Intracardiac and Intravascular Potentials Resulting from Electrical Activity of the Normal Human Heart

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Electrical records were made from the interior of the right heart and attached vessels in 14 normal subjects. Technics differed somewhat from those previously reported in that simultaneously recording string galvanometers were used exclusively, and tracings were made whenever the catheter-electrode was moved within the heart. Previously unreported large positive QRS deflections were found in records from the lower part of the right atrium, from the pulmonary artery, and in two instances from within the right ventricle itself close to the base. The possible anatomical origin of these is discussed.

Several papers are available on the nature of the electrical records obtained from the interior of the human heart. The additional verbal reports being made with increasing frequency attest to the interest in the possible answers these records may supply to many questions in fundamental electrocardiographic theory.

Intracardiac leads in man were begun in this laboratory in 1946. This report is concerned with the findings in 14 subjects with normal hearts as determined clinically. Emphasis will be placed on those aspects of our studies which differ from or supplement those previously reported.

Methods

The catheters* used were 100 cm. long and ranged in caliber from 6 F to 9 F. They consisted of a core of fine copper wire connected at the distal end to a silver electrode and at the proximal end to an ordinary electrical plug. All of the wire and the proximal, flanged end of the electrode were encased in specially impregnated radiopaque nylon or silk. The exposed tip of the electrode was 3 mm. in length. In early experiments this was coated with silver chloride but the covering was later discovered to be unnecessary with the electrical circuit used in recording. "Springiness" of the catheter could be modified by changing the caliber of the copper wire core.

Actual introduction of the catheter into the heart was by technics previously described. We have found the left median basilic vein the best point for insertion for two reasons: (1) the curve described by the left subclavian and innominate veins, the superior vena cava, and the right atrium seems to favor easy entrance of the electrode into the right ventricle and pulmonary artery; (2) an opportunity is afforded to explore the interior of the thorax along the veins mentioned.

Electrocardiograms were made with two Hindle string galvanometers recording simultaneously. In circuit with the first (upper string shadow in all illustrations) was an originally designed single stage amplifier with balanced plate circuit. This amplifier, which maintained an output impedance match of 2000 ohms with the string at all times, had a frequency response which was flat from 0 to 100 cps when used with the string under conditions simulating those of the experiments (Fig. 1).† No reso-

* Cambridge Instrument Co. Models No. 2 and No. 3.
† This desirable frequency feature of our recording instrument may explain some of the differences in our records when compared with those previously published. The latter, with few exceptions, have been made with oscillographic types of instruments the sinusoid voltage response of which is known to be 100 per cent of the equivalent square wave response only between 12.5 and 25 cps. The former is the minimum natural frequency of an electrocardiograph defined in a somewhat different way by Lewis and Gilder although it is noteworthy that all of Lewis' experiments were done with an instrument the

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* Manufactured according to specifications by the U. S. Catheter and Instrument Company, Glens Falls, New York.
nant peaks were discovered following the input of sinusoid voltages up to 300 cps. Actually only the last ten patients were studied with this circuit. With the first four (JRu, JNe, MSh, and JCo) an amplifier with less desirable characteristics was used. Its principal defect was a resonant peak in the stroke of S sometimes encountered in leads from the base of the right ventricle. This was easily recognized. Such deflections were omitted from the few calculations made on the leads of these four patients.

The second electrocardiograph was used in the ordinary way to record a variety of external leads

range 50 to 60 cps. This occasionally resulted in considerable exaggeration of the notch on the down-

natural frequency of which was "approximately 100–200 (cycles) per second under ordinary working conditions." Einthoven specified that an instrument having a rise time (deflection time) equal to 0.01 second for a 1 cm. rise (equivalent to a frequency of 25 cps) would have an adequate frequency band-width for cardiographic recording. Holzer, on the basis of studies on standard leads, has established a flat frequency response to 200 cps as a requirement of an instrument. Reid and Caldwell, by a Fourier analysis of normal standard leads, discovered some important frequency components up to 125 cps.

We have observed intracardiac deflections with a frequency in excess of 60 cps with the string galvanometer and amplifier used. Such deflections will be obscured or completely unrecorded by the oscillographic instruments referred to, and others will be exaggerated if resonant peaks exist in the frequency response curve. The ideal recorder for intracardiac potentials is not certain but from these data it would seem undesirable to have its natural period longer than 0.01 second.

as desired (lower string shadow in all illustrations). The extremity, precordial (Leads Vi to V6), external thoracic (Leads V6, Vp, Vn, Vcr, and sometimes others*) and intracardiac potentials were obtained by connecting one end of the string to the central terminal of Wilson, Macleod, and Barker. The circuit was arranged to augment extremity potentials. The galvanometric connections were so made that in the finished tracing a downward deflection represented relative negativity of the exploring electrode.

The string sensitivity used was quite variable, especially for the endocardial leads, and is indicated on each illustration.

At first the intracardiac potentials were recorded from random locations in the heart. As teamwork improved we attempted to place the electrode initially in the pulmonary artery or one of its branches, usually the right. Its

* Chest position 8 was in the left midscapular line; B, in the midline posteriorly. Both were at the same horizontal level as position 5.
frontal projection was determined with the central ray of the fluoroscope and the point was marked on the chest. It was customary to record this intra-arterial potential first with Lead I, then with a precordial lead when the precordial lead, and this whole procedure repeated. By this means it was usually possible to explore quite thoroughly the right pulmonary artery; the pulmonary stem; the conus, outflow tract, apex, inflow tract and tricuspid region of the right ventricle; all levels of the right atrium; the superior vena cava; and the left innominate, subclavian and axillary veins. In addition, it was possible to obtain records during transit between the pulmonary artery and right ventricle, the right ventricle and right atrium, and the right atrium and superior vena cava. Although

**Fig. 2.**—Teleroentgenograms of four normal subjects. The identifying initials are in the lower left hand corner of each illustration. The single circles with a figure within indicate the approximate location of the intracardiac electrode at the time the electrocardiographic record was made. A double circle with a figure enclosed indicates that the electrode was in the pulmonary artery or its right branch. In patient APy, point 4 was directly under precordial lead V3. In each instance the location of the electrode when recording precordial leads V5 and V6 is shown for comparison.
it was usually easy to identify such transitions electrically, there were occasions when pressure tracings would have provided additional reassuring information.

Tracings were particularly difficult to make when the electrode was in a vein. The string shadow was often distorted by fine oscillations of such type as probably came from electrical activity of the muscle in the venous walls.

**TABLE 1.—Pertinent Data on 14 Patients With Clinically Normal Heart**

<table>
<thead>
<tr>
<th>Patients</th>
<th>Age</th>
<th>Sex</th>
<th>Color*</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>JRo</td>
<td>70</td>
<td>M</td>
<td>W</td>
<td>Subdeltoid bursitis</td>
</tr>
<tr>
<td>JNe</td>
<td>55</td>
<td>M</td>
<td>N</td>
<td>Pituitary neoplasm</td>
</tr>
<tr>
<td>MSh</td>
<td>45</td>
<td>M</td>
<td>N</td>
<td>Lobar pneumonia, type XII, convalescent</td>
</tr>
<tr>
<td>JCo</td>
<td>49</td>
<td>M</td>
<td>W</td>
<td>Lobar pneumonia, type VI, convalescent</td>
</tr>
<tr>
<td>JJa</td>
<td>56</td>
<td>M</td>
<td>W</td>
<td>Psychoneurosis; latent syphilis</td>
</tr>
<tr>
<td>JMa</td>
<td>43</td>
<td>M</td>
<td>W</td>
<td>Multiple sclerosis</td>
</tr>
<tr>
<td>APy</td>
<td>60</td>
<td>F</td>
<td>W</td>
<td>Bronchopneumonia, convalescent</td>
</tr>
<tr>
<td>WLCa</td>
<td>53</td>
<td>M</td>
<td>N</td>
<td>Grippe, convalescent</td>
</tr>
<tr>
<td>PAq</td>
<td>49</td>
<td>M</td>
<td>W</td>
<td>Grippe, convalescent; latent syphilis</td>
</tr>
<tr>
<td>SAT</td>
<td>58</td>
<td>M</td>
<td>W</td>
<td>Pneumococcus pneumonia and meningitis, convalescent</td>
</tr>
<tr>
<td>QWi</td>
<td>44</td>
<td>M</td>
<td>N</td>
<td>Post-traumatic epilepsy</td>
</tr>
<tr>
<td>EFR</td>
<td>49</td>
<td>M</td>
<td>N</td>
<td>Cerebrovascular syphilis</td>
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<tr>
<td>JPHe</td>
<td>52</td>
<td>M</td>
<td>W</td>
<td>Intermittent anomalous A-V excitation; lobar pneumonia, convalescent</td>
</tr>
<tr>
<td>JRo</td>
<td>61</td>
<td>M</td>
<td>W</td>
<td>Adenoecarcinoma of prostate.</td>
</tr>
</tbody>
</table>

* White or Negro.

We have frequently encountered the positive displacement of the RS-T segment and the accompanying distortion of QRS which results from pressure of the intracardiac electrode on the endocardium.\(^{20}\) Comparable modifications have also been seen in the atrial deflections. Usually such distortion of the tracing can be recognized; rarely it is difficult to detect. Unfortunately, distorted records of this kind have already appeared in the literature as representative of normal endocardial potentials.

After the completion of an experiment, lead markers were placed on the skin at the various locations of the intracardiac electrode. A tele-

roentgenogram was then made, in the beginning with the patient first recumbent and again standing, but later only standing. The reason for this was that little difference was found in the location of the markers on the x-ray films made in the two positions. Further, the films obtained with the patient standing were far better, and displacement of the markers was less likely to occur (Fig. 2).

The clinical data pertinent to the 14 patients included in this report are incorporated in table 1.

All measurements were made with the Elliott comparator manufactured by the Cambridge Instrument Company. The error of measurement with this device, when interindividual variations are excluded, is less than ±0.003 second.

**Records from Within The Pulmonary Artery**

In half of the 14 normal subjects studied, the exploring electrode could be placed in the pulmonary artery or its right branch. The records obtained during electrical activity of the atria and of the ventricles were quite variable as might be expected from the considerable length of the tributaries, and the variable relations particularly of the right branch to the different chambers of the heart. These relations are summarized in the following paragraph from Morris’ Anatomy.\(^{21}\) The italics have been put in by us to emphasize the relation of the pulmonary artery and its right branch to the left atrium, and of the former to the left auricle as well.

**Anatomical Relations of the Pulmonary Artery and the Right Pulmonary Artery**

The pulmonary artery has a course of approximately 5 cm. within the pericardium before dividing into the right and left branches. In front it is covered by the remains of the thymus gland and the pericardium. Behind it lies successively upon the ascending aorta and the left auricle. To the right are the ascending aorta, the right auricle, the right coronary artery, and the cardiac nerves. To the left are the pericardium, the left pleura and lung, the left auricle, the left coronary artery, and the cardiac nerves.

The right pulmonary artery passes almost horizontally under the arch of the aorta to the root of
the right lung where it divides into three branches, one for each lobe. In its course to the lung it has in front of it the ascending aorta, the superior vena cava, the phrenic nerve, the anterior pulmonary plexus, and the reflection of the pleura. Behind are the right bronchus and the termination of the azygos vein. Above is the arch of the aorta, and below are the left atrium and the upper right pulmonary vein.

The nature of the atrial deflections observed in the right pulmonary branch depended on whether the exploring electrode was low or high in this vessel or one of its main tributaries.

In the pulmonary artery the initial atrial deflections varied greatly from predominantly negative to predominantly positive. In the one latter instance (patient JPHe, Fig. 3) the end of an easily recognized extrinsic deflection was quite late, almost at the end of the P wave in Lead I, suggesting that the electrode was being affected by the left atrium or auricle (appendage) which are in close proximity to the pulmonary artery.

The nature of the ventricular deflections varied also. In the right branch these were usually similar to comparable deflections in the lead from the right arm, with minor variations. The combinations observed were rSr', QR (with slurred, broad R); QS (with one or several notches), or rS. The T wave in all of these was inverted.

In the pulmonary artery the combinations observed were rSr', rS (with S notched), qRs, rs'r's', and rsR'. The complex, rSr', when present, simulated the initial ventricular deflections in Lead aVr (and presumably in Lead V3); rS simulated what was found in leads from the greater part of the right ventricular cavity. The qRs was not unlike the ventricular deflections recorded in the same patient (SAt, Fig. 9) from the lower part of the right atrium, and rsR' in the one patient in which it was encountered (patient APy, Fig. 4) was similar to potentials recorded from the conus and the tricuspid areas of the right ventricle, and from the lowest part of the right atrium (see below). In the one instance (patient PAq, Fig. 5) of a quadriphasic record (rsr's') unusually high positive QRS deflections were encountered in the tricuspid region of the right ventricle, and in the lower part of the right atrium. Further reference will be made to these important positive deflections later. The T wave in all the records from the pulmonary artery was inverted.

These data may be summarized as follows: the nature of the galvanometric deflections encountered as a result of action currents in atrial and ventricular muscle are more variable when recorded from the main stem than from the right branch of the pulmonary artery. In the former the atrial deflections are apparently determined by the relative closeness of the
FIG. 4.—Patient APy. A variety of internal and external leads recorded simultaneously with Lead I (I), or with a lead (Vp) taken with a precordial electrode directly over the intracardiovascular electrode. The records in the first two rows have been arranged approximately in the order of their occurrence as a line is followed on or in the thorax from the right arm to the left side of the precordium. Each lead is indicated by the symbol V followed by a subscript of letters and a number. The letters indicate the structure from which the lead was made. The number corresponds to the number of the intracardiac point as seen in figure 2. For example: V SVC 5 means potential of the superior vena cava at point 5; VRPA 2, potential of the right pulmonary artery at point 2; VRV 1, potential of the right ventricle at point 1; VA, potential of the right atrium at an unknown location. The number written at the right end of each trace denotes the sensitivity of the string at which it was recorded. If left blank it means that the sensitivity of the string was normal (1 mv. = 1 cm.). Time lines occur every 0.2 sec.

The remaining numbers on the records indicate the time of the adjacent QRS deflection with respect to the beginning of QRS in Lead I. Those waves which are simultaneous with the large positive deflection encountered in leads VRV 1 and VRV 2 at the base of the right ventricle are underlined. In contrast to records of other patients (Figs. 5 and 6) the wave under consideration in lead V SVC 4 is a negative notch on the ascending limb of S, and cannot be identified at all in lead aVR. The intraventricular leads differ from others, too, in the occurrence of a positive T wave in leads VRV 1 and VRV 2.

The lowest row shows a continuous record made simultaneously with Lead I as the electrode was withdrawn rather rapidly from the apex of the right ventricle through the tricuspid orifice into the right atrium. The time of the S wave at the apex of the right ventricle (first complex) is almost identical with the time of the positive deflection seen as soon as the electrode has entered the right atrium (third complex). Location of the electrode in the latter chamber at the time is deduced from the initial occurrence of a diphasic P wave in the record.

upper part of the right atrium or auricle on the one hand and the left atrium or auricle on the other. The initial ventricular deflections may simulate those seen in the lead from the right branch and hence the lead from the right arm. However, in three of seven instances late
FIG. 5.—Patient PAq. Records have been arranged much as in Fig. 4, and symbols and figures have the same significance.

In this record the large positive deflection in the lower right atrium (VRA7) and in the tricuspid region of the right ventricle (VRv6) consists of two peaks, 0.036 and 0.051 sec. after the beginning of QRS in Lead I. The analog in vascular and surface leads (aVR, V5CVS, VRPA1, VRPA2, VPA3, VRA8, and V6) has a time of 0.046 or approximately the mean of the previous two figures.

To be noted is that the deflection in question in a precordial lead (VP) over point 1 (VRPA1) is positive and over point 2 (VRPA2) it is a positive notch on the S wave, but in leads farther to the left (VP of VPA3, not shown, and VRA7) it is negative. Reference to Fig. 2 will demonstrate that points 1 and 2 are quite far to the right at the level of the third rib, while the remaining points may be regarded as closer to the region of the pulmonic conus.

The small deflection in the middle of the P-R segment of lead VRA8 is probably a second intracardiac deflection with origin in a remote part of the atrium (see Fig. 10).

The lowest row shows a continuous record made as the electrode was withdrawn from the pulmonic artery (VPA3) into the conus (VRv4, last complex). Standardizations (1 mv. = 0.4 cm.) distort the third complex. The change of the late positive to a completely negative deflection is easily seen. The simultaneous lead is Lead I.

PHENOMENA OBSERVED WITH CONTINUOUS RECORDING WHILE MOVING THE ELECTRODE FROM THE PULMONARY ARTERY TO THE CONUS OF THE RIGHT VENTRICLE

In six instances a record of the intracardiovascular potential was made simultaneously with Lead I as the electrode was slowly withdrawn from the pulmonary artery to the pulmonic conus of the right ventricle. The change observed was sometimes abrupt, sometimes gradual. In the latter instance it was not possible to say exactly when the electrode had entered the right ventricle.

The changes seen were those involving the P wave, the QRS-T, premature systoles, the development of an ectopic rhythm, and the occurrence of S-T elevation as the electrode made pressure on the ventricular endocardium.

If the P wave was originally inverted in the
pulmonary artery, it became shallower, isoelectric or slightly diphasic in the right ventricle. If it was diphasic to begin with, the wave would become isoelectric.

If the QRS was originally the rS or rSr' type, the r would become more prominent, the S deeper, and the r' would become a negative peak on the ascending limb of S. T also became deeper. The voltage of all deflections was quite variable (Table 2) but in this transition (pulmonary artery to conus) it increased approximately twofold.

In two instances the change in QRS was quite striking in that a large positive deflection appeared in the ventricle with a positive T wave. One of these patients displayed anomalous atrioventricular excitation\textsuperscript{26} at times; the other was a woman, 69 years old, but with no clinical or electrocardiographic evidence of heart disease (Fig. 4). The composition of QRS in these differed from the other. In the first it was qRS and in the second it was rsR'.

In four of the six instances a characteristic change in P and QRS occurred at the same time as the electrode entered the right ventricle; in one instance all deflections were obscured by the development of an irregular ventricular rhythm; and in another the exact time of entry of the electrode into the ventricle could not be determined electrically.

Atrial premature systoles were observed once during movement of the electrode. It is likely that they were produced by pressure of proximal parts of the catheter in the right atrium. An irregular ectopic rhythm and premature systoles of ventricular origin were each seen once. In the former instance when the rhythm stopped spontaneously the intraventricular action potential was found to be distorted by an elevated S-T segment ("injury potential,"\textsuperscript{26,27} "demarcation potential."\textsuperscript{28})

**Records from within the Right Ventricle**

From the first few experiments in which the electrode was placed in random fashion in the normal right ventricular cavity, it was our impression, as reported by others\textsuperscript{3-4, 7, 10}, that the right intraventricular deflections during

<table>
<thead>
<tr>
<th>Lead</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>aVR</th>
<th>aVL</th>
<th>aVF</th>
<th>V1</th>
<th>V2</th>
<th>V3</th>
<th>V4</th>
<th>V5</th>
<th>V6</th>
<th>VR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Min.</td>
<td>2.0</td>
<td>4.0</td>
<td>2.0</td>
<td>1.9</td>
<td>0.0</td>
<td>1.9</td>
<td>4.0</td>
<td>6.0</td>
<td>10.0</td>
<td>14.0</td>
<td>9.0</td>
<td>6.0</td>
<td>4.0</td>
</tr>
<tr>
<td>Max.</td>
<td>14.3</td>
<td>36.0</td>
<td>13.0</td>
<td>18.0</td>
<td>10.0</td>
<td>17.0</td>
<td>38.0</td>
<td>50.0</td>
<td>42.0</td>
<td>44.0</td>
<td>28.0</td>
<td>24.0</td>
<td>54.0</td>
</tr>
<tr>
<td>Mean</td>
<td>7.5</td>
<td>11.7</td>
<td>6.7</td>
<td>9.0</td>
<td>4.4</td>
<td>7.7</td>
<td>14.2</td>
<td>22.0</td>
<td>21.0</td>
<td>25.5</td>
<td>18.3</td>
<td>11.2</td>
<td>18.0</td>
</tr>
</tbody>
</table>

\textbf{Table 2.—Size of the RS (or QR) Deflection in Various Leads of 14 Patients without Heart Disease as an Index of the Relative Size of the Voltages Developed within the Right Heart and at the Surface of the Body (in tenths of a millivolt).}
The size of this R wave varied in different parts of the chamber. In some instances it was larger at the base than at the apex; in others the reverse was true. Between the region of the conus and the region of the tricuspid valve there were often considerable differences in record was not different from what was seen in the other patients.

The initial positive deflection has been ascribed to early depolarization of the left side of the interventricular septum. In general the area concerned is believed to be near the

**FIG. 6**—Patient JRo. A variety of leads arranged as in Figures 4 and 5 in the lower two rows. The upper row shows the standard leads and the augmented extremity potentials (I, II, III, aVR, aVL, aVF). The simultaneous lead is Lead I or a point on the precordium (Vp) over the internal electrode. In this figure only the deflections simultaneous with the late positive deflection in the atrium (V_{RAS} and V_{RAS}) are labelled with the figure 0.060, the time in seconds it occurred after the beginning of QRS in Lead I. In this instance the deflection in question was negative in all leads below the tricuspid orifice and on the front of the chest; it was positive in leads from the atrium, the superior vena cava, the pulmonary artery (not shown) and its right branch, and the right arm. Times of other QRS deflections may be found in Table III. The initial Qa wave in the auricular complexes of leads V_{RAS} and V_{RAS} preceded the onset of the P wave in Lead I by 0.002 sec.

this deflection. When present, it was usually slurred or notched.

The two exceptions were patients PAq and JPHe. The former showed a depression preceding a large, late, notched R when the electrode was in the tricuspid orifice (Fig. 5, V_{RV6}); the latter, already referred to, displayed a Q wave in leads from the pulmonary conus and from the apex but in the tricuspid region the base of this structure; the findings we have presented neither support nor deny this. The variables involved, such as the distance of the electrode from the endocardium, the curvature of the septum and consequent tangential effects of wave fronts, and others still unknown, make the precise origin, on the basis of these experiments, uncertain.

The time of the R peak was early (0.004 to
0.024 second before the beginning of QRS in Lead I. It was usually simultaneous with an early R in Lead aV_R; an R or a notch on its ascending limb in Lead V_1 or Lead V_2; a Q wave in Lead I when present; a Q wave in leads from the left side of the precordium; an initial positive deflection recorded in the right atrium and in the pulmonary artery or its right branch played one or more distinct notches on its descending or ascending limbs. The latter has already been mentioned as being simultaneous with an r’ in a lead from the pulmonary artery (Fig. 6, V_{RPA} and V_{RV}). A distinct notch or slur on the descending limb was often found. This was usually simultaneous with the peak of the R wave in leads from the left side of the precordium (Fig. 4, V_{RV} and V_4), although not invariably so. For this reason this notch along with the descending portion of the S wave preceding it, was believed to be created by events occurring in the free wall of the left ventricle.

The peak of the S wave varied in time in two principal ways. Sometimes it was simultaneous with the peak of the R wave in left-sided pre-

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<table>
<thead>
<tr>
<th>Lead</th>
<th>Q</th>
<th>Notch</th>
<th>R</th>
<th>Notch</th>
<th>S</th>
<th>Notch</th>
<th>R'</th>
<th>S'</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>0.010</td>
<td></td>
<td>0.035</td>
<td></td>
<td>0.060</td>
<td></td>
<td>0.078</td>
<td></td>
</tr>
<tr>
<td>II</td>
<td>0.010</td>
<td></td>
<td>0.035</td>
<td></td>
<td>0.058</td>
<td></td>
<td>0.035</td>
<td>0.050</td>
</tr>
<tr>
<td>III</td>
<td>0.010</td>
<td></td>
<td>0.003</td>
<td></td>
<td>0.025</td>
<td></td>
<td>0.060</td>
<td></td>
</tr>
</tbody>
</table>

(Table 3). The tacit assumption is made that all of these deflections had the same origin, namely early excitation of the interventricular septum from left to right.*

The S wave varied in depth and usually dis-

* Simultaneity of deflections may be fortuitous. But if, in addition, they have a direction in multiple leads ascribable to a single spatial electromotive force, the likelihood of identical origin is very great.
cordial leads (Table 3 and Fig. 6). In such instances there was usually a notch or slur on the ascending limb which had a time similar to the nadir of the S wave in the left precordial leads and the late R or R' in lead aVR. In others the nadir of S was simultaneous with the S wave in leads from the left side of the precordium (Fig. 4, V_RV and V_4). These findings support the contention that the S wave in the right ventricular cavity is made up of several components, and its deepest portion has at least two different origins in different subjects.

A late large R followed by an S wave was encountered in the upper ventricle of two patients (Paq and JPHe). In both of these it was preceded by a Q (Fig. 5, V_RV). In a third patient (APy) there was a large R' preceded by an rs (Fig. 4). In this patient the R' varied in size from moment to moment presumably caused by movement of the electrode.

With two exceptions the T wave was negative in the cavity. The two exceptions again were patients APy and JPHe. The former showed a large R' and positive T wave at the basal region, although an inverted T wave was encountered at the apex (Fig. 4). The latter, in whom exploration was quite detailed, showed a positive T wave in all parts of the right ventricle except in the region of the tricuspid valve. This patient showed anomalous atrioventricular excitation intermittently and perhaps cannot be regarded as normal in the electrocardiographic sense.

Since all of the patients with a negative T wave in the right ventricle displayed a positive T wave in the lead from a precordial point over the intraventricular electrode the conclusion would seem reasonable, though not absolute, that a gradient usually exists across the free wall of the right ventricle of such orientation as to suggest that repolarization takes longer on the endocardial than on the epicardial surface.

**Premature Systoles.** As expected, premature systoles, both of atrial and of ventricular origin, were more frequent when recordings were made while the electrode was being moved in the heart. Undoubtedly they were caused by mechanical stimulation of the walls by the catheter but it could not be assumed that the tip of the instrument was always the stimulating agent. It is true that ventricular premature systoles were more frequent when the electrode was in the ventricle, and atrial premature systoles more frequent when it was in the atrium. However, the reverse occurred, and of the two possibilities it was more usual to encounter atrial premature systoles while the electrode was still in the ventricle. Only once did the opposite set of events occur (patient JNe).

The form of QRS of the ventricular extrasystoles was predominantly negative but this varied; the accession deflections of the premature atrial systoles were always negative.

There did not seem to be any particular region in which extrasystoles were more frequent since they were seen with the electrode anywhere. There was the impression that they might be a little easier to produce in the conus just below the pulmonic valve but this might have been more apparent than real. With the electrode in the pulmonary artery the catheter makes a rather sharp curve backward, to the left, and then to the right. As it is withdrawn into the conus the uncoiling process probably results in considerable pressure either on the free wall of the conus or on the adjacent interventricular septum. The impression may be based on this variable.

**Ectopic Rhythms.** These were seen in 3 patients as the electrode was moved in the outflow tract. In two instances a rhythm indistinguishable from anomalous atrioventricular excitation (Wolff-Parkinson-White syndrome) developed, once while the electrode was still in the pulmonary artery, and once while being withdrawn through the right ventricle. These phenomena, which reawaken interest in the "double rhythm" theory of the syndrome, have been reported elsewhere.

In one instance runs of ventricular premature systoles with coupling appeared after moving the electrode within the right ventricle.

**Demarcation or Injury Potentials.** Elevation of the trace to a new level immediately after the inscription of the deflections resulting from depolarization was observed frequently in the ventricles. This displacement was always positive in intracardiac leads, a fact ascribed by
current theory to a subnormal degree of depolarization at the site of pressure by the electrode. However, the pressure could be such as to change the direction only of the normally inverted T wave and to prolong slightly the duration of electrical systole. In such instances it is probable that pressure and subsequent incomplete depolarization was sufficient to balance or slightly overbalance the normal gradient in repolarization presumed to exist from endocardial to epicardial surfaces. Further, the more marked S-T elevations were accompanied by alterations in QRS occasionally of such extreme nature as to make the curve monophasic (Fig. 7) as described by others.

The P wave in the right ventricle was usually a low positive deflection. At the base, both in the pulmonic and tricuspid regions, a diphasic nature could rarely be made out (one instance in each region). As expected the atrial regression deflections were poorly recorded from within the right ventricle.

**Records from within the Right Atrium**

Records were made from the right atrium in 12 of the 14 subjects. With regard to the gross features of these records there seems to be fairly good agreement in available reports.\(^5\,\,^7\,\,^9\,\,^{10}\) The initial rapid or accession deflections are most often diphasic in character. There is a tendency for the initial positive wave to be larger as the electrode is placed farther from the sinoatrial node. However, with regard to the details of these initial rapid deflections and the presence or absence of others, there is some difference of opinion. Further, the true nature of the regression deflections in the human atrium seems to have escaped most students of the subject.

**Atrial Accession Deflections.** At the outset it must be kept in mind that the electrode in the atrium, as in the ventricle, may be at a variable distance from the endocardium with a variable amount of blood intervening.\(^*\) Further it un-

\(^*\) In general the interval between the maximum and minimum peaks of the diphasic accession deflections is greater than the actual interval which separates the source and sink of the doublet responsible for them. This difference is greater the longer the distance between the electrode and the

doubtedly is shifted from moment to moment by systole of the chamber and by movement of the blood. Both of these possible variables must be taken into account when making interpretations.

**Fig. 7.—Patient JCo. Leads from adjacent points in the right ventricle showing the “monophasic” distortion which occurs (VRV) when the electrode makes pressure on the endocardium. The similarity of the earliest parts of QRS in both records is to be noted. The simultaneous lead is I, recorded at somewhat less than normal sensitivity with VRV2. Time lines, 0.2 sec.**

The nomenclature available for atrial deflections is multiple and unsatisfactory. Lewis\(^28\) designated the initial deflections as extrinsic muscle.\(^29\) It has been demonstrated analytically and experimentally by McLeod\(^20\) that when the distance which separates the elements of the dipole is equal to or greater than the length of the muscle being studied it is accurately measured by the interval between the maximum and minimum peaks of the electrogram. In the only measurement of a similar kind made in an abnormal atrium of man\(^21\) on the assumption that the rate of atrial conduction was 2000 mm. per sec. the length of the train of doublets of accession was calculated as 10 mm. and of the doublets of regression as 150 mm. If the usual figure of 1000 mm. per sec. is accepted as the rate of atrial conduction (and our later experiments to be reported favor this) these figures become 5 mm. and 75 mm. respectively. Since the distance of the electrode from the endocardium was unknown it is probable that either figure for the duration of increasing activity is too high, and that errors may be expected in the determination of this interval from the distance between the two ends of the intrinsic deflection thus recorded. On the other hand the duration of the regression process, when measurable, is probably independent of the distance of the electrode from the endocardium because of its length.
and intrinsic depending on whether they were caused by electrical events distant from or immediately under the electrode in direct leads from the dog's atrium. Although the term extrinsic was used to describe a positive deflection preceding the intrinsic one, it is possible to think of extrinsic effects on an exploring electrode of an electrically negative nature occurring after the intrinsic deflection. Hering designated the deflection resulting from the atrial recovery process as the Ta wave. Hurst Brown on the basis of studies on esophageal leads recognized Sn, a, e, i, and o waves resulting from the atrial excitatory process. The Sn and a waves were negative deflections. The first, ascribed to sinoatrial activation, occurred quite consistently almost 0.018 second before the onset of the P wave in Lead II; the second, really a continuation of Sn but occurring after the onset of P, was said to represent "the summation of activation effects of parts of the auricle distant from the electrode." The e was the positive deflection comparable to the extrinsic deflection, and i was the rapid change from positive to negative called by Lewis the intrinsic deflection. The deflection o was the gradual return of the trace toward the baseline after the end of the intrinsic deflection. No mention was made by Hurst Brown as to the possible partial origin of this deflection from events during recovery. Battro and Bidoggia designated the terminal slow portion of this o deflection u and believed it to be caused by repolarization effects. In a masterful analysis of the nature of the T wave Macleod was able to show in direct leads from a strip of frog's auricle two peaks, one positive and one negative, during the depolarization, or accession process as it was called, which he labelled a1 and a2. During the repolarization or regression process there were also two gradual peaks with reversed polarity which he labelled r1 and r2. Hect has suggested that the atrial deflections all be indicated by P with a subscript to indicate the similarity of the wave to ventricular deflections. Hence there would be a Pn, Pm, Pa, and Pn. Levine and his associates have expanded Hering's terminology to the atrial accession deflections speaking of a Q, Rm, and Sm.

There are advantages and disadvantages of each type of terminology. In the present study it has been possible to identify the intrinsic deflections from each atrium in the record made from the right atrium only, which complicates the matter further. The nomenclatures of Lewis, Hurst Brown, and Macleod are all based more or less on the supposed physiologic origin of the deflections, whereas Hering's and Hecht's are arbitrary as were Einthoven's original designations for the ventricular deflections. The latter approach is perhaps the most desirable at a time when the physiologic origin of all the deflections is still not entirely certain. Perhaps the method of Hering expanded by Levine and his group has a little advantage over Hecht's method, although the choice is largely one of personal preference.

An initial positive deflection preceding the P wave, such as was obtained by Hecht and called a "pre-auricular deflection" because it occurred 0.05 second before the P wave in a simultaneously recorded Lead I or precordial lead, was not observed in the present series. However, both of Hecht's patients had disease of the heart.

An initial negative deflection (Q, not QS) was encountered in 3 subjects. In two it was found in leads from the upper part of the atrium; in one it was found in leads from upper and lower levels but was inconstant. It occurred between 0.002 and 0.012 sec. before the simultaneously recorded P wave in Lead I. The concept, based on measurements in the dog, is that this early negative deflection is caused by activation of the sinoatrial node itself. It does not seem likely that this structure is capable of giving rise to an electromotive force which can be recorded by the string galvanometer. A. Q, occurred in at least 1 subject almost simultaneously with the beginning of P in Lead I. Further, that some part of it occurs before the P wave in a bipolar lead does not necessarily favor its sinoatrial origin because the beginning of the latter may be quite late for obvious reasons. Assuming it is analogous to the Sn of Hurst Brown, the constancy of its timing (0.018 sec. before P in Lead II) in esophageal leads is a more cogent argument in favor of its sinoatrial origin.
From the few observations we have made it would appear that the electrode is near the sinoatrial node when this deflection appears but the mechanism of its creation may be simply a matter of orientation of the electrode.

![Diagram](image)

**Fig. 8.**—A simplified, schematic representation of a frontal section through the atria just anterior to the venae cavae to illustrate how the intratra
torial P wave may begin with a negative deflection (solid angle—\(\omega\)), and later be characterized by a positive deflection as a dipole approaches the electrode through the septum (\(t_2''\)). The effects of dipoles at \(t_1\) and \(t_1''\), being relatively distant \((r_2 \text{ and } r_2'')\) are extrinsic and negative. The diagram serves to illustrate the cause of such bizarre P waves as seen in complexes 10 and 11 of figure 9. Not illustrated are the effects of the regression process. In making the diagram, a rate of conduction of 1000 mm per sec. was assumed.

The times \(t_1, t_1'' = 0.002\) sec. and \(t_2, t_2'' = 0.006\) sec. from the beginning of the atrial excitation. Potentials of the electrode are determined by the equations \(V = \mu \cos \theta / r^2\) and \(V = \phi \omega\).

It is visualized that at first the electrode is dominantly affected by the active side of an umbrella-like wave of excitation preceding in all directions from it (Fig. 8, solid angle subtended at the electrode at times \(t_1\) and \(t_1''\)). Later the electrode is affected by an oppositely oriented wave front on another side (septal) of the atrium (\(t_2''\)). The figure is exaggerated but it is conceivable that something of a similar nature may account for a Qm which precedes the beginning of the P wave in the standard leads.

The figure also helps to explain later intrinsic-like deflections probably arising in the left atrium occasionally seen when the electrode is near the mouth of the superior vena cava and rarely in other locations (Fig. 9, complexes 10 and 11). In one instance during the transition from the right atrium to the superior vena cava a slow slurred initial depression occurred 0.022 second before P1. This was considerably earlier than in any other complex of the strip and may have been due to sinoatrial activation.

Although in general the e wave (Rm) was taller in leads from lower than in leads from higher levels this was not invariably the case. There are several reasons why this might be so, possibly the most important being the distance of the electrode from the endocardium in each instance and the relative orientation of the electrode with respect to extrinsic effects which as noted may be negative as well as positive.
The o wave (ascending limb of $S_a$ or $Q_S$) is most prominent in those leads which show a small or absent extrinsic deflection. It is broad and slurred and is probably in large part caused by excitation of atrial muscle in a direction away from the electrode. This statement is based on the fact that when the electrode is placed near the left auricle (appendage) in the pulmonary artery, the comparable deflection is sharp and rapid, not slurred (Fig. 3, $V_{PA5}$).

Further, its termination is simultaneous with the termination of the o in the lead from the region of the sinoatrial node in the right atrium (Fig. 3, $V_{RA5}$). Although these data support its designation as an accession event, nevertheless, there are certainly included in it the earliest negative portions of the regression process. This point can be seen in Figure 9 where the dipolar nature of recovery can be made out in part by progressive elevation of the P-R segment as the intrinsic deflection becomes deeper (complexes 5 to 8).

Atrial Regression Deflections. In the normal subject the recognition of either $r_1$ or $r_2$ is difficult. The former is obscured by o, the latter by the advent of ventricular excitation. Nevertheless, in records made from near the sinoatrial node, the P-R segment is usually slightly above the baseline (Fig. 9), and in records made distant from this site the reverse displacement of the segment occurs, an observation made by Battro and Bidoggia.3 This fits in well with what Macleod22 has predicted by regarding the regression process as essentially dipolar in nature. Recognition of the initial part of this diphasic deflection is occasionally possible in human records (Fig. 9), especially if there is some degree of atroventricular block. Further, there is a fifth deflection in these abnormal records which is late, positive, and gradual and is comparable in every way to the U wave encountered at times in records of the ventricular action potential.

Duration and Rate of Conduction of Action Currents in the Atrium. Measurements have been made on the duration of the excited state in the atria of animals22 and of man.31 In a group of normal human subjects such measurements cannot be made easily because the terminal portions of atrial regression are obscured by the deflections resulting from ventricular excitation. Considerable error may be made in the measurements, especially on the duration of the accession process, if the electrode is at a distance from the endocardium (see page 21). Nevertheless an attempt was made to approximate the duration of accession by measuring the time between the beginning and end of the intrinsic deflection in representative records made from different levels of the atrium. The maximum, minimum and mean values obtained were 0.003, 0.025, and 0.010 sec. respectively. It is probable that the minimum value is the closest to the true value.

On several occasions, both in normal subjects and in patients, a calculation of the rate of conduction in the atrium was attempted. This was done by measuring the distance on the chest wall between the frontal fluoroscopic projections of the electrode at two different points in the right atrium. The difference in time between the beginning of the intrinsic deflection in leads from these two points was then taken as the time consumed as excitation traveled over the measured distance. In retrospect such measurements are extremely crude, particularly the measurements of the distance, and involve certain assumptions which in the light of our later experience with a double electrode catheter are not justified. Until the newer techniques have been explored further it is probably best to assume that the rate of conduction in the human atrium is similar to that in the dog, namely 1000 mm. per second.

The mean difference between the peaks of $R_a$ in upper atrial records and in lower atrial records was 0.032 sec.; between the nadirs of $S_a$ waves in the same records it was 0.033 sec. This crude measurement means that excitation reaches the lower levels of the right atrium about 0.033 sec. later on the average than it reaches the upper levels.

Demarcation Potentials in the Atrium. Presumably from pressure of the electrode on the atrial muscle a positive displacement of the baseline before the completion of the intrinsic deflection occurred on one occasion (Fig. 10). This is suspected of being caused by an injury potential or demarcation potential27, 28 developed as a result of block, either partial or
complete, of the depolarization process near the pressure-making electrode such as is seen in direct leads, and as was seen in the ventricle (Fig. 7).

It was immediately felt that some practical use could be made of this phenomenon to make an electrographic diagnosis of atrial septal defect. A record with a late intrinsic deflection and elevated P-R segment would be expected if pressure was made with the electrode on the endocardial surface of the left atrium, and indeed such a record has been obtained in a patient with an atrial septal defect. However, a similar one has been encountered in a patient with a normal sized heart and suspected pulmonary arterial hypertension. Further, such electrograms have been encountered in normal subjects by Levine and his associates although these investigators felt that an orifice of some kind existed in the interatrial septum and that the electrode was in the left atrium. In the light of our experience and of more recent findings the electrode was undoubtedly in the coronary sinus or one of its venous branches. Under such circumstances the intrinsic deflection is later, by virtue of the electrode’s proximity to the left atrium. Further, sufficient pressure clearly may be exerted through the sinus wall to cause impairment of depolarization of atrial muscle and consequent displacement upward of the P-R segment. The finding, therefore, cannot be regarded as diagnostic of persistent atrial septal defect.

Ventricular Deflections Recorded from the Atrium. In the 12 subjects with records available from the atrium the QRS began with a positive deflection in seven, with a negative deflection in four, and in one with either a positive deflection or a negative deflection depending upon the location of the electrode in the atrium.

The initial positive deflection, when present, was always simultaneous with a similar wave observed in leads from the tricuspid region of the right ventricle, the superior vena cava, the right branch of the pulmonary artery, and the right arm. An initial Q, when present, was usually simultaneous with the nadir of a similar deflection in the leads from the last three locations just mentioned, and with the nadir of S or the notch on its descending limb in the lead from the cavity of the right ventricle. A late R’ might be small in all atrial locations and simultaneous with a similar deflection in records from above the atrium including the right arm, or with a notch on the ascending limb of S in the ventricular electrogram from the tricuspid region. On two occasions (patient QWi and patient SAT, Fig. 9) this deflection became quite large as lower atrial levels were approached, only to disappear as soon as the electrode was placed in the ventricle. In one of these (SAT) it was also large in the pulmonary artery. In two other instances (patients APy and PAq, Figs. 4 and 5) already mentioned, this late R’ was still prominent as a large positive deflection in a lead from the tricuspid region of the right ventricle, and could also be identified in the lead from the pulmonary artery.

In general the records obtained in the upper part of the right atrium were simulated quite
faithfully by leads from the right arm, but this relationship was not close in the case of leads from the lower atrial levels.\textsuperscript{2}

In summarizing what was found in the atria it may be stated that to begin with the electrical processes in atrial muscle consist of two parts, excitation and recovery, and that each of these, from experimental data, are dipolar in nature. An electrode in the atrium may be affected by both of these processes from one portion of the chamber and at a slightly later time by one or both in distant parts of the chamber. This overlap makes a precise separation of the resultant deflections difficult. It is suspected that initial negative deflections are the result of the electrode being near the sino-atrial node and on the active side of myocardium from the very beginning of atrial excitation. It cannot be stated with certainty from the data presented that activation of the sinoatrial node itself causes any part of initial negativity of the right atrial cavity, although this is possible.

With regard to ventricular deflections, an initial positive wave was usually simultaneous with other deflections ascribable to early depolarization of the left side of the interventricular septum. A large negative deflection, regardless of its location, was usually simultaneous with the peak of the R wave in leads obtained from the left side of the precordium and with the S wave or notch on its descending limb in the right ventricular lead suggesting its origin as being from excitation of the free wall of the left ventricle. The late positive deflection of large size in four subjects, and of moderate size in five others, was ascribable to some considerable mass of muscle excited from below upward and from left to right to be discussed below.

\textbf{Records from Within the Superior Vena Cava and Other Veins of the Thorax}

Records were made from the superior vena cava in nine subjects. In all the P wave was inverted, and usually bore several notches. The QRS in five consisted of the \textit{r}S type, in three of which a distinct notch was present on the ascending limb of \textit{S}. In two QRS had the form of \textit{Q}r, in one of \textit{q}R, and in one of \textit{r}Sr'. The T wave in all was inverted. The similarity to the lead from the right arm was striking in all except for the lower amplitude and the occasional slurring rather than notching of the ascending limb of \textit{S} in the latter. In only two was a simultaneous record made from the precordium with an electrode directly over the intravenous one. In both of these (patients JRo, Fig. 6, V$_{ SVC10}$, and SAt) the P and T waves were similar in the internal and external leads but the QRS was different, in one instance markedly so.

Toward the end of each experiment the electrode was placed fluoroscopically well out to the left border of the thorax (Fig. 2), usually, as far as could be judged, in the left axillary vein. In general, records from this region were similar to what was obtained in leading from the left arm although there were a few exceptions. Other records between the superior vena cava and this point were often attempted but were usually technically difficult because of large, irregular electromotive forces presumably of venous origin.

\textbf{The Large Positive Deflection at the Base of the Right Ventricle}

What is believed to be one of the most significant observations in this study was the large positive deflection found in the lower right atrium of four subjects and a similar deflection of moderate size in several others (Figs. 4, 5, 6 and Table 3). The simultaneity of this wave with a variety of others in leads from within the heart and from the surface of the body have been noted. In brief it appears that the electromotive force responsible for the deflection is large enough to be reflected in leads from the surface of the body and this force, regarded as a vector, seems to be, at least in the cases studied, pointing to the right and upward. Whether its direction is backward or forward appears to depend in part on the position of the heart in the thorax and undoubtedly on such other factors as the relative size of the ventricles, their exact orientation with respect to each other, the orientation of the muscle responsible for the deflection with respect to various points on the surface, and
possibly the architecture of the Purkinje system. When the force is pointing well forward, it is the cause of the r’ in the rSr’ complex sometimes encountered in normal subjects in a lead from the right sternal edge or from points farther to the right on the thorax. We have had the opportunity of confirming this in one subject not included in the present series. Also, when directed forward, it may cause a negative notch on the ascending limb of the S wave in the lead from the superior vena cava (Fig. 4, VSVC).

In the past this deflection or something similar to it has been ascribed, largely from the results of direct leads in dogs, to late excitation of the “conus.” No statement has been made as to the particular part of the conus concerned although it has been generally assumed that it was the free wall. It has also been stated that a late positive deflection (r”) may be found frequently in precordial leads taken over the conus.

There is much evidence which tends to refute both of these concepts. With regard to the dog it is significant that records may be obtained from the free wall of the conus which have a configuration, rsR’S, Fig. 3E, not unlike what was seen in an internal lead from the conus in patient APy (Fig. 4, VRVI and VRVII). The possibility exists that the peak of r really represented the early arrival of excitation at the epicardial surface of the conus and that R’ was transmitted from deeper-lying muscle which was excited quite late. We have made no observations in animals but the point is worth investigating.

With regard to man, in the only direct lead made from the conus it was almost the earliest surface point to be excited (0.0143 sec. after the beginning of R in Lead II), and at all ventricular points, including many on the posterior surface of the left ventricle, the intrinsic deflection began before half of the QRS interval was completed. The deflection we have observed had a time usually well beyond the midpoint of the total ventricular depolarization interval. Further, it was positive in the lower atrium and in the pulmonary artery, and negative in leads from the precordium near the conus (Figs. 5 and 6), both almost impossible to explain on the basis of late excitation of the free wall of the conus either radially outward, or longitudinally toward the pulmonic valve. Lastly, the free wall of the conus is a relatively thin structure, and when it differs quite markedly in a physiologic way from the cardiac muscle, or is particularly devoid of Purkinje fibers, it should not be excited very late.

The particular muscle mass which gives rise to the deflection under consideration must be, in the light of our present knowledge, conjectured. A specific observation regarded as important in localizing it is that the deflection, with two exceptions, underwent an abrupt change from a summit to a depression as the electrode slipped through the pulmonic valve on its way to the right ventricle. A similar but reverse behavior of the record was seen as the electrode traversed the tricuspid valve on its way from the right ventricle to the right atrium (Fig. 4). These data suggest that the particular muscle concerned was common to both orifices. Lastly, the structure must be at the base of the right ventricle, a statement supported by what has already been presented and also by the observation that the late deflection, when encountered in the ventricular cavity, was at the base of that chamber.

An anatomical structure which meets these requirements is the crista supraventricularis in the roof of the right ventricle. It is a thick muscular ridge, 12 to 15 mm. high, which extends forward and to the left from the atrioventricular to the pulmonic orifice. The ridge divides the upper part of the ventricular cavity into an inflow tract and an outflow tract, the latter being the conic interior of the right ventricle recognized on the surface as the conus arteriosus. At the anterior septal end of the ridge the origin of the moderator band (trabecula marginalis) may be found.

If this ridge is adequately supplied by subendocardial Purkinje fibers its anatomical position favors its mean direction of excitation upward, and to the right, a conclusion reached earlier from the direction of various deflections presumed to have their origin in this particular muscle band.

This crista supraventricularis undergoes con-
siderable hypertrophy in diseases causing increased work of the right ventricle, and it is suspected that the electromotive force to which it gives rise normally may be the same one responsible for some of the late positive deflections found in various external leads, especially on the right side of the precordium, in right ventricular hypertrophy. A preliminary survey of our observations on patients with right ventricular hypertrophy suggests that this is so, but a precise statement will depend upon a more detailed analysis of our records than we have thus far been able to make. Whatever the area concerned, it is by no means the last to be excited in the ventricles, for the deflection or deflections resulting from its excitation occur during the third quarter of the QRS interval.

The matter has importance because of the considerable bearing it has on several fundamental electrocardiographic problems. One of these is the problem of the origin of the late R wave or R' in leads from the right side of the precordium in right ventricular hypertrophy and in right bundle branch block already mentioned. Another is the applicability of a vector type of recording of the human electrocardiogram as a possible means of reducing the number of leads that need be taken for clinical purposes. The observations presented suggest that the reduction of all the ventricular electromotive forces to a single vector is probably too gross for practical value. However, it would appear that there are three principal vectors, so far as QRS is concerned, which would give practically all the information desired at least in the normal heart, and possibly in the abnormal heart too. These vectors, briefly, are the three responsible for the “Q”, “R”, and “S” respectively. In terms of what is known and what has been presented, the “Q vector” is produced by early excitation of the left side of the septum; the “R vector” by excitation principally of the apical free wall of the left ventricle; and the “S vector” by excitation probably of muscle in the roof of the right ventricle in a direction which is variable but dominantly upward, backward and to the right.

Studies are under way in which vectocardiograms and surface null potentials of the three principal vectors are being recorded in an effort to delineate their precise orientation in the thoracic space, and to test their usefulness in clinical diagnosis.

Relations between the QRS of Intracardiac and of Precordial Leads

Of particular interest in the normal precordial electrocardiogram has been the origin of the deflections in leads especially from the right side.

From what has gone before it is clear that in Leads V₁ and V₂ both the R wave and the S wave have dual origins. In high-speed records the R wave is usually notched either on its ascending or descending limbs. Either the notch on the ascending limb or the peak may be simultaneous with the peak of the R wave encountered in the lower portions of the right ventricle. In the former instance the peak, and in the latter instance a notch on its descending limb, occur between 0.01 and 0.02 second later. It is suspected that the initial part of the summit in Lead V₁ is septal in origin, and its later part is ascribable to excitation probably of the free wall of the right ventricle. This point has been made previously.  

The S wave in Leads V₁ and V₂ may show a notch on the descending limb simultaneous with the peak of the R wave in leads from the left side of the precordium. Under such circumstances the nadir is simultaneous with the nadir of S in Leads V₁ or V₂ or occurs at a time which is somewhere between the time of R and S in the left-sided leads. Although it occurs, it is exceptional for the nadir of S in leads V₁ and V₂ to be simultaneous with the peak of the R wave in Leads V₄ and V₅. It appears, therefore, that in the normal subject the S wave, both in leads from the right and left sides of the precordium, is produced largely by the electromotive force which gives rise to the large positive deflection often found in the right atrium and pulmonary artery and occasionally inside of the base of the right ventricle.

As to the leads from the left side of the precordium, the origins of Q and R are fairly well proven. The origin of the S wave has been suggested by observations and discussion given above.
Summary

1. Simultaneous galvanometric records were made of the potentials encountered in the right heart and attached vessels in 14 patients without clinical evidence of heart disease.

2. An analysis of these records and their temporal relationships to semidirect and indirect leads has been presented. The relationship of precordial to intracardiac QRS deflections has been elaborated particularly.

3. The significance of multiple deflections in the P wave recorded in the atrium, and of large positive QRS deflections encountered in proximity to the base of the right ventricle has been discussed.

4. It is suggested that the crista supraventricularis of the right ventricle may be the source of a large, moderately late electromotive force which is reflected not only in intracardiac but also in surface leads.

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Intracardiac and Intravascular Potentials Resulting from Electrical Activity of the Normal Human Heart

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