives the direction of the line along which these variations are greatest." Abildskov and associates,36 combining this concept with a display format similar to that used for isopotential mapping, reported QRST isoarea maps depicting the distribution of local variations in recovery. It would thereby be possible to detect abnormal degrees of heterogeneity in recovery. As this finding has been shown37 to predispose to ventricular dysrhythmias, isoarea distributions may permit detection of clinical states at high risk.

Six patients had QRST isoarea distributions with multiple extrema (fig. 9). This is in contrast to dipolar patterns recorded from normal subjects and is in accord with the presence of marked variability in ventricular recovery,36 which may predispose to dysrhythmic events. Whether these subjects represent a high-risk subset for dysrhythmias during the acute or convalescent phase is unknown.

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D M Mirvis

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