THE POSTMORTEM RIGOR OF THE MAMMALIAN HEART AND THE INFLUENCE OF AN ANTE-MORTEM STIMULATION OF THE PNEUMOGASTRIC NERVES UPON ITS DEVELOPMENT.[1]

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Part I.

RIGOR MORTIS OF THE HEART.

The main object of the investigations to be reported in this paper was the study of the influence of electrical stimulation of the pneumogastric nerves upon the onset and development of postmortem cardiac rigor in mammals. We shall state later the considerations which led up to the investigation of this problem. However, the study of the changes which cardiac rigor may undergo as a result of stimulation of the vagi, presupposes a definite knowledge of the course of cardiac rigor under so-called normal conditions. Here we were soon confronted with the fact that such knowledge is still far from being complete. In pursuing our main studies we were, therefore, obliged to study carefully at the same time the course of cardiac rigor in animals whose vagi were not stimulated. We have thus collected a number of facts pertaining to the onset and development of rigor of the mammalian heart in normal animals. These observations we intend to embody in the present paper. Before giving the report of these findings we wish to review briefly the experimental data upon which the present knowledge of cardiac rigor is founded.

Historical.—Our knowledge of cardiac rigor is, as stated before, far from being complete. While the studies of rigor of the skeletal

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muscles are very numerous and date back at least a hundred years, rigor mortis of the heart has been studied but slightly, and its systematic investigations are practically of only recent date. The obvious reason for this difference is this: whereas general rigor presents itself after the death of man and beast without any special preparation, the condition of the heart can be observed only after special dissection. Furthermore, autopsies being usually made only for special purposes—pathologic or forensic—were frequently performed some time after death, when the rigor had perhaps passed off. Then, too, the character of rigor which permits of hardly any other interpretation when it is present in skeletal muscle, is capable of being confounded with other states when it occurs in the heart. Rigor mortis is characterized by a contraction; a shortening of all the dimensions of the heart; and by a thickening of its walls. These features, however, may be met with also either in so-called concentric hypertrophy of the heart, or when the heart remains in systole at death. As long as fifty years ago Kussmaul called attention to the possible confusion of rigor with either of these states, the actual existence of which he doubted. He thought concentric hypertrophies as well as cardiac systole after death were possibly nothing but cardiac rigor. Systematic studies of rigor mortis of the heart began only about two decades ago.

Strassmann[2] investigated in 1889 the question of whether in some cases the heart may indeed stop in systole during death. He examined the amount of blood present in the right and left ventricles in a number of dogs killed by potassium cyanide, asphyxia, exsanguination, etc. Examinations were made in some cases immediately after death, and in still others after twenty-four hours. He came to the conclusion that during death the heart stops in diastole, and that the heart muscle gets into a state of rigor which sets in quite early. In a later study,[3] 1896, he compared the dimensions of the hearts of dogs immediately after death, and twenty-four hours later. The measurements were taken by means of drawings and by the insertion of needles into various parts of both ventricles and comparing the distances between them as they presented themselves at each examination. The conclusion drawn from these observations was that during the development of rigor the heart becomes smaller in all dimensions.

Brouardel,[4] 1889, studied the condition of the hearts of animals post-mortem,

[2] Strassmann, *Vierteljahrssch. f. gerichtl. Medizin*, 1889, Ser. 2, li, 300.
[3] Strassmann, *idem.*, 1896, Ser. 3, xii, Suppl., 110.
[4] Brouardel, *Gazette des Hopitaux*, 1889, lxii, 653.

by connecting them with a manometer. The main object of the investigations of Brouardel as well as of Strassman seems to have been to prove from a forensic point of view, that the heart after death passes into a state of rigor—and this less than twenty years ago.

Meirowsky,[5] 1899, reports the experiments of Ludloff on heart rigor which were carried out in the physiological laboratory of L. Hermann. Hearts of cats and rabbits were removed from the body, all blood vessels ligated, except the aorta, into which a cannula was tied, and after filling the ventricle and tubes with a physiological salt solution, they were connected with a manometer containing chloroform. The hearts were kept at various temperatures in solutions of normal saline. The level of the colored chloroform in the manometer was observed every few minutes. According to Meirowsky the beginning of the reduction of volume of the ventricle, as shown by the manometer, which was taken as being exactly proportionate to the onset of rigor, begins in most cases immediately after death, and attains its maximum at the earliest in fifty minutes, and at the latest in two and a half days.

R. F. Fuchs,[6] 1900, removed the heart from the body, tied cannulas in the ventricles through the aorta and pulmonary artery and in the auricles through the corresponding veins. He found that soon after death, solutions injected through the ventricles would pass unhampered through the auricles, which showed that at this period the atrio-ventricular valves were patent. About an hour and a half later no liquid could pass from the ventricles to the auricles. Fuchs assumed that this impassability was due to the development of rigor of the heart. A similar observation was previously made by L. Krehl,[7] 1889. Fuchs seems to have been the first to establish the fact that rigor of the heart appears before the occurrence of general rigor, and he ascribes it to the preceding continuous activity of the heart.

MacWilliam,[8] 1901, was the first to publish a tracing presenting the development of rigor of the left ventricle. The tracing was obtained by tying a cannula into the aortic orifice of the heart of a cat just excised, and connecting the cannula with a mercurial manometer, the oscillations of which were transmitted to a slow-moving drum. The pressure within the ventricle, caused by the contraction of the rigor, may, so MacWilliam states, become as high as 25 to 30 mm. Hg. and may be maintained at this high level for a considerable time. No special data are given for the time of onset, and further development of the rigor after death. Judging by the tracing, it seems that the maximum of rigor is attained in less than an hour and a half, and that at least one-third of the contraction occurs almost immediately after connection with the manometer, since a good part of the ascending limb of the curve is nearly vertical. In this particular case it is not stated how soon after death the ventricle was connected with the kymograph. MacWilliam points out that there is no special parallelism between cardiac and skeletal muscle in regard to the occurrence of rigor, its intensity, etc. Irritability may still persist after the beginning of

[5] Meirowsky, *Pflüger's Archiv*, 1899, lxxviii, 64.
[6] R. F. Fuchs, *Zeitsch. f. Heilk., Abt. f. pathol. Anat.*, 1900, i, 1.
[7] L. Krehl, *Arch. f. Anat. u. Physiol., Physiol. Abt.*, 1889, 253.
[8] MacWilliam, *Jour. of Physiol.*, 1901–1902, xxvii, 336.

cardiac rigor and some slow and rapid oscillations occur in some hearts during the course of rigor.

An extensive investigation on post-mortem changes in the form of the heart was carried out by J. Rothberger,[9] 1903. He examined the hearts of two hundred dogs which were killed in various ways or which had died from various causes. The hearts were removed from the body, the blood content washed out by a physiological salt solution and cannulas tied in the vena cava, left auricle and aorta. The ventricles and connecting tubes were filled with salt solution and connected with a manometer containing water. By means of a float, the changes of level of the water in the manometer were marked on a slow-moving drum. In a number of experiments, hearts taken immediately after death, and twenty-four hours later, were hardened in formalin; four circular sections were made of each, and by drawings, the size and outlines of these sections of the ventricles were compared. Rothberger brought out numerous details of which we shall mention here only the following few statements.

All hearts are, during death, in a state of diastole; some, however, are very relaxed, wide and filled with blood; others are also relaxed, but are small and empty. The latter hearts are slightly tonically contracted. This difference in the behavior of the hearts depends upon the different modes of death. The tracings obtained show various phases. Immediately after filling the heart and connecting it with the kymograph, a rapid rise occurs which is followed sooner or later by a primary fall; then another rise occurs to be followed finally by a secondary fall. The first rise, Rothberger thinks, is a vital phenomenon—a tonic contraction brought on by distension of the heart through the filling of its cavities, and aggravated by the increased irritability of the ventricles, due to the stimulating effect of the solution of sodium chloride. With the gradual loss of irritability the tonus subsides and the primary dilatation takes place. Then the rigor sets in and the secondary rise appears. Finally the resolution of the rigor is the cause of the secondary fall. Rothberger believes that a similar sequence of events takes place in the heart after death, even when it remains under natural connections within the chest cavity. He assumes, with Bayliss and Starling,[10] and with MacWilliam,[11] that at the time of death there remains enough positive intravascular pressure to distend the heart and thus cause, soon after death, a tonic contraction. This contraction, however, under natural conditions, passes over into rigor without an intervening primary dilatation. According to Rothberger, then, the heart very soon after death becomes contracted, but this contraction which develops quite abruptly is a "vital" phenomenon and is brought on by distension. With the loss of irritability of the heart, the "vital" contraction gives way and passes over gradually into the developing rigor. The rigor systole, according to Rothberger, is less strong than the preceding one and appears, Rothberger states, in most cases, two or three hours after death.

This is, in brief, a review of all the systematic investigations made on the subject of cardiac rigor, as far as we know them.

[9] Rothberger, *Pflüger's Archiv*, 1903, xcix, 389.

[10] Bayliss and Starling, *Jour. of Physiol.*, 1894, xvi, 166.

[11] MacWilliam, *loc. cit.*

Neither investigations nor the established facts are very numerous. All writers agree that during death the heart stands still in diastole and all seem to agree that in general the rigor sets in early after death. As to the exact time, Meirowsky (Ludloff) states that the rigor begins in most cases immediately after death, and the tracing of MacWilliam seems to agree with this statement. Fuchs gives the time for the beginning of rigor as the middle of the second hour, and Rothberger speaks of two to three hours. Meirowsky, MacWilliam and Rothberger employed practically the same method —the connection of the cavity of the heart with a manometer. There is one point which comes out with uniformity in all three investigations—namely that soon after the manometer is connected with the cavity of the ventricle, a fairly abrupt rise in the manometer occurs indicating a contraction of the ventricle. Meirowsky and MacWilliam ascribe this to the onset of rigor. Rothberger, who apparently studied the question more extensively, assumes that the contraction occurring immediately after the connection with the manometer is not rigor, but is a vital phenomenon, artificially brought on through the distension of the ventricle by filling it with the solution of sodium chloride. This interpretation of the phenomenon seems to be plausible indeed. But then Rothberger assumes further that such a " vital " contracton takes place also in the normal connections of the heart within the dead body, and this because there is, according to him, at the time of death enough intravascular pressure to cause a contraction similar to that which occurrs when connecting the excised heart with the manometer. This last assumption contains very unusual elements. In the first place, it would distinguish the rigor of the heart strikingly from the rigor of a skeletal muscles, inasmuch as the rigor of the latter is not preceded by any "vital" contractions except under the influence of certain poisons. In the second place, this assumption would practically deny the statement that the heart is in diastole during death. Rothberger has seen the vital contraction setting in immediately after connecting the ventricle with the manometer, which was about fifteen minutes after the death of the animal, because, as Rothberger states, it took so long to make all the necessary preparations. In the normal connections of the heart, such a " vital " tonic con-

traction ought to occur immedately after death, since, according to Rothberger himself, there is enough intravascular pressure at death to produce such a tonic contraction. This would mean that at death immediately after the last normal diastole the heart would contract and remain tonically contracted. But this means, in other words, that at death from any cause the heart stands still in a state of systole, lasting often over two hours. We cannot see how Rothberger can avoid drawing such a conclusion from the second assumption which he makes.

The results, therefore, which were obtained by Meirowsky (Ludloff), MacWilliam and Rothberger by means of a manometer can hardly be accepted as presenting the actual conditions as they occur in the heart normally connected within the body.

The investigations of Brouardel, Ludloff, Fuchs, MacWilliam and Rothberger were carried out by means of connecting the ventricle with a manometer. This method, as we have seen, introduces an artificial element which permits of more than one interpretation. Strassmann, who studied the appearance and outlines of the heart while it remained undisturbed within the thoracic cavity, has seen each heart at the utmost twice for a brief time. He could only state that there was a change of form at a certain time, but was unable to state when rigor sets in and how long it takes until it reaches its maximum.

For the rigor of skeletal muscles which is plainly visible without any special preparation, simple observation was amply sufficient to establish firmly the main facts of the phenomenon. Would not such a simple method be of great advantage also in the study of cardiac rigor? It is possible that the heart was watched by one or the other of these observers in its natural connections within the body for many hours after death, but there is no record of it—at least such a record did not come to our knowledge.

Our Own Observations.—As stated in the introduction, the chief object of the present research was to study the effect of vagus stimulation upon the course of cardiac rigor. Our studies of cardiac rigor under normal conditions were made only incidentally from the necessity for having careful extensive observations upon control animals. In the present research each experiment consisted

in parallel observations made on the onset and development of the rigor of both ventricles simultaneously in two animals: one whose vagi were stimulated and another whose vagi remained untouched. Forty-six double experiments of this kind were made, which means that the course of normal cardiac rigor was studied in forty-six animals. The foregoing analysis of the status of the present knowledge of our subject makes it evident that further studies of it are very desirable. Our report will demonstrate, we believe, that the method of simple observation is as well capable of shedding light upon the normal course of cardiac rigor, as it has been upon the rigor of skeletal muscles.

Method.—The control observations were made on five rabbits, fifteen cats and twenty-six dogs. With three exceptions death was brought about in all animals by the same method—namely exsanguination. The animals were etherized, tracheotomized and both carotid arteries exposed and severed. When bleeding ceased and the convulsions and respiration had stopped completely, the sternum and large sections of most of the ribs were rapidly removed, the pericardium freely dissected away, and the heart thus rapidly exposed to full view. In a number of experiments the animals were etherized for thirty to forty-five minutes, and longer, in order to be comparable with the animal whose vagi were stimulated for half an hour or more, while under the influence of ether. In such cases ether was administered through a tracheotomy tube.

In a few instances we made an attempt to study the cardiac rigor by the graphic method. The results were very unsatisfactory. The results which we are going to state here were obtained by direct observation, *i. e.*, by ocular inspection and by palpation. In thirty-nine cases the heart remained throughout the entire time of observation in its natural position and connections within the animal body: in six experiments (on dogs) the heart, after ligating all the blood vessels, was excised and suspended in a transparent bottle filled with Ringer's solution or a solution of 0.9 per cent. sodium chloride. From the time of exposure the heart remained under constant observation for many hours, or until the rigor reached its maximum. Except in a few cases we have made no special study of that stage when the rigor goes into solution. The criteria

by which we judged the occurrence of rigor were the changes in size and shape, and the changes in consistency tested by palpation. The change in color we found to be an unreliable criterion. According to the rapidity of the course of development of rigor the heart was tested more or less often, but rarely oftener than once every two minutes, or less often than ten minutes. In many experiments the heart was covered in the interval between observations with a very thin layer of absorbent cotton, saturated with a solution of sodium chloride or Ringer's solution. In other experiments the hearts remained uncovered, but were moistened with Ringer's or saline solution now and then to prevent drying. The room temperature during the entire course of this investigation varied between 19° C. and 25° C.

From our extended experience we can make the positive claim that, as in the case of skeletal muscle rigor, the method of direct observation gives most satisfactory results and permits the drawing of conclusions in a safer way than the method of connecting the cavities of the ventricle with a manometer. The changes in the diameters of the heart and in its configuration indicate satisfactorily the onset, progress and maximum of the rigor. A still better indicator is the change in the consistency of the heart muscle. By palpation one is capable of reliably recognizing the beginning of the change as well as its progress. This is especially true after one has acquired some experience with this method.

We may add that our data were derived from observations made by two observers independently of each other.

Results.—For most of our experiments we may divide the interval between the death of the animal and the attainment of maximum cardiac rigor into three periods. Before going further we wish to say that we considered the animal to be dead when the respiration had ceased completely; bleeding and all convulsions usually subsiding before. Furthermore, after respiratory death, some time was taken up by the opening of the thorax and the proper exposure of the heart. This time is included in the first period. The three periods are (1) the period during which the heart was still pulsating, (2) the period during which the heart was quiet and relaxed, and (3) the period taken up by the devel-

opment of rigor. We shall employ these periods as a basis for the report of our observations.

PULSATION OF THE HEART AFTER DEATH.

In every one of the control (normal) animals the heart continued beating after stoppage of the respiration, and in nearly all of them the heart was found beating on opening of the thorax. As a rule all compartments took part in the beating which first presented itself. We shall, however, deal with the right and left ventricles separately, since there is quite a marked difference between the two with reference to the length of time during which the pulsation continued.

Left Ventricle. Dogs.—For the twenty-six normal dogs the average time during which the left ventricle continued to beat after respiratory death was twenty-seven minutes. This average includes also the six hearts which were suspended in Ringer's solution or normal saline. The average time for the twenty hearts which remained *in situ* in the thorax was about ten minutes—two minutes being the shortest and twenty-two minutes the longest time the left ventricle was beating after death.

The average time for three hearts (of dogs) which were suspended in normal saline immediately after death was sixty-nine minutes, and for the three hearts suspended in Ringer's solution about ninety minutes. These hearts were not washed out at all and the blood vessels were ligated before suspension of the hearts in the solution, so that none of the solutions could enter the cavities of the heart or the coronary arteries.

While the number of three experiments for each solution is too small to give sufficient weight to the absolute figures thus obtained, these experiments nevertheless demonstrate clearly enough the considerable influence which a bath in a salt solution is capable of exerting upon the vitality of the excised hearts. And we may point out again that since the cardiac vessels were all ligated, the solutions could reach the tissues of the heart only through the epicardium.

Etherization.—Of the twenty dogs whose hearts remained in the thorax fourteen received ether for at least thirty or forty minutes longer than the remaining animals. Grouping the animals

according to this principle we find a marked difference between averages of the two groups. While the average time for the ether dogs amounts to about fourteen minutes, the hearts of the dogs which received ether for only a short time, continued to beat for an average of only about six minutes. Could the prolonged etherization have had the effect of prolonging the pulsation of the left ventricle? We shall draw no positive conclusions from this meagre observation, nor shall we enter into a discussion of this point. We wish, however, to call to mind here that a good many practitioners have employed subcutaneous injections of ether as a cardiac stimulant.

Cats.—In fifteen normal cats the average time during which the left ventricle continued to beat amounted to about fourteen minutes. In this average are included seven cats which besides the usual etherization received atropine. Their average alone, *i. e.*, that of the atropinized animals, amounts to thirteen minutes, which increases the average of the remaining eight cats to a little over sixteen minutes.

Rabbits.—The average duration of the pulsation of the left ventricle in five rabbits amounts to thirty-two minutes. Four of these rabbits were killed by asphyxia and only one by bleeding. In this one the left ventricle continued beating for forty-seven minutes. The asphyxia consisted in three in opening the chest freely under ether and one animal was killed by pithing which means perhaps asphyxia plus internal bleeding. In this animal the left ventricle continued beating for thirty-six minutes.

Right Ventricle. Dogs.—The average duration of the pulsation of the right ventricle for all the twenty-six dogs amounts to forty-five minutes. Excluding the six animals whose hearts were suspended in solutions, the average for the remaining twenty hearts is about thirty-one minutes. Dividing these animals again according to the duration of etherization the average for the fourteen dogs which received ether for thirty or forty minutes and longer amounts to about thirty-five minutes. The average for the remaining six dogs is only about twenty-one minutes. The average time for the right ventricle of the three hearts suspended in Ringer's solution amounts to about one hundred and thirty-one minutes, and for the hearts suspended in a physiological solution of sodium

chloride it amounts to sixty-four minutes. Here again there is a striking difference between the hearts remaining *in situ* and those suspended in a salt solution; and the difference between the hearts suspended in a Ringer's solution and those suspended in a physiological solution of sodium chloride, is for the right ventricle even greater than for the left. The favoring effect of a prolonged etherization comes out quite distinctly also in the duration of pulsation of the right ventricle.

Cats.—For the right ventricle we have notes on only fourteen cats. The average time for the entire number amounts to forty-three minutes. For the seven atropinized cats the average time of pulsation amounts to only thirty-three minutes. This increases the average time of the remaining seven non-atropinized cats to fifty-three minutes.

Rabbits.—For the length of time during which the right ventricle continued beating in rabbits we have complete notes in three experiments only, and those are for the rabbits which were killed by asphyxia. The average time here amounts to fifty-one minutes.

The various data regarding the average time of pulsation of left and right ventricles after death are embodied in Table A, which follows:

TABLE A.

Shows the Average Time the Left and Right Ventricles Continued Beating after Death from Exsanguination in Dogs, Cats and Rabbits.

(L. V. = left ventricle; R. V. = right ventricle.)

Number and Species of Animals.	Conditions of Experiment.	L. V.	R. V.
26 dogs.	Total of dogs.	27 minutes.	45 minutes.
20 dogs.	Total of dogs, with heart *in situ*.	10 "	31 "
14 dogs.	Hearts *in situ* and prolonged etherization.	14 "	35 "
6 dogs.	Hearts *in situ* and short etherization.	6 "	21 "
3 dogs.	Hearts suspended in Ringer's solution.	90 "	131 "
3 dogs.	Hearts suspended in 0.9 per cent. NaCl.	69 "	64 "
15 cats.	Total of cats.	14 "	(1) 43 "
7 cats.	Atropinized.	13 "	33 "
8 cats.	Non-atropinized.	16 "	(2) 53 "
5 rabbits.	Total of rabbits.	32 "	(3) 51 "

(1) 14 cats only. (2) 7 cats only.

(3) Complete notes only on three rabbits.

The above table brings out the difference between the left and right ventricles. In all conditions except one the right ventricle continues beating considerably longer than the left. The exception occurred when the entire heart was suspended in a 0.9 per cent. solution of sodium chloride.

The table brings out some more facts of interest. It shows in the first place that in dogs which were subjected to a longer etherization the left as well as the right ventricle continues to beat longer than the ventricle of dogs which had undergone only a brief etherization. Furthermore it shows that in atropinized cats both ventricles stop beating sooner than in non-atropinized. Finally there is a striking difference between the hearts (of dogs) which remained *in situ* and the hearts which were suspended in a salt solution. In the latter case both ventricles continued to beat much longer than in the non-excised hearts.

During the period of pulsation after death the ventricular beats do not, of course, remain of uniform rhythm and strength. They gradually diminish in vigor and frequency. The rate may fall as low as half a dozen beats a minute or even less—the contractions becoming gradually very weak and slow.

We made the interesting observation that the pulsations do not stop in the entire ventricle at once. The part nearest the base stops first; the entire ventricle is seen to be pulsating except the basal portion. Then the quiescent part gradually increases in extent downward until only the apex contracts rhythmically. Later only the very tip of the apex continues beating alone, and finally this stops also. Both ventricles show the same course: the uppermost part stopping first and the lowermost part stopping last. The successive dropping out of the various parts of the ventricles from regular participation in the systolic contractions takes place gradually and spreads much slower in the right ventricle than in the left. The entire process is more sharply pronounced in the left ventricle than in the right. We have to add that with regard to the latter we find in one instance a note reading: "the last part to stop beating was the portion nearest the right auricle."

In a number of cases we observed that the last motion to occur was in the form of fibrillary contractions. It is noteworthy that

the fibrillary contractions occurred mostly in the right ventricle, and the two instances in which fibrillary contractions appeared in the left ventricle occurred while the heart was suspended in a solution of sodium chloride, the right ventricle also showing fibrillary contractions.

Auricles.—For various reasons we were obliged to neglect the systematic study of the behavior of the auricles. We have, however, collected some data, part of which may be given here. In most cases the auricles seem to beat longer than the ventricles, and the usual order in which they stop beating seems to be: L. V., R.V., L.A., R.A. The right auricle frequently beats very long. In the heart of a rabbit we observed the auricle to pulsate for more than twenty-four hours after death.

When the activity of the heart began to lose its vigor, the phenomenon of heart block sometimes made its appearance, *i. e.*, both ventricles continued to beat rhythmically, but only with every second, third or fourth auricular beat. We have noted occasionally such unusual rhythms as 1 : 10 and 1 : 20. However, such heart blocks in which both ventricles were concerned simultaneously were rare. More frequently the left ventricle would beat rhythmically with every second, third, etc., beat of the right ventricle and the two auricles together. We have notes on instances in which the left auricle and ventricle were pulsating properly together, but both beating synchronously only with every second beat of the right auriculo-ventricular rhythm. We have further notes in which the left auricle stopped beating, while the left ventricle continued beating in the previous rhythm; also a note on a similar relation between the right auricle and ventricle; and finally notes on instances in which both auricles stopped beating long before both ventricles. In short, during the cardiac pulsations after death various dissociations take place between the auricles and ventricles. A systematic study of these dissociations might perhaps be of assistance in the study, during life, of the phenomena of dissociation of the normal cardiac rhythm.

In reporting above that, on opening the chest, the heart was found beating, we expressed it by saying that the heart was found beating in "nearly" all the control animals. There were a few exceptions to the rule. These exceptions are as follows: In Rabbit A (control), of Experiment 4, artificial respiration was used, and on stopping it the respiratory convulsions set in. When these ceased the heart was found to have stopped too. But two minutes later the heart began to beat again and continued to beat for over ten minutes. In Rabbit A (control), of Experiment 5, the heart was beating when it was first seen and it stopped after one minute. However, after thirty seconds, the heart began to beat again.

In Dog A, of Experiment 36, twelve minutes passed after respiratory death before the heart was completely exposed. It was then found to be already quiet.

In Dog A, of Experiment 39, the left ventricle had stopped when it was first completely exposed, which was about five minutes after respiratory death.

A behavior different from the other hearts was observed in those which were immersed in salt solutions. One heart was found quiet when first seen; the beats of another heart were at first very feeble; in a third the beats were

feeble and became rapidly slow; and a fourth heart, after an initial pulsation stopped beating completely. In all four cases the *ventricles soon began to beat again and became gradually fairly vigorous,* the pulsations lasting, as stated above, much longer than in the other control animals whose hearts were left *in situ.* In one instance the pulsations appeared in groups of ten or more with a pause of considerable length between each group. While the hearts which remained *in situ,* without exception, gradually decreased in vigor and frequency, nearly all of the hearts in the salt solution improved for a certain period in both vigor and frequency of pulsation.

PERIOD OF RELAXATION.

Judging by the available data in the recent literature reviewed above, it would appear that at least in the great majority of cases the heart, soon after death, passes into a state of contraction or rigor without the intervention of an appreciable period of relaxation.

From our observations upon normal animals we must claim that just the opposite is true. In the great majority of our control animals there was a definite, fairly long period in which the ventricles were completely relaxed. For the twenty-six dogs there were only two exceptions: for the fifteen cats the exceptions were one for the left ventricle and five for the right ventricle. The records on the five rabbits do not permit of such a summary statement. By the " period of relaxation " we mean the period in which the last trace of a spontaneous movement of any form in any part of a ventricle was gone, and no sign of a beginning of rigor had yet made its appearance in any part of the same ventricle. This does not preclude the condition that one ventricle may be at complete rest while the other may be still beating or be already even in advanced rigor. There were however such periods in a goodly number of cases in which both ventricles were completely relaxed at the same time. The appearance of the heart in absolute relaxation is very characteristic. It looks broadened and flattened. Especially is the transverse diameter visibly larger and the antero-posterior diameter strikingly shorter. *The heart in this intermediate period is distinctly more relaxed than at any diastole of a fairly normal rhythm.* This complete relaxation, however, does not come on abruptly. It develops gradually with the gradual slowing down of the rhythm. What was said above of the entire heart is true

also of each ventricle; they flatten down by their own weight when they are completely relaxed. It is also true for parts of a ventricle. The flabbiness of the upper (basal) part of a ventricle while the lower (apical) part is contracting is an instructive phenomenon.

In the following table (B) the average duration of the intermediary period (period of rest) is given for both ventricles in the different animals, under various conditions. In making up the averages, those cases which had no relaxation period (*i. e.*, where rigor began in some part of a ventricle immediately after the last part of the ventricle stopped beating) were included as zero. In those few cases where the "period of rigor" overlapped the "period of pulsation" (*i. e.*, where rigor began at the base while the apex was still beating) the period of relaxation was considered as a negative quantity, and the amount of negativity was deducted from the sum total. (Except in fractions of 0.5 round numbers are given; fractions larger than 0.5 were added as 1.0 to the figures.)

TABLE B.

Shows the Average Time of the Period of Relaxation in the Ventricles.

Number and Species of Animal.	Conditions of Experiment.	L. V.	R. V.
26 dogs.	Total of dogs.	28 minutes.	(1) 69 minutes.
20 dogs.	Hearts *in situ*.	23 "	(2) 66 "
14 dogs.	Prolonged etherization.	26 "	59 " (?)
6 dogs.	Short etherization.	24 "	(3) 82 " (?)
3 dogs.	Hearts in Ringer.	23 "	(4) 8 "
3 dogs.	Hearts in 0.9 per cent. NaCl.	48 "	147 "
15 cats.	Total of cats.	16 "	21.5 "
7 cats.	Atropinized.	11 "	19 "
8 cats.	Non-atropinized.	19 "	25 "
3 rabbits.		23 "	(5) 130 "

(1) 25 dogs only. (2) 19 dogs only. (3) 5 dogs only.

(4) In one experiment there was a period of relaxation of 25 minutes' duration; in the other two experiments there was no period of relaxation.

(5) For the right ventricle there was, for two animals, no record at all and on two others the periods of relaxation were very long and the rigor began during the night. In the only animal in which it was ascertained the period of relaxation was about 130 minutes.

Table B shows that the periods of relaxation are in certain respects similar to the periods of pulsation. They are much longer in the right than in the left ventricle; they also seem to be some-

what prolonged by etherization and shortened by atropinization. The influence of the bath is rather peculiar. The Ringer bath has no special influence upon the length of the period in the left and seems rather to shorten it in the right ventricle. The sodium chloride bath on the other hand, seems to prolong greatly the period of rest especially in the right ventricle.

IRRITABILITY OF THE HEART DURING THE PERIOD OF REST.

During the period of rest the ventricles were frequently palpated for the purpose of testing the oncome of rigor. Such a palpation though carried out gently frequently brought out a complete pulsation of the entire ventricle. A review of these observations brought out some interesting results with reference to the irritability of the heart during the period of rest. This question was then studied in six dogs more systematically by means of electrical stimulation. The results are very instructive. Briefly stated they are as follows: At the beginning of the period of rest, stimulation of any part of the ventricle will bring out a contraction of the entire ventricle, and sometimes even of both ventricles. With the advance of time the effect becomes diminished in two directions. In the first place the extent of the contraction becomes gradually lessened. From the contraction of the entire ventricle or a good part of it, the response becomes gradually lessened until the contraction is finally reduced to the point just around the electrode. Before it reaches that minimum, the response comes sometimes in the form of fibrillary contractions. And again, like the spontaneous contractions this occurs oftener in the right than in the left ventricle. When the stimulation by single electric shocks no longer elicits some kind of a response then a tetanizing stimulus might still cause some sort of a contraction, but finally even those stimuli remain without any effect.

Then there is a gradual loss of irritability in a topographical sense. Like the spontaneous contractions the irritability is lost first in the part of the ventricle nearest the base, and remains longest at the very tip of the apex. It was quite a frequent phenomenon to find that the upper part had lost all irritability while the apex was still responding to a stimulus with a fair contrac-

tion. In fact there were instances in which the apex was still beating spontaneously after the upper part of the ventricle had lost all irritability.

We may mention that in one instance when all irritability was completely lost in the left ventricle, circumscript mechanical stimuli applied strictly to the left ventricle brought out complete contractions of the resting but still irritable right ventricle. While this is only a single instance it demonstrates an important principle. It shows that a tissue which has lost all irritability may still be capable of receiving and transmitting stimuli to distant responsive tissues. We can thus understand how for instance a quiescent and unresponsive right auricle might still be capable of receiving stimuli from a source higher up and transmitting them to the ventricles below—a point of contention between the neurogenic and myogenic camps.

THE PERIOD OF RIGOR.

Cardiac rigor as a rule develops slowly. Its very beginning we recognized by a change in the consistency of the tissue. Soon, however, there was also a change in the shape. Strassmann called attention to the shortening of the transverse and long diameters of the heart in rigor. We wish to call attention to the *increase of the antero-posterior diameter* which is at least as striking as the shortening of the transverse diameter and surely more striking than the shortening of the baso-apical diameter. These changes are manifested in a striking way in the development of rigor of the left ventricle. But they are also present and unmistakable in in their manifestations in the right ventricle. At the beginning of our studies—we began with rabbits—we had some misgivings about our capability of judging the phases of rigor in the right ventricle. However, we soon learned that there was little difficulty in recognizing the beginning, progress and maximum of rigor of the right ventricle.

The maximum rigor we have simply judged by the absence of further increase in the consistency as well as in the change in size and shape. When both ventricles are in maximum rigor, the interventricular groove is deeper than normal and the right ven-

tricle is visibly smaller than the left. It looks more like a lateral appendage of the left ventricle.

The observation of the *progress of the rigor* brought out the interesting fact that it also *advances topographically from the base toward the apex of the heart.* The uppermost part of the left ventricle gets into a state of rigor first and appears at the angle formed by the anterior descending ramus, and the ramus circumflexus of the left coronary artery, like a lump raised above the level of the remainder of the heart.

The time consumed in the advancement of rigor is spent not only on an increase of the intensity, but also on the topographical advance toward the apex.

During the topographical march of the rigor it frequently happened that the apex was still quite irritable while the basal portion was in unmistakable rigor. In fact, in a few cases it even happened that the apex or its lowest part was still *beating,* while a portion near the base was already in rigor.

We have not made a single observation in which a region of the heart already in distinct rigor still responded to stimulation. This is of interest considering the statement made concerning skeletal muscles while in rigor.

We made no direct observation on the question of whether in each section of the ventricle the entire thickness of the wall gets into rigor at once, or whether there is also in this respect a certain progressiveness. We are inclined to assume the latter and believe that the rigor begins on the inside and advances toward the outer surface. In support of this assumption we can offer the following observation: when the irritability in any part of the ventricle was on the point of disappearance, *an electrical stimulation sometimes caused a contraction of only the superficial muscle fibers of a circumscribed area.* In these cases apparently the inner muscle fibers had already lost their irritability. Since we now have sufficient reasons to assume that rigor sets in first where the irritability first disappears, it seems that we are justified to suppose that the loss of irritability, as well as the onset of rigor, begins on the inside and ends on the outside surface of the ventricular muscle.

Time of Onset of Rigor.—From what was said above about the

periods of pulsation and relaxation, it is evident that even in the most susceptible parts of the left ventricle rigor does not set in immediately after death. We may now say definitely that under normal conditions there was not a single exception to that rule.

The following is a table of the average time which elapsed between death and the onset of rigor for both ventricles of all the normal animals upon which we have made such observations.

TABLE C.

Showing the Average Time Elapsed Between Death and the Beginning of Rigor.

Number and Species of Animals.	Conditions of Experiment.	L. V.	R. V.
26 dogs.	Total of dogs.	55 minutes.	(1) 115 minutes.
20 "	Hearts *in situ*.	33 "	(2) 96 "
14 "	Prolonged etherization.	39 "	(3) 105 " ?
6 "	Short etherization.	31 "	99 " ?
3 "	Hearts in Ringer.	114 "	138 "
3 "	Hearts in 0.9 per cent. NaCl.	117 "	212 "
15 cats.	Total of cats.	30 "	64 "
7 "	Atropinized.	23 "	55 "
8 "	Non-atropinized.	34 "	78 "
5 rabbits.	Total of rabbits.	56 "	(4) —

(1) 25 dogs. (2) 19 dogs. (3) 13 dogs.

(4) The data on R. V. in rabbits do not permit the giving of exact figures.

The data on the right ventricle were incomplete on certain points. The long duration of the periods made some observations less definite than the observations upon the left ventricle. Some discrepancies in this table have their origin, partly at least, in the fact that in some cases rigor began in one part of the ventricle while another part was still beating. The time during which this took place had to be deducted from the sum total.

The foregoing table (C) shows definitely that the right ventricle in all animals begins to pass into rigor distinctly later than the left ventricle. Even for rabbits, in the experiments of which our data do not permit the giving of exact figures, we can state positively that rigor in the right ventricle begins much later than in the left. The table indicates also, in a general way, that etherization and salt solutions retard, and that atropinization hastens more or less the onset of rigor. The retardation of the onset of rigor by the salt baths is rather marked.

The Growth of Cardiac Rigor.—The following table gives the average time which elapsed between the onset of rigor and the attainment of its maximum.

TABLE D.

Showing the Average Time Elapsing from the Beginning of Rigor to the Attainment of its Maximum.

Number and Species of Animals.	Conditions of Experiment.	L. V.	R. V.
26 dogs.	Total of dogs.	145 minutes.	(1) 143 minutes.
20 "	Hearts *in situ*.	138 "	(2) 153 "
14 "	Prolonged etherization.	121 "	(3) 139 "
6 "	Short etherization.	150 "	184 "
3 "	Hearts in Ringer.	98 "	108 "
3 "	Hearts in 0.9 per cent. NaCl.	251 "	153 "
15 cats	Total of cats.	43 "	(4) —
7 "	Atropinized.	31 "	(4) —
8 "	Non-atropinized.	54 "	(4) —
5 rabbits.	Total number of rabbits.	45 "	(4) —

(1) 25 dogs. (2) 19 dogs. (3) 13 dogs.
(4) Data on right ventricle incomplete.

The figures in Table D bring out some differences between the period of growth of rigor and the two preceding periods. In the first place it appears that the time required for the development of rigor is not so strikingly different between the left and right ventricles as is undoubtedly the case in the periods of pulsation and relaxation. The average time for all the twenty-six dogs—for which there were also complete data on the right ventricle—is, for the period of growth, nearly the same in both ventricles. This, however, is essentially due to the fact that in the *saline experiments the period of growth of rigor is much longer in the left ventricle than in the right.* Furthermore, there appears to be a difference also with regard to the effect of etherization. While the periods of pulsation and relaxation are lengthened by prolonged etherization (*i. e.,* in other words etherization delays the *onset* of rigor), the *growth of it is apparently hastened by prolonged etherization.* Atropinization, on the other hand, appears to hasten both the onset and the growth of rigor. Suspension of the heart in a Ringer solution hastened the growth of rigor (using hearts which remained *in situ* as a standard), while suspension of the heart in a 0.9 per

cent. solution of sodium chloride retarded the growth of rigor considerably.

The growth of rigor—as its onset—progresses, as already stated above, topographically from the base towards the apex, and probably also from the endocardium towards the epicardium. The progress is from section to section, and presumably also from muscle fiber to muscle fiber within each section.

Possibly there is a progression of the onset of rigor even within each individual fiber, that is, every part of an individual fiber may not get into the same state of rigor simultaneously. The time which passes from the beginning of cardiac rigor until its full development is consumed, in part at least, by its topographical progress. There is probably also a growth in the intensity of the rigor within each part. However, we have no experimental facts to help us in an intelligent discussion of these surmises.

DISCUSSION OF THE RESULTS.

According to the last and most extensive work on cardiac rigor, that of Rothberger, the heart very soon after death passes into a state of more or less strong contraction. Rothberger considers this a state of tonic contraction. Other investigators who studied the subject by the same method as Rothberger—the connection of the heart with a manometer—have apparently observed the same condition, and consider it a state of rigor.

Our observations bring out an entirely different result. Except in a single doubtful case, in all our normal (control) animals we did not meet with a single instance in which the heart soon after death was in a state of contraction. After the termination of the life of the animal, in the majority of cases, the beginning of cardiac rigor was preceded by two periods: the period of postmortem cardiac pulsations and the period of complete cardiac relaxation. At any rate our direct observations show that, practically in all cases, rigor was separated from death by a fairly long period of complete relaxation.

The setting in of rigor, or of a continuous tonic contraction soon after death, which was observed by the other experimenters is, we have reason to assume, an artificial phenomenon brought on by the method they employed. The mechanical effect of the distension and the chemical effect of filling up the ventricular

cavity with saline, etc., brings on in the still very irritable heart a tonic contraction which in some cases might indeed hasten the development of rigor. We agree, then, with Rothberger that the distension of the heart after death may cause tonic contraction, but on the basis of our observations we must disagree with him that such a rigor-like tonic contraction occurs after death when the heart remains in normal conditions.

Our observations showed further—to bring this point out separately—that in nearly all animals after exsanguination and respiratory death, the heart continued to beat for a fairly long time. For the left ventricle the average time in rabbits was thirty-two minutes in dogs twenty-seven minutes, and in cats sixteen minutes. The right ventricle was beating in all instances much longer than the left. It must be remembered also that the hearts were exposed to room temperature. In the half dozen experiments on hearts of dogs which were left in baths of salt solutions (room temperature), the pulsation continued in some instances for longer than two hours. This is the more noteworthy as none of the solutions could enter through the coronary arteries.

The literature contains very few statements as to the length of time the heart, when left alone, may continue to beat after death.

MacWilliam speaks of "oscillations which look like systoles and diastoles." But they were infrequent, irregular and occurred as late as two hours after death. These occasional contractions apparently have nothing in common with the pulsations which we have observed to occur regularly for some time after death. Rothberger says that post-mortem pulsations occur frequently in death, after exsanguination, but gives no intimation as to the length of time they may last. He also says that pulsations often accompany the "primary dilatation." From his statements it seems that he has not observed any pulsations during the period of tonic contraction which appears immediately after connecting the heart with the manometer. In one case in which there were some slow pulsations at the beginning of the manometric observations, the connection was made two hours after death. From all these facts it appears that in these experiments *the intraventricular distension causes a continuous systole which thus wipes out the regular pulsation.*

This is a very interesting fact. While in normal life, or soon after death when the circulation of the coronary arteries is re-established, intraventricular pressure, when it acts as a stimulus, can at the utmost start pulsations or effect their character, but can never produce a continuous systole—a tetanus of the heart; we see here that soon after death the heart without a coronary circulation responds to an intraventricular pressure with a tonic contraction—a continuous systole. Furthermore, later, when the irritability of the heart becomes more decreased, distension of the heart without the aid of coronary circulation, does not cause a tonic contraction but is capable of bringing out some rhythmical pulsations. These are significant facts, into a further discussion of which, however, we shall not enter here.

Our observations have shown also—to point out again—that there has been in all animals, with a very few exceptions, an outspoken difference between the left and right ventricles with regard to the length of the periods of pulsation and of relaxation. In all the observations on the three species of animals the right ventricle did beat longer, persevere afterward in a state of complete relaxation and lose its irritability much more slowly than the left ventricle. In other words, in all cases the rigor set in later in the right than in the left ventricle.

For the time taken up by the growth of rigor the difference between the two ventricles is not so striking as it is in the other periods.

From the forensic literature we learn that a great deal of importance used to be attached to a post-mortem finding in which the right ventricle was still in a state of diastole, while the left ventricle was already in rigor. According to our observations such findings could be met with in every case. In fact, in some cases it occurred that the left ventricle was already in fair rigor while the right was still beating fairly rhythmically.

While the fact that rigor sets in later in the right ventricle than in the left is surely not an entirely new observation, no special attention was paid to it in any of the recent studies on the subject. We find statements saying that the heart passes into rigor at this or that time, without defining which part of the heart is meant. In

fact it is in the nature of the method which was employed in most of these studies that in one animal as a rule only one ventricle was studied. But it seems that in some instances the results obtained from both ventricles were not kept separately.

The difference between the two ventricles seems to be based essentially upon greater perseverence of the vitality and irritability of the right ventricle over the left. As to the origin of this difference we wish to call attention to some conditions which possibly serve as causal factors. The right ventricle, as a part of the venous end of the circulation contains, as a rule, soon after death—even when death is caused by bleeding—more blood than the left ventricle. There are good reasons for the assumption that this blood can reach the muscle fibers of the right ventricle and exert some nutritive (or other favorable) influence upon them. Porter[12] and Pratt[13] have shown that nutritive solutions can reach the muscle fibers within the ventricular wall from the cavity of the right ventricle through the foramina Thebesii. Besides, we have seen in our experiments that simple bathing of the heart (with ligated vessels) improves and prolongs the pulsations and the irritability of the heart. This effect can only be brought about by the liquid reaching the muscle fibers through the epicardium. Why could we not assume that the nutritive and stimulating liquid within the ventriclar cavity may also reach the muscle fibres directly through the endocardium? Now, since the right ventricle contains, as a rule, a good deal more blood than the left, the muscular tissue of the right ventricle will be better and longer provided with nutritive and stimulating material than the left—venous blood being at least as good a medium as a Ringer solution. Furthermore, the thinness of the wall of the right ventricle might be another cause of its great vital resistance. In our experiments the favorable influence of the saline baths was generally more pronounced upon the right ventricle than on the left, on account, as we assume, of the lesser bulk of the right ventricular wall, the solutions being able to reach a greater percentage of its constituent muscle fibers than in the wall of the left ventricle. This ought to be true also for the venous blood within the right ventricle. To recapitulate: the greater perseverance of the vital activity and irritability of the right ventricle over the left may be due to (1) that the blood within the cavity of the right ventricle is capable of reaching the muscle fibers of the wall through the foramina Thebesii and also directly through the endocardium; (2) that on account of the thinness of the wall a greater percentage of the fibers can be reached by the nutritive medium than in the bulky wall of the left ventricle; and (3) the right ventricle, as a rule, contains more blood after death than the left ventricle.

Our observations revealed the interesting fact that the various post-mortem changes which precede complete rigor progress topographically from the base towards the apex. The spontaneous

[12] Porter, *American Jour. of Physiol.*, 1898, i, 71.

[13] Pratt, *idem.*, 1898, i, 86.

pulsations do not cease in all parts of the ventricle at once, but stop at the uppermost part first and in the tip of the apex last. The same topographical succession takes place in the disappearance of irritability of the ventricles during the period of relaxation. Finally, rigor appears first in the upper part of the ventricle and spreads gradually to the apex. As we have stated before, we have reason to assume that the rigor wanders also from the inner surface towards the epicardium. This wandering of post-mortem changes is confined to each ventricle separately and it sometimes happened that the apex of the left ventricle was already in rigor while one part of the right ventricle was still beating.

L. Krehl[14] and F. R. Fuchs[15] have established, as stated before, that some time after death, liquids injected into the ventricular cavities cannot pass into the auricles. This is due, according to Fuchs, to the onset of rigor in the ventricles. Fuchs considers the impassability of liquids through the venous ostia as an early phenomenon of rigor and states that it may occur before the ventricles show any other signs of rigor. Connecting these observations with our own, we might assume that rigor begins with the innermost muscle fibers at the uppermost part of the ventricle and around the venous ostia, thus producing early a stenosis of the corresponding orifices which sometimes might be capable of preventing the passage of liquid from the ventricle into the corresponding auricle. This is perhaps the cause of the not infrequent occurrence that the right ventricle while already in full rigor still contains some blood.

Strassmann[16] has called attention to the early appearance of cardiac rigor. Fuchs[17] has pointed out that rigor appears in the heart before it appears in the skeletal muscles. Both speak of the heart in general, which is inadequate, inasmuch as the rigor of the skeletal muscles might be already advanced before it had made its appearance in the right ventricle of the heart. However, the statement of Fuchs seems to be true with regard to the onset of rigor in the left ventricle. In this regard we may say in general

[14] L. Krehl, *loc. cit.*

[15] F. R. Fuchs, *loc. cit.*

[16] Strassmann, *loc. cit.*, 1889.

[17] Fuchs, *loc. cit.*

that rigor was noticed in a part of the left ventricle before it was noticed in any group of skeletal muscles. That this is not due exclusively to the constant activity of the heart, as Fuchs assumes, is evident from the fact that rigor of the right ventricle, which works as constantly as the left, sets in rather late.

Finally, we might recall the fact that in our observations *ether delayed the onset and hastened the growth of cardiac rigor, while atropin accelerated both phases.*

[NOTE.—Summary of Part I will follow with Part II of this paper.]