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Importance of the electrocardiogram in early pulmonary embolism diagnostic

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Analysis and evaluation of the various diagnostic methods of pulmonary embolism

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In recent years a great incidence of Acute Pulmonary Embolism has been recorded all over the world and, of course, in Uruguay.

The number of deaths that Pulmonary Embolism causes as a result of late diagnosis is a matter of major concern in present-day Medicine. Too late a diagnosis also implies the impossibility of adequate treatment at the right time, an essential requirement not only in life saving but also in shortened hospitalizations and consequent reduction of the expenses involved in the care of these patients. Knowledge of the clinical manifestations and pathogenesis of Pulmonary Embolism began with Virchow (1856). Cohn (1860), and Flint (1867). In the last decade various diagnostic techniques such as pulmonary enzymogram, simple chest radiography, pulmonary scintillogram and pulmonary arteriography have increased the number of diagnosis of Pulmonary Embolism.

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However, it is emphasized that, often, P.E. diagnosis is not made during the life of the patient or, if so, it is carried out too late, as shown by postmortem studies. Smith et al. state that Pulmonary Embolism is diagnosed during life only in 50 % of cases, and Vernant notes that P.E. is responsible for 25 % of postoperative deaths and 30 % of the mortality among people with heart disease.

After a fairly comprehensive review of the different methods of Pulmonary Embolism diagnosis, the importance of early serial electrocardiographic study with the original interpretation of the authors of this article is pointed out. We think that the diagnosis of Pulmonary Embolism will be greatly facilitated if early and serial electrocardiograms are taken and indirect signs of paroxysmal right atrial enlargement (reduction of QRS potentials in V1, V2, V3) are found to be an expression of acute overload of the right cavities in those cases where P.E. is suspected (even in the absence of the so called typical signs), in surgical operations, recent labors, patients confined in bed over long periods of time or in persons with abruptly uncompensated heart insufficiency. This has been corroborated by 91,4 % of positive findings in the clinical cases (35 cases) we have studied as and confirmed by pulmonary scanning, and in 100 % of our experimental cases. A survey of the above findings as well as their wise evaluation enables us to consider the indirect signs of paroxysmal right atrial enlargement as the earliest most frequent and sensitive sign, as a detector of an acute pulmonary hypertension resulting in the diagnosis of Acute Pulmonary Embolism and allowing for the proper therapeutic measures and follow-up of the patient.

Bases of our original concepts on the importance of the electrocardiogram in the diagnosis of early pumonary embolism

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The importance of an early serial electrocardiographic study in cases of presumable A.P.E. production is pointed out.

The reliability and earliness of the procedure is not comparable with the present management of most or all cases. Such management is made dependant on:

1) The development of clinical signs that may make it presumable; 2) radiographic studies, which are generally late and, only in a few cases, confirmatory; 3) enzymographic study; 4) Pulmonary scintillogram, always delayed and requiring a simple previous radiographic study for its correct interpretation; 5) Arteriography of the pulmonary artery, always delayed, risky and only possible in well organized and fully equipped centers with competent staffs; 6) Electrocardiographic studies that are generally neither early nor serial and always interpreted partially or through secondary signs, have led to understimate and hence neglect the essential need for and execution of an easy method which is neither risky nor bothersome and within the possibilities of any health care center.

ELECTROCARDIOGRAPHIC CHANGES ASSOCIATED WITH A.P.E.

1935 Mc Ginn-White: Pattern S3-Q3-T3.
1938 Love et al: RST segment depression and T wave inversion.
1939 Durant, Grinsberg, Roesler: Transient Right Bundle Branch Block.
1940 Sokolow et al: reported that it is frequently absent along with the electrocardiographic changes in A.P.E.
1941 Wood: T wave inversion (V1, V2, V3).
1947 Wilson et al: Clockwise rotation of the ventricular mass in A.P.E.
1947 Katz: P pulmonale.
1950 Pseudoinfarction pattern: prominent q wave associated with RST segment or T wave abnormalities.
1958 Cutforth and Oram: RST segment depression, T wave inversion in precordial or Limb leads.
1966 Pseudoinfarction pattern: q wave or QS V1, T wave inversion in V1, V2, V3.
1971-72 Stein-Bruce; Lynch-Stein-Bruce: left anterior fascicular block.
1972 Dalen: the atrial flutter or atrial fibrillation in A.P.E. would be limited in patients with previous heart disease.
1972 Chatterjee, Sutton, Miller: observed reduction of the right ventricle intracavitary potentials in massive A.P.E.
1974 Isasi M. E., Isasi E. S.: pointed out the importance of the Indirect Signs of Paroxysmal Right Atrial Enlargement (paroxysmal reductions of the QRS potentials of the right precordial leads as the most frequent electrocardiographic sign in A.P.E.: 86 % of 22 cases).
1975 Stein et al: point out the low voltage QRS complexes in frontal plane (limb-leads) in A.P.E. as a feature not described so far but fail to give a physiopathological explanation of the phenomenon.
They find it in 6 % of cases.

Love et al, in 1938, reported that the right ventricle dilatation caused the electrocardiographic changes in A.P.E.

In 1941 Wood concluded that: a) the most important factor causing the electrocardiographic changes in the A.P.E. was the *mural stress* of the right ventricle; b) the electrocardiographic changes depended on the size of the embolus.

On the basis of our clinical and experimental studies we contend that the electrocardiographic changes in A.P.E. are secondary to the acute overload of both right cavities.

1) The acute overload of the right atrium evidenced in the electrocardiogram by the Indirect Signs of Paroxysmal Right Atrial Enlargement is the main electrocardiographic detector of Acute Pulmonary Hypertension, i.e., it reflects the severity of the hemodynamic changes.

The serial electrocardiographic records of V1, (V1, V2, V3) are the elements that allow us to assess the severity of the case, its regression earlier than or parallel to the clinical radiological and scintillographic improvement, and to detect the recurrence of the disturbance.

2) The acute overload of the right ventricle would account for: i) Primary RST segment and T wave abnormalities, ii) other less common signs (S1, Q3, T3, right bundle branch block, right axis deviation...)

PHYSIOPATHOLOGY

A) *Changes in the repolarization of ventricular myocardium*

The vascular flow depends on the pressure gradient between the ascending aorta and the venous sinus, and the resistance of the coronary artery involved.

Generally, the venous sinus pressure is low and its cyclical variation with the heart activity is negligible.

There normally exists a cyclical modification of the vascular resistance (different for each coronary artery) dependent upon the cardiac cycle due to the vascular contraction. In A.P.E. the aorta venous-sinus pressure gradient drops due to the hypertension in the right atrium and venous sinus, and the fall of artery pressure.

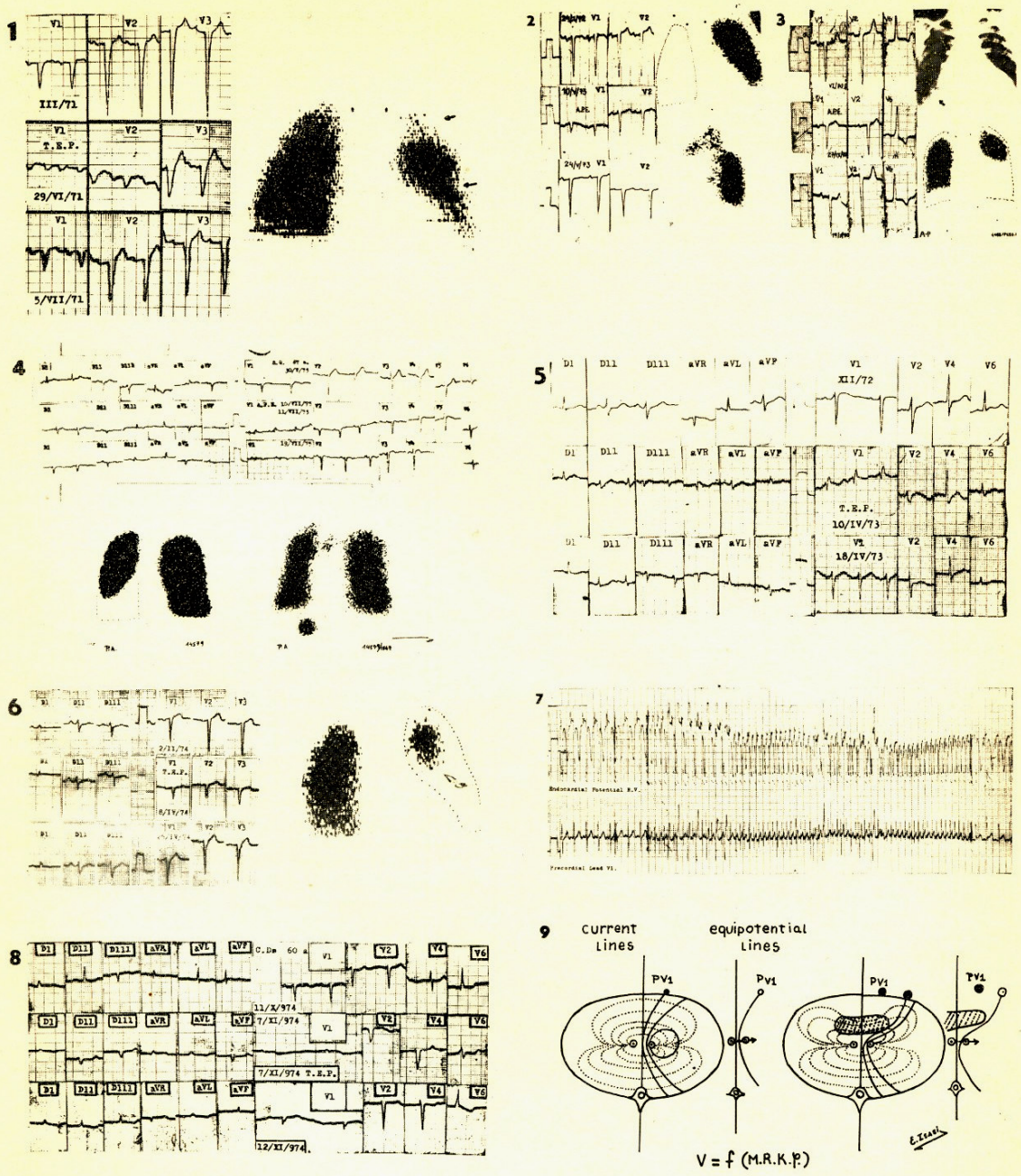
On the other hand there occur changes in the vascular resistance; the coronary arterioles from the right coronary artery modify their diameter due to the mural stress of the right ventricle (mural stress = mural tension/thickness).

The right coronary flow is naturally continuous (systolic-diastolic).

In A.P.E. the drop of the output of the right coronary artery is due to

a) Retrograde repercussion of the pulmonary embolism as exhibited by:

1) Fall of the coronary $\triangle P$ due to the Hypertension in the right atrium and venous sinus.



$$\Delta P = \frac{\text{ascending Aorta pressure}}{\text{venous sinus pressure}}$$

2) The elongation of the right coronary artery by dilatation for the right cavities, with reduction for diameter and increase in resistance.

b) Anterograde repercussion fall of the left ventricle output (fall of artery pressure).

It is then understandable that these cases exhibit in ECG a systolic overload of the right ventricle with T waves of ischemic type and angor (even in the absence of arteriosclerotic pathology of the coronary vessels). Angor Ceruleus or Right Angor.

B) *Transient Right bundle branch block. Intermittent Right bundle branch block*

Atrial Extrasystoles with phasic aberrant ventricular conduction with B R D configuration manifested in A.P.E. are phenomena secondary to the overload of the right cavities.

Mural Tension = Pr.R Pr — intracavitary pressure.

$$\text{Mural Stress} = \frac{Pr.R}{e}$$

R — Radius of the right ventricle.

e — Thickness of the right ventricle.

FIG. 1 — 45 frs Dyspnea - Pleural Pain - Cough. Perfusion Lung Scan 16/VII/71 there are two areas of decreased perfusion in left Lung (upper and lateral) with well ventilation. 29/VI/71 E.C.G. on day of A.P.E. showing the indirect signs of paroxysmal right Atrial enlargement.

FIG. 2 — 49 yrs Dyspnea - Cyanosis - Pleural effusion. 10/V/73 E.C.G. on day of A.P.E. showing the indirect signs of paroxysmal right atrial enlargement. (Reduction of QRS potentials in V_1-V_2). 24/V/73 Observe the recovery of QRS potentials. Perfusion and Ventilation lung scan: Multiple Pulmonary Embolism (25/V/73).

FIG. 3.— Correlation electrocardiographic, radiographic and scintillographic. Observe the reduction of QRS potentials, in the right precordial leads in A.P.E. (27/IX/70).

FIG. 4.—30/V/75 E.C.G. sinus-rythm. Left Anterior Fascicular Block. 11/VII/75 E.C.G. on the day of A.P.E. Sinus rythm. Right axis deviation. Indirect signs of paroxysmal atrial enlargement. Incomplete right bundle branch block absence of r in V_2. 19/VII/75 E.C.G. 8 days later. Partial recovery of the potentials in the right precordial leads.

FIG. 5.— 64 yrs Dyspnea - cough - hemoptysis. 10/IV/73 E.C.G. on day of A.P.E. showing the indirect signs of paroxysmal atrial enlargement, low voltage QRS complexes in frontal plane and transient right bundle branch block.

FIG. 6.— 65 yrs Dyspnea - Pleural pain. 8/IV/74 E.C.G. Indirect signs of paroxysmal right atrial enlargement. 8/IV/74 perfusion lung scan showing hypoperfusion at the left lung base.

FIG. 7.— Gas embolization in dogs, showing the behavior of the myocardial potentials in the exo and endocavitary records.

FIG. 8.— 60 yrs. Dyspnea - cough-hemoptysis. 11/X/74 sinus rythm. RST abnormalities. 7/XI/74 A.P.E. atrial fibrillation. Left anterior fascicular block. qR V_1. Indirect signs of paroxysmal right atrial enlargement. Low voltage QRS complexes in frontal plane. 12/XI/74 Sinus rythm. Recovery of the QRS potentials in the right precordial leads.

FIG. 9.

In Acute Pulmonary Embolism we observe an increase in mural tension and a great increase of the mural stress of the right ventricle due to increase of intracavitary pressure, increase of the radius (by dilatation of the right ventricle) and diminution of its wall thickness. The result of the great mural stress both on the free wall of the right ventricle and the septum, is the compression of the right branch of the bundle of His with subsequent alteration of intraventricular conduction, which, due to the transient characteristic of the phenomenon, has been analogized to a Neuropraxia. Some authors refer to it as neuroapraxia of the right branch of the bundle of His.

C) *Indirect Signs of Paroxysmal Right Atrial Enlargement*

The lead directly facing to right atrium in a normal situation is the right precordial lead V1 as observable in anatomic section.

The indirect signs of paroxysmal right atrial enlargement would be produced by the R.A.E. interposing between the dipole (generating source of heart currents) and the exploratory electrode V1 (V1, V2, V3) facing it.

The reason for the voltage reduction would be the shunt effect causing the increase of blood mass in the right atrium that would shortcircuit. the current lines of heart source displacing the equipotential lines.

Hence, from V2 to V6 or V3 to V6 where the electrode explores the ventricular myocardium, it registers a normal-sized QRS.

...Many characteristics of the clinical situation face the practitioner with a differential diagnosis with acute coronary occlusion, especially when dyspnea and chest pain (of precordial topography) are accompanied by manifestations of diminution of the left ventricle output (anterograde manifestations of A.P.E.), particularly, marked artery hypotension.

In these cases the clinician generally requests an urgent electrocardiogram.

We consider that an early serial electrocardiogram is indicated in this case not only to discard the acute coronary occlusion (which has to be ruled out) but to detect the acute overload of the right cavities particularly elicited by paroxysmal atrial enlargement.

...When the hypertensive state secondary to A.P.E. occurs in subjects without previous heart disease, the changes in the cardiovascular silhouette are very important.

It is not so when dealing with patients with previous heart pathology (patients with cardiac insufficiency) where a greater cardiovascular dilatation is of difficult evaluation.

It is in these cases, among others, that we consider that serial E.C.G. is of great interest in the disclosure of the increase of dilatation or its reduction according to the course undergone by patients.

The poor diagnostic significance that many authors ascribe to the E.C.G. is due to the fact that

1) They do not recognize the indirect signs of paroxysmal right atrial enlargement (as indicative of the great acute stress of the right chambers).

2) They do not take serial E.C.G. at an early stage. The early electrocardiographic record is a must since, according to our observations, the electrocardiographic signs of acute overload of the right cavities may disappear within 36 hours (in the case with the fastest recovery in our study).

Experimental acute cor pulmonale in dogs

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It is intended to show in this report the results attained in Experimental Acute Cor Pulmonale.

The study shows the behavior of the myocardial potentials in the *exo* and *endocavitary* records, the only electric variable to be analyzed.

Our concern arose from the observation of myocardial potentials recorded with the conventional serial electrocardiograms in the clinical cases studied.

We have analyzed the myocardial potentials obtained by the *exo* (right precordial lead V1) and *endocavitary* records from a bipolar electrode catheter placed in the right ventricle. 38 experiments in 12 animals were performed. The electrocardiographic cardiocirculatory changes present in Acute Pulmonary Embolism were experimentally reproduced by:

a) gas embolism; b) clamping of one of the branches of the pulmonary artery or of the pulmonary trunk; c) steel-pellet embolization. We have performed the recording of Pre-Intra and Post gas embolization.

The basal records were prolonged for ten minutes. The record was continuously performed for 30 minutes after the embolism (the time set for recovery).

The experiment was repeated several times in every animal after a recovery period.

In every case the amplitude or size of the QRS was analyzed (taken as the total sum of the QRS deflections pre-intra and post-gasembolism).

The electrocardiographic changes obtained can be summarized as follows:

1) Reduction of exocavitary potentials (right precordial leads) in 100 % of the provoked embolisms.

2) Reduction of potentials in endocavitary records of the right ventricle in 100 % of the cases.

3) Sinus tachycardia and supraventricular and ventricular extrasystoles recorded immediately after gas injection.

4) Isolated or run ventricular extrasystoles. Cardiac death was preceded by ectopic ventricular rhytm that led to ventricular fibrillation. All the animals studied died from ventricular fibrillation.

5) Classical direct signs of right atrial enlargement.

6) Simultaneous presence of indirect signs of paroxysmal right atrial enlargement and development of a right bundle branch block (complete, incomplete, transient, intermittent).

7) Changes in the ventricular repolarization.

To Sum Up

Our experimental study has shown that in A.P.E. the *exo* and *endocavitary* potentials undergo the following changes.

I) Reduction of QRS potentials.

II) The reduction is paroxysmal.

III) The recovery times of the *exo* and *endocavitary* potentials are different, the latter occurring in a shorter time.

Clinical Applicability

We ascribe the indirect signs of paroxysmal right atrial enlargement a great value in A.P.E. because:

1) They are the most frequent electrocardiographic diagnostic feature found in A.P.E.

2) They indirectly permit determining the hemodynamic modifications caused by the pulmonary embolism.

3) They make it possible to determine the course of the overload on the right cavities (through serial electrocardiographic records).

The recovery of potentials in the right precordial leads is earlier than or parallel to clinical, radiographic and scintillographic improvement.

4) They are easy-to-detect signs (that is why early and serial electrocradiographic records are necessary) which can be taken with the patient in bed and away from big health centers.

5) It is a low-cost diagnostic and follow-up procedure.

Bioelectric interpretation of the changes in exo and endocavitary patentials in pulmonary embolism

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The theory of the electric currents produced by a dipole placed into a conducting volume would enable the interpretation of the voltage variations occurring in the QRS complex of the E.C.G. in Acute Pulmonary Embolism. Accordingly, the voltage at a point *P* of a conducting medium is a scalar function of:

1) The Modulus of the dipolar moment vector $|\vec{m}|$.

2) Medium conductivity (K).

3) (r, ϑ) geometrical factors determining the position of point P with respect to the dipole.

Consequently: $V_P = f\ [|\vec{m}|, K, r, \vartheta]$.

The "f" function has been mathematically determined by different authors for particular cases (infinite and homogeneous medium, finite media, plates, spheroidal surfaces, etc.). All the expressions obtained exhibit an inverse dependance on the conductivity and a potency of distance "r" of the natural exponent "n"

$$V_P = \frac{1}{K} \cdot \varphi\ (r^n),\ \psi\ [|\vec{m}|, \vartheta]$$

$\varphi\ (r^n)$ an "r" monotonously diminishing function.

The preceding expressions will suffice to interpret, through a variation analysis of the variables, the changes observed in the E.C.G. of A.P.E.

Interpretation of the EXO and ENDOcavitary records

1) *Exocavitary:* in the basal situation, the variables ($|\vec{m}|$, K, r, ϑ) determining the voltage in the precordial leads take up values that enable a QRS record of normal *sixe*. Let $|\vec{m_0}|$ K_0 R_{10} Q_{10} be those values. ° subscript refers to basal; 1 subscript refers to each lead.

In A.P.E., it is necessary to study the possible variation in the values of the variables with respect to the basal values. The fact that no electrocardiographic changes appear in the left precordial leads (at least up to the embolization degree experimentally attained) suggests that, within those limits, the variables maintain their basal values in A.P.E. Let us now consider lead V1 in situation of A.P.E.:

In accordance with what has been previously stated we may assume that: $|\vec{m_0}| = |m_1|$; $r_{10} = r_{11}$; $\vartheta_{10} = \vartheta_{11}$.

Thus only a local variation of conductivity in the region close to the right precordial lead would allow us to interpret the value descents observed.

The increase of blood volume contained in the right atrium would be, in the authors' opinion, the primary cause of descents in QRS potentials.

This increase of blood mass of high conductivity would act as a shunt short-circuiting the cardiac currents, provoking a redistribution of the equipotential surfaces in the conducting medium and, as a result, the decrease of the voltage absolute values in the right precordials leads.

As an example, a very simplified scheme of what has been expressed, appears in Fig. 9.

2) *Endocavitary:* simultaneous *endo* and *exocavitary* records show sudden decreases in the total voltage of the QRS complex in A.P.E.

In order to interpret this finding, we shall again analyze the variables determining the voltage at the registering point, now located within the right ventricle.

a) What we have expressed in the case of the exocavitary records allow us to admit that for this case $|\vec{m_0}| = |\vec{m_1}|$.

b) Although the redistribution of equipotential surfaces that enabled us to interpret the exocavitary records also acts in this case as a voltage decreasing factor, we do consider it to be a primary one.

There is also the possibility of a variation in the blood composition with the subsequent variation of the conductivity K, a factor that we have not studied and which, if produced, should be quantified in order to determine its influence on the findings recorded.

c) So far as the distance factor is concerned, let us remember that the function $\varphi\ (r^n)$ is monotonously decreasing and that for small values of the variable (as it occurs in the intracavitary records) small $\triangle r$ increments produce increases of the function, to be taken into consideration.

The variation of "r" is thus regarded to be the most important cause in the decrease of the QRS voltage. We admit, for such a purpose, that ventricular filling flow maintains, in basal conditions, the light recording catheter in a medial position within the right cavity, an electrocardiographic record of normal size being obtained.

In A.P.E. the catheter would be displaced towards the ventricular wall, increasing the value of the variable "r", and as a result, decreasing the voltage obtained. When reaching a certain degree of embolization, the catheter would lie beside the ventricular wall, a finding that would allow us to interpret the curve in fig.

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