Dog hearts were taken without loss of blood under 3 different conditions: (1) normally beating hearts, (2) hearts that were arrested by potassium chloride for varying lengths of time and (3) potassium chloride-arrested hearts that were reperfused with blood after varying lengths of time. In normal hearts counts of open capillaries containing erythrocytes showed a uniform distribution throughout the right myocardium and a gradient decreasing from the epicardium toward the endocardium in the left myocardium. Potassium chloride-arrested hearts showed a reverse gradient in the left myocardium. In normal hearts, erythrocytes are oriented in capillaries so that their flat sides lie against the nearest myocardial cell. In the arrested hearts, one sees few or no erythrocytes with this orientation. The erythrocytes in the arrested hearts are swollen, rounded, or packed in clumps in the capillaries. Arrested hearts exhibit a weakening of the connective tissue elements of the myocardium, evident upon fixation or on perfusion of the myocardium with blood. The weakening increases as the length of myocardial arrest increases.

The use in recent years of potassium chloride perfusion of the coronary circulation to arrest human hearts in order to yield dry and static fields for surgery has led to the comparison of the metabolism of beating and arrested hearts of dogs. The arrested-perfused heart uses only about 25 per cent of the oxygen consumption of the beating heart. Severe disturbances in the carbohydrate metabolism of the arrested heart have also been observed. Arrest of the coronary circulation for varying lengths of time followed by reperfusion of the coronary circulation, results in a gradual diminution in myocardial oxygen consumption. Temporary myocardial ischemia and anoxia also lead to negative myocardial balances of pyruvate and lactate which persist even after reperfusion of the heart. The vascular supply of the arrested heart has been previously investigated by Wearn and his co-workers. They found that there was a definite rearrangement of capillaries in the arrested heart as compared to the beating heart, and concluded that the Thbesian vessels play an important role in the circulation of blood through the arrested heart.

This present report deals with a morphologic study of the functional capillary vascular beds of hearts obtained under the following 3 conditions: (1) the heart still beating, (2) the heart arrested for varying periods of time, and (3) the heart arrested for varying periods of time and then reperfused.

The results show that the functional capillary bed of beating hearts differs significantly from the other 2 states. Observations have been made also upon a unique orientation of the circulating erythrocytes to the myocardial tissues as well as upon the fragility of the tissues in the arrested hearts, especially demonstrable in those that have been reperfused.

**METHODS**

The experiments were performed on a total of 38 mongrel dogs. Of these, 16 experiments had to be discarded because the coronary sinus blood contained blood from sources other than the perfusate.
entering the aortic sinus (sinus of Valsalva). This "extracoronary" blood probably arises from the ventricular cavities or other extracardiac sources. When this admixture appears in coronary vein blood in large quantities, calculation of myocardial extraction of oxygen and substrates is impossible. In order to estimate the quantity of admixture and to eliminate it as a source of error, a known amount of dextran was added to the coronary perfusate. Since this material is not utilized by the muscle, therefore, the proportion of dextran in coronary vein blood as compared with that in the arterial perfusate permits a quantitative estimation of that quantity of coronary blood which did not originate in the perfusate. Only those experiments were selected in which the extraction of dextran was above 80 per cent.

The animals were anesthetized with intravenous sodium pentobarbital (30 mg/Kg. of body weight). Heparin (50 mg.) was injected intravenously to prevent clotting. A no. 7 Birdseye catheter was then inserted into the coronary sinus under fluoroscopic control through an external jugular vein. A special triple lumen catheter equipped with 2 inflatable balloons was then inserted into the left internal jugular and the coronary sinus for approximately 3 to 5 min., 3 or 4 blood samples from the inflow tubing and the coronary sinus for approximately 3 to 5 min., 3 or 4 blood samples from the inflow tube and from the coronary sinus were collected.

Perfusion was then interrupted for periods of time varying from 30 min. to 4 hours. Coronary perfusion was then commenced again and continued for approximately 30 min. During this time, repeated samples of arterial and coronary vein blood were collected. Results of the metabolic studies are reported elsewhere.3

### Histologic Methods

In order to study the essential morphology of the coronary circulation, the technic of Chacko and Reynolds,4 and Reynolds5 was employed. In this technic, the organs are examined histologically with the blood contained in their vessels by a simultaneous clamping of all vascular channels leading into and from an organ. In this way, erythrocytes are trapped in all vessels in which blood is flowing at the moment of clamping. Use of a stain specific for acidophilic materials including hemoglobin permitted the study of arteriole, capillary and venule relationships through the distribution of erythrocytes within the blood vessels as it existed when the tissues were taken. The hearts were removed immediately after perfusion, care being taken to preserve the blood within the vessels. This was done by firmly ligating the coronary sinus, the superior and inferior vena cava, the pulmonary artery and the aorta. The heart then was removed and immediately fixed in a saline solution containing 10 per cent formalin. Sections of the heart were cut sagittally and transversely at 24 μ. Staining by the Crossman method6 was then carried out. This procedure results in brilliant red staining of all erythrocytes in situ. Tissues were counterstained faintly with methylene blue and hematoxylin. Hearts reperfused after circulatory arrest from ½ to 5 hours were also examined with this technic. The sections were compared with those obtained from hearts that had remained in situ for the same lengths of time without reperfusion.

For study, serial sections were obtained, cut sagittally from the endocardium outward. Each slide contained 3 to 6 serial sections, the distance between any slides for one heart being 200 μ for the right ventricle and 400 μ for the left ventricle. Three areas of myocardium were available for study in each section, one from the inner third, one from the middle third and one from the outer third, beneath the epicardium. In addition, one slide of each ventricle was prepared from a cross section of that ventricle. The first group of slides was prepared from hearts whose vessels were clamped off while the heart was still beating. These served as control preparations. In the second group of slides, the experimental hearts were arrested and perfused as shown in table 1.

### Table 1.—Duration of Arrest and Perfusion

<table>
<thead>
<tr>
<th>Heart 1</th>
<th>Arrested</th>
<th>Perfused</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart 6</td>
<td>1 hr. 1 min. 30 sec.</td>
<td>28 min. 30 sec.</td>
<td>1 hr. 30 min.</td>
</tr>
<tr>
<td>Heart 7</td>
<td>1 hr. 30 min.</td>
<td>36 min. 15 sec.</td>
<td>2 hr. 6 min. 15 sec.</td>
</tr>
<tr>
<td>Heart 4</td>
<td>3 hr. 3 min. 20 sec.</td>
<td>20 min. 30 sec.</td>
<td>3 hr. 32 min. 50 sec.</td>
</tr>
<tr>
<td>Heart 5</td>
<td>3 hr. 27 min. 30 sec.</td>
<td>15 min. 15 sec.</td>
<td>3 hr. 38 min. 45 sec.</td>
</tr>
</tbody>
</table>

Histologic Methods. In order to study the essential morphology of the coronary circulation, the technic of Chacko and Reynolds,4 and Reynolds5 was employed. In this technic, the organs are examined histologically with the blood contained in their vessels by a simultaneous clamping of all vascular channels leading into and from an organ. In this way, erythrocytes are trapped in all vessels in which blood is flowing at the moment of clamping. Use of a stain specific for acidophilic materials including hemoglobin permitted the study of arteriole, capillary and venule relationships through the distribution of erythrocytes within the blood vessels as it existed when the tissues were taken. The hearts were removed immediately after perfusion, care being taken to preserve the blood within the vessels. This was done by firmly ligating the coronary sinus, the superior and inferior vena cava, the pulmonary artery and the aorta. The heart then was removed and immediately fixed in a saline solution containing 10 per cent formalin. Sections of the heart were cut sagittally and transversely at 24 μ. Staining by the Crossman method6 was then carried out. This procedure results in brilliant red staining of all erythrocytes in situ. Tissues were counterstained faintly with methylene blue and hematoxylin. Hearts reperfused after circulatory arrest from ½ to 5 hours were also examined with this technic. The sections were compared with those obtained from hearts that had remained in situ for the same lengths of time without reperfusion.

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A third group of slides represents a second set of control tissues. These hearts were arrested for lengths of time which compare with the total time of arrest and perfusion in the experimental hearts. In addition, hearts were obtained for study at immediately and 1/2 hour after arrest.

RESULTS AND DISCUSSION

The results of the detailed study of all available material is summarized in a very general way in table 2. Examination of all tissues was made at low (100) power and high (430) power and, where necessary, under oil immersion at 970 power. Each section was examined for its general characteristics to determine its typical features. This included the extent to which there was separation of the muscle bundles, capillary size, incidence of arteries, arterioles, venules and veins, areas of hemorrhage into the tissue, the arrangement of erythrocytes within the vessels, and the shapes and sizes of erythrocytes. In addition, representative areas of the cross sections of each ventricle were selected for the purpose of counting the capillaries. Such counts were made using an ocular grid which was calibrated with a stage micrometer scale. It was found necessary to use high power for the counting so that, by optical dissection with the fine adjustment on the microscope, no capillary would be counted more than once as can occur with thick sections that permit focusing along some length of each capillary if it was not cut squarely across. Experience showed that if repeated counts were made of the number of capillaries in 20 squares of the grid, good agreement of the counts was obtained. It was necessary to be sure that well-fixed, unseparated areas of myocardium were used. For this reason, adjacent squares on the grid could not always be used for counting.

The number of capillaries counted was far less than that in the study by Wearn who made a thorough study of the entire capillary bed in human, cat, and rabbit myocardial tissues. Checks of counts from comparable areas in different sections in each heart showed that counts of the same order of magnitude were obtained.
The notable morphologic features are briefly described in the following paragraphs.

**Tissue Maceration**

Some maceration of tissues was to be expected because of the mode of fixation: the entire heart was dropped into the fixative and allowed to stand until hardened. Nevertheless, maceration was by no means uniform in the 3 groups of hearts.

The normally beating hearts showed good fixation. The right ventricle of one heart was markedly macerated, although the left ventricle was well fixed. All other sections from beating hearts showed good fixation with only isolated areas of muscle bundle separation. In contrast, the right ventricles of all arrested-only hearts showed considerable to extreme bundle separation. This was seen in left ventricles of the same hearts only at the 3 hours and 4 min. and later period of cardiac arrest. The experimental tissues (arrested-perfused hearts) showed progressive maceration in the right ventricle from slight at 1 hour and 23 min. to extensive at 3 hours and 55 min. and, except for the last (3 hours and 55 min.) heart, the left ventricles generally showed far less maceration.

It appears that prolonged cardiac arrest renders the tissue increasingly susceptible to maceration under the stress of fixation. This is true whether or not the heart is perfused. However, perfusion of the coronary vessels before fixation clearly contributes to more extreme bundle separation. The experimental tissues (arrested-perfused hearts) showed progressive maceration in the right ventricle from slight at 1 hour and 23 min. to extensive at 3 hours and 55 min. and, except for the last (3 hours and 55 min.) heart, the left ventricles generally showed far less maceration.

Capillary Size

Measurements of capillary diameters were made in representative areas of many sections. In the normal hearts, the diameters were from 3 to 5 \( \mu \) but usually 4 \( \mu \). The arrested-only hearts showed many capillaries of comparable size, although some ranged from 6 to 8 \( \mu \). One heart taken at 2 hours and 5 min. showed many dilated capillaries, particularly in the right ventricle.

The arrested-perfused hearts showed general capillary dilatation in hearts studied at 2 hours and 5 min. and later. They tended to be from 6 to 10 \( \mu \) in diameter. In one heart (3 hours and 55 min.) the vessels were enormously engorged and dilated.

Cardiac arrest causes little change in capillary size, unless the heart is subsequently perfused.

**Erythrocyte Arrangement**

One of the striking observations of this study is the unique orientation of the erythrocytes in normal capillaries. The capillaries run along the muscle cells, as Wearn described, and there are about one to a muscle cell. However, we have seen that they arrange themselves in a particularly flattened position, presenting their flat surfaces to the muscle cell (fig. 1). The capillaries normally cannot be round in cross section, therefore, but must be elliptical. This arrangement in the normal hearts we have called trains since, in section, the red cells look like small cars on a track, some abutting on the ones next, without overlapping (fig. 2).

When the capillaries are dilated, some degree of overlapping of erythrocytes is seen, as if the red cells had been tumbling along inside the larger capillary. This arrangement we have called "packs" since it appears that the cells have packed in the vessel in a random manner (fig. 3).

A third arrangement is also seen. In this, the flat surfaces of the red cells lie against each other, so that when many are so arranged they resemble a stack of coins. These are called rouleaux.

Rouleaux and packs are seen in all of the arrested hearts, except in the immediately arrested. However, trains may also be seen to varying degrees in hearts arrested for a short
Fig. 1 Top Left. Section of left ventricle, normally beating heart. Capillaries running along syncytial muscle bundles. Erythrocytes oriented in trains with flat sides apposed to muscles in flattened capillaries. (X 107).

Fig. 2 Bottom Left. Part of section shown in figure 1. No overlapping or random arrangement of erythrocytes. They are arranged in orderly trains. (X 417)

Fig. 3 Top Right. Dilated capillaries in arrested heart, showing erythrocytes packed together, filling the dilated lumens. (X 417)

Fig. 4 Bottom Right. Section of arrested heart (2 hours and 5 min.). Erythrocytes no longer biconcave discs but are swollen into spheroidal shapes. (X 107)

period of time (table 2). Later, none are seen. At times the rouleaux and packs are seen in rather isolated areas of a section.

The observation, that red cells normally slide along a capillary with the flat side presented to the muscle cell, has not been made, because all other previous studies of myocardial capillaries have been undertaken by the use of injection media perfused through the circulatory system. In usual histological preparations of the heart, pieces of myocardium are excised, permitting red cells to escape from the cut capillaries. This arrangement of the red cells would appear to be a most efficient mode of erythrocyte circulation, allowing for the most rapid and effective exchange of oxygen.

Erythrocyte Shapes

The flattened, biconcave shape of the normal erythrocyte is well known. This shape of cell only has been seen in the normally beating heart. It occurred in arrested hearts, but with variable frequency (fig. 4). The arrested-perfused hearts showed primarily normal erythrocytes at 1 hour and 23 min., as did the immediately arrested-only heart. However, after that, fewer were seen. For the most part the erythrocytes became rounded, or if packed in a capillary, square
and angular. This was most marked in the arrested-only hearts, especially after 2 hours and 5 min.

The significance of the large, rounded, and square or angular, red cells is a matter for speculation. It may be related to the fact that the potassium chloride is used to stop the heart, and that the potassium ion moves into the cells, causing them to act like minute osometers, so that they become swollen. If they are not crowded in a vessel they become round. However, if they are close together, they become compressed into distorted shapes.

**Capillary Hemorrhages**

Careful examination was made of all available tissues to look for evidence of capillary rupture and hemorrhage. Only regions free of fixation maceration were examined for hemorrhage. The thickness (24 μ) at which the sections were cut rendered the search difficult. Even so, areas of capillary rupture were identified in both groups of arrested hearts, but not in the normally beating hearts. None was seen in the 1 hour and 23 min. arrested-perfused heart or in the immediately arrested-only heart.

Areas of rupture were characterized, when small, by red cells lying free and at random in otherwise normal-looking regions (fig. 5). No endothelium was ever seen under oil immersion in such situations; however, it usually may be identified in favorable situations when the capillary is intact.

A suggestion may be made concerning the cause of these hemorrhages. They were seen most frequently in sections of the right ventricle. As noted before and shown in table 2, bundle separation is more marked in right, rather than left, ventricles. The tearing of capillaries may be secondary to the changes in the tissue incident to fixation. Even so, hemorrhages were not seen in beating heart no. 3, the right ventricle of which exhibited considerable maceration. Perhaps maceration and long-standing hypoxia both contributed to the result. By far the most extreme hemorrhage was seen in the arrested-perfused heart of greatest duration (3 hours and 55 min.). See fig. 6. This may have resulted from the trauma of pressure upon reperfusion of greatly weakened capillaries.

**Counts of Capillary Density**

The results of making counts of the capillary population in different regions of a heart and in the different hearts is given in table 3. The counts were a measure of the subjective impression gained from a study of the material available (fig. 7).

The base line with which the experimental data must be compared are the counts made
### TABLE 3.—Counts of Minute Vessels per Square Millimeter of Cross Section in Areas of Myocardium

<table>
<thead>
<tr>
<th>Condition of heart</th>
<th>Left ventricle</th>
<th>Right ventricle</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normally beating</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. 1</td>
<td>1600</td>
<td>340</td>
</tr>
<tr>
<td>No. 2</td>
<td>1200</td>
<td>900</td>
</tr>
<tr>
<td>No. 3</td>
<td>1300</td>
<td>200</td>
</tr>
<tr>
<td>Arrested—perfused (hours)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 23/60</td>
<td>1700</td>
<td>500</td>
</tr>
<tr>
<td>2 5/60</td>
<td>700</td>
<td>300</td>
</tr>
<tr>
<td>3 32/60</td>
<td></td>
<td>500</td>
</tr>
<tr>
<td>3 55/60</td>
<td></td>
<td>500</td>
</tr>
<tr>
<td>Arrested only (hours)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0/60</td>
<td>800</td>
<td>340</td>
</tr>
<tr>
<td>30/60</td>
<td>950</td>
<td>650</td>
</tr>
<tr>
<td>1 23/60</td>
<td>1250</td>
<td>1250</td>
</tr>
<tr>
<td>2 5/60</td>
<td>500</td>
<td>800</td>
</tr>
<tr>
<td>3 4/60</td>
<td>800</td>
<td>350</td>
</tr>
<tr>
<td>3 35/60</td>
<td>950</td>
<td>400</td>
</tr>
<tr>
<td>3 55/60</td>
<td>950</td>
<td>500</td>
</tr>
</tbody>
</table>

in the normally beating hearts. Only in one of these hearts was fixation of the cross section of the right ventricle good enough to permit counts to be made with confidence.

The densities of erythrocyte-filled capillaries was not the same throughout the left ventricle, as was found in the right ventricle. The count of erythrocyte-filled capillaries near the epicardium was of the order of 4 to 6 times or more that near the endocardium. The midmyocardial region was also low in erythrocyte-filled capillaries. In the right ventricle the count is about the same throughout, but less than in the left ventricle near the epicardium.

The reason for this situation is not explained. It might be recalled, however, that the principal coronary arteries pass over the surface of the myocardium and penetrate the substance of the muscle. We have observed that wherever there is a relatively large artery, there are more erythrocyte-filled capillaries. This may occur in isolated areas deep in the myocardium. Under low power of inspection, the counts appear to be representative and free of such artefacts as tissue disorganization and the clear presence of an "island" of tissue of filled capillaries. With the exception of islands of capillaries, then, there appears to be a far richer supply of subepicardial capillaries that are filled than there are in the deeper tissues. This applies to the left ventricle and only possibly to the right.

In the second group of hearts (arrested-perfused) the one at 1 hour and 23 min. shows the same situation with respect to the density of capillary population as in the normally beating hearts. Later, at 2 hours and 5 min. there was a rich, subendocardial supply of erythrocyte-filled capillaries, and a lower density near the epicardium. The right ventricles of these hearts also generally exhibited high counts, especially in the epimyocardial and midmyocardial regions. Hemorrhage and tissue disorganization were too extensive to permit counts in the 3 hour and 32 min. and 3 hour and 55 min. hearts (fig. 5), except for a part of the right ventricle of the former.

The arrested-only hearts show a quite different picture. The vascular bed near the epicardium contains fewer erythrocyte-filled capillaries (with one exception) than in the normally beating groups, and the subendocardial area tends to be quite rich in such capillaries (fig. 8). The right ventricular muscle is generally low in such vessels until after 3 hours and 4 min., when the count rises. Large arteries and veins are seen near the subendocardial myocardium in arrested hearts (fig. 9). These may be part of the Thebesian system. Conduction tissue is avascular (fig. 10).

The meaning of these distributions is not, of course, clear in respect to their relation to function or to the conditions of the experiment. Wearn, in his two classic papers on the capillaries and blood vessels of the heart, showed a rich and uniform bed of injectable capillaries throughout the right and left ventricles. His counts are of the order of 1,000 capillaries per 1,000 muscle fibers. However, his values of counts, which were made under rigorously standardized conditions, are about 4,000 to 6,000/mm. in human,
cat and rabbit hearts. The lower figure obtained by us may probably be ascribed to the entirely different conditions under which the counts were made. Wearn carefully perfected his perfusion technic until he obtained as nearly uniform and complete injection with india ink or Berlin blue as was possible. We have counted only those capillaries that contain erythrocytes when the heart is fixed, without losing blood. However, the connec-
tion of the myocardial capillary system with the venae minimae cordis (thebesian veins) permits escape of blood into the chambers of the ventricle, as Wearn has shown. This could explain the gradient of a diminishing number of filled capillaries proceeding from the epicardium to the endocardium in normally beating hearts. The equalization of counts or even a sort of reversal in the left ventricle is difficult to explain, unless it be by the fact that the heart, being stopped in full diastole when fixed, shrinks slightly upon fixation and moves red cells in toward the thebesian veins where they remain. However, Ogniev, Savvin and Savaleva7 have recently stressed the rich blood supply of the ventricular septum, relating it to right, proportionate or left variants in distribution of the coronary supply of the heart. It is possible, therefore, to expect variable densities of filled capillaries according to which part of the coronary blood supply contains blood to the septum under the conditions of fixation.

We note, in conclusion, that when the heart is normally beating the vascular pattern of filled capillaries is generally quite different from that in the arrested hearts. Arrested hearts generally exhibit more fixation maeeration, more hemorrhages, fewer trains of erythrocytes, more packs, more rouleaux and more obviously abnormally shaped erythrocytes than do the normal hearts. The picture one sees in the normal hearts could, therefore, bear a relation to what one might find in the myocardium at the end of strong systole.

Differentiation Between Arterioles, Capillaries and Venules

All that has been said above refers specifically to the capillary vascular system of the ventricles of the heart. In order to make such observations, it was necessary to distinguish between the blood capillaries and the smallest precapillary arterioles and postcapillary venules. After careful and extended study of the material, this became possible.

When, for example, an arteriole in the normally beating heart is found to have a clear muscular coat, one may follow it, in serial sections, to the point of ultimate branching. Several characteristics of the terminal arteriole are evident in these preparations. First, there is always a packing of the erythrocytes in the lumen although the red cells in normal
hearts are always biconcave disks. A second feature of the minute arteriole is the presence of an endothelium with distinguishable, smooth muscle cells running along its wall although these are not always seen uniformly. The third characteristic is that the arteriole definitely gives rise to a number of capillaries that continue in the same direction as the arteriole, and these run parallel to each other, when seen in the sagittal heart sections (fig. 11). The number of capillaries emerging from any one such terminating arteriole is small in any one section. When the capillary level is reached, the erythrocytes lie in the trains described above.

An aggregation of collecting venules looks quite different. The capillaries, with trains of red cells, come from opposite directions along the muscle fibers to join with each other, and then through further merging unite in a postcapillary venule. This vessel rapidly increases in size as it is joined by other similar tributaries which give rise, by virtue of their diameters and the presence of a recognizable muscular wall, to a venule. The postcapillary venules do not show smooth muscle in their walls in the thick (24 μ) sections studied. Finally, in the collecting venules and small veins, the erythrocytes are jumbled and packed upon one another in a random fashion (fig. 12).

The distinguishing features, therefore, of both precapillary and postcapillary vessels in normally beating heart preparations are the size of vessels and the absence of trains of erythrocytes. Further distinctions of these two groups of vessels are the presence of a muscular wall which is visible in the arteri-
oles but not in the venules, and the characteristic arrangement of the distribution of the vessels.

**Summary**

1. Study has been made of the microcirculation in hearts of dogs that were (a) normally beating, (b) arrested by potassium chloride for varying lengths of time, and (c) arrested followed by perfusion for about half an hour. Blood was trapped within the circulation and the red cells stained in order to reveal the functioning pathways at the time the heart was removed.

2. Arrest of the dog heart appears to weaken the connective tissue elements so that the myocardial fasciculae may become separated upon fixation. This is more accentuated with the duration of arrest and more prominent in the right than in the left ventricle.

3. The characteristic erythrocyte arrangement in capillaries of the normally beating heart is that they lie along the muscle bundles, presenting their flattened surfaces toward the muscle, and the capillaries are flattened. The appearance is that of trains of red cells.

4. In arrested hearts, some trains may be seen, but, with time there is more rouleaux formation and swelling of erythrocytes causing them to be rounded, and if packed tightly, to become squarish. This may be due to the osmotic effect of the potassium chloride used to arrest the hearts, or it may be the result of metabolites in the arrested hearts.

5. Capillaries in arrested hearts generally have larger diameters than in the normally beating hearts.

6. Arrested hearts show focal capillary ruptures, the number increasing with time. Arrested-perfused hearts show profound tissue hemorrhage upon reperfusion of the capillary system. Hemorrhages are not seen in normally beating hearts.

7. The density of erythrocyte-filled capillaries in normally beating hearts is greater near the epicardium than in the endocardium, especially in the left ventricle. In arrested hearts, there is deviation from this. The highest counts of filled capillaries is generally near the endocardium. The relation of these distributions to the coronary arteries and the Thebesian veins is discussed.

8. Characteristics of the precapillary arteriole are described, and differentiated from both true capillaries and collecting venules.

9. We conclude that arresting of the heart by potassium chloride for the purpose of yielding a dry field for operative procedures on the heart, greatly alters the distribution of functioning capillaries, the sizes and shapes of erythrocytes and the resistance of the connective tissue elements of the heart to stress. Such stress is that induced by fixation of the heart for histological study, or that resulting from pressure of blood within the vessels upon reperfusion of the coronary system with blood at the conclusion of a period of cardiac arrest. This increased fragility of the tissue is accentuated with time.

**Summario in Interlingua**

1. Esseva studiato le microcirculation in cordes canin que (a) pulsava normalmente, (b) esseva arrestate per medio de chlorum de kalium, e (c) esseva similmente arrestate sed alora perfusionate durante un periodo de circa un medie hora. Sanguine esseva trappate intra le circulation, e le erythrocytos esseva tiiicturate pro revelar le vias in function al tempore del excision del corde.

2. Le arresto del corde canin pare debilitar le elementos de histo conjuctive, de manera il occurre que le fasciculas myocardial se separa post le manovra fixatori. Iste pheno-meno deveni plus marcate con le duration del arresto, e illo es plus prominentin in le ventriculo dextere que in le ventriculo sinistre.

3. Le arrangiamento characteristic del erythrocytos in le capillares del corde que pulsa normalmente es que illos jace al longo del fasces muscular, presentante lor superficies applattate verso le muscolo e dante le impression de un "traino" cellular. In iste situation, le capillares offere un apparentia applattate.

4. In cordes arrestate, certes del supra-describite "trainos" es ancora incontrate, sed
in le curso del tempore, le arrangiamento del erythrocytos es plus frequentemente illo de rouleaux. Il occurre un turgescenția del erythrocytos, rendente los rouleaus. Ei illos es pacate densemente, illos deveni plus o minus quadratic. Iste phenomeno es possibilemente un effecto osmotic del chloruro de kalium usate pro arrestar le corde, o illo es le resultato del metabolitos in le corde arrestate.

5. Le capillares in le corde arrestate ha generalmente plus grande diametros que illos in cordes que pulsa normalmente.

6. Cordes arrestate monstra focal rupturas capillar. Le numero de iste rupturas cresce in le curso del tempore. Le re-perfusion del sistema capillar de cordes arrestate resulta in profunde histo-hemorrhagias. Tales non es incontrate in cordes que pulsa normalmente.

7. Le densitate del capillares plenate de erythrocytos, in le caso de cordes que pulsa normalmente, es plus grande proxime al epicardio que in le endocardio, specialmente in le ventricular sinistre. In cordes arrestate le situation es differente. Le plus alte numeracion de capillares plenate es generalmente proxime al endocardio. Le relation de iste distributiones al arterias coronari e al venas thebesian es discutite.

8. Es describite le characteristicas de arteriolas pre-capillar in differentiation ab ver capillares e ab venulas collectori.

9. Nos conclude que arrestar le corde per medio de chloruro de kalium (como on lo face pro effectuar un campo sic pro objectivos de chirurgia cardiaque) altera grandemente le distribution del capillares functionante, le dimension e le configuration del erythrocytos, e le resistencia del elementos de histo conjuctive al effecto de omne generes de stress. Le stress poter esser le effecto del fixation del corde pro studios histologic o etiam le effecto del pression de sanguine intra le vasos quando le systema coronari es re-perfusionate con sanguine al fin del periodo de arresto cardiaque. Le fragilitate del histo deveni plus marcate con le passage del tempore.

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Functional Capillary Beds in the Beating, KCl-Arrested and KCl-Arrested-Perfused Myocardium of the Dog
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