Excitation of Human Auricular Muscle and the Significance of the Intrinsicoid Deflection of the Auricular Electrocardiogram

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The technic of endocardial electrocardiography has been useful in applying certain basic concepts to the interpretation of an electrocardiogram. One of the earliest intra-auricular records, obtained in 1944, is here analyzed in detail. It led to a reaffirmation of the dipole concept of cardiac excitation. A number of selected auricular electrocardiograms recorded by simultaneous tracings from the chest wall and the esophagus demonstrate the direct clinical usefulness of the theoretic postulates.

Electrocardiograms of human subjects obtained from the right auricular and ventricular cavities have stimulated re-examination of the dipole approximation to the interpretation of the membrane theory. The shape of the electrocardiograms thus obtained has tended to confirm previous concepts which considered cardiac action potentials to arise from a band of dipoles moving over the heart. This interpretation is not generally accepted by those who view cardiac excitation and recovery in terms of an "interference theory" which assumes that an electrocardiogram represents the summation of two somewhat asynchronous monophasic action potentials. A discussion of suitable direct leads from the auricular surface of the human heart in situ with observations concerning the clinical application of such studies therefore appeared desirable. One of many records obtained from the right auricular cavity was found to be especially suitable for further analysis of the dipole concept.

The record (fig. 1) was obtained from M. S., a 58 year old Greek-American laborer who suffered from arteriosclerotic heart disease with sclerosis of the coronary arteries and right bundle branch block. A catheter containing the electrode was inserted into the right auricular cavity through the right antecubital vein. To avoid injury effects on the electrocardiogram, the tip of the electrode did not coincide with the tip of the catheter (whistle-tip catheter). When an attempt was made to pass the catheter into the right ventricular cavity it was deflected upwards and posteriorly and came to rest against the posteromedial portion of the interauricular septum about halfway between the sinoauricular region and the annulus fibrosus. The location was confirmed by roentgenograms obtained with the patient in the anteroposterior and in the right anterior oblique positions. A Sanborn Tribem electrocardiograph was used and Lead I was recorded simultaneously with the endocardial records.

The auricular electrocardiogram of figure 1 consists of three parts: (1) a very small deflection, Pp, (2) an activation phase which appears as a simple diphasic potential and is designated P\textsubscript{QRS}, and (3) a recovery phase, P\textsubscript{T}, which is represented by a slow low-amplitude deflection. P\textsubscript{QRS} averaged about 3.2 millivolts in

*The preauricular deflection P\textsubscript{p} (or "O") which is occasionally present in examples of human endocardial electrocardiograms has been described for human heart by one of us and may also be seen in records published by Sodi-Pallares. It is tempting to assign this deflection to the excitation of the sinus node. It is recalled that in cold-blooded animals the bulbus venosus gives rise to a striking preauricular wave and that a distinct deflection preceding P has been described for the mammalian electrocardiogram in leads taken from the vicinity of the sinus node.
height and 65 microseconds in duration. The deflection $P_{QRS}$ representing auricular activation is similar to experimental records from the exposed auricular muscle of the dog obtained with a unipolar recording system by Wilson, Macleod, and Barker and to the tracings of Craib from excised turtle heart muscle, striated muscle, and nerve. The human record under discussion is contrasted in figure 2 with direct unipolar leads from frog hearts showing a spontaneously beating bulbus venousus ($C, D$) and auricle in situ ($B$), an electrically stimulated piece of auricular muscle ($E$), and ($F$) a strip of spontaneously beating ventricular muscle. Preparations $E$ and $F$ were submerged in turtle ringer solution. The similarity of the animal records to the human tracing is inescapable.

**Theoretic Considerations**

Because of its syncytial nature human auricular muscle may be regarded as if it were a large single cell. An exciting impulse arising at the sinus node exceeds the threshold of the surrounding auricular muscle and a wave of excitation spreads radially from the center of excitation. The wave of excitation invades the polarized, resting muscle and leaves behind a region of depolarized tissue. The tissue is restored to its normal resting stage by a wave of repolarization which does not necessarily and presumably in normal heart muscle seldom arises from the same focus of origin as the excitatory wave. This wave of excitation may be considered as a band in which the membrane is being depolarized according to the law of excitation. The band is not sharply demarcated but shades from complete polarization on the leading edge to complete depolarization on the following edge. Its effective width is appreciable and may range from a few millimeters to several centimeters. Since closely adjacent areas are in electrically different states (polarized or depolarized) a voltage difference of about 50 to 100 millivolts will exist between the two regions and consequently a current will flow. For the sake of simplicity and without inducing a large error it can be assumed that the current flows between two lines one at the leading edge and one at the following edge of the band of depolarizing activity. This is equivalent electrically speaking to two line charges of opposite sign the effective width of the band apart and submerged in a volume conductor.

![Diagram](http://circ.ahajournals.org/)

**Fig. 1.** A human auricular electrogram. Upper tracing, Lead I; lower tracing, endocardial electrocardiogram from interauricular septum. Arrow points to small preauricular deflection ($P_p$). Ventricular QRS complexes characteristic of right bundle branch block. Time lines 0.040 second. (See text.)
The two line charges may be considered to be formed of an infinite number of dipoles placed side by side (see fig. 5). It must be emphasized that the symbol is not the reality and that the dipole concept is only a convenient symbolization of the membrane theory of activation.

The two line charges when submerged in the volume conductor of the body fluids give rise to a current flow and to the accompanying lines of equipotential distributed according to the laws of volume conduction.

For auricular muscle a simple geometrical picture of the voltage lines about the source and the sink may be constructed. Such a representation, familiar since Waller's original discussion, is shown in figure 3. This figure demonstrates the lines of equipotential about two line charges in a conduction medium whose dimensions are large compared with the distance between the source and the sink. The two line charges and their equipotential lines extend throughout the conducting fluid of the body. Midway between the two charges is a line of potential which is equal to half of the voltage difference between the two. This line is often referred to as the zero line and is used as a reference for measuring the other voltages.

If an electrode is placed in or on a volume conductor but at an appreciable distance from the source and the sink it will be effectively on, or very close to, the zero line (a "neutral" or "indifferent" electrode). An examination of figure 3 will show the truth of the statement. If now another electrode is placed so that the field about the line charges moves under or very close to this electrode, or if the electrode is moved along the line S-S from the extreme right of figure 3 to the extreme left, a diphasic wave will be recorded. In figure 4 a diphasic wave that was obtained in this fashion is illustrated. The resemblance to the diphasic action potential of figure 1 reproduced beside it is obvious and it is reasonable to believe that the PQR deflection of figure 1 arose from some such mechanism.

Figure 5 is a schematized version of two line charges viewed from above and passing across an exploring electrode with the other (neutral) electrode situated between the two charges. Again it is obvious that the passage of current over the electrode results in a plus-minus deflection. The distance between the two peaks of the recorded tracing of a direct lead represents the distance between the source and the sink and defines the width of the excitation wave, i.e., the band of tissue undergoing depolarization. The rapid downstroke of a record such as is illustrated in figure 1 may therefore be taken as an approximate measure of the width of this band. It is roughly identical with the so-called intrinsic deflection of a direct unipolar electrocardiogram. Given a rate of transmission of the action current through auricular muscle of 1,000 mm. per second, the width of the band or the distance between the...
source and the sink in the record presented here would approximate 10 millimeters. If recent figures on the rate of conduction through the human heart are substituted, the width of the band would measure 4.5 to 5 millimeters. The width of the band in a dog’s auricle calculated by Wilson, Macleod, and Barker measured 3.6 to 4 millimeters. It must again be remembered that these values are only first approximations and that the actual width of the band of depolarization is indefinite.

PRACTICAL CONSIDERATIONS

The sudden reversal of voltage as the source and sink pass the electrode has been termed the “intrinsic deflection” by Lewis, Meakins, and White. If the exploring electrode is located at a place distant from the source and sink beneath the exploring electrode, serves as a valuable diagnostic aid.

The conclusion, however, that the intrinsicoid deflection is always the same as the intrinsic deflection must be viewed with caution. When the electrode is directly on the surface of excitable tissue, effects of adjacent areas undergoing excitation are small. With the electrode at some distance from the source and sink, other areas of excitation will materially
and appreciably contribute to the field at the electrode and the result represents the sum of all fields at this point. The observed intrinsicoid deflection may thus not represent the actual passage of the wave of excitation. This appears to be one of the reasons for the absence of a delay in the intrinsicoid deflection over the right ventricle in certain examples of right ventricular hypertrophy and for the appearance alteration of excitation over the right ventricle in instances of left ventricular hypertrophy and left bundle branch block. However, when the electrode is close to excited tissue, and when this tissue is belatedly activated, the intrinsicoid deflection is probably a valid indication of the passage of the excitatory wave.

Macleod, Wilson, and Barker were unable to obtain an intrinsicoid deflection from auricular muscle comparable to that observed in leads from the vicinity of ventricular musculature. They suggested that this was due to the difference in the manner in which the excitation process spreads over ventricular as compared to auricular muscle. Deflections similar to the one under discussion, however, and clearly showing an intrinsicoid deflection have since been demonstrated with an electrode placed in the esophagus and almost in contact with left auricular tissue. A semi-intrinsic deflection is observed with such electrodes even when auricular flutter is present. Auricular endocardial records, not necessarily in contact with cardiac tissue, are likewise characterized by a diphasic component. The similarity between endocardial auricular and esophageal (epicardial) auricular records is in contrast with the striking difference that exists between records taken from the ventricular cavities when compared with epicardial ventricular leads. This confirms the theory that the order of excitation of auricular muscle, though not excitation itself, is different from that of the ventricles. In the auricles, excitation spreads radially from the sinoauricular node over the sheet of auricular tissue; in the ventricles, direct penetration of ventricular muscle by the excitation wave from within outward and its simultaneous emergence at the surface at several points are generally accepted.

Fig. 4.—A comparison of calculated and recorded action potentials. A (left), artificial action potential calculated by placing an electrode on I of figure 3 and moving the other electrode along the line S-S from the extreme right of the diagram to the extreme left. The voltage lines crossed are plotted as a function of the distance along S-S and according to the laws of volume conduction. (See text.) B, Enlarged P of figure 1. The essential similarity to A is apparent.

Fig. 5.—The dipolar concept of cardiac action currents and the intrinsic deflection. P defines a point representing the location of the exploring electrode. Electrical potentials recorded at P will show increasing positivity as the action current advances in the direction of P. As the dipole passes over P the potentials at this point will swing from maximum positivity to maximum negativity and the distance between the peaks will be proportional to the width of the dipolar band of cardiac accesion. The interconnecting line between the two maxima of the curve is closely related though perhaps not identical with Lewis' intrinsic deflection.
Auricular esophageal leads reveal an intrinsicoid deflection which may be assigned to the activation of left auricular tissue. No intrinsicoid deflections other than those obtained from the endocardium by blood contact have ever been demonstrated for the right auricle. The true intrinsic deflection from this chamber apparently is hidden by the potentials from other
regions because of the distance of the electrode from the auricular surface. A lead usually considered to represent the effects of activation of the right auricle (V1) shows a small, upright, rounded deflection quite different from the deflection of the left auricular esophageal lead (figs. 6 and 7). It may be argued that the absence of a distinct diphasic deflection obtained from an electrode in this position indicates that of excitation never completely passes under the region being explored. A chest lead in Lead V1 position, usually directly opposite the right atrium, need not necessarily be considered a semidirect auricular lead. It is of interest, however, that the onset of right auricular activation, evidence of which appears simultaneously in leads from the right auricular endocardium and in V1, definitely precedes the onset of

![Diagram of electrocardiogram](image)

**Fig. 7.—Normal and abnormal activation of left auricular musculature.** Lead V1 and esophageal auricular leads recorded simultaneously. A, B, and C were obtained from a 34 year old normal subject; D, E, F, and G from a 59 year old man with a markedly stenotic and calcified mitral valve, left auricular and right ventricular hypertrophy and dilatation (autopsy). The onset of the intrinsicoid deflection of all auricular esophageal leads in D, E, F, and G is definitely delayed and coincides with the final segment of the typically biphasic P wave in V1. A single upright esophageal P deflection characterizes left auricular hypertrophy in the regions where the intrinsicoid deflection is most delayed while a biphasic P wave is usually seen in all auricular levels of the normal esophageal electrocardiogram. Film speed 75 mm. per second; time lines 0.2 second. Arrows and figures as in figure 6.

the electrode lies always in the field of one side of the dipole, that is, sharply to the left or to the right of the diagram of figure 3 and that the center line of the dipole never crosses the region under the electrode. This would be possible if the impulse spreads radially over the auricular musculature. A rounded, upright P wave in V1 would be so for the same reason that leads from the sinus region and from the area cephalad to it (upper auricular region and vena cava) are always uniformly negative: the wave positivity in left auricular (esophageal) leads of normal subjects (figs. 7 and 8) and that the intrinsicoid deflection recorded from these esophageal regions coincides approximately with the final portion of the small, rounded deflection of the P wave in V1 or in any of the standard limb leads (figs. 6, A, and D, and 7, A, B and C). It is exaggerated in left auricular hypertrophy and enlargement, where a true delay in the intrinsicoid deflection similar to that observed over hypertrophied and di-
lated ventricular muscle, is common. In instances of this kind, V_{1} or a lead taken one intercostal space higher, reveals a sharp, biphasic deflection remotely resembling the biphasic plus-minus deflection of figure 1 (figs. 6, 7, and 8). This was first noted by one of us^{30} and has frequently been reported since. Standard limb leads usually reveal a broad and the electrode. Records of this kind should not be considered as representing an intrinsicoid deflection caused by hypertrophy of the right auricle which has frequently been proposed.^{32-36} The biphasic pattern of the P wave in V_{1} occasionally encountered in normal subjects may likewise be considered as a remote effect of auricular muscle distant from the exploring electrode. It is of interest to recall that the mechanical contraction of the right auricular muscle slightly precedes that of the left.^{36}

Because no semidirect thoracic lead exists which reflects the changes of the right auricle, hypertrophy of this chamber appears almost impossible to diagnose electrocardiographically. Originally, a tall and upright P wave in Leads II and III was taken as evidence of right auricular hypertrophy ("P pulmonale"^{31}). In clin-

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**Fig. 8.**—Right and left auricular enlargement with right ventricular hypertrophy. Lead V_{1} and esophageal auricular leads recorded simultaneously. A, B, and C are from a 36 year old man with cor pulmonale following chronic pneumonitis and fibrosis of the entire left lung and of the lower and middle lobes of the right lung. The right ventricle was predominantly enlarged and the right auricle dilated (autopsy.) D, E, and F were obtained from a 24 year old man with advanced mitral stenosis. The electrocardiographic evidence of right ventricular enlargement was almost identical in both subjects. Auricular esophageal leads were normal in A, B, and C but showed definite delay of intrinsicoid deflection in esophageal leads with biphasic P waves in V_{1} in the second case. Film speed 75 mm. per second; time lines 0.2 second. Arrows and figures as in figure 6.
ical examples of this kind, the P wave in V1 is rarely altered (fig. 8, A, B, and C), though it may at times be negative or small and biphasic (fig. 6, G). Esophageal leads from such subjects fail to show a late activation of left auricular tissue (figs. 6, C and G, and 8, A, B, and C). It is therefore postulated that the pattern of P pulmonale is not caused by unilateral auricular hypertrophy but may be primarily the result of abnormal cardiac rotation or of auricular dilatation which allows the electrical effects of both auricles to be deflected backward and toward the left leg. The lack of correlation between right auricular hypertrophy and "P pulmonale" has been frequently noted.34-40 Figure 9 reveals an example of temporary acute cor pulmonale in a patient with chronic disease of the lungs. During the phase of failure, huge "pulmonary P waves" were present which promptly disappeared upon recovery. Observations such as these likewise argue against the concept that views P pulmonale as a manifestation of right auricular hypertrophy. On the other hand, regression of mitral P waves, once present, has never been observed to our knowledge. The two types of P-wave changes are therefore unrelated and arise from entirely different mechanisms.

CONCLUSIONS

1. Direct electrocardiograms obtained from the human auricular muscle in situ allow a re-examination of the dipole approximation of the membrane theory of cardiac excitation.

2. It is assumed that during activation a current flows between two lines, one at the leading edge and one at the following edge, of a band expressing depolarizing activity of muscle units submerged in a volume conductor.

3. The line charges and their equipotential lines extend throughout the conducting fluid of the body and an electrode located in the path of the moving dipole will record a diphasic plus-minus deflection. Employing available formulas for the flow of currents in volume conductors, a record may be plotted that has striking similarities to those obtained from direct leads in animals and man.

4. These considerations are directly applicable to semidirect auricular leads. An electrode placed in the esophagus may be considered to lie in the field of the moving dipole.

5. The intrinsicoid deflection of such leads may be used to estimate left auricular hypertrophy. From these the remote effects of auricular activation on standard limb leads and on
certain precordial leads can be interpreted with reasonable accuracy.

6. The "mitral" P wave and sharply biphasic P waves in $V_1$ may be considered to result from left auricular enlargement. Right auricular hypertrophy on the other hand cannot be diagnosed from the electrocardiogram and examples of "pulmonary" P waves may be caused by abnormal cardiac rotation allowing the effects of both auricles to be deflected backward and toward the left leg.

REFERENCES


27. ———: A review of advances in the study of aurice-

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