Left Precordial Isopotential Mapping during Supine Exercise

DAVID M. MIRVIS, M.D., FRANCIS W. KELLER, JR., B.S.E.E., JOHN W. COX, JR., B.S.M.E., DAVID G. ZETTERGREN, B.S., ROBERT F. DOWDIE, AND RAYMOND E. IDEKER, PH.D., M.D.

SUMMARY  Junctional depression is often observed during physical exercise in overtly normal subjects. To explore its pathogenesis, 15 normal volunteers were studied during supine, bicycle ergometer, submaximal stress tests. Electrocardiograms were simultaneously recorded from 42 electrodes on the left anterior precordium at two minute intervals at rest and during exercise. Data were used to construct isopotential maps throughout the P-QRS-T intervals. At rest, maps throughout the ST segment were dominated by a single maximum along the upper left sternal border. During exercise, all subjects developed junctional depression that was maximal along the lower left sternal border. Exercise maps during the early to mid-ST segment showed an intense minimum along the lower left sternal border that was continuous with terminal QRS forces in both intensity and location. Later in ST, this minimum decreased in strength and was replaced by a maximum located in the same area as that observed at rest. These observations suggest that junctional depression is the result of competition between two effects, one being normal repolarization which is obscured in the early ST segment by the second, possibly representing delayed terminal depolarization forces.

TRANSGENIC ELECTROCARDIOGRAPHIC ABNORMALITIES during spontaneous angina pectoris were first reported by Bousfield in 1918.1 These observations, coupled with the then obvious difficulty in continuous monitoring of the electrocardiogram in patients with episodic chest pain, led to the development of stress tests designed to induce angina pectoris while the subject was being monitored. Thus, in 1939, Master2 reported a simple tolerance test for circulatory efficiency which currently bears his name. Within a few years, investigators such as Goldhammer and Scherf3 were able to document electrocardiographic changes in half the subjects with ischemic heart disease who were tested. Over the next half century, exercise testing evolved into an invaluable component of the clinician's diagnostic armamentarium.

The value of the exercise stress test in the diagnosis of covert coronary artery disease remains moot.4-12 One difficulty limiting the efficacy of exercise tests is the common occurrence of electrocardiographic abnormalities in normal subjects during exercise.5-12 Changes in depolarization and repolarization phases of both atrial and ventricular complexes have been described. Two of the most frequently encountered "functional" aberrations are PR segment and J-point depression. The former distorts a commonly used baseline level while the latter leads to ST-segment depression easily mistaken for true ischemic abnormalities.

Although the importance of these repolarization changes is apparent, little is known of their pathogenesis. Lepeschkin7 suggested that an augmented atrial repolarization wave during exercise may cause junctional depression. Although this hypothesis has been accepted by many, recent vectorial studies16 have suggested that this may be only a partial explanation. Other proposed,7 but unproven, mechanisms include transient hyperkalemia, altered hematocrit, changes in intracardiac blood volume, and terminal depolarization alterations. We have approached this problem using a computerized system for acquiring data from multiple precordial electrodes during exercise. Results, displayed as left precordial isopotential maps, portray the thoracic distribution of depolarization and repolarization changes during exercise in normal subjects.

Methods

Study Group

Fifteen male volunteers were recruited from the housestaff and faculty of the University of Tennessee Center for the Health Sciences. All were under 35 years of age and free of cardiovascular disease, as determined by a detailed history, physical examination and resting electrocardiogram. Subjects with lipid or carbohydrate metabolic defects, hypertension, cardiac murmurs, or family histories of atherosclerotic coronary artery disease were excluded, as were those taking any cardiovascular medication. Voluntary informed consent was granted prior to study.

Exercise Protocol

Exercise stress tests were performed using a supine, bicycle ergometer protocol. All subjects initiated exercise at a load of 300 kg-m/min. Subsequently, the load was incremented by 100-150 kg-m/min at two minute intervals until 85% of predicted maximum heart rate13 was reached and maintained for two minutes. Loads were increased without interruption of exercise.

Experimental Lead System

A total of 49 electrodes were placed on each subject. Electrodes were constructed from pure silver discs, one-half inch in diameter and four-hundredths of an inch thick, recessed from one side of a cast resin housing three-fourths of an inch in diameter. Prior to use, all electrodes were chloridized.

Seven equally spaced strips of six electrodes were placed parallel to the sternum, extending from the right sternal...
border to the left posterior axillary line. The upper edges of
the four medial columns were aligned with the sternoclavicular joints while the remaining two were placed as high under the left axilla as was feasible. The lower borders extended to the inferior rib margins. Additional electrodes were placed proximally on each of the four extremities, and at the H, I and M sites of the Frank lead system.

**Data Acquisition System**

Each of the 42 precordial grid electrodes was hardwired to
one input of a variable gain (1000 to 16000X), variable time-
constant, differential amplifier. The four limb electrodes were hardwired together to form a buffered Wilson central terminal; the output of this central terminal served as the second input to each differential amplifier. Thus, the output of each channel was the potential recorded at each electrode position relative to that of the Wilson central terminal. Other amplifiers received signals from the limb electrodes in pairs
representing the standard bipolar limb leads, while others amplified signals from the VCG electrode sites. As typically employed, gains of 1000X and time constants of two seconds were selected. Amplifier noise levels were determined to be 4 µV, peak-to-peak.

Outputs from these 48 amplifiers were digitized on-line at
a sampling rate of 1000 samples per channel per second using
a laboratory oriented computer system (PDP-7, Digital Equipment Corp). Digital data were passed directly to disc and then copied on digital tape during the interval between data acquisition periods.

Electrocardiograms recorded from each lead were exam-
ined prior to exercise on an oscilloscope to determine
noise levels as well as to confirm the normality of ST-
segment and T-wave morphologies. One electrode, approx-
imating a V4 location (V'4) was selected for continuous
monitoring during exercise and for use as a reference channel in data analysis procedures.

Data were collected from all channels for fifteen second periods before and at two minute intervals during exercise. The fifteen second periods of data acquisition during exercise corresponded to the final fifteen seconds of work at a given workload. Exercise was continued throughout the sampling period.

**Data Analysis Procedures**

The fifteen seconds of data stored on disc were copied to
magnetic tape during the 105 seconds between data acquisi-
tion periods. A series of offline steps followed. First, the channel designated as the V4 reference lead was scanned and all beats with cycle lengths within three standard deviations of the mean value were averaged. Each beat was then numerically compared to this averaged complex using an automated autocorrelation method. This technique computed a "waveform index," which deviated from unity as the two waveforms became less similar in morphology. Beats with indices of 0.8 to 1.2 were selected and averaged to compute a final averaged waveform for each lead.

Next, an expanded plot of single representative beats from the unipolar lead and from leads approximating V1 and V4 were used to manually select onsets and offsets of the P wave, QRS complex and T wave. A section of the T-P seg-
ment was also selected as a baseline, or zero potential line.
All such determinations were made by the same observer to reduce interobserver variations in technique.

Leads not directly recorded or recorded with excessive noise were interpolated from neighboring lead potentials. These included the standard precordial leads as well as the Frank vectorcardiographic leads. Programming to "fix" leads not properly recorded, due primarily to electrode instability, permitted construction of complete isopotential maps from incomplete data sets. Finally, isopotential maps were computed from the averaged waveforms for each lead using a bilinear interpolation routine to derive potentials at loci between electrodes.

**Results**

Data will be presented as left precordial isopotential maps with a standard format. The location of the sternal notch is identified by ∠, and that of each of the six standard pre-
cordial electrode sites is represented by E. The positions of the forty-two precordial grid electrodes are marked by '+' and '-' symbols, reflecting the sign of the potential recorded from the electrode. Because all subjects demonstrated qualitatively similar patterns both at rest and during exercise, maps from one study will be presented.

**Rest Maps**

Maps depicting potential distribution during the PR se-
gment, the terminal portions of the QRS complex, the ST seg-
ment and the T wave are illustrated in figures 1 and 2. The PR segment (fig. 1A) was characterized by a single, stationary minimum, i.e., a single zone of peak negative potential, located near the V4 site. Potential distributions during the terminal 20 msec of the QRS complex (fig. 1) also demonstrated a single minimum, located caudal to the V4 position. As the J-point was approached (fig. 1C), this minimum decreased in intensity but remained stationary in location. Within 4-8 msec after the end of QRS (fig. 1D), this minimum was lost, with only an area of low amplitude negative potential remaining. An area of positive potential with a single maximum, however, now appeared above the V4 position (fig. 2A-C). This maximum increased in intensity but remained fixed in location on maps drawn progressively later in the ST segment.

The rising portion of the T-wave was characterized by
further increases in potential strength of this fixed location maximum. Near the summit of the T-wave (fig. 2D), however, the maximum often moved laterally while continu-
ing to increase in strength.

**Exercise Maps**

High quality, low noise electrocardiograms were recorded from all subjects throughout the exercise period. The effect of the averaging routines is illustrated in figure 3. Single beat recordings (left column) were relatively noise free. Averaging of fifteen beats, however, reduced random noise to produce a finer grained signal (center column). The degree of reproducibility of successive beats was typically high, as confirmed by the stability of the computed waveform index (right column).
All fifteen subjects reached predicted submaximal heart rates and maintained this rate for a minimum of two minutes. Resting heart rate for the group was 65.0 ± 10.9 beats per minute (mean ± 1 standard deviation). Peak rate was 169.0 ± 7.3 beats per minute, which was reached after 11.3 ± 2.2 minutes of exercise.

PR segment maps during exercise (fig. 4A) demonstrated a single, stationary minimum located as at rest, i.e., over the left lower sternal border. Its magnitude increased progressively as heart rate rose; at peak heart rates, its amplitude at QRS onset was −354.6 ± 60.4 uV. PR intervals, measured from lead V₁, uniformly decreased with exercise, from 154.0 ± 12.6 msec at rest to 127.3 ± 16.1 msec at peak exercise.

The terminal 20 msec of the QRS complex during exercise was likewise characterized by a single minimum, located to the left and caudal to that observed at rest (fig. 4B-D). Magnitude of this extreme 20 msec before the J-point was greater than that observed at rest in six subjects, equal to it in four and less than at rest in five subjects. At 5 and 10 msec before the J-point, the minimum was of greater magnitude during exercise than at rest in all subjects.

Early portions of the ST segment during exercise (fig. 5A-C) were characterized by a distinct minimum not present at rest. Its intensity and location were continuous with those of the terminal QRS minimum. Thus, junctional depression recorded from individual leads was greatest in the lower mid-precordium, caudal to V₃, and ranged from 190 to 320 μV at peak exercise. At the V₅ position, magnitudes varied from 70 to 210 μV, and exceeded 100 μV in four subjects. Zones along the upper sternum demonstrated positive potentials at the J-point, corresponding to junctional elevation. The minimum during the ST segment (fig. 5A) was lateral and caudal to that observed during the PR segment (fig. 4A).

Further into the ST segment, this minimum progressively weakened and finally became undetectable. Its duration and

---

**FIGURE 1.** Left precordial potential distribution in one subject prior to exercise. Locations of the sternal notch (♦) and the six standard precordial electrode sites (X) are identified. Each of the 42 precordial grid electrode locations is designated by a '+' or ' −', corresponding to the sign of the potential recorded by that electrode. The zero potential line is overdrawn and appears darker than the remaining isopotential lines. A) Isopotential distribution 10 msec prior to onset of the QRS complex, demonstrating a single anterior minimum. Contour lines are drawn at 10 μV intervals. B-D) Potential distributions during the terminal QRS, drawn with contour lines at 40 μV intervals. An intense single minimum is present near V₅ 15 msec prior to the end of the QRS. B) At 5 msec prior to the end of the QRS (C) this minimum has decreased in strength but has remained stationary, while positive potentials appeared superiorly and to the left. At the J-point (D), only low level positive and negative potentials persist.

---

**FIGURE 2.** Isopotential distribution at rest during the ST segment and T wave. Within 8 msec after the J-point (A), the distribution becomes dominated by a single anterior maximum. This potential extreme increased in intensity further into the ST segment (panel B-20 msec into ST; panel C-40 msec into ST) and during the T wave (panel D-72 msec into T-wave) with little change in location. Contour lines in A-C are at 10 μV intervals, and in D are at 40 μV intervals.
intensity increased as workload increased; at peak workload, it was clearly definable up to 80 msec into the ST segment in all cases.

Temporally coincident with the decrease in the strength of this "exertional minimum," a positive potential zone emerged with greatest intensity near the V_{2;3} position, just leftward and inferior to the position of the repolarization maximum observed at rest. It increased in intensity and, in some cases, moved laterally during the T-wave (fig. 5D), as did the maximum at rest. The intensity of the maximum, however, was always less than at rest for a given instant in the ST-T wave.

Potentials recorded during peak exercise load at the J-point were subtracted from those sensed 40, 60 and 80 msec into the ST segment. These differences were plotted as isopotential "difference" maps (fig. 6) to portray the magnitude and direction of ST-segment slope. In this context, a '+' sign indicates an upward sloping ST segment, a '-' a downward sloping segment and the zero line an isoelectric or flat one. "Isopotential" lines indicate slope intensity; in figure 6C, each contour line represents a slope of 0.5 μV/msec. Results demonstrated that the ST-segment slope was directly related to the direction and amplitude of the junctional deviation. In zones with the most intense junctional depression, the ST segment had the greatest upsloping velocity. Areas along the upper sternum where junctional elevation was noted demonstrated a negative ST-segment slope.

Difference maps were also constructed by subtraction of potentials recorded at rest from those sensed at peak exercise. The resulting distribution depicts the field generated by exercise. During the PR segment, these maps were dominated by a minimum located caudal to V_{4} and by a maximum cephalad to it (fig. 7A, B). As the QRS was approached, the minimum intensified but remained stationary; the maximum, in contrast, became less intense causing the zero potential line to move superiorly. During the first 10 msec of the QRS, this pattern persisted with an intense minimum dominating the left precordium. Maps during the last 20 msec of the QRS demonstrated a single minimum located caudal to the V_{4} position. This wavefront decreased in strength but moved little in maps corresponding to the J-point through the first 40 msec of the ST segment (fig. 7C, D).

Potential distributions during the terminal QRS and during the ST segment at peak exercise were examined after subtraction of potentials recorded at the onset of QRS. This, in essence, converted the mapping baseline from the T-P segment to the P-R segment. Results (fig. 8) again demonstrated the occurrence of exertional minimum located over the left lower precordium (fig. 8C, D) not present at rest (fig. 8A, B), that was continuous with terminal QRS potential intensity.
patterns. Maximal J-point depression with this baseline was recorded in the left lateral precordium, caudal to V_{6-4} positions.

**Discussion**

Clinical electrocardiology strives to semiquantitatively define the physiologic state of the heart from the electrical potentials it generates. The use of complex surface electrode configurations coupled to sophisticated recording networks has facilitated this effort. One resultant technique is that of isopotential body surface mapping,^{17-20} by which the electrical potential distribution on large portions of the body may be quantitatively depicted at each instant during the cardiac cycle. In this report we have described initial ex-

---

**Figure 5.** Potential distribution during exercise at instants during the ST segment and T wave. An intense minimum located near V_{4} is observed 8 msec (panel A) and 18 msec (panel B) into the ST segment, with peak amplitude of 350 μV and 300 μV, respectively. By 40 msec into the ST-segment (panel C) the minimum has shifted leftward as a positive field becomes prominent. Later (panel D, 15 msec into the T-wave), a maximum appears near V_{9}. Contour lines are drawn at 10 μV intervals in panels A-C, and at 15 μV intervals in panel D.

**Figure 6.** A) Potential distribution during exercise as recorded at the J-point. Contour lines are at 20 μV intervals. Map is identical to that in figure 5D but for the contour interval. B) Averaged electrocardiograms recorded from electrodes labeled in panels A and C. Waveforms 1 and 2 were recorded from the first electrode (left) of the top row and the second electrode (from the left) of the second row of the grid, respectively. C) Isopotential difference map constructed after subtraction of potentials recorded 40 msec into the ST segment. Thus, contour lines connect to positions of equal ST-segment slope, here 0.5 μV/msec per contour line. A positive value corresponds to an upsloping ST segment and a negative one indicates a downsloping segment. Areas demonstrating marked J-point depression (electrode sites 3-5) also demonstrated greatest upward sloping of the ST-segment while electrodes recording junctional elevation (electrode site 1) demonstrated a downsloping ST segment. Electrodes near the zero potential line (electrode site 2) or in zones of low level junctional depression (electrode sites 6 and 7) recorded minimal or intermediate ST-segment slopes, respectively.
experiences applying this technique to the study of exercise-induced electrocardiographic changes.

Construction of isopotential maps during exercise requires solution of technical difficulties not present for mapping at rest. First, all leads must be sampled simultaneously, requiring the use of lead amplifiers and/or complex multiplexing circuitry.22 Sequential sampling of groups of leads, as is commonly practiced for rest mapping, would be inappropriate during a physiologically unstable period, as during exercise. Noise reduction is also more critical during exercise.23 Mechanical artifacts and muscle tremor must be dampened as much as possible, amplifier and environmental noise. Because beats occurring during a fixed time interval were averaged, more beats were averaged and noise reduction was greater at the higher heart rates reached at higher exercise loads, when muscle artifact would present a more significant problem.

A supine position was also selected to improve the signal-to-noise ratio of data collected during exercise. Pilot studies with upright ergometer or treadmill exercise systems documented the poorer signal quality, especially as recorded from lower chest sites, than illustrated here using a supine position. Several physiologic studies20-23 have compared the upright to the supine position, and concluded that supine exercise represents an equal or greater stress to the circulatory system than does upright exertion.

The significance of the potential distributions recorded during and after exercise relates to two advantages of isopotential mapping over conventional electrocardiographic and vectorcardiographic techniques. First, potentials at all surface sites covered by the electrode grid may be determined, either by direct measurement or by interpolation.16,18 Previous studies have documented that diagnostically critical information may be recorded only at
sites distant to those elected in routine electrocardiography. For example, standard anterior precordial leads may yield identical waveforms in both normal subjects and in patients with right ventricular hypertrophy; leading from the right precordium may, however, separate these groups.  

Several reports have discussed the selection of proper leads for exercise stress tests. Mason et al. described positive tests in 56 of 174 patients using twelve standard leads. In nineteen, abnormalities were recorded in only one lead. Of these, two were abnormal only in V₄ and six tests were positive only in the right precordial leads. Thus, if only V₄ had been recorded, sensitivity of the test would have been reduced from 84% to 58%. Blackburn and Katigbak similarly described an 11% incidence of truly positive tests that would have been falsely negative if only V₄ had been recorded. Call et al., documenting the value of exploration of the posterior thorax, reported a 24% incidence of exercise tests positive in a left subscapular unipolar lead but negative in anterior chest leads. Thus, published data suggest a role for expanded thoracic leading for the accurate interpretation of exercise ECG records.

Results presented here confirm these suspicions in regard to junctional aberrations. First, junctional depression was most marked along the lower left sternal border, i.e., in a zone not normally sampled during exercise. Second, whereas all subjects had greater than 1.0 mm of junctional depression in this area, in only four would this change have been detected if only leads V₃ and V₄ were recorded. Third, the slope of the ST segment varied from site to site, being directly correlated with the magnitude and direction of the J-point deviation.

A second advantage of surface mapping is that the recorded array of electrocardiograms does contain "proximity potentials" reflecting local myocardial electrical events. Thus, regional abnormalities may be evaluated by leading from torso zones spatially related to the underlying epicardial phenomena. Conversely, surface potential distributions may be physiologically interpreted in terms of cardiac activation and recovery patterns. In addition, multiple simultaneously occurring phenomena may be detected. This would not be possible with methods, such as the vectorcardiogram, which are based on a single dipole model of cardiac electrical activity.

In this context, junctional depression may be viewed as the result of the presence of a new effect, projecting negative potentials over the left lower sternal border during early repolarization. Upsloping the ST segment results from a rapid decrease in the intensity of this effect, with the subsequent appearance of a second force projecting positive potentials to the left precordium. These observations are consistent with those of Blomqvist, Horsten, Rautaharju and Simoons; all described a rightward and superior shift of the mean ST-segment vector during exercise. This would cause the negative pole of the presumed dipole to project leftward and inferiorly, i.e., the location of the observed minimum. Extensive evaluation of the dipolarity of the observed patterns is prohibited, however, as potentials on only one thoracic quadrant were sensed.

The late ST-segment maximum emerged at a location and with an intensity similar to those of the repolarization maximum at rest. This suggests that the generator responsible for this phenomenon during exercise is the normal repolarization dipolar source. Its effects early in the ST segment are obscured, however, by the superposed electrical field generated by the earlier appearing source projecting the left lower sternal border minimum.

More speculative is the origin of the early ST-segment minimum. Its surface distribution was continuous with the minimum observed during the terminal QRS in both location and intensity. A similar continuity in exercise-induced changes of terminal QRS early ST-segment vectors has been described by Blomqvist and by Simoons and Hugenholtz. These findings suggest that the appearance of this "exercise minimum" and, hence, of junctional depression may result from altered sequences of ventricular depolarization and/or repolarization. Exercise-induced P-wave changes have been attributed to a similar mechanism by Irisawa and Seyama and by Rautaharju et al. Vectorial studies of ventricular activation have likewise suggested depolarization alterations. Blomqvist reported both QRS prolongation and a rightward and superior shift of terminal QRS vectors. Simoons and Hugenholtz noted widened S-waves with rotation of terminal vectors superiorly and to the right.

Activation data from revived human hearts have demonstrated that the basal left and right ventricles have depolarized late during the QRS complex. Boineau et al. further suggested that it is activation of these areas that is responsible for the late anterior precordial minimum observed in canine studies. Epicardial and intramural isopotential mapping during depolarization and repolarization also demonstrated temporal overlapping of activation and recovery near the J point. During the final 10 to 18% of the QRS, scattered intramural areas of positive potential, representing repolarization forces, coexisted with continued excitation wavefronts in the posterobasal left ventricle and interventricular septum. The dual fields described here may represent surface reflections of this phenomenon. Thus, delayed activation of basal areas, leading to accentuated depolarization-repolarization overlap, may result in physiologic junctional depression.

Another more commonly voiced hypothesis as to the cause of junctional depression is that augmented atrial repolarization forces (Ta) produce early ST depression. Objections to this concept have been raised. For example, vectorial directions of these forces differ from those of the ST vector during exercise and no correlation could be found between junctional and P-wave amplitudes. This latter relationship would be expected according to gradient theory, if the two were casually related. These studies suggest that factors other than, or possibly in addition to, atrial repolarization must be operative.

Isopotential patterns relate to this hypothesis as well. The PR segment during exercise was dominated by a stationary anterior minimum that differed in location from that observed during the ST segment. This difference is emphasized in the subtraction maps illustrated in figure 7; exercise induced an anterior minimum and maximum during the PR interval but only a minimum during the ST segment that differed in location from the PR-segment minimum. These observations suggest that the cardiac sources responsible for these fields differed in orientation and/or in location. A shift in the orientation or a change in the nature of atrial
repolarization forces during the QRS cannot be excluded, however.

Also noted was an inconsistent intensification of the terminal QRS minimum despite a uniform appearance of an ST-segment negative wavefront. If atrial repolarization forces persisted through the early ST segment to depress it, they should also produce greater negative potentials during the QRS complex.

A final factor mediating against Ta as a sole cause of J-point depression is the prolonged persistence of the abnormal exertional minimum. A distinct negative wavefront was observed at peak exercise load in maps drawn up to 80 msec into the ST segment. This, coupled with the observed lesser reduction in PR-interval duration, would require a prolongation of atrial depolarization-repolarization times if the minimum were due to Ta. Microelectrode studies in animals, in contrast, have documented shortening of the atrial action potential and epicardial repolarization events with increased stimulus frequency. Similarly, clinical studies have reported reduction in atrial effective and functional refractory periods with pacing-induced changes in cycle length, suggesting shortening of repolarization. Adrenergic enhancement during exercise would be expected to accentuate these changes, although direct measurements during exercise have not been reported.

Projective interpretations of surface patterns are not, however, without hazard. Potential distributions generated by an epicardial source are dependent not only on the location, intensity and character of the generator but also on the electrical and geometric properties of intervening tissues. For example, electrical inhomogeneities may cause multiple dipolar or multipolar sources to project dipolar patterns. Likewise, the field generated by one source may be altered by the simultaneous activity of another wavefront; the net result may be a single maximum reflecting the combined activities of multiple sources.

References

33. Scher AM, Young AC: Ventricular depolarization and the genesis of the QRS. Ann NY Acad Sci 65: 768, 1956
34. Spach MS, Barr RC: Ventricular intramural and epicardial potential distributions during ventricular activation and repolarization in the intact dog. Circ Res 37: 243, 1975
Left precordial isopotential mapping during supine exercise.
D M Mirvis, F W Keller, Jr, J W Cox, Jr, D G Zettergren, R F Dowdie and R E Ideker

Circulation. 1977;56:245-252
doi: 10.1161/01.CIR.56.2.245

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1977 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on
the World Wide Web at:
http://circ.ahajournals.org/content/56/2/245

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally
published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the
Editorial Office. Once the online version of the published article for which permission is being requested is
located, click Request Permissions in the middle column of the Web page under Services. Further
information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/