Body-surface Maps of Heart Potentials

Tentative Localization of Pre-excited Areas in Forty-two Wolff-Parkinson-White Patients

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SUMMARY Heart potentials were recorded from the entire chest surface in 42 patients suffering from Wolff-Parkinson-White syndrome. We were able to identify six types of surface maps, according to the location of the potential maximum and minimum during the delta wave. For each of these types we suggested the most likely location of the pre-excited region around the A-V rings (types 1 to 5) or in the interventricular septum (type 6). In 13 patients belonging to Types 1, 2, 3, 5 and 6 our hypotheses were in agreement with intracardiac recordings, epicardial maps or surgical results obtained by others. Isopotential surface maps provide more information on the location of the pre-excited area than conventional ECGs, particularly when these exhibit intermediate features between Types A and B.

The Wolff-Parkinson-White (WPW) syndrome is generally considered to be due to the early excitation of some portions of the ventricles through anomalous conduction pathways. A number of anatomical and electrophysiological studies have proven the existence of these pathways. Accessory conducting pathways can be found in various sites in the heart: between the atrial and ventricular heart muscle anywhere along the fibrous rings (Kent bundles); between the atrial septum and the His bundle (James fibers); and between the His bundle (or bundle branches) and the septal myocardium (Mahaim fibers).1-24 Accordingly, pre-excitation spreads through the ventricles from different points and various potential patterns can be expected to appear at the surface of the body. Conventional ECGs and VCGs provide useful information on the orientation and direction of spread of the pre-excitation wavefront, but they do not usually enable the location of the pre-excited area to be determined reliably.25 The limited number of body-surface isopotential contour maps recorded to date in WPW patients have shown that much more information on the probable location of the pre-excited area can be gained by exploring the entire chest surface.26-30

In this investigation we have studied the potential distribution on the body surface in 42 cases of WPW syndrome with a view 1) to describing the surface potential maps in these patients and 2) to establishing whether the location of the pre-excited region can, at least approximately, be deduced from chest potentials, on the basis of accepted biophysical principles governing the current and potential distribution in volume-conducting media.

Locating the pre-excited area from body-surface measurements is clinically important. It may help the surgeon to choose the most appropriate access and it may enable him to limit epicardial mapping to a small area.

Methods

We studied 42 patients, 23 males and 19 females aged 10 to 63 years. Several subjects were asymptomatic and the presence of WPW syndrome was revealed only by an occasional ECG recording. Other patients had recurrent episodes of paroxysmal tachycardia or more complex arrhythmias. In nine patients another heart disease was also present, i.e., mitral insufficiency (four cases), ventricular septal defect (one case), primum atrial septal defect (one case), coronary disease without evidence of myocardial infarction (three cases). The ECGs showed the characteristic features of Type A WPW syndrome in 18 patients and those of Type B in 13 patients, according to Rosenbaum's criteria.37 In 11 cases the ECG patterns were intermediate between Type A and Type B or in some instances frankly atypical. After the patients gave informed consent for the recording of body-surface maps, between 180 and 230 electrodes were applied to the anterior and posterior chest surface by means of vertical rubber straps, as shown in previous papers.38, 39 The ECG signals from all the electrodes were recorded simultaneously using an automated 240-channel instrument which performs on-line amplification, multiplexing at 120 kHz and digital conversion of the electrocardiographic signals.39

The digital data were recorded on a high-speed IBM 2401/5 magnetic tape unit, and were processed off-line on an IBM 360/44 system.30 The maps were printed in tabular form by the computer; equipotential lines were drawn manually. The procedure employed yields one map for every 2 msec interval during the cardiac cycle. Each map illustrates the distribution of equipotential lines on the chest surface at one time instant. Wilson's Central Terminal was taken as the reference point for measuring chest potentials.

Results

Before describing the maps recorded in our patients it may be useful to recall briefly the essential features of normal QRS maps.36, 37 In normal adults studied so far, at the onset of ventricular excitation a potential maximum usually appeared in the sternum area and a minimum was located in a lower position on the left thoracic wall (fig. 1A) or in the dorsal region. These phenomena coincided in time with septal excitation, which occurs in a predominantly left-to-right direction.38 Later on the minimum migrated dorsally (fig. 1B) and then toward the right shoulder, finally appearing anteriorly in the right clavicular area, while the maximum moved downward to the left (fig. 1C). At this time excitation was spreading in an endo-epicardial direction through the walls of both ventricles. Thereafter, in about 60% of the
cases, a new minimum appeared in the midsternal region (fig. 1D). In adults, this new minimum appeared between 14 and 44 msec after the onset of ventricular excitation. This minimum is considered to be the surface manifestation of right ventricular breakthrough. In the following instants the sternal minimum and the right clavicular minimum merged to form a single broad anterior minimum (fig. 1E). In about 40% of the adults the sternal minimum did not appear as a separate entity and the midsternal area became negative as a result of the migration of the clavicular minimum. Later on, the maximum moved toward the left thoracic wall and then dorsally (fig. 1F). This can be ascribed to the main excitation wavefront spreading through the left ventricular wall, pointing first to the left, and then posteriorly. In 56% of the adults a new maximum appeared in the upper sternal area during the last third of the QRS interval (fig. 1G). The sternal maximum was attributed to excitation of the crista supraventricularis and pulmonary infundibulum. In the vast majority of cases the new maximum appeared while the dorsal maximum was still present. The time overlap was 10–30 msec. The time relationships between the two maxima provided some indirect information about the time-course of excitation in the posterior left ventricular wall and upper part of the right ventricular myocardium.

Our patients exhibited a great variety of surface potential patterns throughout the QRS interval. We attempted to group them according to the location of the potential maximum and minimum during the delta wave. We chose this criterion because we considered that during the delta wave the locations of the surface minimum and maximum must necessarily be correlated with the site of the pre-excitation wave in the heart (see discussion). According to this criterion, we divided the patients into six types (fig. 2). Although there is some degree of continuity from one type to another, our division proved useful for description and interpretation.

Type 1

Twenty-three subjects were labeled Type 1 (fig. 2A). In 17 patients the ECG showed the characteristic features of Type A WPW syndrome; in six patients the ECG exhibited in-
termediate patterns between Types A and B, with an rS ventricular complex in lead $V_1$.

At the onset of the delta wave the chest maps showed a potential maximum on the left mammary or submammary region and a minimum on the back in all 23 patients (fig. 2A). During subsequent stages, in 15 patients the minimum moved upward and reached the upper anterior chest wall at 40 to 75 msec (figs. 3B and C), or remained on the back, while the negative area expanded upward toward the upper anterior chest wall. This upward migration generally occurred when the minimum was initially located at a higher horizontal level than the maximum. In eight patients the dorsal minimum moved downward or remained confined to the lower half of the back (fig. 4). In 21 of 23 subjects a second minimum appeared in the midsternal area (figs. 3D and 4D) 40 to 90 msec after the onset of ventricular excitation. The anterior chest surface also became negative in the two remaining cases not as a result of the appearance of a new minimum but due to a migration of the dorsal minimum, which traversed the left or the right lateral wall of the thorax and finally reached the precordial area.

In all 23 patients of Type 1 the maximum remained in the left mammary or axillary region during the first two-thirds of the QRS interval (figs. 3 and 4). During the final stages of ventricular activation the potential maximum did not migrate to the back, as it does in most normal subjects, except in one case. In 17 subjects the maximum remained on the pre-apical area until it gradually disappeared; meanwhile a new maximum appeared on the upper sternal area. In the remaining five patients no separate sternal maximum appeared, but the upper sternal area nevertheless became positive as the pre-apical maximum moved to that region.

Type 2

In the eight subjects we labeled as Type 2 the ECG showed the Type B pattern. At the onset of ventricular activation the minimum was located on the inferior half of the right anterio lateral chest wall and the maximum in the sternal or left mammary area (figs. 2B and 5B). The potential maximum was generally located superiorly in relation to the minimum or at the same horizontal level. The distance between the two extremes was comparatively large in all cases except one (No. 5 in fig. 2B). The potential distribution underwent little change during the first 45 to 75 msec. The minimum then moved toward the sternal area and the maximum toward the left axillary region (figs. 5C, D). In six subjects the maximum stayed in this area and did not migrate to the back, as it did in the two remaining patients; in the majority of the cases a potential maximum appeared on the upper sternal area at the end of the QRS interval (figs. 5D, E).

Type 3

We labeled as Type 3 four patients whose ECG showed a Type B WPW syndrome; in one of these patients, however, the delta wave was clearly positive in lead $V_1$.

The first signs of ventricular excitation consisted of a potential maximum in the sternal region and a minimum or a large negative area (with very low potentials) over the upper portion of the back (fig. 6B). Subsequently, a clear-cut minimum appeared in the right clavicular area at 10, 14, 25 and 40 msec, respectively, after the onset of ventricular activation, while the minimum stayed at the same location (fig. 6C). At this stage, the distance between maximum and minimum was comparatively short (about 10 cm) (fig. 2C).

During the following phases of ventricular activation the potential maximum and minimum gradually increased in voltage and slowly moved leftward and inferiorly; the minimum reached the sternal region and the maximum moved to the left lateral chest wall (fig. 6D) or to the back. In none of the four patients did a second minimum, indicating a normal right ventricular breakthrough, appear in the sternal area. In one of them, however, the second
minimum did appear during a normal beat (fig. 6E). During the final stages of the QRS interval, no secondary maximum appeared in the upper regions of the anterior thorax as occurs in many normal subjects.

Type 4

Two patients were labeled as Type 4. Their ECGs showed intermediate features between Types A and B in one case (fig. 7) and a Type A pattern with an atypical small Q wave in lead V1 in the other (fig. 8). At the onset of ventricular excitation a minimum appeared in the upper sternal area while a maximum lay on the left mammary region (fig. 2D). In the first patient (fig. 7) the potential maximum and minimum maintained their position for about 60 msec. At 44 msec, a second rapidly growing minimum appeared in the lower sternal region (fig. 7C). These two separate minima persisted for about 20 msec; the upper minimum then merged with the sternal minimum. In the following stages the two extremes increased in strength and the maximum shifted gradually to the left lateral chest wall (fig. 7D).

In the second subject the location of the potential maximum and minimum (fig. 8B) showed little change for a hundred msec after the onset of ventricular activation. During this interval a second minimum did not appear on the sternal area, as in the first patient. Subsequently the minimum migrated to the left mammary region, while the maximum moved to the right and then reached the right subclavicular area (fig. 8C). This late behavior of the maximum was similar to that observed in patients with right bundle branch block.38

Type 5

This type was represented by a single subject, whose ECG showed intermediate features between Type A and B (fig. 9). At the onset of ventricular activation the potential minimum was located in the left axillary region (fig. 2E and fig. 9) and the potential maximum was on the inferior part of the left anterior chest wall. Thereafter the minimum shifted slightly to the left while the maximum migrated toward the midline. The potential pattern did not change significantly until 70 msec after the onset of ventricular excitation. At this time a new minimum appeared in the right clavicular region, while the axillary minimum was disappearing. Subsequently a normal sequence of potential patterns was observed.

Type 6

Type 6 was also represented by a single patient. The ECG showed unusual features in the precordial leads (fig. 10A).
The ventricular complex in V₁ had a qRS pattern without a clearly visible delta wave (fig. 10B); in V₃ and V₆ it showed a very small positive deflection, hardly detectable on reproduced tracings, followed by a negative one and then by an R wave of normal voltage. The patient suffered from frequent bouts of tachycardia and was admitted to the Cardiology Division at the Policlinico Hospital at Pavia. Here a complete electrophysiological study was carried out. A sudden clearcut change in the surface ECG and His bundle electrogram was observed during ajmaline administration (fig. 10B, C, D). The Q wave in lead V₁ disappeared and the H-R interval rose from 15 to 40 msec. These findings suggested that ventricular pre-excitation was present before but disappeared after ajmaline.

At the onset of ventricular activation the body-surface potential distribution was characterized by a minimum at the right lower sternal border and a maximum on the left scapular area (figs. 2F and 11A). This pattern was similar to that characterizing Type 1, but with reversed polarity; during the following instants the maximum moved to the left axilla (fig. 11B) and reached the sternal area 25 msec after the onset of ventricular excitation (fig. 11C). In the meantime the minimum migrated downward and waned while another minimum appeared on the lower part of the back (fig. 11C). During the following phases of ventricular activation the sequence of potential patterns was within normal limits (fig. 11D).

Indeterminate Cases

In three subjects out of 42, the location and motion of the potential maximum and minimum during the delta wave were different from those observed in Types 1 to 6. Their ECGs showed a Type B WPW. These patients were not placed in any particular group for reasons we shall deal with in the discussion.

Discussion

Our data clearly show that various surface potential patterns occur in the WPW syndrome. Interpreting these patterns is difficult because the distribution of heart potentials on the body surface depends not only on the site of the excitation waves, but also on the geometry of the chest and the conductivity of body tissues. However, an attempt has been made to correlate surface potentials with intracardiac events. In agreement with Wilson's criteria, the presence of a potential maximum in a given area on the chest surface indicates that an intracardiac excitation waveform is pointing toward that area. A potential minimum indicates that the negative aspect of a waveform is seen from that area on the chest surface where the minimum appears, or that a hole (breakthrough or infarction) is present in an advancing waveform. However, when several wavefronts are simultaneously present in the heart, the relationships between surface extremes and waveform locations become more com-
plex. Further information on the location of a wavefront can be obtained by taking into account the following elementary principles governing the distribution of currents and potentials in homogeneous conducting media:12,14

1) When a dipole is centrally located in a spherical homogeneous conducting medium, the potential maximum and the minimum have identical absolute values in relation to the dipole mid-potential and they appear on the surface of the medium at the ends of a diameter. 2) If the dipole is located eccentrically and oriented orthogonally to the radius, the distance between the surface maximum and minimum becomes shorter; the two extremes have identical absolute values and the line joining them indicates the orientation of the dipole. 3) If the dipole is located eccentrically and has a radial direction, the surface maximum and the minimum are again located at the ends of a diameter but one extreme will exhibit a higher absolute potential value and will be surrounded by higher potential gradients than the other. Similar criteria can be used for interpreting intermediate cases. The use of these simple rules is justified, within limits, during the delta wave, because we can assume that a single wavefront is present at that time. However, we are fully aware that locating intracardiac sources from surface maps in humans by means of these criteria will entail a certain amount of error since the human trunk is neither spherical nor homogeneous. On the other hand, the probability of locating the pre-excited area correctly is enhanced by the topographical constraint resulting from our knowledge that in WPW patients the anomalous pathways must be located somewhere along the A-V rings (annulus fibrosus) or, in case of Mahaim fibers, in the upper portion of the interventricular septum.

Type 1

The maps of all the patients belonging to Type 1 exhibited some common features suggesting that: 1) the site of pre-excitation was located deep in the thorax roughly halfway between the anterior and posterior chest wall and most likely lay on the postero-basal wall of the heart (fig. 12); b) the pre-excitation wave was spreading in a postero-anterior direction. These conclusions were supported by the fact that during the delta wave the potential maximum was on the precordial area and the minimum was on the posterior chest wall: the absolute potential values of both extremes, with few exceptions, were fairly close. In addition, the appearance of a second minimum on the sternal area at a later stage, due to right ventricular breakthrough (see fig. 1D), suggested that the right anterior ventricular wall was excited through the normal pathways and was therefore not affected by pre-excitation. Moreover, in 22 out of 23 patients the

Figure 5. Patient belonging to Type 2. Distribution of equipotential lines at 10 (B), 62 (C), 84 (D), 102 msec (E) after the onset of ventricular excitation. The distance between the upper and lower ends of the maps is 48 cm. The ECG shows a Type B WPW pattern.
maximum did not migrate to the back at the end of the QRS interval. This would seem to suggest that the posterobasal regions of the heart had already been excited. However, the validity of this criterion is not absolute since in three of 50 normal subjects previously studied the maximum did not move to the back at the end of ventricular activation. We are unable to give a satisfactory interpretation of the two different migration routes of the dorsal minimum (upward or downward) (figs. 3 and 4). Possibly the pre-excited area, which we believe was in the postero-diaphragmatic wall, was larger when the minimum moved downward than when it moved to the upper regions. On the other hand, the heart position could also influence the behavior of the minimum. Eight patients in this group were independently examined by another team in the Division of Cardiology at the Policlinico S. Matteo Hospital, Pavia. Electrogroms were recorded from the His bundle, the right atrium and at various points along the coronary sinus and the vena cardia magna, during sinus rhythm and reciprocating tachycardia with atrioventricular conduction over the A-V node-His pathway and with ventriculoatrial conduction over the accessory pathway. The results have been published.21 Patients Nos. 9, 10 and 12 through 17 in figure 2A correspond to Nos. 1, 2, 3, 9, 11, 15, 16, 17 in that study.21 The data obtained with intracardiac recordings showed that the pre-excited area was in the posterior wall either to the left or to the right of the crux, in agreement with our hypothesis. However, body surface maps did not enable us to clearly differentiate left posterior from right posterior A-V bridges, while intracardiac recordings appeared to yield more detailed information in this respect.

Type 2

In the patients belonging to Type 2 the location of the minimum to the right of and generally below the maximum on the right anterior chest wall (fig. 2B) suggests that the pre-excitation wave moved leftward and upward and probably spread from the lateral-inferior portion of the A-V groove through the right ventricular wall (fig. 12). In no patient did a second minimum appear on the sternal area. This supported our view that the right ventricular wall was not activated through the normal conducting pathways. The scattering of the minima on the chest surface (fig. 2B) was probably related 1) to the different sites along the right A-V groove from which ventricular excitation started and 2) to the different positions of the heart in the thorax. In one patient (fig. 5; No. 18 in ref. 23) intracardiac recordings sup-

Figure 6. Patient belonging to Type 3. Distribution of equipotential lines at 12 (B), 28 (C) and 98 (D) msec after the onset of the delta wave. In (B) the negative potentials are very low (in the order of 25 μV): the dashed line encircles the most negative area. (E) In the same patient, during normal beats a second minimum appeared 26 msec after the beginning of QRS, indicating normal right ventricular breakthrough. The ECG shows a Type B WPW pattern.
FIGURE 7. Patient belonging to Type 4. Distribution of equipotential lines at 12 (B), 52 (C) and 76 msec (D) after the onset of ventricular excitation. The ECG shows intermediate features between Types A and B.

ported our view that pre-excitation probably originated from the lateral-inferior portion of the right A-V groove.

Type 3

In the 4 patients belonging to Type 3 the position of the potential maximum and minimum on the right anterior chest wall during the second half of the delta wave (fig. 2C) suggested that an excitation wave was spreading through the anterior wall of the right ventricle from the upper part of the right A-V groove (fig. 12). The dorsal negativity at the onset of the delta wave can probably be ascribed to the pre-excitation wave spreading initially within the thickness of

FIGURE 8. Patient belonging to Type 4. Distribution of equipotential lines at 8 (B) and 124 (C) msec after the onset of ventricular excitation. In (C) the dashed arrow indicates the migration of the potential maximum during the last stages of the QRS interval. The ECG shows a Type A WPW pattern.
BODY-SURFACE MAPS IN WPW/De Ambrogi, Taccardi, Macchi

the right ventricular wall, near the basal border, in an endoepicardial direction. We suggest that after the excitation wave had reached the epicardial surface, it started spreading downward and to the left. At that moment the potential minimum moved to the right anterior chest wall. A second minimum did not appear in the sternal area in this group either, suggesting that the right ventricular wall was not excited through the normal pathways.

One patient in this group (No. 3 in fig. 2C) had an intermittent WPW syndrome. In this patient the sternal minimum appeared only during the normal beats (fig. 6E) and was located about 3 cm below the pre-excitation maximum (figs. 6B and C). This showed that the pre-excited region was located above the normal site of right ventricular breakthrough. In another patient (fig. 13 and No. 1 in fig. 2C), a 10-year-old girl suffering from severe mitral insufficiency, the location of the pre-excited area was confirmed at surgery. An epicardial map showed that excitation spread from a small area near the upper right A-V groove into the right ventricular wall (fig. 13C). After an incision divided the anterior aspect of the right atrium from the right ventricle, the excitation was found to spread from the area trabecularis toward the A-V groove (fig. 13D), as in normal hearts, and the delta wave disappeared from the ECG (fig. 13B). In the fourth patient the potential minimum remained on the back for a rather long time (40 msec). This behavior could suggest pre-excitation of the posterior wall of the heart. However, this time interval was shorter than that observed in Type 1 (44–106 msec). Furthermore, the potential patterns observed during the later stages of ventricular excitation were typical of Type 3. This interpretation was supported by intracardiac recordings (case No. 14 in ref. 23).

Type 4

In the two patients belonging to Type 4 the potential maximum and minimum were both located on the left anterior chest wall during the delta wave (fig. 2D). This potential distribution suggested that pre-excitation probably started in the anterior portion of the left A-V groove, spreading toward the apex of the left ventricle (fig. 12). A posterior location of the pre-excited area could be ruled out because of the anterior position of both extremes and the comparatively short distance between them. Similarly a right location was unlikely since neither extreme was on the right chest surface.

In agreement with this interpretation, the appearance of a second minimum on the sternal area in the first patient (fig. 7C) indicated a normal right ventricular breakthrough and showed that pre-excitation did not involve the anterior wall of the right ventricle. In the second patient the sternal minimum did not occur. The maximum moved from the left mammary region to the right anterior chest surface and remained there until the end of the QRS interval, which lasted 160 msec. This behavior of the maximum indicated delayed excitation of the right ventricle. A persisting maximum on the right anterior chest surface during the second half of the QRS interval has been observed in all the patients with right bundle branch block studied in a previous investigation.39

Type 5

In the single patient belonging to Type 5 the location of the minimum and maximum on the left anterior thorax (figs. 2E and 9) indicated that pre-excitation most probably spread through the left ventricular wall starting from the anterolateral portion of the left A-V groove and then moving downward and to the right (fig. 12). This localization was in good agreement with that indicated by intracardiac recordings obtained during cardiac catheterization.

Type 6

In the patient belonging to Type 6 the location of the minimum on the anterior chest wall and the maximum on the back (figs. 2F and 11A), indicated that the pre-excitation wave was probably spreading in an anteroposterior direction, leftward and slightly superiorly. The greater strength of the anterior minimum as compared to the posterior maximum suggested that the pre-excitation wavefront was closer to the anterior chest surface than to the posterior. Its location could be either in the right ventricular wall (with an epicardial direction) or in the right side of the interventricular septum. The right ventricular location is unlikely because of the subsequent appearance of signs of a normal right ventricular breakthrough (fig. 11D). We therefore sug-

Figure 9. Patient belonging to Type 5. Potential distribution at 14 msec after the onset of ventricular excitation. The ECG shows intermediate features between Types A and B.
FIGURE 10. Patient belonging to Type 6. (A) The ECG shows an atypical pattern of ventricular pre-excitation. Surface leads and His bundle electrogram before (B), during (C) and after (D) ajmaline administration (50 mg i.v.). Note the clearcut changes during the ajmaline infusion (C).

FIGURE 11. Same patient as in figure 10. Potential distribution at 5 (A), 16 (B), 25 (C) and 48 (D) msec after the onset of ventricular excitation. In (A) the positive potentials are very low (in the order of 10 μV). The reference tracing below each map was recorded from a point close to the V1 position.
gest that a septal pre-excitation may have started from the right side of the interventricular septum. The most likely mechanism for right septal pre-excitation would be through Mahaim fibers, stemming from the right bundle branch close to the bifurcation of the His bundle. According to Lev and to Rossi, Mahaim fibers can originate not only from the His bundle, but also from "the first or second portion of the right bundle branch to the ventricular septum." Clearly a pre-excitation wavefront of this kind would spread only for 20 to 30 msec before normal excitation took over. In our patient the early appearance of normal surface potential patterns, which occurred 20 to 25 msec after the onset of ventricular excitation, was in agreement with the above hypothesis.

Indeterminate Cases

In these three cases the maximum and minimum were located on the anterior chest wall. This suggested that pre-excitation did not involve the posterior heart walls. However, we were unable to propose satisfactory hypotheses about the location of the pre-excited area. For this reason we did not put these patients into definite categories.

Conclusions

By taking into consideration the location of the potential extremes during the delta wave we were able to recognize at least six different types of WPW. We could also suggest the most likely location of the pre-excitation wavefront in each type. In three subjects out of 42 we were unable to suggest a convincing location. Our division into groups may be partly arbitrary and certainly does not include all possible cases, but it proved useful for the description and interpretation of our data. This study shows that body surface maps provide more information on the probable location of the pre-excited area than can be obtained from 12-lead ECGs. This
is particularly true where the precordial ECG shows intermediate features between Types A and B. These subjects exhibited surface potential maps that suggested different locations of the pre-excited area in the heart: the posterior basal wall (type 1, fig. 14), the anterior wall of the left ventricle (type 4, fig. 7) and the lateral wall of the left ventricle (type 5, fig. 9). In another of our patients, electromaps showed that a right ventricular delay was associated with pre-excitation in the left ventricle (fig. 8). This delay could not be clearly detected from the conventional ECG which revealed only a type A WPW syndrome (fig. 8).

Though based on accepted biophysical criteria our interpretations are still tentative. However, in a number of patients belonging to Type 1, 2, 3, 5 and 6 the site of pre-excitation as deduced from surface maps was in good agreement with that independently established by others using intracardiac recordings, epicardial maps or surgical results. When more comparisons of this kind are available, the reliability of locating pre-excited areas from body surface maps, a noninvasive method, will progressively increase. In this respect it is worth mentioning that the knowledge of potential values on the entire chest surface is a prerequisite for solving the “inverse problem of electrocardiography,” that is, determining the location, strength and orientation of intracardiac current sources from surface data, by means of mathematical procedures. Since there is only one waveform at the beginning of pre-excitation, the localization of this front by means of mathematical methods can be expected to be easier than in other conditions.

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Two to Three Years of Failure-free Testing of a Rechargeable Pacemaker in Experimental Complete Heart Block

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SUMMARY Six hermetically sealed single cell rechargeable mercury-zinc pacemakers (B.T.) that will run continuously for over 4 years between recharging have paced dogs with complete heart block for 2 to 3 years. To maintain full cell capacity (over 1,000 mA hours) requires recharging for from 2–3 min/day to 60 to 80 hr once every 4 years, with any variation between these extremes being acceptable. Six realtime bench tests continue after over 7 years and accelerated tests have simulated a minimum of 50 years continuous pacing. Battery voltage is assessed by direct telemetry, eliminating the risk of patient intrinsic rhythm-pacemaker competition which is present with all current indirect (stimulation rate change) battery assessment techniques. The B.T. is an excellent 4–5 year primary pacemaker, fully rechargeable after several 4 year periods of battery rundown. A clinical test series has been initiated.

RECHARGEABLE MERCURY-ZINC PACEMAKER/Hughes, Brownlee, Tyers 263

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SINCE THE FIRST USE OF CARDIAC PACEMAKERS, the objective has been to develop long-lived units. Alternatives to nonrechargeable mercury-zinc cells have included nuclear, solid state, and rechargeable power sources. Nuclear pacemakers do have a potential ten year life; however they are expensive and have a radiation hazard.4,5 Although studies are preliminary, solid state power sources (for example lithium iodide) appear to be reliable and long lasting; however, their low energy density necessitates a relatively large pacemaker to produce a unit lasting five to ten years.4 Early attempts by several investigators, and some recent efforts, utilized rechargeable nickel-cadmium cells4 and at present there is a commercially available rechargeable nickel-cadmium pacemaker.6 Basic disadvantages of the nickel-cadmium cells include a low initial capacity (less than 200 mA hours), high self-discharge rate, the necessity for frequent recharging for relatively long periods of time (90 min/day), and failure after a relatively short period of time (6–8 weeks) if not recharged.6 In addition, some investigators have felt that patients would object to recharging a pacemaker, as this act “reminds them of their disability” but this has been shown to be an unfounded concern, even with rechargeable units that have the limitations of a nickel-cadmium cell.9 Anxiety regarding the prospect of the next replacement operation, which often begins soon after implantation of a primary (nonrechargeable) pacemaker, is a far greater and more oppressive patient concern.

The objective of this study was to test in animals, a low drain, hermetically sealed, long lived, rechargeable pacemaker capable of years of pacing between rechargings and capable of a total life in excess of 20 years, so as to essentially eliminate reoperation for pacemaker pulse generator replacement. Marked reduction of recharging time was also sought to decrease patient inconvenience and increase physician acceptance. Using a high capacity rechargeable silver modified mercuric oxide-zinc cell and low current drain, low voltage circuitry, sealed in a hermetic container achieved all design objectives.6,10 This report details the results of chronic rechargeable pacemaker implantation in a series of dogs with surgically-induced complete heart block.

Methods

The pacemakers used in this study were of the fixed rate (asynchronous) type, although ventricular programmed units have also been developed.11 All electronic components were hermetically sealed in a seamless, stainless steel container, 3 by 1.5 by 0.5 inches including the connector (fig. 1). The rechargeable mercury-zinc cell and the induction recharging coil were hermetically sealed within a separate internal stainless steel case to prevent exposure of the other electronic components to either cell electrolyte or hydrogen
Body-surface maps of heart potentials: tentative localization of pre-excited areas in forty-two Wolff-Parkinson-White patients.
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