ABSTRACT OF THE MAIN THESIS

An Experimental Study on Myocardial Infarction of the Dog - Especially an Investigation from View of Clinico Electrocardiography

Wataru Yoshiike

Dep. of Veterinary Surgery, Azabu Vet. College

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The aim of this study has thoroughly investigated myocardial infarction on the dog -- mainly from view of clinical electrocardiography.

It was necessary that I first should examine the state of distribution of coronary arteries and veins, and that of their blood flow to observe myocardial infarction accurately on the dog. From the reason of this, I tried to extract the heart of healthy dog and to inject Polyester resin into the coronary arteries and veins and to make the model of the coronary blood vessels.

From the view experimented above, I found out the following result.

The right coronary artery arised from the right Valsalva sinus of the aorta and sent the right atrial branches directly and then divaricated the dorsal branches. Moreover, four to nine right ventricular branches were emerged from the right coronary artery and supplied the blood to the right ventricular free wall. The dorsal branches changed into the right branches, from which a large number of right ventricular branches emerged, and then extended in the direction of the apex of the right ventricle. The left coronary artery arised from the left Valsalva sinus, from which septal branches emerged. What is more, the cranial descending branches of the circumflex which the left coronary artery terminated in ran round the base of the left ventricle, branching the left atrial branch and several left ventricular branches, and became the dorsal interventricular branch. It was divided into several left ventricular branches to become dorsal and left marginal branches. From this fact, several left ventricular branches were divided and became dorsal and left marginal branches. The left atrial branches were distributed in the left atrium, while the left ventricular branches were distributed on the free wall of the left ventricle. The dorsal interventricular branches and the left marginal branches were comparatively large and sometimes anastomed.

The cranial descending branch which branches several right and left ventricular branches, descended toward the middle of the ventricle and the apex of the ventricle, and became ventral interventricular branches.

From these vessels, septal and left ventricular branches were divaricated. The right and left ventricular branches of the cranial descending branch were distributed to the anterior wall of the ventricle. Many anastomoses of the cranial descending branches and the left marginal branches were also recognized.

After observing the distribution of these coronary vessels, I directly tried to inject $B_a\ O_6\ Fe_2\ O_3$ (Ferrite) into the cardiac myocardium to create myocardial injury artificially. And I observed, in process of time, the experimental process on electrocardiogram of body surface, using various leads.

In consequence, the following was proved. Electrocardiogram of these leads, that is, is lead A-B$_{II}$ of the A-B leads, leads II and III of the standard limb leads, leads aV_R and aV_L of the augmented unipolar limb leads, leads C_2, C_4 and C_5 of the precordial leads and leads M of the supplementary precordial leads, etc.

These changes in the ST segment were not so clear but the change from 0.2 to 0.3 milivolts was only observed. In fact, this damage of

experimental myocardial infarction was very limited. Therefore, I think that Ferrite acted on non-inflamatory against the tissue of myocardium and that a limited injury was comparatively unimportant from the histologic point of view.

On the basis of the experimental result, I tried the following.

I produced the ~~non-thoracotomy~~ occlusive infarction of the coronary artery artificially without thoracotomy and decided to examine it, from view of electrocardiography. From the reason of this, non-corrosive metal (in this case, ball bearing) was injected into the coronary artery. After it was finished the occulusive infarction, the ischemic myocardial infarction revealed there was observed by electrocardiogram of body surface.

As a result, the electric potential of the myocardial infarction -- the change in the ST segment -- was shown in the case of standard limb leads rather clearly than that of A-B lead, and in the case of the precordial leads, the change was shown up the lead of left ventricular side more clearly than that of the right ventricular side. In the supplementary precordial leads, the change of the ST segment, in the left ventricular apex of leads M_3 and M_4 was quite apparent, but the region of the occlusive infarction was limited, so the change in the electric potential on the electrocardiogram of body surface

were not shown remarkably. In the result of these experiments, I found it necessary to observe this change of the electric potential more clearly, so I directly tried to reveal the myocardial infarction by tying up the coronary artery.

It was necessary that the study of methods should open the chest and expose the heart and tie up the coronary artery. When carrying out an operation of this sort, I previously had to examine the effect which was caused by opening the chest and closing the chest, in process of time after an operation. As the preliminary experiments, I operated on opening the chest without tying up the coronary artery and also increased the investigation on electrocardiogram. As a result, the effect in each wave which was caused by this surgical attack on the dog was not shown a remarkable change. And then the chest of the dog was opened and its heart was exposed. The circumflex and cranial descending branches of the left and right coronary artery were ligated. Moreover, myocardial infarction was made artificially and the change in the electric potential was observed through electrocardiography of body surface.

When the right coronary artery was tied up, a clear elevation or depression of the ST segment was shown in the following leads: Lead A-B_{III} of the A-B leads, lead A-B aV_L of the A-B augmented unipolar

leads, lead II of the standard limb leads, leads aVL and aVF of the augmented unipolar limb leads, leads C_3, C_6 or C_I of the precordial leads, and leads M_3, M_4 and M_2 of the supplementary precordial leads.

Of these leads, the ones that showed the most conspicuous change ST segment in the electric potential of myocardial infarction of the right ventricular free wall were lead A-B_{II} of the A-B leads, leads II of the standard limb leads and lead C_6 of the precordial leads.

When the circumflex branch of the left coronary artery was ligated, an elevation or depression of the ST segment which indicated an ischaemic myocardial infarction with the epicardial electrocardiogram was clearly revealed.

In the electrocardiography of body surface, there were leads A-B_I and A-B_{II} of the A-B leads, leads A-B aVL of the augmented unipolar A-B leads and leads C_3, C_4 and C_5 of the precordial leads, etc. The electric potential in the ST segment indicating the myocardial infarction of the free wall of the left ventricle was observed. In the case of the standard limb leads and the augmented unipolar limb leads, the change in the ST segment was considerably small. In all case of leads of the supplementary precordial leads, the change in the ST segment was not clear.

When the cranial descending branch of the left coronary artery was bound, the electrocardiography of epicardium showed a marked elevation or depression of the ST segment, indicating the occurrance of an ischaemic myocardial infarction in the area of blood vessels below the spot of ligation. The electrocardiography of body surface recognized an apparent change in the ST segment in lead A-B_I of the A-B leads, lead A-B aV_R of the augmented unipolar A-B leads and lead C_5 of the precordial leads. However, in the case of the standard limb leads, the augmented unipolar limb leads and the supplementary precordial leads, the change in the ST segment was not clear.

The change in the electric potential on the electrocardiogram of body surface by means of the myocardial infarction became most conspicuously around the third day of the infarction, but, with the passage of time, the dog tended to recuperate gradually. This is guessed that, considering the correlative study of the pathological anatomy, the patholohistology of the infarctional region and angioarteriography of the coronary artery, the myocardium of the infarctional part which was rapidly displosed by means of ligation of the coronary artery plunged from the state of ischaemia into that of necrosis and was finally replaced by either granulated tissues or connective tissue. And also, the coronary arteries surrounding the infarctional part

developed a large number of collateral blood vessels, which played an important role in the replacing the new tissues instead of the infarctional ones.

From the result of these experiments, it has been nearly proved that a clinical diagnosis of myocardial infarction on the dog was possible to observe the potential change of the ST segment on the electrocardiogram of body surface. It was also ascertained that the clearest change in the electric potential was observed through unipolar leads in face of infarction region and bipolar lead that had electric field corresponding to infarction.