Electrocardiographic Effects of Changing the Nature of the Contact Between the Exposed Heart and the Body

By John J. Sayen, M.D., Aaron H. Katcher, M.D., and George Peirce, M.S.

For studying localized changes in myocardial electrical activity, and especially localized ventricular ischemia, the value of electrocardiographic leads with one electrode directly on the heart surface has long been realized. Intramyocardial extracellular lead-points for the exploring electrode provide more prompt and sensitive responses to ischemia and permit correlation of the bioelectrical phenomena with the comparably rapid and responsive polarographic oxygen and muscle contraction changes. Other precise indices of electrical activity not obtainable by direct heart-body leads, such as cellular action-potential sampling (necessarily limited to the superficial muscle layers and short periods) or closely spaced differential lead-points for timing excitation process arrival, cannot for practical purposes be used to predict the extracellular potential differences from which body-surface signals originate. Provided the investigative aim is to study the electrical activity of the relatively undisturbed, working heart or make comparison of open-chest situations with body-surface lead behavior, it is essential to obtain direct heart-body leads and important to make certain that the electrocardiographic representation of localized physiological changes in the heart is reproducible and unambiguous.

It is well known that, at the approximately X/20 standardization required for direct heart-body leads, changes in location or composition of a reference electrode on the body surface do not alter electrocardiographic signals significantly. It is not as well realized that changes in the areas of heart surface which are in contact with the body grossly alter the configuration of direct heart-body leads from all areas of the heart. Our studies, have shown that when the contact between the body and the myocardial generators of ventricular potential is limited to the ventricular cavity blood and the ventricular walls adjacent to the valve rings, as may be accomplished by insulating the heart from the body save for its vascular pedicle, the ventricular R waves (compared with a body or atrial reference) are consistently of greater amplitude than when any portion of the ventricular surface is also permitted to be in electrical contact with the body. Furthermore, induced changes of electrical activity at a ventricular surface in contact with the body will be represented in every direct heart-body lead by a uniform alteration of pattern, opposite in polarity, smaller in amplitude, but similar in configuration to the change that may be recorded from the ventricular surface in question when it is insulated from the body.

Consideration of these phenomena is relevant to the interpretation of the electrocardiographic representation of any open-chest heart situation and must be further discerned in comparing different experimental preparations, lest confusion arise from apparent inconsistencies of behavior. For elucidation of these relationships, direct-coupled amplification* and intramyocardial electrocardiograms

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*An A.C. (capacitor coupled) amplifier converts all deviation of baseline or RS-T segment into net RS-T segment deviation, failing to distinguish, for example, a millimeter of baseline depression from a millimeter of RS-T elevation. All unqualified references to baseline or RS-T segment behavior in this paper are to absolute levels, as obtained from a D.C. amplification system. We distinguish A.C.-amplifier "RS-T segment" data either by quotation marks or by prefixing the word "net."
are necessary in addition to the usual requisites of an open-chest heart study.

Methods

Procedures

Six dogs were studied in association with thoracotomy, suspension of the heart in a pericardial cradle, and the recording of both capacitor-coupled (A.C.) and direct-coupled electrocardiograms from the ventricular surface and the underlying walls, utilizing techniques previously described. The retropericardial space was routinely packed with saline-soaked gauze, since this eliminated variations due to differing dog-chest configurations, drying of the pericardium, degree of lung inflation, and other irrelevant factors. For insulation, a thin sheet of polyethylene film was slipped under the heart to separate completely the ventricles and the atria from the rest of the body, except for the basal vascular connections. This insulation could be withdrawn without disturbing the heart or the electrode positions. Epicardial records were taken from the portions of the ventricular walls in contact with the pericardial cradle, before and after placement of the insulation. For surface records, and some intramyocardial leads, small silver, silver chloride electrodes were used with direct-coupled amplification. The usual recorder was a two-channel Brush ink-writing oscillograph, but a few records have been recorded with a Hathaway oscillograph (frequency response 1,500 c.p.s.) for more precise analysis of QRS configuration and time relationships. Care was taken to minimize and recognize any electrode drift. Amplifier drift was negligible. In four animals, an array of platinum myocardial electrodes was inserted and after stabilization for 45 to 60 minutes, the QRS configuration was measured before and after withdrawal of the insulating polyethylene. D.C. amplification was not feasible for these latter situations because platinum electrodes do not maintain themselves at a fixed potential relative to surrounding tissues.

Terminology

By a direct heart-body lead is meant an electrocardiogram recorded (necessarily at about N/20 standardization) when the exploring ("LA") electrode is directly in contact with the epicardium, myocardium, or underlying cavity of a portion of the heart wall of which the surface has been widely exposed to the air or otherwise insulated from the body—the reference ("RA") electrode being placed at some extracardiac point. We have usually employed the left leg for this reference, but at N/20 standardization it does not matter what extra-cardiac site (or combination of sites) is selected. A maximally insulated heart has its entire atrio-ventricular surface, exclusive of the area of vascular connection at the base, exposed to the air or separated from pericardial supporting structures by insulation. Direct heart-body leads from such a heart are conveniently referred to as free heart-body leads, to distinguish them from the more general category of direct heart-body leads from exposed or insulated portions of less completely insulated hearts. By a local electrocardiographic effect we mean that portion of the electrocardiographic change produced by a localized physiological alteration of the myocardium which has an unequal distribution in or on the heart. In general, this is confined to the vicinity of the area of physiological change. By a reciprocal effect we mean that portion (if any) of the electrocardiographic change produced by localized physiological change which is present in all leads from the heart. By a localized electrocardiographic change is meant a change not associated with reciprocal effects.

Results

Effects of Changing Contact Between the Unaltered Heart Surface and the Body

Limb leads were taken at normal standardization (1 cm. = 1 mv.) before and after opening the pericardium, and after suspending the heart in a cradle. There was little change of amplitude or of QRS configuration in lead II until the sutures holding the cradle were drawn up to suspend the heart. Then QRS amplitude diminished greatly (fig. 1, lower left). This diminution was greatest in dogs with narrow, deep chests in which a higher cradle was needed for bringing the heart up to convenient height for experimental manipulations. Packing normal-saline-soaked gauze beneath the pericardial cradle restored the amplitude for lead II (fig. 1, lower right). Lifting the heart by its apex to free the ventricular surfaces completely from contact

*Splodip film type 120p4 (Goodyear Tire and Rubber Co., Inc.).

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with the pericardial cradle produced a negative QRS complex in lead II (fig. 1, middle of lower row). Lead I was small and was insignificantly altered by any of the maneuvers.

For direct heart-body leads, the standardization of the electrocardiograph was reduced to one-twentieth of normal (1 cm. = 20 mv.). At this level the effect of small potential differences of the order of magnitude of the largest limb lead deflections (range: 1 to 2 mv.) became insignificant, as compared with large heart-body lead potentials from the exposed ventricular surfaces, the underlying walls, and the cavities (range: 20 to 40 mv.). There was also no more than a small potential difference between the body surface and the pericardial area of contact between the posterior atrioventricular surface and the pericardial cradle. Direct heart-body leads from the exposed atrial surfaces and the atrial cavities immediately beneath them likewise showed insignificant potential differences of ventricular origin. In addition to the two aforementioned extreme ranges of ventricular-complex amplitude, potentials of intermediate amplitude were present in leads from those portions of the ventricles which were close to the area of contact with the pericardial cradle. Packing the space beneath the cradle with conducting material enlarged the area of small-amplitude signals at the expense of the areas of intermediate voltage, resulting in a sharper demarcation and a more reproducible preparation.

Insulation of the heart except for its vascular pedicle changed all signals in direct ventricular heart-body leads from the previously low-voltage area of posterior atrioventricular-pericardial contact (and any intermediate-amplitude signals in the short-circuited heart-body leads from the edges of the area) to large-amplitude complexes comparable to those of the exposed surface or the ventricular cavities. In association with the insulation procedure, the ventricular areas that had originally been exposed showed taller R waves and shallower S waves, as well as measurable alteration of all other components. Signals of ventricular origin recorded from the atria were not altered in amplitude to any important degree, although their configuration changed considerably. The amplitude of lead II diminished to less than 0.2 mv. with insulation, but usually the QRS complex did not become inverted.

Arrays of intramyocardial platinum electrodes were inserted into the anterior surface of maximally insulated hearts. When the insulation was withdrawn, the most conspicuous and consistent change was a decrease of absolute R-wave amplitude at all electrodes, and usually an increase of absolute S-wave amplitude (fig. 2, upper and middle bands). The net RS-T segment level and the T waves were also measurably changed. If the exposed portion of the left ventricular apex was then connected to the pericardial cradle by a saline-soaked wick, thus further increasing the area of ventricular surface contact with the body (but not short-circuiting the heart surface directly overlying any of the myo-
Effects on the intramyocardial ventricular complexes of maximally insulated hearts of removing insulation, and of connecting the apex with the pericardium. An AC-amplifier records from platinum electrodes. Standardization: 1 cm. = 20 mv. in this and subsequent figures. See text.

Cardiac electrode positions, the R-wave amplitude diminished still more (fig. 2, lower band). The changes of QRS configuration are shown for six experiments (four dogs) in figure 3. Preliminary studies with high-frequency response records showed striking differences not only in R-wave height but in the form of the extrinsic deflections preceding the inscription of the intrinsic* deflection. When insulation was withdrawn, the exact time of the onset of the (considerably altered) extrinsic deflections in left ventricular myocardial leads was less easy to discern (fig. 4, left and center). When the apex was also connected to the pericardium, the onset might be significantly delayed (fig. 4, upper right).

Effects of Changing Contact Between Altered Heart Surfaces and the Body

Myocardial Changes Limited to the Outer Muscle Layers

Fifty per cent procaine HCl was applied to a limited portion of the posterior ventricular surface, maximally insulated as described above. Before removing the insulation, time was allowed for the appearance

*Identified by Dürre et al. as the most rapidly inscribed portion of the descending limb of the ventricular complex of an epicardial or myocardial heart-body lead, since this "steep" part of the QRS (when present) was found to be simultaneous with the spike of a differential lead.
of upward RS-T segment deviation in D.C.-amplifier recordings at the site of topical application.* Free heart-body leads elsewhere on the heart were unchanged. With removal of the insulation any direct heart-body lead from the surface or substance of the exposed myocardium showed negative RS-T segment displacement as compared with the control levels. The magnitude of such reciprocal RS-T shifts was not as great as the local change originally recorded in heart-body leads from the insulated posterior wall, where diminution both of signal amplitude and relative RS-T elevation resulted from removal of the insulation. Lead II (at normal standardization) showed greatly increased net RS-T elevation when insulation was withdrawn. Figure 5 illustrates a characteristic series of events. Topical 5 per cent potassium chloride produced analogous reciprocal effects, mainly affecting the baseline, but also to a lesser degree the RS-T segment, T wave, and QRS complex (fig. 6). The reciprocal effects of applying procaine or potassium could be introduced or heightened at will by establishing or improving electrical contact of the physiologically altered area with the pericardial cradle (fig. 7). Altering posterior-wall temperature produced reciprocal T-wave changes unless the heart was insulated, in which case only localized changes resulted.

**Localized (Transmural) Ischemia**

Ischemic areas involving the posterior ventricular-wall muscle of insulated hearts did not produce net RS-T depression on the anterior surface inverse to the local effects (net RS-T elevation)† recorded over the distribution of occluded left circumflex coronary branches. With removal of the insulation, however, inverse changes appeared in almost every anterior lead and the severity of the local anterior abnormality (when the anterior apex was in the distribution of the occluded branch) diminished (fig. 8). The amplitude of inverse change varied considerably with different electrode sites. Anterior ischemic areas large enough to involve the left ventricular apex and reach its contact with the pericardial cradle produced inverse changes in anterior leads from uninvolved muscle, as well as from any exposed nonischemic portions of the posterior wall. Insulation abolished these changes. In contrast, ischemic areas confined to the exposed heart surface, as in the case of muscle in the distribution of a medium- or small-sized branch of the left anterior descending coronary, did not produce reciprocal effects, whether or not insulation was used. With ischemia of longer duration, the Q waves recorded over ischemic areas (common within 10 to 30 minutes at central or border muscle, particularly in the deeper myocardial substance and with small-area electrodes) were smallest (but not abolished) if the posterior atrioventricular surface was insulated from the pericardial cradle, and largest when there was good contact of heart and cradle with the posterior mediastinum.

**Discussion**

We have been attempting, in this as in earlier studies,1-3 to obtain a localized electrocardiographic representation of localized myocardial changes whenever possible, and hence have been concerned to avoid or recognize reciprocal information, since this tends to cancel local changes in direct heart-body leads from physiologically disturbed muscle and introduces spurious changes in leads from unaffected areas. The studies reported here inves-

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*Application of procaine to the exposed anterior ventricular wall at first produces a combination of epicardial RS-T elevation and baseline depression (mainly the latter), but within 5 to 10 minutes RS-T elevation predominates. Changes of progressively lesser amplitude occur in the outermost myocardial layer, but are not measurable more than a few millimeters beneath the surface. No changes are found elsewhere in the heart, whether the atrioventricular portion of the heart-body contact be insulated or not. The response to topical procaine thus differs somewhat from the effects of intracoronary cocaine injection observed by Alazamora-Castro et al.4 They did not describe the effects of topical application.

*Only A.C.-amplifier records were obtained in these particular ischemia studies. The absolute baseline and RS-T segment behavior of such situations is described elsewhere.*
Investigated the direct electrocardiographic effects of reducing the ventricular surface contacts of the heart and body below what is ordinarily the case for an open-chest heart suspended in a pericardial cradle. We thus proceeded in an opposite direction from the two other investigations which may be said to have examined pertinent phenomena.

Wood and Wolferth, attempting to improve the sensitivity of the body-surface electrocardiogram in reflecting experimental ischemia, demonstrated early and consistent local surface electrocardiographic changes resulting from coronary occlusion and then studied the effect of improving the surface contact of ischemia areas with the body. Pruitt and Valencia's turtle-heart studies were concerned primarily with the ventricular cavity representation of surface lesions and the surface representation of endocardial lesions in hearts of which the posterior wall of the ventricle remained in contact with the retrocardiac structures and a saline-filled carapace or bath. The main electrocardiographic interest of both groups was in representation of myocardial electrical phenomena by indirect leads rather than direct heart-body leads.

We have not attempted to alter the nature of the intracardiac portion of the heart-body contact area since there seems no method of so doing which does not produce profound disturbances of heart and circulatory function. The interesting recent work of Conrad and Cuddy, which includes partial replacement of heart-cavity blood with CO₂ gas, is thus not comparable with our procedures. These investigators did not attempt to increase the degree of isolation of the partially exposed heart, but demonstrated the large effects on the myocardial QRS complex of short-circuiting the overlying ventricular surface: We have not attempted to alter the boundary conditions for the sites of exploring electrode placement, but have confined our studies to direct heart-body leads from exposed or insulated portions of the heart as defined previously.

The differences among open-chest preparations may be considered as lying along a scale of degrees of completeness of heart-surface insulation. Animals with extensive contact...
between pericardial cradle and posterior mediastinum, as well as between posterior ventricular surface and pericardial cradle, represent one extreme. Animals with hearts insulated as far as the great vascular attachments or with ventricles lifted clear of the pericardial cradle are at the other extreme. We have confined our studies to these two extreme situations for the sake of simplicity. Random experimental open-chest preparations will show a great deal of variation between the extremes. Wide-chested dogs tend toward the large-ventricular-surface-contact situations while narrow, deep-chested dogs (requiring a high pericardial cradle to bring the heart within convenient investigative reach) tend toward more complete heart exposure. A further source of experimental variation stems from a tendency for some animals to show delayed or slight ischemic surface electrocardiogram changes, while others show marked changes almost as early as the more sensitive and extensive intramyocardial disturbance. If any portion of an area where there are ischemic surface changes is in contact with the body, large reciprocal effects will be found in all direct heart-body leads.

The effects of these variations in pericardial cradle-body contact and heart-pericardial contact may account in considerable part for divergent reports regarding QRS configurations in myocardial electrocardiograms, net RS-T depression frequency in localized ischemia situations, T-wave behavior variations, and the relative contribution of RS-T segment elevation and baseline depression in D.C.-amplifier studies of early massive ischemia. Indeed, intracellular potentials are not independent of reference electrode placement, since their amplitude is not more than twice that of free heart-body lead potentials.

Direct heart-body leads from hearts insulated less completely than the free heart-body leads we have described may have only a slightly reduced R-wave amplitude, but leads from those portions of the ventricular surface which lie at or near areas of contact with the body show potentials of intermediate or even low amplitude. Localized physiological changes will produce localized electrocardiographic changes only if they spare these partially short-circuited ventricular areas. Otherwise, the electrocardiographic representation of the spatially localized physiological changes will contain both local effects (partially canceled) and generalized reciprocal effects.

To avoid reciprocal effects with direct heart-body leads from less-than-maximally insulated hearts, care must be taken to confine experimental changes to completely exposed heart areas; for example, avoiding the run-
Local and reciprocal effects of procaine application to the posterior ventricular surface. The control baseline level for these direct-coupled amplifier records is indicated by a heavy line at each end of each strip. In dog no. 180, procaine was applied to the uninsulated posterior wall, while an anterior-surface heart-body lead was recorded continuously (uppermost strip). Baseline elevation appeared, reciprocal to the immediate local effect of topical procaine, which was baseline depression. The heart was then insulated, and a few minutes later another anterior-surface heart-body lead was recorded during withdrawal of the insulation, which required about five seconds. The beginning of the withdrawal is indicated by an arrow in this and the subsequent figures. Baseline elevation and RS-T segment depression appeared (second strip), reciprocal to the more fully developed local posterior-wall effects of the procaine. In dog no. 181, procaine had been applied to the insulated posterior surface several minutes before the third strip was recorded. With removal of the insulation, an anterior-surface heart-body lead again showed baseline elevation and RS-T segment depression. When the procaine effect had subsided, lead II was recorded (A.C. amplifier and normal standardization) after another application of procaine to the insulated posterior wall (lowermost strips). Slight net RS-T elevation and diminished QRS amplitude were recorded as compared with a control recorded with the heart uninsulated. With removal of the insulation, a greatly increased net RS-T segment elevation appeared, together with a comparatively slight increase of QRS amplitude. The epicardial QRS responses in these dogs (lower R, deeper S) are not mainly procaine effects, but are related to the removal of insulation, resembling those in figures 2, 3, and 4.

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Local and reciprocal effects of topical 5 per cent KCl. Application to the exposed anterior surface depressed the baseline and reduced QRS amplitude of the epicardial electrocardiogram, but did not alter the myocardial electrocardiogram 5 mm. beneath (dog no. 183). Application to the insulated posterior wall at its contact with the pericardium introduced reciprocal effects (mainly baseline elevation) in a myocardial heart-body lead from the anterior wall when insulation was removed (dog no. 177).

That localized electrocardiographic representations of physiological changes limited to the outer myocardial layers may be invariably obtained provided that the affected surface is not connected to the body might have been predicted from simple electrical circuit analogies. A locally altered area of exposed ventricular surface would not be expected to change direct heart-body leads unless it were made part of a circuit, either by application of an exploring electrode or by connection with the body. The magnitude of the reciprocal effects introduced by connecting an altered ventricular-surface area to the body is not predictable. Our experimental observations show these effects to be surprisingly large. The general nature of the relationship, however, can be inferred from the work of Pruitt and Valencia.7

That transmural ischemic areas should produce only local effects in direct heart-body leads, however, is by no means predictable from familiar electrical circuit or image analogies. It has been generally assumed that reciprocal effects are unavoidable with transmural ischemia because of cavity information transmitted via the atria and the vascular pedicle. Pruitt and Valencia7 demonstrated net RS-T segment elevation in direct endocardial (or adjacent cavity) heart-body leads from localized ischemic areas in the dog. Obstruction of relatively large dog coronary branches results in extensive areas of net RS-T depression over portions of the heart remote from the ischemic zone as well as at its borders.10 On the other hand, we had long been aware that small- or moderate-sized ischemic areas which happened to be confined to the exposed anterior heart surface pro-

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Effect of connecting a topical procaine application site to the pericardium. A saline wick had been placed in position before recording the two simultaneous upper strips from the anterior electrode positions indicated on the map. Removal of the wick demonstrated that this connection with the pericardium had been shifting the RS-T segment negatively in both the epicardial and the myocardial leads, as well as reducing R-wave, and slightly increasing S-wave amplitude. The lowermost strip compares the responses to topical potassium and procaine, using a sweep technique. The response of area (A) (not studied because it had to be short-circuited by the wick) was assumed to be similar to that of area (B): RS-T segment elevation with insignificant baseline depression.

duced electrocardiographic changes only in the vicinity of the ischemic zone,1-3 and Rakita et al.14 failed to find net RS-T segment negativity on the surface or in the myocardium of locally ischemic hearts.

It has now become obvious that insulation or exposure of those portions of the locally ischemic heart where net RS-T segment elevation has appeared will abolish all or most of the net RS-T segment depression found elsewhere. In maximally insulated hearts, moderate-sized anterior or posterior ischemic areas can be produced without any electrocardiographic changes remote from the ischemic zone. However, in the case of very large ischemic lesions not all remote electrocardiographic effects can be abolished by surface insulation. We have recently found that left main circumflex occlusion produces epicardial-surface RS-T depression and baseline elevation far beyond the apparent extent of the lesions; and these changes persist in part even when the heart is maximally insulated.

One would expect atrially recorded potentials of ventricular origin to be small when the ventricles are completely exposed, but they do not become any larger when portions of the surface are also connected with the body. Under neither circumstance do direct atrium-to-body lead QRS configurations resemble ventricular cavity-to-body lead configurations. Like the consistent absence of reciprocal effects from regional transmural ischemic areas, these observations indicate that ventricular-cavity information is very poorly conducted to the body via the vascular pedicle. If the ventricular cavity thus forms less of the effective heart-body contact area than is ordinarily supposed, the perivalvular ventricular muscle bands of the atrioventricular interface presumably must make a larger contribution. Futhermore, any ventricular
Direct and reciprocal effects on the anterior heart surface of posterior descending coronary occlusion. The occluded branch of the circumflex supplied the apex as well as the posteroseptal wall. With insulation, net RS-T elevation was present over the area of visibly disturbed function (positions K, I, L, and possibly J) but no net RS-T depression was recorded. With the removal of insulation, reciprocal RS-T depression appeared at (I, D, C, and B), along with partial or complete canceling of the local RS-T segment abnormalities in (K, I, and J). Insofar as changes were not equal in all leads, they were attributable to altered local behavior and the lack of exact similarity of epicardial electrode positions.

Surfaces that are added to the heart-body contact area have been shown to have unexpectedly large effects. While great caution is necessary in making inferences from open- to intact-chest situations, it appears that heart-surface information must play a very large part in the formation of body signals and that a number of widely-accepted assumptions, especially that certain combinations of body-surface lead points can provide direct ventricular-cavity information, require reappraisal.

Summary

In dog studies designed to improve the accuracy of direct extracellular heart-body leads, the electrocardiographic representation of the heart when maximally insulated (connected to the body only by its vascular pedicle) was compared with common open-chest experimental situations in which portions of the normally behaving ventricular surface are allowed to remain in contact with the body. The addition of any ventricular surface to the heart-body contact area (by removal of insulation) lowered the R-wave amplitude in all direct heart-body leads from the ventricular surface or wall.

The effects of locally altering the physiological behavior of portions of the left ventricle were also studied. Any portion of the ventricular surfaces of maximally insulated hearts could be altered by locally applied agents to show heart-body lead changes entirely confined to the area of demonstrably local physiological disturbance. Small- or medium-sized areas of transmural regional ischemia likewise produced electrocardiographic changes only in the vicinity of the ischemic muscle. In the case of less completely insulated hearts, direct heart-body lead changes consequent to localized physiological change...
or ischemia were found only in the vicinity of altered areas, provided that the affected area was widely exposed or otherwise insulated from the body. Whenever ventricular surfaces at which electrocardiographic changes had been induced were brought into contact with the body, reciprocal changes appeared in all direct heart-body leads, partially canceling local changes and making physiologically unaltered myocardium appear to be the source of electrocardiographic change.

The nature and extent of the ventricular surfaces contacting the body grossly alter open-chest, direct heart-body lead electrocardiograms. Experimental study of hearts which are the sites of locally altered ventricular electrical activity requires insulation or wide exposure of the overlying surface to avoid or minimize reciprocal effects. These can account for important discrepancies among the findings of different investigative groups. Their measurement requires routine comparison of leads from maximally insulated hearts with the findings for less completely insulated hearts. That medium-sized ischemic areas do not produce reciprocal effects, provided their surfaces are insulated from the body, suggests that less ventricular-cavity information is transmitted via the atria and the vascular pedicle than is ordinarily supposed.

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JOHN J. SAYEN, AARON H. KATCHER and GEORGE PEIRCE

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