SPECIAL ARTICLE

The Role of Necrosis in the Origin of Electrocardiographic Alterations Characteristic of Myocardial Infarction

By M. G. Udelnov

Action of Necrotic Tissue on the Intact Myocardium and the Method of Study

THE METHOD of monophasic or unipolar leads is used in all the diverse fields of electrophysiology: one electrode is placed on an altered section of the myocardium, nerve, or muscle. However, the reason for the registration of unipolar or monophasic waves by this method has yet to be explained. Injured tissue is a good physical conductor and therefore should, it seems, be in itself a continuance of the registering electrode and thus insure the registration of the action potential from the intact tissue, neighboring the altered region.

The injured tissue also does not have rectifier properties. It is therefore impossible to explain the "indifference" of the electrode placed on the injured tissue by the physiologic properties of the latter. We therefore decided to try to explain this phenomena of unipolar leads by the action of the necrotic tissue on the adjacent intact myocardial structures.

To study this problem in simple and easily controllable experimental conditions we decided against employing the usual method of forming a section of necrotic tissue by injuring the myocardial structures. Instead a strip of necrotic tissue was placed on the surface of the intact myocardium. This strip of tissue was originally taken from a skeletal muscle or from the ventricle and they were permanently injured beforehand. One of the registering electrodes was placed on the surface of this necrotic strip, now "glued" to the myocardium; the second electrode was placed on the surface of the atrium or ventricle (fig. 1).

We used this method for the following reasons: if the necrotic tissue of the altered region during the usual recording procedure produces unipolar recording by its action on neighboring intact structure then the foreign strip of necrotic tissue placed on the myocardium should also give a monophasic record in the electrocardiogram.

The very first experiments done on a frog heart in situ showed that the placing of necrotic tissue on the surface of the intact heart ventricle of a frog is accompanied by the appearance of monophasic waves (fig. 1): The monophasic tracing remains unchanged as long as the strip of necrotic tissue stays in contact with the myocardium. Control tests showed that the monophasic electrocardiograms recorded in these conditions were in amplitude, form, and duration very much the same as monophasic electrocardiograms recorded by the usual electrophysiologic method.

After the necrotic tissue was removed and the heart was washed with Ringer solution, a normal electrocardiogram was again recorded (fig. 1). Therefore, we may say that necrotic tissue has no permanent damaging effect on the myocardium.¹

However, the functional changes in that part of the myocardium in contact with the necrotic tissue were very pronounced and disappeared only very slowly if the necrotic tissue was in contact with the myocardium for

¹ From the Institute of Therapeutics of the Academy of Medical Sciences of the U.S.S.R., Moscow.
Figure 1

Electrographic alterations of a frog's heart when necrotic tissue is applied. Conditions of electrographic recording are schematically depicted above the record. I, normal electrogram; II, after the application of necrotic tissue to the apex of the ventricle; III, soon after the removal of the necrotic tissue. During application the electrogram is monophasic; after removal of the necrotic tissue, it is diphasic.

a few minutes. In such cases monophasic traits characterized by deviation of the RS-T segment and deformation of the T wave persisted for as much as 10 minutes after the necrotic tissue was removed.\textsuperscript{2, 3} It is important to note that the monophasic electrocardiogram appeared only when the necrotic tissue was in contact with the myocardium. If the necrotic strip were placed under the recording electrode in the electric field of the heart, the electrocardiograms did not change. This phenomenon emphasizes that necrotic tissue in itself has no physical or chemical properties that would allow the electrode in contact with it to relay potential waves. We also observed that the necrotic tissue also produced a marked weakness of the ventricular contraction (fig. 2) : the section of the myocardium under the necrotic tissue did not take part in the ventricular systole. It remained relaxed and bulged slightly during the systole. While the contracting part of the ventricle was of a pale color, this section remained dark red.

There was no systole in this section for quite a period after the necrotic tissue was removed. This fact, noted by Kyandjuntseva\textsuperscript{2, 3} made possible the filming of these phenomena at high speed. Consecutive films (fig. 3) illustrate the development of the ventricular systole and the state of the myocardium at the apex in contact with necrotic tissue for a certain time. The bulging of the apex grows noticeably during the progressive development of the systole.

The area of the relaxed part of the myocardium is usually greater than the area of the necrotic strip. During the ventricular systole a small band of myocardial tissue lying close to the necrotic strip is also in a relaxed state.

No one can suppose that this section of the myocardium loses its property of excitation. Experiments with necrotic tissue placed on a section of the myocardium\textsuperscript{4, 5} and on a spontaneously contracting trabecula of the atria,\textsuperscript{7} which function as linear conductors of excitation, showed that the action of necrotic tissue blocks excitation.

The loss of excitation and its blockage by necrotic tissue was particularly noticeable when a strip of necrotic tissue was placed
on the bare atrioventricular funnel. We call a section of the myocardium which has, under the action of necrotic tissue, lost its property of excitation a near necrotic zone (NNZ). The tissue of an NNZ does not take part in the general bioelectric activity of the heart. This fact may be proved by the appearance of a monophasic electrocardiogram of the ventricle in contact with necrotic tissue. Moreover, the action of necrotic tissue is accompanied by changes in the diastolic potential of the electrocardiogram. The direction of this change (change of the demarcation potential) points to a marked depolarization of the myocardial sector acted upon by the necrotic tissue. The change of the diastolic level of the electrogram is approximately equal to half the amplitude of the monophasic electrogram. In certain cases the changes equaled two thirds of the amplitude of the monophasic electrogram but they were never greater.

These experiments suggest that necrotic tissue not only depolarizes the neighboring myocardium but plays an active role in making it negative. Probably the depolarizing action of necrotic tissue is the cause for the loss of excitation and contraction of the myocardial elements forming the NNZ. The functional paralysis of the myocardium in the NNZ is not permanent.

As we have mentioned, the electrograms lose their monophasic quality and become normal shortly after the removal of the necrotic tissue. The no-systole state of the NNZ also disappears, while its excitability and contractility become normal. The time for this is, however, far greater than the time of action of the necrotic tissue. It depends largely on the state of the heart and its muscles. Exhaustion, hypoxia, and poor blood supply lengthen this process. This restoration follows a normal and consecutive sequence.

Figure 4 does not reflect all the possible dynamic variations of the restoration of the cardiac electrogram. It does, however, show the general scheme of this process and its most important features. A gradual deflation in the plateau of the monophasic electrogram reflects the appearance of an action potential of the tissue of the NNZ.

At the beginning the action potential has a small amplitude and is very short. Later the amplitude and duration of the action potential of the NNZ increase and then become

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**Figure 2**

The alterations of a mechanogram (above) and electrogram (below) of a frog's heart due to the application to it of necrotic tissue. The conditions of electrographic recording are schematically depicted above the records: I, normal electrogram; II, after application of necrotic tissue to the apex; III, after its removal. After the application of necrotic tissue to the heart's apex, the electrogram becomes monophasic while the contraction amplitude becomes smaller. These changes disappear after the necrotic tissue has been removed.
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Figure 3

Functional changes in the myocardium under the effect of necrotic tissue. Photographs of a frog's active heart, taken at the speed of 8 to 10 frames per second. a, diastole of the intact heart; b, systole of the intact heart; c, systole of the heart during application of necrotic tissue; d, e, f, g, h, and i show that during systole the region in contact with necrotic tissue is relaxed. It bulges slightly toward the end of systole.

normal. At this time the RS-T segment reaches an isopotential level while the T wave regains its normal direction and form. The contractile properties of the myocardial NNZ are regained steadily and follow a definite law. After the necrotic tissue has been removed the myocardium does not contract and takes no part in several systoles. After a short period almost unnoticeable contractions may be seen in this area. These contractions begin with each new ventricular systole. These short contractions of the myocardial NNZ are followed by periods of relaxation, each of which takes place while the ventricular systole continues.

After some time the systole of the altered myocardial sector gradually becomes longer and longer until it becomes the same as the whole ventricular systole.

This recovery of the properties of the myocardial NNZ after the removal of necrotic tissue probably takes place by normalization processes in the ionic metabolism. The basis for such a statement will be shown in another part of this paper.
Inhibition of the Action of Necrotic Tissue on the Intact Myocardium

We began studying the internal mechanism of action of necrotic tissue on the myocardium by finding out if the effect of this action is due to the nature or type of injured tissue and to the method of necrosis.

Kyandjuntseva tested the action of necrotic tissues on the myocardium with necrotic tissues from muscles, lungs, liver, stomach, skin, and blood clots taken from the living organism and from the ventricle of another animal.

All these tissues in which necrosis was produced by different methods, such as burning, ligatures of alimentary organs, etc., had the same action on the myocardium. They all paralyzed its functional properties with the gradual development of the NNZ and monophasic electrogram.

If the monophasic extent in the electrogram is used as an indicator of the depth of depression of the myocardial NNZ, then the area of this zone and the extent of functional depression of its tissue structures are seen to depend upon the extent of injury done to the tissue.

The skeletal muscle of a frog after 45 minutes of isolation may, when put in contact with the myocardium, cause typical monophasic traits of the electrogram, such as changes in the RS-T segment.

The similar action of necrotic tissues taken from different organs shows that they all have properties causing a functional depression of the myocardium. Control tests and experimental data show that this factor or property of necrotic tissues does not lie in the action of some substance such as "necrosive" (Menkin and other authors).

We did not forget about the very much higher concentration of intracellular potassium compared to the potassium in the tissue fluid and plasma, nor about the possibility of its leaking out in great amounts after tissue injury. Accordingly, we compared the action of necrotic tissue and potassium chloride on the myocardium and found them to be the same. Most important was the same depolarizing action of necrotic tissue and potassium chloride. Golovshchikov and Keder-Stepanova similarly found the potassium ratio between the necrotic tissue and the myocardium to be important in the development of the necrotic tissue action.

Salmanovich showed in frog and rabbit hearts that isolated muscle tissue depleted of most of its potassium also loses its property of paralyzing the myocardium. He also found that other ions and active substances in the necrotic tissue do not play any role in the mechanism of paralyzing the myocardium.

The potassium ions do not, however, fully explain the effect of necrotic tissue on the myocardium. The quick negative effect of necrotic tissue differs from the action of a 0.5 per cent solution of potassium chloride. In the action of necrotic tissue the active movement of potassium is an important factor.

We also tested certain pharmacologic substances and physiologic effects that stop or limit parabiotic action of necrotic tissue on the myocardium. As was shown by Golovshchikov and Keder-Stepanova atropine counteracts the effect of necrotic tissue on the myocardium, sharply limiting the area of the NNZ. We think that the effect of atropine is due to the stabilization of polarity of the cellular membranes. Atropine, also, slightly hyperpolarizes the membrane.

The paralytic action of necrotic tissue is limited to a great extent by the excitation of the vagus nerves. It was found that the vagus nerves when inhibiting the heart cause a hyperpolarization not only in the physiologically normal heart tissues, but also in the NNZ. Not only that: in the depolarized tissues of the NNZ a slightly positive change in the resting potential may be noticed when compared to the growth of the demarcation potential in healthy tissues.

At the same time during vagus nerve action the NNZ grows smaller, while the heart electrogram and electrocardiogram become more normal.

Yastrebtseva et al. showed that the irritation of the sympathetic nerves, on the con-
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Contrary, increases the effect of necrotic tissue, perhaps because of the quicker and stronger heart beats.

Finally one must note that the depressing effect of necrotic tissue is probably universal; functional depression from necrotic tissue action takes place not only in heart tissues but equally in nerve structures. Yastrebtsova and Udelnov14 pointed out that necrotic tissue in the frog brain (medulla) makes all reflex activity of this part of the brain impossible. Necrotic tissue on the vagosympathetic trunk blocks conduction.

Role of Necrotic Tissue on the Origin of Electrocardiographic Alterations Characterizing Focal Disease of the Myocardium (Infarction)

The foregoing experimental results suggest that a necrotic focus in the myocardium will always have a paralyzing action on the myocardial tissue adjoining it. This action should be accompanied by pathophysiological alterations very much like those in the NNZ of the myocardium under the influence of necrotic tissue brought in from a foreign body. Accordingly we may assume that the method described in parts I and II may serve as an experimental model of certain pathophysiological conditions that take place in the heart with the appearance of necrosis.

Since the electrocardiographic effect of necrotic tissue on the myocardium is much the same as the electrocardiographic alterations in cases of focal disease, we may again assume that this model permits reproductions of the electrocardiographic components of myocardial disease and thus helps solve the problem of its genesis.

Since necrotic tissue causes reversible alterations of the myocardial functional properties, we had in mind the reproduction of those electrocardiographic alterations that disappear during the recovery period when the normal electrocardiogram appears.

The problems formulated earlier were studied in experiments on frogs, cats, and rabbits in acute and chronic conditions.

The results of acute experiments2 are illustrated by figures 5, 6, and 7.

The application of necrotic tissue is accompanied by the displacement of segment RS-T, alterations of T, and the first part of the ventricular complex (figs. 5 and 6). The degree of these alterations depends on the size of the necrotic tissue. By changing the size of the necrotic tissue we aimed at fixing the measurements of the smallest NNZ, which may be reflected in the electrocardiogram. We also assumed that the variation in size of necrotic tissue and the corresponding alterations of the electrocardiogram would make it possible to reproduce more or less accurately the electrocardiographic features corresponding to the different sizes of focal disease.

Electrocardiographic characteristics of focal disease of different localization were reproduced in the same way. Figure 6 illustrates alterations of the electrocardiogram of a cat, depending on the place of application of the necrotic tissue. The three standard leads are used. Discordant displacements of the RS-T segment are noticeable. When the necrotic tissue was placed on the left side surface closer to the ativoventricular boundary (the base of the left ventricle), the following changes occurred: an upward displacement of the RS-T segment while RS-T_{2,3} move down, waves T_{2} and T_{3} are negative, and there are changes in the QRS complex.

The application of necrotic tissue to the apex of the ventricle causes contrary displacements of RS in leads I, II, and III. Sometimes a Q wave is markedly pronounced.

The application of necrotic tissue to the right ventricle closer to the base is accompanied by a downward displacement of interval RS-T while intervals RS-T_{2} and RS-T_{3} move upward. In certain cases a Q wave is very deep and pronounced, while waves S_{2} and S_{3} are larger than usual.

Figure 7 (tracing 2) illustrates the electrocardiographic alteration of a cat (with standard leads) after the necrotic tissue has been removed. Columns C, D, and E show the successive stages of recovery of the electrocardiogram after the removal of necrotic tissue. Recovery goes through the stage of a marked coronary T wave (C), the RS-T
Figure 5
The dependence of the electrographic alterations of a frog's heart on the size of the necrotic tissue applied to the normal myocardium. First column. The electrogram is normal. Second column. Electrocardiographic alterations when a piece of necrotic tissue measuring 2 mm. is applied to the apex of the ventricle. Third column. The same, when the necrotic tissue measures 4 mm. Fourth column. The same, when the necrotic tissue measures 8 mm.

Figure 6
Electrocardiographic alterations of a cat depending upon the localization of necrotic tissue on the myocardium. First column. Normal electrogram. Second column. Electrocardiogram after the application of necrotic tissue to the left-hand base of the ventricle. Third column. After the application of necrotic tissue to the apex. Fourth column. Application to the base of the right ventricle. The experiment was conducted on the same heart.
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Figure 7
Electrocardiographic alterations of a cat when necrotic tissue is applied and after its removal. First column. Normal electrocardiogram. Second column. Electrocardiogram recorded when necrotic tissue was applied to the apex of the heart. Third column. Electrocardiogram recorded 2 minutes after the injured tissue was removed from the myocardium. Fourth column. The same after 3 minutes. Fifth column. The same after 4 or 5 minutes.

Gradually returns to its normal level (D), and at last the negative T wave disappears (E).

Experiments have thus shown that alterations of the electrocardiogram and their discordance, their dependence on the localization and size of the necrotic tissue in the area of the NNZ, are the same as those alterations that are characteristic of focal pathology in man.

The alterations in the electrocardiogram, noticeable after the removal of necrotic tissue, remind one to a certain point of the dynamic changes in the RS-T interval and T wave during the recovery period.

Alteration of Impulse Conduction in the Atrioventricular Area of the Heart under the Effect of Necrotic Tissue

Cases of depressed atrioventricular conduction accompanying necrotic foci has been described by Pardee, 1942; Katz, 1946; and Nezlin, 1951. In certain cases deep changes in atrioventricular conduction were not accompanied by pathologic symptoms of the conducting systems. This was shown by post-mortem study of the heart.

The explanation of this fact usually is based on the assumption that together with the blocking of the blood stream in the damaged area ischemia of the conducting system occurs. Not only is this explanation completely a priori but it is hard to use in cases when the depressed atrioventricular conduction is combined with atrial infarction, and also with such depression of atrioventricular conduction for 10 or more days.

We thought it possible that such changes in atrioventricular conduction may depend on the influence of the necrotic area and involvement of the conducting system in the NNZ.

To check this assumption the necrotic tissue was placed on the bare conducting funnel of a frog spread out in the form of a plate, or on the surface of a cat’s heart, lying next to the atrioventricular bundle. In this way a great variety of reversible forms of atrioventricular conduction defects can be seen, beginning with the syndrome of Wolff, Parkinson, and White, on through Wenkebach’s periods and finishing in complete block.8,15 The results of these experiments done on the spread-Gramenitsky hearts of frogs are given
in figure 8. The alteration of atrioventricular conduction when necrotic tissue is placed on the epicardial surface near the atrioventricular bundle, is illustrated in figure 9 A and B.

We may conclude that reversible disturbances of atrioventricular conduction that take place during myocardial infarction of a definite locality may be due to the effect of the necrotic tissue in the damaged area and to the transformation of the conducting system into the NNZ.

Analysis of the QRS Complex Alterations, Characteristic of Myocardial Infarction

Our experimental model was further studied by Keder-Stepanova and Udelnov in long-term animal experiments in which the electrocardiographic alterations after the placing of necrotic tissue on the intact myocardium were compared with blocking of the coronary arteries.

Fifty-eight rabbits were used. In 33 of them the alterations of the electrocardiogram were studied when necrotic tissue was applied to the intact ventricular myocardium under completely sterile conditions. Four rabbits composed the control group.

The comparison of the electrocardiographic alterations during the application of necrotic tissue to the base, middle, and top of the left ventricular frontal wall (12 rabbits) to the same during the blocking off of the anterior descending artery (9 rabbits) showed the following. In four, myocardial infarction measuring 1.5 cm.² was found in the area of the base and middle of the frontal wall. In the five other cases myocardial infarction of approximately the same size covered the middle and anterior surface of the left ven-

Figure 8
Consecutive stages of development of atrioventricular conduction disturbance resulting from the action of necrotic tissue on the conducting system. The arrow shows the moment of application of necrotic tissue. (Explanation in text.)

Figure 9
Alterations of atrioventricular conduction during the application of necrotic tissue to the epicardial surface of a cat's heart. A. The electrogram of a 6-day-old kitten; lead II. 1. Normal; intervals: P-P, 0.62 second; P-R, 0.1 second, complex QRS, 0.017 second. 2. After the application of necrotic tissue to the region close to the coronary sinus (see schema). P-P, 0.65 second; P-R, 0.13 second; complex QRS, 0.03 second. 3. After the removal of necrotic tissue; P-P 0.63 second; P-R, 0.1 second; complex QRS, 0.017 second; B. Electrogram of a cat; lead II. 1. Normal electrogram; P-P, 0.4 second; P-R, 0.075 second; complex QRS, 0.04 second. 2. After the application of necrotic tissue to the lateral surface of the right ventricular base (see schema); P-P, 0.44 second; P-P, 0.04 second; complex QRS, 0.04 second. 3. After the removal of necrotic tissue; P-R, 0.75 second.

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Figure 10

From the top to bottom in each tracing I, II, and III are standard leads, and CR₁, CR₂, and CR₃ are chest leads. a. Rabbit electrocardiogram before necrosis and operation. b. Rabbit electrocardiogram immediately after necrotic tissue has been applied to the frontal wall of the left ventricle close to the interventricular septum, and the shutting off of the pericardium. c. Electrocardiogram of the same rabbit 18 hours after operation. d. Electrocardiogram of another rabbit immediately after ligation of the anterior descending artery and the shutting off of the pericardium. e. Electrocardiogram of the same rabbit 8 hours later. f. Electrocardiogram of the same rabbit 2 days later. The schema shows where the zone of damage is situated.

developed, and when this rabbit was dissected 6 months later it was found that the myocardium was covered with cicatrices in the base and center of the left ventricular anterior wall. The electrocardiograms registered after the artery had been blocked are shown in the last three columns. In accordance with the localization and size of the infarcted area in this rabbit, a piece of necrotic tissue was placed on the heart of another rabbit (approximately 1.5 cm.² and 1 mm. thick on the anterior wall of the left ventricle in the base and center region—closer to the interventricular septum).

The electrocardiogram of this rabbit before the application of necrotic tissue and after its application are shown in columns a, b, and c. The figure shows that, immediately after
necrotic tissue is applied and the pericardium is shut off, an alteration occurs in the QRST complex characteristic of an infarct in this location.

According to the localization of the "infarct" the chest leads are the most altered in this electrocardiogram, since they most clearly reflect damage in such locations. There also are alterations in the electrocardiograms of the standard leads.

In the other 26 rabbits we compared the alterations of the electrocardiogram that occurred when necrotic tissue was applied to the posterior lateral and posterior walls of the left ventricle, and when ligatures were placed on the corresponding arteries. (Ligatures were placed on different ramifications of the circumflex coronary artery and the posterior descending.)

The alterations of the coronary were very much alike in both groups. At the same time there was a difference in the time needed for the development of alterations and for the normalization of the electrocardiogram. The electrocardiographic alterations after ligatures have been placed on arteries take place much later than the electrocardiographic effect of necrotic tissue. After the ligation of the circumflex branch of the left coronary artery (electrocardiograms were recorded every 30 minutes) it was found that only during the third hour after the operation sharp alterations of the electrocardiogram did appear. These differences in the time needed for the development of alterations are of great interest.

It appears that until necrosis develops the electrocardiogram shows no radical changes in the QRST complex, characteristic of myocardial infarction, even if the artery is occluded. Probably sudden and deep ischemia does not have an immediate effect on the alterations of the QRST complex. In other words, it does not cause these alterations independently from necrosis. The changes develop only after the tissue becomes necrotic. Together with this, electrocardiographic recovery appears earlier when under the effect of necrotic tissue than when the arteries are blocked by ligatures.

One may assume that the cause of this is the intensive circulation of cardiac lymph in the pericardial cavity, which relatively quickly begins to wash away the potassium of the necrotic tissue. However, the speedier recovery of the electrocardiogram in the case of necrotic tissue effect cannot serve as a basis for doubting the high activity of necrotic tissue as a factor changing the conditions of formation of electrocardiographic alterations.

Conclusion

This paper gives experimental data permitting us to suppose that electrocardiographic signs of myocardial infarction beginning at a definite stage of the disease, may be due to the paralyzing effect of necrotic focus on the surrounding myocardium.

Experimental modeling of certain stages of development of myocardial focal damage makes it possible to reproduce all the alterations of the electrocardiogram characteristic of myocardial infarction in the acute period as well as during the renovation periods (this is done by placing a piece of necrotic tissue on the intact myocardium).

The discordant alterations in the electrocardiogram are also reproduced together with displacements in the QRST complex (chest leads), which characterize a definite localization of the myocardial infarction. At the same time, the experimental model copying the effect of a necrotic focus on the intact myocardium allowed us to find important supplementary pathophysiologic characteristics of the heart in cases of myocardial damages.

It was found that the necrotic focus due to ischemia or other factors causes by its effect functional paralysis of that myocardial region, which surrounds the contact of necrotic tissue and is much larger in size.

This myocardial region, which we have called the near-necrotic zone (NNZ), does not take part in the general systole of the heart and is excluded from the summary bioelectric activity of the myocardium.
Depending upon the localization of the focus of damage and, accordingly, depending upon what structures of the heart are included in the NNZ, either the contracting myocardium or main structures of the conducting system are reversibly paralyzed.

The consequence of such different localization of the focus of damage may be depression of the contracting functions of the heart or the reversible depression of excitation and conduction. According to this hypothesis, different reversible depressions of atrioventricular conduction were reproduced with the help of the mentioned experimental model. These reversible changes are typical of myocardial infarction with a definite localization.

The experiments of different scientists, cited in this paper, revealed that the tissue of the NNZ is depolarized and, accordingly, is electro-negative in respect to the normal myocardium. The depolarization of structures is the direct cause of functional depression of the myocardium in the NNZ.

Experiments have shown that necrotic tissue causes a depolarizing and depressing effect on the surrounding structures of healthy myocardial tissue by means of potassium ions. After injury the cellular structures cannot retain their intercellular potassium. Potassium, flowing out after the cell’s injury, has a depolarizing effect on the myocardium of the NNZ.

The presence of this depolarized NNZ, excluded from all bioelectric activity in one sphere of leads, is the condition responsible for "unipolar" leads and the monophasic electrogram of the heart.

The region of necrosis and the NNZ must have a certain minimum size so as to ensure the "indifference" of the registering electrode placed on it.

This condition also determines the monophasic symptoms (displacement of segment RS-T) and the alterations in the ventricular complex in the electrocardiogram recorded in standard and chest leads.

Experiments showed that nervous effects, the conditions of blood flow, and certain other factors may change the size and length of the NNZ.

The depth of functional alterations of structures in the NNZ may also be changed.

However, wide compensating possibilities of the organism such as nervotrophic effects and adaptive changes in the coronary blood flow, as was shown in our experiments on mammals, may bring about changes but cannot exclude the appearance of the NNZ as a consequence of the direct influence of necrotic tissue on the myocardium.

The effect of necrotic tissue may, up to a point, be limited by the action of atropine.

References


The following corollaries are the result of my observations of inflammatory diseases of the chest, studied under the sign of morbid resonance:

1. The duller the sound, and the more nearly approaching that of a fleshy limb stricken, the more severe is the disease.

2. The more extensive the space over which the morbid sound is perceived, the more certain is the danger from the disease.—From *On Percussion of the Chest*. Published in 1761. Translated by John Forbes, M.D. In: *Classics of Medicine and Surgery*. New York, Dover Publications, Inc., 1959, p. 130.
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M. G. UDELNOV

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