Origin of Body Surface QRS and T Wave Potentials from Epicardial Potential Distributions in the Intact Chimpanzee

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SUMMARY Epicardial and body surface QRS-T wave potential distributions were measured during normal and ectopic sequences in intact chimpanzees. Epicardial potential distributions were used because they provide a comprehensive picture of total cardiac electrical activity for relating heart and body surface events during both ventricular activation and repolarization. When the epicardial potential gradients existed over a distance greater than that to the recording points on the body surface, e.g., as occurred during the overlap of terminal ventricular activation and early repolarization, the epicardial events were mirrored well on the anterior chest surface. However, when the recording points were at a distance greater than that over which the epicardial potential gradients existed, the details of the epicardial events disappeared and their effect was to produce distinct changes in the low-level potentials over broad distant areas. The major manifestations on the body surface of selective epicardial events frequently were changes in the distant low-level potential areas while there was no change in the pattern near the maximum or minimum. The ST-T wave body surface distributions were as useful as the QRS patterns for localizing the ventricular ectopic foci presented. A direct experimental basis is provided for explaining T wave notches which occurred during normal and ectopic beats and resembled U waves. It should be possible to achieve as precise an understanding of ST-T waves on the basis of epicardial potential distributions as has thus far been achieved for QRS on the basis of isochrones.

The long-term electrocardiographic goal is to understand the sequence of electrical events in the total heart that produce body surface potentials and to explain the relationship between the two on the basis of experimental measurements for any instant during the cardiac cycle. At present there are several major limitations to achieving both components of this goal. The first problem is that there are little experimental data to provide the accompanying heart electrical information to account for measured body surface potential distributions. Second, as noted by Taccardi et al.,1 it is “often an arduous process to grasp the relationship” between the cardiac excitation waves represented by isochrones2-3 and the body surface potential distributions. Third, there has been no way to characterize and to measure directly the cardiac electrical events of ventricular repolarization. This void has restricted studies of the relationship of the electrical events in the total heart and those on the body surface to the QRS complex. A final limitation has been the lack of experimental capability to measure almost simultaneously both the potentials surrounding the heart and the corresponding body surface potential distribution in the same biological specimen with the volume conductor intact, a requisite for verifying the origin of body surface potentials throughout QRS-T waves.

This paper considers the use of potential distributions surrounding the heart (potentials on the epicardial surface of the heart) as a way to characterize cardiac electrical activity for relating heart and body surface events. For study of ST-T waves, measurement of ventricular intramural and epicardial potential distributions in intact dogs4-6 has been shown recently to provide a method previously lacking to describe in detail the events of repolarization of the heart in a way that is analogous to the use of the isochrone method7 for describing in detail the events of depolarization of the total heart.

Our goal was to examine experimentally the relationships of heart and body surface events during both ventricular excitation and repolarization. For this we used the intact chimpanzee to achieve the closest experimental approximation to the human. In this paper we present the results for normal beats and go on to study ectopic foci at the apex of each ventricle. These sequences were selected because they provide a good test of how changes in cardiac electrical events in normal ventricular muscle affect the total body surface potential distribution, and they illustrate QRS and T wave features for distinguishing one ectopic site from another.

Methods

We initially used the same intact dog preparation that was previously reported for measuring ventricular intramural and epicardial potential distributions throughout the heart6-7 except that only epicardial electrodes8 were implanted. In 20 intact dogs we were unable to document the presence of prominent T wave notching during normal sequences,7,9 all of which were recorded at heart rates greater than 90 beats per minute. Also, there were differences between the dog and human body surface maps, and we considered these differences due to heart orientation and torso shape variations. Therefore, we undertook a systematic examination of the heart-body surface anatomical relations of numerous primates to achieve an experimental preparation that was as close an approximation to the human as possible.

Selection of the Preparation

We first identified the major geometric differences between the human and the dog. The cross-sectional anatomy of the adult human thorax at the ventricular level is shown in figure 1, A1 as taken from Pernkopf’s atlas.8 At the completion of the study of several dogs the carcass was frozen, cross sections of the thorax made, and from direct imprint drawings of each cross section the geometry of the

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thorax and its contents were documented (fig. 1, B). Similar studies were conducted in the two intact chimpanzees of this report (fig. 1, A2).

The cross sections of figure 1, A1 and B illustrate well known differences in thoracic anatomy of humans and dogs that become important in relating heart to body surface potentials. The shape of the adult human thorax, i.e., the transverse diameter greater than the anterior-posterior one, is reversed in the dog due to the elongated oval shape of its thorax. Also, the position of the heart is more caudal in the dog as compared to the human and the relative distance between the anterior epicardial surface of the heart and the skin overlying the sternum is greater in the dog than in the human. Finally the orientation of the ventricles is different: in the human the two ventricles are oriented in a transverse direction with an anterior-posterior relationship whereas in the dog this orientation is primarily a vertical one.

Examination of numerous animals revealed that only in the advanced primates that conduct locomotion in the most orthograde manner of any group, the superfamily Hominioidea (apes and man), does the cardiac geometry achieve a transverse orientation of the two ventricles similar to the human. Also, the torso shapes of the chimpanzee (fig. 1, A2) and of the gorilla are more similar to that of the human than that of the dog. Further considerations of primates and a comparison of dog and chimp potential distributions are given in Appendix A.

We next recorded body surface maps in ten chimpanzees. In none of these were U waves or clearly notched T waves found. However, all of these chimps were estimated to be younger than three years old, weighed less than 40 pounds, and had heart rates above 100 beats per minute. We then obtained the largest male chimpanzee that we could, one that weighed 80 pounds and was estimated to be nine years old. Surface mapping revealed prominently notched T waves which were distinguished from U waves by the criteria described by Lepeschkin. We elected to study this chimpanzee as a counterpart of the adult human and selected a three-year-old male chimp that weighed 35 pounds to study as a counterpart of a school-age child.

The Preparation

In each chimp, control preoperative QRS-T wave isopotential body surface maps were recorded under the same general anesthesia that was used at the time of final study. After extensive testing for tuberculosis and observation for six weeks, each animal underwent surgery. Through a median sternotomy 75 epicardial electrodes were implanted with approximately equal spacing for each ventricle to provide extensive coverage of the ventricles and the atria. The electrodes were the type previously used in intact dog preparations and consisted of insulated silver-coated copper wire with an exposed 1 mm tip anchored to the epicardium by a superficial stitch. Bipolar pacing electrodes were su-

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FIGURE 1. Cross-sectional thoracic anatomy of the human, chimpanzee, and dog. The thoracic section of the adult human (A1) was drawn from Pernkopf's atlas.* That of the chimpanzee (A2) was made from direct photographs of the frozen section of the thorax of the 9-year-old chimpanzee of this study, as were the sections of the dog (B). Cross section B1 of the dog was 3 cm cephalad to cross-section B2; both are shown to illustrate that the ventricles of the dog have a vertical orientation; only the right ventricle was transected in B1, and in B2 the section included the lower portion of the right ventricle and the main portion of the left ventricle. The numbers at the surface of each thoracic cross-section indicate the location of the 15 columns used for positioning the electrodes to record the body surface potential maps. (Permission to use the drawing of the human thoracic cross-section was kindly given by W. B. Saunders Co., Philadelphia, from Figure 134 of Pernkopf: Atlas of Topographical and Applied Human Anatomy.)
tured to the upper right atrium in the sinus node area. To approxi-
mate naturally occurring ectopic foci that would occur in the
immediately underlying Purkinje system at the apex of each
ventricle, the noninsulated portion of the pacing wires was sutured transmurally in these areas where the ventricular walls were only 2 mm thick (as determined at nec-
ropsy). The insulated wires were looped within the chest, and the
distal ends brought out superficially, implanted beneath the
skin of the lower abdomen, and exposed at the time of study.

Postoperatively both chimps survived, did well, and ap-
peared to be quite active, vigorous, and healthy. When
repolarization had returned to normal 14 days after surgery, as judged from visual comparison of body surface maps, each chimp underwent study.

Protocol for Measuring Heart and Body Surface Potentials

At the time of study each chimp was premedicated with 8
mg/kg i.m. of ketamine hydrochloride and intubated with a
cuffed endotracheal tube. Thereafter, each was maintained
with 0.5–0.8% halothane which was a level of anesthesia just
sufficient to depress the cough reflex. Ventilation was con-
trolled with a Harvard respirator at a minute volume
necessary to maintain a PaCO2 of 35–45 torr. A mixture of
nitrous oxide and oxygen was used to maintain a PaO2 of
100–200 torr. The resulting preparation was quite stable for
15 hours; maintenance fluids were administered in-
travenously throughout the recording procedure. The wires
from the heart were exposed and attached to individual con-
nector cards. These were inserted into a switching box that
allowed rapid sequential recording of the waveforms at each
of the 75 epicardial positions with each of 15 epicardial
points being recorded simultaneously. Body surface poten-
tials were recorded from 150 sites in reference to Wilson’s
central terminal. The central terminal was used for body
surface recordings to maintain conformity with the current
widespread practice of this reference for surface maps. The
epicardial potentials, however, were measured in reference
to the left leg. As can be seen from the maps in the results,
the difference in reference, which would be the left leg
voltage, is insignificant relative to the voltage present on the
heart for all instants. A grid composed of 15 columns and 10
rows was drawn on the torso (fig. 2A) with the interelectrode
spacing between the columns greater in the back than on the
front. Needle electrodes were inserted at each point and con-
ected to a switching box for rapid sequential sampling of 20
body surface leads simultaneously. To record a complete set
of waveforms required approximately 12 minutes for the
body surface and 10 minutes for the epicardium.

Since changes in heart rate produce well known T wave
changes,7, 12, 13 the heart rate was held constant throughout
each sequence, and the same rate was used for different se-
tuations to allow comparisons of the different sequences at
the same rate. The spontaneous rates were 92 and 105 beats
per minute for the large and small chimp; to control cardiac
rate, atrial pacing was carried out at fixed rates of 96 and
112 beats per minute, respectively. Pacing stimuli were 1
msec impulses at 1.5 times the threshold value. Since the
atrium had to be controlled so that atrial currents did not in-
terefer with the interpretation of ventricular repolarization
events, the heart was controlled during ventricular pacing by
simultaneously pacing the right atrium and the ventricular
ectopic site with the ventricular stimulus delivered 40 msec
after that to the sinus node area. Frequent checks of the P-R
interval with right atrial pacing alone ensured that there was
no influence from atrioventricular transmission (fusion) when
evaluating ectopic foci, since ventricular activation
was completed before the P-R interval ended. This pacing
procedure avoided retrograde atrial activation and produced
a stable rate throughout.

The pacing sequence was controlled by a PDP-11/20
computer which synchronized the pacing stimuli with data
recording. The design of the computer system14 for measur-
ing cardiac electrical signals while simultaneously con-
trolling heart rate and rhythm comprised five basic tasks: (1)

![Figure 2](http://circ.ahajournals.org/content/55/2/270/F2.large.jpg)
synchronizing the system with events occurring in the heart (cuing); (2) controlling heart rate and rhythm by appropriately timed stimuli; (3) continuously displaying the waveforms; (4) displaying the status of the computer and the experiment; and (5) receiving, checking, and executing commands from the investigator. The recording ensemble consisted of 24 a-c amplifiers; the output of each amplifier was sampled at a rate of 1000 samples/sec. The computer stored the data and displayed the waveforms immediately on a Tektronix 4002 display unit. Once it was established that each beat was free of artifact, the waveforms were recorded on digital tape. In each chimp, the normal sequence was recorded during sinus rhythm followed by recording the sequence during pacing of the right atrium; both sequences produced the same QRS-T wave potential distributions. Thereafter, numerous ventricular ectopic sequences were produced. At the end of the experiment, a repeat recording was made of the normal sequence to ensure that there had been no change in the normal QRS-T wave events.

After each experiment the carcass was frozen and serial sections of the torso were made from the level of the neck to the lower abdomen. Direct imprint drawings were made of each section. The position of each of the 150 body surface electrodes was documented on the separate cross sections. Finally, the sections of the heart, each 1.5 cm thick, were dissected to document the location of each of the 75 epicardial electrodes. Figures 1 and 2 illustrate the relation between various parts of the heart and the body surface electrodes.

A second computer program was used to redisplay the waveforms and photograph them from the Tektronix display unit. To ensure the absence of baseline shifts throughout the QRS-T wave, only waveforms that were judged to have minimal linear drift, or no detectable baseline drift, were used. After baseline points were selected at instants just before the onset of two consecutive P waves to include the QRS-T wave for analysis, the computer program applied linear interpolation to adjust each potential value from the first baseline instant to the second one. The common reference waveforms were used for time-alignment purposes, and potential maps were printed for each 1 msec interval throughout ventricular activation and for each 4 msec interval throughout repolarization. The heart-body surface relationships during the P wave were not analyzed. However, checks were performed for each ectopic sequence to exclude any detectable overlap of atrial and ventricular activation potentials during early QRS.

A special computer program displayed the epicardial and body surface potential maps with the contour lines drawn in final form, and automatically photographed each map for subsequent instant-by-instant and motion analysis. Finally, the unipolar waveforms were utilized to construct isochrone epicardial activation sequences. Previous atrial and ventricular activation studies showed that the same isochrones were produced by analysis of either unipolar or bipolar waveforms.

**Evaluation of Methods**

Epicardial repolarization positive potentials occurred during the ST segment over wide areas of the ventricles during normal beats and occurred in the area of early excitation with ectopic beats. Therefore, it was important to ensure that these positive ST-segment shifts were not due to local injury caused by the electrode. Each electrode was checked to rule out the presence of local injury as described in detail previously.4 Pacing at varied ventricular sites altered the ST segments so that they became negative or isoelectric whenever the recording point was located in the area of terminal ventricular excitation; only at two electrode sites in the young chimp did the initial ST segment remain positive and these two points were discarded. Because of the above considerations, we consider the damage produced by the electrode insertions to be insufficient to alter the overall excitation and repolarization distributions as depicted. Also, because the body surface T waves returned to the preoperative state and since there was marked similarity between the final epicardial distributions and those recorded in intact dogs, we believe that repolarization could be considered normal at the time of final study.

Since changes in the metabolic state can alter T waves, continuous monitoring throughout each 15 hour recording procedure ensured a steady metabolic and hemodynamic state as evidenced by frequent measurements of arterial pH (range 7.40 to 7.51) and potassium (range 3.3 to 3.8 mEq/L); arterial blood pressure was monitored continuously via a femoral artery catheter with a P23AA pressure transducer and was 120–140/75–80 mm Hg in the older chimp and 85–100/55–60 mm Hg in the small chimp.

We were not able to repeatedly measure potential distributions to evaluate the effects of surgery in the chimps. However, we evaluated the evolution of epicardial and body surface T wave changes postoperatively in 12 intact dog preparations. For four days after surgery the lower torso was occupied by negative T wave potentials and the upper chest by positive potentials, the opposite of the normal control state.4 The body surface distributions returned to normal within five to nine days postoperatively. The ventricular epicardial T waves for the first four days were predominantly negative. As the body surface distributions returned to normal, the epicardial T waves changed to those previously described for normal sequences, i.e., there were predominantly positive potentials over the ventricle throughout the ST-T wave.4 Local injury potentials were present for all electrodes for 9 to 12 hours postoperatively. In one-fourth of the electrodes local injury potentials persisted for five days, i.e., the ST segments did not become negative or isoelectric when the recording point was in the area of terminal excitation of ectopic beats. When the body surface distributions had returned to normal, the precordial ST-segment elevations on the body surface occurred just as they had in the preoperative control state suggesting that epicardial and body surface ST-segment positive potentials were not artifacts. Thirty intact dogs followed two to ten weeks after surgery have shown stable body surface maps after the tenth postoperative day.

**Results**

The major features of the epicardial potential distributions to be presented were consistent for each chimp. The differences occurred only in the normal sequences during which there were prominent notches in the downstrokes of some T waves in the large chimp but not in those of the small one. Also, prominently positive T waves occurred over the right ventricle (RV) in the large chimp, as occurs in adult
dogs, while lower level positive or slightly negative right ventricular T waves occurred in the small chimp. Because of the marked similarities of the potential distributions produced by comparable atrial and ventricular ectopic foci for the two chimps, the results are presented only for the large chimp to allow comparisons between the different sequences for similar time instants.

Figure 3A shows the 150 body surface locations for which potential values are presented in each map. Column 15 is duplicated to produce the same potentials at each side of the map. The location of the heart is shown in reference to that of the anterior body surface leads; these geometric relationships were determined from the cross sections of the thorax and from the chest X-rays. The location of the 75 electrodes used to measure the epicardial potential distributions is shown in figure 3B.

**Normal Sequence (figs. 4 and 5)**

**Potential Distributions during Ventricular Activation.** The general sequence of epicardial changes was similar to that of the dog. Initially positive potentials covered most of the ventricles except for negative potentials on the lateral left ventricle (LV). On the body surface this produced an anterior maximum and a posterior minimum. Subsequently there were rapid changes due to right ventricular break-through which was apparent on the epicardium for approximately 5 msec before a saddle distribution typical of that seen in humans occurred on the body surface. During the latter half of ventricular activation the epicardial patterns became complex due to multiple isolated excitation waves, and these complexities were not reflected in the same patterns on the body surface. Changes on the epicardial diaphragmatic surface produced little change on the body surface in the area of the maximum and minimum but produced major changes in the distant low-level potentials, especially over the right lower torso.

5 msec (fig. 4): On the epicardium a maximum was present over the septum anteriorly and a minimum at the LV base. The body surface pattern reflected this with an anterior maximum over the one on the epicardium and with a minimum on the back.

11 msec: Epicardial positive potentials extended over the apex of the LV and a second maximum developed on the diaphragmatic surface. The minimum remained stationary. On the body surface the positive potential area expanded inferiorly while the minimum did not move.

16 msec: Negative potentials had developed on the anterior RV due to epicardial breakthrough which had occurred at 11 msec. The maxima on the LV increased in magnitude. On the body surface there was a saddle distribution with a minimum over the upper sternum where there had been positive potentials. The positive potential area had expanded inferiorly on the torso.

28 msec: Epicardial breakthrough on the diaphragmatic surface of the LV produced a minimum there, and the RV was enveloped in negative potentials. Positive potentials covered most of the LV on which there was a single maximum. The body surface distribution was more simple. The minimum was over the lower sternum in front of the epicardial minimum, and the maximum was on the left precordium directly over the left ventricular epicardial maximum. Positive potentials formed a broad base around the lower torso.

44 msec: Two excitation waves produced isolated positive potential areas superimposed on a predominantly negative epicardial pattern. Scattered positive potentials due to repolarization appeared. On the body surface the major change was the development of negative potentials on the right lower torso with little change in the maximum and minimum.

**ST-T Wave Potential Distributions.** During the upstroke of the T wave there was a stable epicardial pattern with positive potentials over most of the ventricles and negative potentials over the atria; there was a minimum at the base of the LV where excitation had ended. On the body surface there was also a stable pattern with an anterior maximum and a posterior minimum. However, during the downstroke of the T wave there were sequential epicardial changes; i.e., the apex of the LV became negative after which there was a shift of positive potentials into that area during terminal repolarization. On the body surface there were associated sequential changes that produced notched T waves.

110 msec (fig. 4): On the epicardium there was an anterior
and a posterior maximum during early repolarization, and positive potentials covered most of the ventricles. However, there were two isolated areas of negativity that corresponded to the areas of late ventricular activation, one near the base of the LV and one on the diaphragmatic surface. Negative potentials occurred over most of the atria. On the body surface there was an anterior maximum and a minimum was in the back.

218 msec (fig. 5): The epicardial pattern remained the same as the magnitude of the maxima increased. These epicardial events were associated with a stable body surface pattern.

286 msec: During the downstroke of the T wave rapid changes occurred on the epicardium with a minimum developing at the apex of the LV. On the body surface the minimum shifted toward the left precordium, and the maximum did not move.

322 msec: The epicardial pattern continued to change with positive potentials completely replacing the previously negative ones at the apex of the LV. Negative potentials persisted over the atria. On the body surface the maximum decreased while the magnitude of positive potentials in the lower left precordial region increased. There was marked expansion of the positive potential area inferiorly. These events coincided with the inscription of T wave notches on the lower left precordium.
all of the lower torso became negative. The body surface minimum remained in the same place and in about the same relation to the maximum despite the change in cardiac events.

95 msec: On the epicardium, positive potentials due to repolarization occurred over the LV with a repolarization maximum at the ectopic stimulus site. Positive potentials on the anterior RV near the AV ring continued due to excitation. The body surface pattern was not a simple projection of the epicardial one; however, note the extension of positive potentials downward on the left chest as a result of the epicardial repolarization positive potentials. Excitation ended at 109 msec (not shown).

**ST-T Wave Potential Distributions.** The epicardial potentials were basically patterns having an initial minimum near the ectopic focus at the apex of the LV and a minimum near the site of terminal excitation on the RV. However, as in epicardial sequences in the dog, during the downstroke of the T wave the fundamental pattern went through a series of subtle, but definite, phases with two maxima developing from the initial one. The epicardial patterns produced simple body surface potential distributions which also changed in a definite way during the latter part of the T wave.

131 msec: The initial epicardial repolarization maximum was at the ectopic site. Its magnitude was considerably greater than that of the minimum which was near the site of terminal ventricular excitation. The body surface pattern was similar to that of the epicardium with the maximum located on the left precordium immediately over the ectopic stimulus site.

235 msec: The epicardial maximum remained stationary and increased in magnitude. The steepest potential gradients were near the maximum. This produced a similar distribution on the body surface, with an expanded area of positive potentials inferiorly on the torso.

330 msec: The single epicardial maximum now had evolved into two maxima both of which were closer to the minimum. The area of positive potentials increased. On the body surface the maximum and area of positive potentials shifted into the upper sternal region.

**Ectopic Focus at Left Ventricular Apex (fig. 6)**

**Potential Distributions during Ventricular Activation.**

Pacing the left ventricular apex produced the general sequence of epicardial potential changes that was expected, i.e., a series of reasonably simple patterns, beginning with a minimum at the ectopic focus and positive potentials away from the ectopic site. The body surface pattern had a minimum directly overlying the ectopic site. As excitation progressed, the boundary between positive and negative potentials moved away from the pacing site. This boundary was roughly in the same place as the excitation wave determined by the isochrone measurements. As excitation continued, it produced an increasing area of epicardial negative potentials and a decreasing area of positive potentials; these epicardial changes produced similar changes on the body surface. During the last 15 msec of QRS the epicardial pattern changed due to a repolarization maximum at the ectopic site; this maximum increased while on the opposite side of the heart there was a decreasing area of positive potentials due to terminating excitation waves. These epicardial events produced similar changes on the body surface with low-level positive repolarization potentials developing over the ectopic site as the excitation maximum disappeared.

21 msec: A prominent minimum occurred near the stimulus site. The isochrone excitation wave was in the negative potential region. Two maxima were on the LV, one anterior and one posterior. The body surface pattern was approximately a projection of the epicardial pattern.

55 msec: The epicardial area of negativity enlarged, and

**Ectopic Focus at Right Ventricular Apex (fig. 7)**

**Potential Distributions during Ventricular Activation.**

The general patterns had the same major characteristics of the previous sequence. An initial prominent minimum was near the right ventricular ectopic site. On the body surface the associated minimum was located immediately over the ectopic site. As excitation continued, the area of negative potentials expanded away from the ectopic site, and the area of positive potentials decreased toward the site of terminal excitation. Similar changes occurred on the body surface. During late ventricular activation the epicardial and body surface patterns became more complex due to repolarization positive potentials near the ectopic site.

25 msec: A prominent epicardial minimum occurred near the ectopic site. There were two maxima, both of lower magnitude than the minimum, on the LV. On the body surface the minimum occurred immediately over the ectopic site. Positive potentials covered most of the torso with a single maximum on the back.

55 msec: The epicardial negative potentials expanded
The two initial maxima was located immediately overlying the epicardial maximum at the ectopic focus during the upstroke of the T wave (131 and 235 msec).

293 msec: On the epicardium the two maxima continued to shift toward the stationary minimum. On the body surface little change occurred except for slight expansion of the positive potential area.

Interpretation of Body Surface T Waves

By knowing the epicardial and body surface potential distributions, it is possible to interpret the shapes of the unipolar waveforms recorded in various areas. Since the relation of the shape of unipolar body surface T waves to total heart electrical events is not known, it seemed appropriate to use the epicardial distributions for analysis of the body surface T waves recorded near the heart. The waveforms recorded at specific epicardial sites and those recorded from the body surface precordial positions of the previous sequences are shown in figure 8.

Elevation of the epicardial ST segments occurred normally (A1), particularly on the anterior LV in the area adjacent to the septum. This area was where the anterior epicardial maximum was located during the ST segment and upstroke of the T wave.

For the left ventricular apical focus the most prominently positive ST-T waves occurred near the apex of the LV (A2). The T waves on the body surface (B2) had the greatest
FIGURE 7. Ectopic focus at right ventricular apex. During early QRS (25 msec) the body surface minimum occurred immediately over the ectopic focus at a location medial to that of the previous sequence. Ventricular excitation ended at 104 msec (not shown). On the body surface the repolarization maximum was located immediately over the ectopic focus; i.e., at the same site where the initial excitation minimum had been (190 msec).

FIGURE 8. Epicardial and precordial waveforms recorded during the normal and ectopic sequences. Epicardial waveforms of the three sequences are shown in A with dashed lines indicating the recording sites. The accompanying precordial body surface unipolar waveforms for rows 4 and 5 are shown in B with the arrows indicating the location of columns 5–9 in relation to the heart. The numbers indicate the sequences: 1 = normal, 2 = left ventricular ectopic focus, and 3 = right ventricular ectopic focus.
magnitude in the overlying region in column 8. For the right ventricular focus the most prominently positive ST-T waves occurred in the septal region (A3) near the apex of the RV. These changes were reflected on the body surface (B3) in the overlying area in column 7. To correlate the site of the ectopic focus with the body surface waveforms for the two ectopic sequences, the T waves showed as good a correlation with the underlying epicardial events as did QRS. In fact, in the precordial leads (B2 and B3) ST-T waves changed more from lead to lead overlying the stimulus site than did the shape or negative amplitude of the QRS. That the shape of the T wave and its amplitude in the body surface waveforms should correlate with the site of the ectopic focus can be explained on the basis of the epicardial potential distributions. These showed that during the upstroke of the T wave the repolarization maximum occurred in the epicardial area of the ectopic focus.

Just as prominently positive epicardial T waves occurred near the ectopic focus, prominently negative T waves occurred near the site of terminal ventricular activation. The epicardial repolarization potential distributions indicate that the polarity of these T waves was due to gradients that extended across the vessels between the ectopic focus and the terminal site of activation. Positive T waves occurred in areas of early repolarization and negative T waves occurred in areas of late repolarization.

In the normal sequence the terminal notch of precordial T waves (B1) was related to similar prominent notchings of T waves on the left ventricular epicardial surface near the apex (A1). In figure 5 the epicardial map at 218 msec shows that before the time of the notch the apex of the LV was positive as a part of the overall epicardial pattern. This pattern of mostly positive ventricular potentials was due to the effect of the transmural gradient produced by the epicardium repolarizing before the endocardium. A secondary effect was the influence of excitation, where negative potentials occurred in the two areas of late ventricular excitation. The map at 286 msec shows that at the time of the low point in the notch, the apex of the LV was negative because the apex was taking longer to repolarize than the left ventricular areas where the maxima were located. The subsequent map at 322 msec shows that at the time of the peak following the notch, the apex had again become positive, because of the tendency of the epicardium to repolarize before the underlying endocardium. This effect became evident because the apex region continued to repolarize as the more basal areas were completing repolarization. The apex-to-base extracellular gradient was more prominent than found in the dog (i.e., indicating a greater difference in action potential durations). This further confirms the suggestion of Burgess et al. from their refractory period measurements in dogs that normally repolarization occurs later at the apex than toward the base.

During ectopic sequences no terminal T wave notches were in the leads where they occurred in the normal sequence but positive deflections following negative T waves appeared in other leads. The left ventricular apical focus resulted in negative epicardial T waves with terminal positive deflections on the anterior right ventricular free wall (fig. 8, A2). On the overlying body surface (B2) similar changes occurred in the precordial T waves on the right chest (B2, row 4). The epicardial potential distributions showed that the terminal positive deflection of these T waves was due to the repolarization maximum and positive potentials that continued to shift away from the ectopic site onto the RV. In figure 6 the epicardial map at 235 msec shows that during the negative portion of the T wave the RV was negative due to later repolarization than the LV. The map at 330 msec shows that during the time of the terminal positive deflection the RV had become positive because of the increasing area of completed repolarization, a process that began at the ectopic site. At that time the right ventricular area near the anterior wall maximum was the region that was most nearly repolarized of the areas still actively repolarizing. A quantitative explanation for these events can be given on an intracellular-extracellular basis. Similar terminal deflections occurred on the lateral left chest (fig. 8, B3), due to the right ventricular apical focus; they were associated with similar terminal positive deflections on the lateral LV.

Discussion

The fundamental emphasis provided by the results is that a comprehensive picture of the relationship between total cardiac electrical activity and body surface potentials during both ventricular activation and repolarization can be obtained on an experimental basis. There were two major features involved in epicardial events being projected to the body surface: a) the magnitude of the potential gradients and the distance over which they existed on the epicardial surface (cardiac sources), and b) the distance to the recording area of the body surface. For any instant, when the potential gradients on the anterior epicardial surface existed over a distance greater than that to the recording points on the anterior chest, the epicardial events were mirrored in some detail on the overlying anterior chest surface. However, when the recording points were at a greater distance as compared to the distance over which the epicardial potential gradients existed, the details of the epicardial events disappeared and their effect was to produce distinct changes in the low level potentials that occurred over broad distant areas. These distance factors were more important than the absolute potential magnitudes on the epicardium in determining the degree to which the body surface potential distribution mirrored that of the epicardium. The above cases are specific applications of general principles of body-surface relationships; other examples correlating the known location of single and multiple pacing sites with regional effects on the body surface in dogs recently were presented by Abildskov et al. Major emphasis in the interpretation of body surface maps has focused on the changing positions of maxima and minima as an indication of changes in the cardiac sources. Possibly this practice relates to the importance, as well as the fascination, of the equivalent dipole concept as the basis of vectorcardiograms. These results indicate that, while the position of the maximum and minimum is important, quite frequently the major manifestations on the body surface of changing events in the heart can be seen in the patterns of the low level potentials while little or no change occurs in the pattern around the maximum or minimum. Particularly, changes on the diaphragmatic surface of the heart often produced little or no change in the position of the body surface maximum and minimum while there were easily seen effects in distant low level potential areas (see fig. 4, 28 and 44 msec). These simple changes in the body surface distribu-
tion usually occurred with no increase in the complexity of the overall pattern.

During the course of the ST-T wave a predictable series of events occurred as evidenced by the changing magnitude and position of the epicardial gradients. These changing gradients existed over a considerable distance during the downstroke of the T wave and produced notches in epicardial and body surface T waves (fig. 8). The results provide a direct experimental basis for explaining T wave notches in normal and ectopic repolarization sequences.

Abildskov et al. recently stated that "the cardiac source-surface potential relationship which is fundamental in electrocardiography is recognized to be complex. Source orientation and strength, volume-conductor characteristics of the body, and source location all are factors in the relationship. This multiplicity of factors makes it difficult to rigidly prove and quantitatively define rules of each." The results of this study indicate that epicardial potential distributions in intact animals provide a way to separate the effects of the cardiac generator from those of the volume conductor and thereby reduce the complexity of the problem. On the one hand, epicardial potential distributions directly characterize the source orientation, strength, and location of the underlying heart events. On the other hand, they afford a basis for mathematical studies during all phases of the cardiac cycle to relate the potentials on the epicardial surface of the heart to those on the body surface in a form that can be verified by experimental measurements. This feature makes it possible to develop a new way to view all the body surface potentials directly in relation to the heart as done recently with computed epicardial potential maps, a feature that should assume increasing importance to extend the clinical use of potential distributions.

Appendix A

Except for the Hominioidea (apes and man), all other primates, including the orangutan,42 the squirrel monkey,43 and the baboon,44 are predominantly pronograde or quadrupedal in locomotion and all have cardiac geometric relationships similar to the dog, i.e., the left and right ventricles are oriented primarily in a caudo-cephalic direction and all have thoracic shapes that are more oval like the dog than like the human. Thus, the geometric relationships of the two ventricles are related to adaptations involved in pronograde versus orthograde locomotion. In addition, Jolly44 points out that a number of other morphological features separating the superfamilies Hominioidea (man, chimpanzee, gorilla, and orangutan) from the superfamily Cercopithecoidea, which includes the rhesus monkey and the baboon, are related "to the fact that the Cercopithecidae are predominantly pronograde and quadrupedal in locomotion, while the living Hominioidea are structurally adapted to orthograde locomotion. . . . The reader is referred to the work of Buettner-Janusch for a review of the classification of primates."

The epicardial potential distributions in the chimps and in the dogs were essentially the same for the ectopic sequences shown, as well as for other ectopic sequences. For the normal sequence both chimps had more prolonged terminal ventricular activation in the anterolateral basal area of the LV than the dogs. Species differences primarily were noted for the body surface distributions. 1) During normal sequences the "saddle distribution" following right ventricular epicardial breakthrough consisted of two distinct minima in the chimps with patterns similar to humans, whereas in dogs two distinct minima of the "saddle distribution" usually were not present. 2) Epicardial left ventricular events (maxima and minima) in the dogs were associated with body surface changes predominantly on the lower torso whereas in the chimps similar epicardial events were reflected in the left axilla and back. 3) Anterior right ventricular events in the dogs were projected predominantly to the right thorax and in the chimps similar events produced changes over the sternum and in the left parasternal areas.

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