Differential electrocardiographic effects of myocardial ischemia induced by atrial pacing in dogs with various locations of coronary stenosis

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ABSTRACT The spatial distribution of abnormal repolarization potentials caused by regional myocardial ischemia was determined in 45 dogs. Ameroid constrictors were placed around the left circumflex artery in 10, the left anterior descending artery in 10, and the right coronary artery in 10. Ten dogs without constrictors served as controls. Electrocardiographic events were determined from body surface isopotential distributions, which were computed from potentials sensed by 84 torso electrodes. In control dogs, pacing to heart rates of 230 to 250 beats/min increased the intensity of positive and negative surface extrema during the ST segment without altering their spatial features. Two weeks after placement of the ameroid constrictors, tachycardia induced abnormal negative potentials during the ST segment. Localization of these ischemic forces varied with the placement of the constrictor in a manner consistent with the affected perfusion territories. However, much of the torso surface was involved by all lesions, and only small zones of ST segment depression unique to specific lesions could be identified. In five additional dogs a constrictor was placed on the right coronary artery 3 months after implantation of a device on the circumflex vessel. ST segment patterns during pacing in dogs with two lesions were consistent with the sum of the two individual lesions. Thus, the regional nature of myocardial ischemia is detectable in the body surface isopotential distributions, but the degree of spatial overlap may limit the value of such techniques in extending the usefulness of clinical exercise-stress electrocardiography.

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ELECTROCARDIOGRAPHIC exercise stress testing is a standard method for detecting clinically significant coronary atherosclerosis. Determination of the location as well as the presence of such lesions would be a major benefit because it would provide a noninvasive safe procedure for assessing, for example, prognosis and possible need for surgical intervention.

Clinical studies have, however, reported various conclusions as to the accuracy of the exercise test in predicting the location of angiographically significant arterial lesions. Several studies have reported good correlations between the distributions of ST segment abnormalities and of arterial lesions. Robertson et al. successfully correlated electrocardiographic changes in inferior and anterior leads with right and left coronary artery abnormalities, respectively. Fox et al., using a 16-point precordial mapping system, were able to identify ST segment depression distributions characteristic of single- and multiple-vessel involvement.

Others, however, have not been able to derive similar conclusions. Dunn et al. reported no differences in the distribution of exercise-induced ST segment depression in patients with isolated left anterior descending artery lesions nor in those with single left circumflex or right coronary lesions. For example, ST depression in both inferior and anterior leads occurred in 43% of patients with left anterior descending artery disease and in 29% of subjects with right or circumflex artery disease. Block et al. were similarly unable to identify coronary lesions by exercise surface-mapping or vectorcardiography.

Many causes for such differences can be suggested. These include patient variables, such as variations in regions of myocardium supplied by given vessels, in the topographic relationship of specific myocardial regions to body surface electrodes, in the hemodynamic consequence of an obstruction, in the proximal or distal position of a lesion, and in collateralization. Oth-
er considerations include variations in recording techniques and exercise protocols.\(^1,4\)

Because of these discrepancies and because of the significance of the problem, we examined the body surface electrocardiographic patterns produced in an animal preparation\(^8\) designed to simulate clinical exercise stress testing and in which many of the previously listed parameters may be controlled. In this preparation, localized progressive coronary arterial narrowing is produced by a surgically implanted ameroid constrictor; myocardial ischemia is then induced by graded atrial pacing, and the resulting electrocardiographic effects are detected by body surface isopotential mapping.\(^10\) It was hoped thereby to determine whether or not myocardial ischemia in specific myocardial zones generated repolarization abnormalities that were projected to characteristic and distinct torso areas under experimentally optimized conditions.

**Methods**

Forty-five adult mongrel dogs weighing 12 to 16 kg who were free of parasites were studied.

**Surgical procedures.** Surgical protocols were completed under sterile conditions after induction of anesthesia with a mixture of halothane, nitrous oxide, and oxygen. A right or left (see below) thoracotomy was performed, and the pericardium was opened. A quadripolar plaque electrode was sutured to the ipsilateral atrial appendage.

Dogs were then subdivided into five subsets. Left thoracotomies were performed in groups A, B, and C. In group A \((n = 10)\), no further procedure was undertaken. In group B \((n = 10)\), the origin of the left circumflex coronary artery was gently dissected and an ameroid constrictor with an internal diameter of 2.77 mm was placed around the vessel. In group C \((n = 10)\), the proximal left anterior descending coronary artery, distal to the origin of the septal artery, was isolated and a similar ameroid constrictor was implanted. In group D \((n = 10)\), a right thoracotomy was performed, and the electrode was sutured to the right atrial appendage. A 2.5 mm internal diameter ameroid constrictor was then placed around the proximal right coronary artery. In group E \((n = 5)\), an ameroid constrictor was implanted on the left circumflex artery as in group B; however, 3 months later a right thoracotomy was performed and a second ameroid constrictor was seated on the right coronary artery as in group D. Locations of ameroid constrictors on each vessel were standardized.

The pericardium and the chest wall were then closed in routine manners. Pacemaker electrode wires were tracked subcutaneously to the nape of the neck and were covered with a sterile dressing. All animals survived surgery and were free of infection at both incision and exteriorization sites.

**Electrocardiographic recordings.** Electrocardiographic signals were recorded from 88 chloridized silver electrodes. Eighty-four of those electrodes were placed on the shaved chests of the dogs and extended from the level of the sternal notch to below the inferior rib margins. The remaining four electrodes were located on the extremities to record standard limb-lead patterns and to derive the Wilson central terminal potential. Positions of all electrodes were marked to permit reproducible placement.

Experimental protocol. Electrocardiographic data were recorded 14 days after surgery. At that time significant coronary obstruction is expected\(^9\) and, as previously shown, tachycardia results in ST segment depression.\(^5\) Animals were sedated with 1 to 2 ml of Innovar (20 mg fentanyl plus 0.4 mg droperidol/ml) and were respired with pure oxygen. Electrodes were positioned and the dogs were placed in an upright posture with a support sling.

We recorded 14 sec of data. Signals were amplified by 88 low-noise differential AC coupled amplifiers (bipolar limb or grid electrode vs Wilson central terminal voltages). Gains were set at 1000 to 16,000 so that the amplifier output filled the input range of the analog-to-digital converter. Analog-to-digital conversion was then performed at a rate of 500 samples per channel per sec.

After surgery the data acquisition protocols were repeated during atrial pacing. Atrial pacing was begun at a rate of 90 beats/min with bipolar 2 msec pulses with an amplitude 25% above threshold. Heart rate was increased by 20 beats/min every 3 min until a rate of 250 beats/min was achieved. Atropine (1 to 2 mg) was administered intravenously if atrioventricular block developed; this uniformly restored 1:1 atrioventricular conduction.

Data were recorded during the last 14 sec at each pacing rate, immediately after, and 10 min after cessation of pacing.

**Data analysis.** Data analysis procedures have been previously detailed.\(^8\) Electrocardiographic waveforms with similar morphologic characteristics, as determined by an autocorrelation routine, were averaged. Onsets and terminations of Q wave, QRS complex, and ST-T interval were next manually determined from plots of root-mean-square potential, and linear baseline drift was corrected. Voltages during the terminal 20 msec period of the PR segment were averaged as a zero potential baseline.

Isopotential distributions were constructed from potentials recorded at 2 msec intervals during the ST-T wave. At high rates, pacemaker artifacts were superimposed on preceding ST segments; only portions of the ST segment before this stimulus were studied. A linear-bilinear interpolation routine was used to draw isopotential contours. All values were expressed as means ± 1 SD and were compared with analysis of variance.

**Results**

**Group A: control.** All animals survived the experimental period. Resting ST segment waveforms were identical in preoperative and postoperative recordings. Because patterns within each group were qualitatively similar, single examples of each will be presented.

Isopotential distributions constructed from data recorded 40 msec into the ST segment are presented in figure 1. In each panel plus and minus signs mark electrode locations, with the sign corresponding to the polarity of the second voltage. The sternal notch is indicated by a ‘‘V’’; the right and left edges of the map correspond to the left and right paravertebral zones, respectively. Contour lines connecting points at equal potential relative to the Wilson central terminal are drawn at zero and at plus and minus 10, 20, 40, 100, 200, and 400 \(\mu\)V levels. Zero isopotential lines are overdrawn for emphasis. Electrode strips and, hence, isopotential patterns are inferiorly displaced in axillary areas.
Patterns in dogs at rest (figure 1, A) were characterized by an anterolateral maximum, or zone of peak positive voltage, with a value of 306 μV. Negative potentials, with a nadir or minimum of −116 μV, were localized to the right posterosuperior torso. During atrial pacing at rates of 160 beats/min (figure 1, B) and 230 beats/min (figure 1, C), the overall spatial features did not change; left anterior positivity and right posterosuperior negativity persisted. Intensity of the extrema did progressively intensify as heart rate was increased. Thus, the maximum increased from 306 to 490 μV at 160 beats/min and to 544 μV at 230 beats/min. QRS durations were not affected by pacing.

The "difference" map illustrated in figure 1, D, was constructed by subtraction of each electrode potential shown in figure 1, A, from the corresponding voltage shown in figure 1, C. Thus, the distribution depicts the difference between the pattern in dogs at rest and that at a paced rate of 230 beats/min and may be considered to reflect the electrical field "generated" by tachycardia. In this map, potentials from electrodes with positive differences voltages were augmented by pacing (increased positivity or reduced negativity), and those with negative levels were reduced (less positivity or greater negativity) in magnitude.

As shown in figure 1, D, voltages over the left inferior thorax were augmented (maximal increase of 295 μV), and those over the superior torso were reduced in strength (maximal decrease of 183 μV). Positions of the extrema and of the zero isopotential contour corresponded closely to those of the isopotential maps of figure 1, A through C. Thus, tachycardia in these normal animals intensified both positive and negative extrema without altering their spatial distribution. This correlated with accentuation of the resting ST segment configuration with increasing heart rate. Neither QRS form nor duration was altered by the tachycardia.

**Group B: left circumflex artery constriction.** All dogs in which an ameroid constrictor was placed around the left circumflex artery survived for 2 weeks, although two died suddenly after the 2 week postoperative recording session. Resting QRS and ST-T interval electrocardiographic patterns were unaffected by the surgical procedure.

Electrocardiographic waveforms recorded from lead II 2 weeks after implantation of ameroid constrictors are presented in figure 2. In dogs at rest, the ST segment is slightly convex up (top). Similar patterns were seen at a rate of 160 beats/min (middle). However, at
rates of 190 beats/min or more, flat ST segment depression typical of that observed in abnormal clinical exercise tests was observed (lower). Within 4 min after ending pacing, ST segment patterns recovered. Note the maintenance of 1:1 atrioventricular and normal intraventricular conduction at the high-paced rates.

Isopotential maps, drawn as in figure 1 and determined from this animal 2 weeks after surgery 40 msec into the ST segment, are shown in figure 3. ST segment patterns at a resting rate of 84 beats/min (figure 3, A) were similar to those described in control animals; an anterior maximum (303 μV) and posterosegmental negativity (−81 μV) were observed. At a paced rate of 160 beats/min (figure 3, B) the spatial pattern was unchanged. Intensity of the extrema did increase, as previously described for control cases.

At rates of 190 beats/min and faster, isopotential maps clearly differed from those of normal dogs. In figure 3, C, which shows recorded maps at a rate of 230 beats/min, positive potentials were recorded over the superior anterior regions, with an anterior maximum of 281 μV. Negative voltages were then registered along the inferior anterior and most of the posterior thorax, with a peak negative voltage of −139 μV. Waveforms recorded from this latter zone demonstrated ST depression typical of myocardial ischemia. The isopotential difference map (figure 3, D) constructed by subtracting that in figure 3, A, from that in figure 3, C, demonstrated negative voltages (peak = −249 μV) over the inferior right and left hemithoraces with superior extension on the left posterior torso.

These patterns were observed in all cases. Thus, these patterns, particularly the difference distribution shown in figure 3, D, characterize the electrical field generated by tachycardia-induced ischemia of the circumflex artery territory. Magnitudes of the abnormal negative voltages in subtraction maps ranged from −174 to −362 μV (−229.6 ± 41.2).

At the end of the experimental period, animals of this and subsequent groups were killed, and the constricted vessels were examined. In all cases in groups B to D, the artery was constricted to a diameter less than 50% of that at more proximal sites.

**Group C: left anterior descending artery constriction.** All animals completed the experimental protocol without dying; one animal died suddenly during the third postoperative week. Resting ST segment patterns remained unchanged, and QRS duration was unchanged from that in preoperative records.

Isopotential maps from one animal, 2 weeks after surgery, are presented in figure 4. At a spontaneous heart rate of 76 beats/min, patterns 40 msec into the ST segment (figure 4, A) demonstrated an anterior maximum and posterior negativity. Differences between this pattern and other control patterns are within the range of spontaneous variability observed in our dogs. Tachycardia again induced abnormal negativity with ST segment depression. At a paced heart rate of 190
beats/min, abnormal negative voltages were recorded (figure 4, B) over the lower torso with a minimum ($-484 \mu V$) located over the sternum. Positive potentials bordered this minimum superiorly. Subtraction patterns (figure 4, C) were characterized by a low central anterior minimum with negative potentials spreading symmetrically around the inferior torso, and with positive voltages spreading superiorly.

These patterns were observed in all cases. The abnormal minimum in subtraction distributions at the peak paced heart rate varied from $-246 \mu V$ to $-606 \mu V (442.7 \pm 49.6)$. These values were significantly greater than those observed with circumflex occlusion ($p < .01$, one-way analysis of variance). Thus, lesions of the left anterior descending artery caused electrocardiographic changes that were more intense and that were spatially more central with less posterior superior extension than were those due to circumflex obstruction.

**Group D: right coronary artery constriction.** As in other groups, all animals were alive at the end of the 2 week experimental period and were without resting QRS or ST segment abnormalities.

Typical isopotential patterns are displayed in figure 5. During spontaneous sinus rhythm (figure 5, A), an anterior maximum (254 \mu V) and posterior negativity were observed. Pacing to heart rates above 170 beats/min resulted in abnormal negativity and ST depression. At a rate of 190 beats/min (figure 5, B), a left anterior minimum ($-401 \mu V$) was registered, with negative voltages extending to the superior posterior torso. Positive potentials were restricted to the upper right chest. Subtraction maps (figure 5, C) were likewise characterized by left anterior and bilateral posterior negativity with a left axillary minimum.

This pattern, consistently observed in all cases, yielded abnormal minima with an intensity of 382.6 $\pm$ 56.4 $\mu V$. This was not significantly different from that in dogs with either the circumflex artery or left anterior descending artery constricted (group B and C, respectively). When topographic extrema of difference maps were compared with those of prior groups, right coronary lesions generated ischemic potentials that were centered more superiorly and to the right than those with circumflex artery lesions, and potentials that were more laterally located than those with left anterior descending artery lesions.

**Intergroup comparisons.** The spatial distributions of negative voltages in the three experimental subgroups (groups B, C, and D) were compared in two ways. First, voltages 40 msec into the ST segment at each

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**FIGURE 3.** Potential distribution, as in figure 1, from one dog 2 weeks after placement of an ameroid constrictor around the left circumflex artery. All patterns correspond to 40 msec into the ST segment. A, Measured at rest (84 beats/min). B, Paced at 160 beats/min. C, Paced at 230 beats/min. D, Difference map computed by subtraction of A from C.
FIGURE 4. Isopotential patterns, 40 msec into ST segment, 2 weeks after a constrictor was implanted around the left anterior descending artery. A, Measured at rest (76 beats/min). B, During atrial pacing at 190 beats/min. C, Subtraction map (B minus A).

FIGURE 5. Maps 2 weeks after implantation of a constrictor around the right coronary artery. 40 msec into the ST segment. A, Measured at rest (82 beats/min). B, During atrial pacing (190 beats/min). C, Difference map (B minus A).
electrode site in different maps (peak rate minus control) were averaged within each group. Electrodes at which this mean value was less than zero and at which the mean plus 1.65 SD was also less than zero were identified. Only 5% of animals would be expected to have positive voltages at such sites. Thus, the distribution of electrodes so identified would correspond to a 95% confidence region for tachycardia-induced ST segment depression.

Regions so defined for each group were plotted as shown in figures 6, A. Much of the torso (40 of 84 electrode sites) was within the confidence regions of all three lesions, and many other regions (20 sites) were within the ranges of two lesions. Of the total chest, only 13 electrode sites defined areas specific to only one coronary lesion, and no zone was specific for circumflex artery obstruction. When a similar analysis was performed with directly recorded potentials at peak rate rather than difference voltages, no unique areas could be defined.

Second, locations of the electrode with peak negative potentials in difference maps (peak rate minus control), 40 msec into the ST segment, were plotted. As shown in figure 6, B, minima with left anterior descending artery lesions were more central, and those with right coronary artery obstruction were more superior and posterior than minima generated by left circumflex artery obstruction. Once again, overlap among lesions was widely observed.

**Group E: dual coronary lesions.** Differences in ischemic patterns after one and after two vessels were narrowed further illustrate these findings. Isopotential patterns are shown in figure 7. The map in figure 7, A, was constructed from potentials sensed in dogs at rest (72 beats/min) 40 msec into the ST segment, 3 months after implantation of an ameroid constrictor on the left circumflex artery. The distribution is as expected with an anterior maximum (303 µV) and posterosuperior negativity (−91 µV). Pacing to heart rates over 190 beats/min resulted in inferior negative voltages (figure 7, B), as previously described for group B (figure 3, C). These patterns recorded 3 months after implantation were virtually identical in spatial and intensity factors to those registered 2 weeks after surgery.

Two weeks after implantation of the second constrictor on the right coronary artery, resting patterns were again unchanged. Pacing at 190 beats/min (figure 7, C) then resulted in negative voltages with two left-sided minima, with extension of low-level negativity superiorly in left axillary areas. This extension was consistent with that of the regions characteristic of right and circumflex artery lesions as in figures 3, 5, and 6.

This is further demonstrated by the difference map
in figure 7, D, constructed by subtraction of voltages recorded during pacing in a dog with an ameroid constrictor on the circumflex artery (figure 7, B) from those recorded during pacing in a dog with dual ameroid constrictors (figure 7, C). The distribution was characterized by a left lateral minimum comparable to that observed with only a constrictor on the right coronary artery (figure 5, C). Thus, the effects of multiple ischemic areas were additive.

Postmortem examination revealed complete occlusion of the circumflex artery and subtotal obstruction of the right coronary artery.

**Discussion**

It was the purpose of this study to determine whether or not regional myocardial ischemia could be reliably detected and localized by body surface isopotential mapping. The hypothesis was based on the premise that surface maps can reflect regional myocardial events.

That this is true has been substantially documented. Wilson et al.\(^{11}\) concluded in 1944 that "Precordial leads of the kind under consideration (unipolar precordial leads) . . . are in reality semidirect leads from the anterior ventricular surface, capable within certain limits of serving the same purposes as direct leads from the ventral surface of the exposed heart." Experimental studies have successfully correlated surface patterns with epimyocardial excitation sequences,\(^{12}\) with the location and orientation of implanted generators,\(^{13}\) and with the location of experimentally induced abnormalities, including cauterization\(^{14}\) and ectopic pacing.\(^{15}\) Clinical studies have similarly related surface and epicardial events in subjects with ventricular hypertrophy\(^{16}\) and the preexcitation syndromes.\(^{17}\)

Multiple simultaneously active effects may also be detected. They may be manifest as multiple pairs of extrema. Under other conditions of distance, eccentricity, and strength, however, only single extrema may be observed,\(^{14,15}\) but the presence of multiple events may be discerned by changes in low-level potential magnitudes and locations. The importance of such differences has been stressed during atrial\(^{18}\) and ventricular\(^{13}\) activation and recovery.

The animal preparation used to explore these relationships has both advantages and constraints.\(^{8}\) First,
the number and location of coronary lesions, and hence the general location of the ischemic myocardium, could be controlled. As mentioned previously, by 14 days after implantation, lumenal diameter would be expected to be less than half of that in control, with a 20% reduction in resting flow. Subendocardial ischemia, causing ST segment depression, may then be provoked by tachycardia because of very limited coronary reserve. Although we did not examine the affected bed microscopically, significant infarction would not be expected; 1 month after implantation, when complete anterograde flow block has occurred, only small (2.02 ± 1.1% of left ventricular volume) zones of patchy subendocardial infarction can be demonstrated.

Second, myocardial stress as determined by heart rate can be controlled. Atrial pacing was used rather than exercise to eliminate the electrical noise generated by motor-driven devices. As shown by Fedor et al., atrial pacing may produce more severe subendocardial ischemia, possibly due to the absence of exercise-induced vasodilatation. Thus, although we did not measure coronary hemodynamics, the observed electrocardiographic effects may reliably be attributed to subendocardial ischemia.

Other limitations are also significant. First, the torso shape of the dog is very different than that of man and this may significantly alter volume conductor properties and, hence, surface electrical distributions. Second, the coronary circulation and, particularly, the involvement of a native and an extensive collateral bed in the dog differs from that of man. However, study only 2 weeks after amioder constrictor placement and existence of abnormal potentials 3 months after surgery suggest that collateral growth may be a limited problem. Third, the necessary use of capacitor-coupled amplifiers lumped both systolic and diastolic injury currents into a single ST segment shift. It may be assumed that this is suboptimal, but the use of direct coupling amplifiers is not technologically feasible at this time. Fourth, there is presently no independent standard defining the surface changes predicted from specific regional abnormalities. This important information could be obtained by a forward mathematical simulation, but this technique will require further development for routine practical application.

The results of the current study do demonstrate that, in this preparation of tachycardia-induced ischemia, the regional nature of the ischemia is reflected in the isopotential surface patterns. Thus, left anterior descending artery obstruction resulted in an abnormal ST segment minimum that was more central, and right coronary artery constriction caused a minimum that was more to the right and superior than did circumflex artery lesions (figure 6). These differences likely correlate with the perfusion territories of the three vessels as defined by Scheel and Ingram. Distinctions among the three lesions were most clearly noted in subtraction patterns, presumably due to removal of the complex and variable resting repolarization potential patterns. The greater intensity of the minimum with left anterior descending artery obstruction than that with a similar lesion of the circumflex vessel probably reflects the position of the former perfusion territory near to the chest wall; this may overcome the larger mass at risk from circumflex underperfusion.

Data from experiments with dual-vessel obstruction extend these observations. Superposition of a right coronary lesion on a preexistent circumflex artery obstruction produced new ST segment depression on the posterior torso, just as in animals with isolated right coronary artery amioder constrictors, as well as a second minimum not seen in any case with single-vessel involvement (figure 7, C and D). Thus, the regional influences were additive. This was evident, as previously discussed, in effects on the high- and low-level potential patterns.

Extracellular currents responsible for the observed ST segment shifts are probably regional in location and transmural in direction. During normal ventricular activation, the ST-T potentials are largely due to transmural differences in action potential durations. Normally, action potentials are longer on endocardial than on epicardial zones, resulting in positive extracellular epicardial potentials. Regional differences in action potential form are less important, due largely to cancellation of oppositely directed forces. Myocardial ischemia, here localized to the subendocardium, reduces resting membrane potential and action potential duration and amplitude. The former causes extracellular epicardial diastolic positivity and the latter, reversing the normal transmural action potential duration gradient, generates systolic epicardial negativity. In AC coupled systems, the net effect is surface ST segment depression, with current flows in a transmural direction.

The differing sizes and locations of torso zones that show electrocardiographic abnormalities with single and multiple coronary lesions may explain the clinical observation that single-vessel disease is associated with normal exercise tests more often than is multivessel obstruction. A small number of electrodes in fixed torso locations as used clinically may not sample the restricted zone with abnormalities. This would be es-
pecially true with circumflex artery and right coronary artery lesions, i.e., lesions associated with particularly high incidences of normal exercise tests, 30 which generate ST segment depression on the undersampled posterior and right chest areas.

Major overlap in the torso zones with abnormal negative voltages and, hence, ST segment depression did occur (figure 6, A). This likely reflects the same biophysical factors (eccentricity, intensity, distance) that cause masking of multiple epicardial events on surface recordings as noted above. 12, 14, 15 These factors and resultant overlap or “smoothing” clearly limit the usefulness of the method, since discrete and unique areas for each myocardial zone could not be identified.

Some data applying surface mapping to clinical exercise testing has been reported based on normal 31, 32 and abnormal 3, 5 populations. The current results are similar to those of Fox et al. 2 in patients with single- and multiple-vessel disease. That study also demonstrated characteristic patterns for single-vessel lesions, but there was considerable overlap. Thus, experimental and clinical evidence support the belief that isopotential surface mapping may have only a limited role in expanding the usefulness of exercise electrocardiography. A more quantitative approach to torso potentials may improve this application.

The occurrence of ST segment depression during pacing 3 months after ameroid constrictor placement, when collateral formation should be extensive, 33 demonstrates that such collateralization is inadequate to prevent tachycardia-induced ischemia. This is consistent with data of Cox et al., 34 Heaton et al., 35 and Tomoike et al., 20 who demonstrated the functional inadequacy of coronary collaterals to protect against exercise- and catecholamine-induced ischemia. These data are not in accord with those of Lambert et al., 36 who reported maintenance of normal transmural distributions of coronary flow during exercise. That the latter study was performed 6 months after ameroid constrictor placement rather than after shorter time periods as in the other and current studies, as well as other methodologic differences, may be responsible for the differences.

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